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Preface

The central aim of many studies in the physical, behavioral, social, and biological sciences is the elucidation of cause–effect relationships among variables or events. However, the appropriate methodology for extracting such relationships from data – or even from theories – has been fiercely debated.

The two fundamental questions of causality are: (1) What empirical evidence is required for legitimate inference of cause–effect relationships? (2) Given that we are willing to accept causal information about a phenomenon, what inferences can we draw from such information, and how? These questions have been without satisfactory answers in part because we have not had a clear semantics for causal claims and in part because we have not had effective mathematical tools for casting causal questions or deriving causal answers.

In the last decade, owing partly to advances in graphical models, causality has undergone a major transformation: from a concept shrouded in mystery into a mathematical object with well-defined semantics and well-founded logic. Paradoxes and controversies have been resolved, slippery concepts have been explicated, and practical problems relying on causal information that long were regarded as either metaphysical or unmanageable can now be solved using elementary mathematics. Put simply, causality has been mathematized.

This book provides a systematic account of this causal transformation, addressed primarily to readers in the fields of statistics, artificial intelligence, philosophy, cognitive science, and the health and social sciences. Following a description of the conceptual and mathematical advances in causal inference, the book emphasizes practical methods for elucidating potentially causal relationships from data, deriving causal relationships from combinations of knowledge and data, predicting the effects of actions and policies, evaluating explanations for observed events and scenarios, and – more generally – identifying and explicating the assumptions needed for substantiating causal claims.

Ten years ago, when I began writing *Probabilistic Reasoning in Intelligent Systems* (1988), I was working within the empiricist tradition. In this tradition, probabilistic relationships constitute the foundations of human knowledge, whereas causality simply provides useful ways of abbreviating and organizing intricate patterns of probabilistic relationships. Today, my view is quite different. I now take causal relationships to be the

fundamental building blocks both of physical reality and of human understanding of that reality, and I regard probabilistic relationships as but the surface phenomena of the causal machinery that underlies and propels our understanding of the world.

Accordingly, I see no greater impediment to scientific progress than the prevailing practice of focusing all of our mathematical resources on probabilistic and statistical inferences while leaving causal considerations to the mercy of intuition and good judgment. Thus I have tried in this book to present mathematical tools that handle causal relationships side by side with probabilistic relationships. The prerequisites are startlingly simple, the results embarrassingly straightforward. No more than basic skills in probability theory and some familiarity with graphs are needed for the reader to begin solving causal problems that are too complex for the unaided intellect. Using simple extensions of probability calculus, the reader will be able to determine mathematically what effects an intervention might have, what measurements are appropriate for control of confounding, how to exploit measurements that lie on the causal pathways, how to trade one set of measurements for another, and how to estimate the probability that one event was the actual cause of another.

Expert knowledge of logic and probability is nowhere assumed in this book, but some general knowledge in these areas is beneficial. Thus, Chapter 1 includes a summary of the elementary background in probability theory and graph notation needed for the understanding of this book, together with an outline of the developments of the last decade in graphical models and causal diagrams. This chapter describes the basic paradigms, defines the major problems, and points readers to the chapters that provide solutions to those problems.

Subsequent chapters include introductions that serve both to orient the reader and to facilitate skipping; they indicate safe detours around mathematically advanced topics, specific applications, and other explorations of interest primarily to the specialist.

The sequence of discussion follows more or less the chronological order by which our team at UCLA has tackled these topics, thus re-creating for the reader some of our excitement that accompanied these developments. Following the introductory chapter (Chapter 1), we start with the hardest questions of how one can go about discovering cause–effect relationships in raw data (Chapter 2) and what guarantees one can give to ensure the validity of the relationships thus discovered. We then proceed to questions of identifiability – namely, predicting the direct and indirect effects of actions and policies from a combination of data and fragmentary knowledge of where causal relationships might operate (Chapters 3 and 4). The implications of these findings for the social and health sciences are then discussed in Chapters 5 and 6 (respectively), where we examine the concepts of structural equations and confounding. Chapter 7 offers a formal theory of counterfactuals and structural models, followed by a discussion and a unification of related approaches in philosophy, statistics, and economics. The applications of counterfactual analysis are then pursued in Chapters 8–10, where we develop methods of bounding causal relationships and illustrate applications to imperfect experiments, legal responsibility, and the probability of necessary, sufficient, and single-event causation. We end this book (Epilogue) with a transcript of a public lecture that I presented at UCLA, which provides a gentle introduction of the historical and conceptual aspects of causation.

Readers who wish to be first introduced to the nonmathematical aspects of causation are advised to start with the Epilogue and then to sweep through the other historical/conceptual parts of the book: Sections 1.1.1, 3.3.3, 4.5.3, 5.1, 5.4.1, 6.1, 7.2, 7.4, 7.5, 8.3, 9.1, 9.3, and 10.1. More formally driven readers, who may be anxious to delve directly into the mathematical aspects and computational tools, are advised to start with Section 7.1 and then to proceed as follows for tool building: Section 1.2, Chapter 3, Sections 4.2–4.4, Sections 5.2–5.3, Sections 6.2–6.3, Section 7.3, and Chapters 8–10.

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J. P.
Los Angeles
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CHAPTER ONE

Introduction to Probabilities, Graphs, and Causal Models

Chance gives rise to thoughts,
and chance removes them.
Pascal (1670)

1.1 INTRODUCTION TO PROBABILITY THEORY

1.1.1 Why Probabilities?

Causality connotes lawlike necessity, whereas probabilities connote exceptionality, doubt, and lack of regularity. Still, there are two compelling reasons for starting with, and in fact stressing, probabilistic analysis of causality; one is fairly straightforward, the other more subtle.

The simple reason rests on the observation that causal utterances are often used in situations that are plagued with uncertainty. We say, for example, "reckless driving causes accidents" or "you will fail the course because of your laziness" (Suppes 1970), knowing quite well that the antecedents merely tend to make the consequences more likely, not absolutely certain. Any theory of causality that aims at accommodating such utterances must therefore be cast in a language that distinguishes various shades of likelihood – namely, the language of probabilities. Connected with this observation, we note that probability theory is currently the official mathematical language of most disciplines that use causal modeling, including economics, epidemiology, sociology, and psychology. In these disciplines, investigators are concerned not merely with the presence or absence of causal connections but also with the relative strengths of those connections and with ways of inferring those connections from noisy observations. Probability theory, aided by methods of statistical analysis, provides both the principles and the means of coping with – and drawing inferences from – such observations.

The more subtle reason concerns the fact that even the most assertive causal expressions in natural language are subject to exceptions, and those exceptions may cause major difficulties if processed by standard rules of deterministic logic. Consider for example the two plausible premises:

1. My neighbor's roof gets wet whenever mine does.
2. If I hose my roof it will get wet.

Taken literally, these two premises imply the implausible conclusion that my neighbor's roof gets wet whenever I hose mine.

Such paradoxical conclusions are normally attributed to the finite granularity of our language, as manifested in the many exceptions that are implicit in premise 1. Indeed, the paradox disappears once we take the trouble of explicating those exceptions and write, for instance:

1*. My neighbor's roof gets wet whenever mine does, except when it is covered with plastic, or when my roof is hosed, etc.

Probability theory, by virtue of being especially equipped to tolerate unexplicated exceptions, allows us to focus on the main issues of causality without having to cope with paradoxes of this kind.

As we shall see in subsequent chapters, tolerating exceptions solves only part of the problems associated with causality. The remaining problems – including issues of inference, interventions, identification, ramification, confounding, counterfactuals, and explanation – will be the main topic of this book. By portraying those problems in the language of probabilities, we emphasize their universality across languages. Chapter 7 will recast these problems in the language of deterministic logic and will introduce probabilities merely as a way to express uncertainty about unobserved facts.

1.1.2 Basic Concepts in Probability Theory

The bulk of the discussion in this book will focus on systems with a finite number of discrete variables and thus will require only rudimentary notation and elementary concepts in probability theory. Extensions to continuous variables will be outlined but not elaborated in full generality. Readers who want additional mathematical machinery are invited to study the many excellent textbooks on the subject – for example, Feller (1950), Hoel et al. (1971), or the appendix to Suppes (1970). This section provides a brief summary of elementary probability concepts, based largely on Pearl (1988b), with special emphasis on Bayesian inference and its connection to the psychology of human reasoning under uncertainty. Such emphasis is generally missing from standard textbooks.

We will adhere to the Bayesian interpretation of probability, according to which probabilities encode degrees of belief about events in the world and data are used to strengthen, update, or weaken those degrees of belief. In this formalism, degrees of belief are assigned to propositions (sentences that take on true or false values) in some language, and those degrees of belief are combined and manipulated according to the rules of probability calculus. We will make no distinction between sentential propositions and the actual events represented by those propositions. For example, if A stands for the statement "Ted Kennedy will seek the nomination for president in year 2000," then $P(A \mid K)$ stands for a person's subjective belief in the event described by A given a body of knowledge K, which might include that person's assumptions about American politics, specific proclamations made by Kennedy, and an assessment of Kennedy's past and personality. In defining probability expressions, we often simply write $P(A)$, leaving out the symbol K. However, when the background information undergoes changes, we need to identify specifically the assumptions that account for our beliefs and explicitly articulate K (or some of its elements).

In the Bayesian formalism, belief measures obey the three basic axioms of probability calculus:

$$0 \leq P(A) \leq 1, \tag{1.1}$$

$$P(\text{sure proposition}) = 1, \tag{1.2}$$

$$P(A \text{ or } B) = P(A) + P(B) \text{ if } A \text{ and } B \text{ are mutually exclusive.} \tag{1.3}$$

The third axiom states that the belief assigned to any set of events is the sum of the beliefs assigned to its nonintersecting components. Because any event A can be written as the union of the joint events $(A \wedge B)$ and $(A \wedge \neg B)$, their associated probabilities are given by[1]

$$P(A) = P(A, B) + P(A, \neg B), \tag{1.4}$$

where $P(A, B)$ is short for $P(A \wedge B)$. More generally, if B_i, $i = 1, 2, \ldots, n$, is a set of exhaustive and mutually exclusive propositions (called a *partition* or a *variable*), then $P(A)$ can be computed from $P(A, B_i)$, $i = 1, 2, \ldots, n$, by using the sum

$$P(A) = \sum_i P(A, B_i), \tag{1.5}$$

which has come to be known as the "law of *total* probability." The operation of summing up probabilities over all B_i is also called "marginalizing over B"; and the resulting probability, $P(A)$, is called the *marginal* probability of A. For example, the probability of A, "The outcomes of two dice are equal," can be computed by summing over the joint events $(A \wedge B_i)$, $i = 1, 2, \ldots, 6$, where B_i stands for the proposition "The outcome of the first die is i." This yields

$$P(A) = \sum_i P(A, B_i) = 6 \times \frac{1}{36} = \frac{1}{6}. \tag{1.6}$$

A direct consequence of (1.2) and (1.4) is that a proposition and its negation must be assigned a total belief of unity,

$$P(A) + P(\neg A) = 1, \tag{1.7}$$

because one of the two statements is certain to be true.

The basic expressions in the Bayesian formalism are statements about *conditional probabilities* – for example, $P(A \mid B)$ – which specify the belief in A under the assumption that B is known with absolute certainty. If $P(A \mid B) = P(A)$, we say that A and B are *independent,* since our belief in A remains unchanged upon learning the truth of B. If $P(A \mid B, C) = P(A \mid C)$, we say that A and B are *conditionally independent* given C; that is, once we know C, learning B would not change our belief in A.

Contrary to the traditional practice of defining conditional probabilities in terms of joint events,

$$P(A \mid B) = \frac{P(A, B)}{P(B)}, \tag{1.8}$$

[1] The symbols $\wedge$, $\vee$, $\neg$ denote the logical connectives *and, or,* and *not,* respectively.

Bayesian philosophers see the conditional relationship as more basic than that of joint events – that is, more compatible with the organization of human knowledge. In this view, B serves as a pointer to a context or frame of knowledge, and $A \mid B$ stands for an event A in the context specified by B (e.g., a symptom A in the context of a disease B). Consequently, empirical knowledge invariably will be encoded in conditional probability statements, whereas belief in joint events (if it is ever needed) will be computed from those statements via the product

$$P(A, B) = P(A \mid B)P(B), \tag{1.9}$$

which is equivalent to (1.8). For example, it was somewhat unnatural to assess

$$P(A, B_i) = \frac{1}{36}$$

directly in (1.6). The mental process underlying such assessment presumes that the two outcomes are independent, so to make this assumption explicit the probability of the joint event (equality, B_i) should be assessed from the conditional event (equality $\mid B_i$) via the product

$$\begin{aligned} P(\text{equality} \mid B_i)P(B_i) &= P(\text{outcome of second die is } i \mid B_i)P(B_i) \\ &= \frac{1}{6} \times \frac{1}{6} = \frac{1}{36}. \end{aligned}$$

As in (1.5), the probability of any event A can be computed by conditioning it on any set of exhaustive and mutually exclusive events B_i, $i = 1, 2, \ldots, n$, and then summing:

$$P(A) = \sum_i P(A \mid B_i)P(B_i). \tag{1.10}$$

This decomposition provides the basis for hypothetical or "assumption-based" reasoning. It states that the belief in any event A is a weighted sum over the beliefs in all the distinct ways that A might be realized. For example, if we wish to calculate the probability that the outcome X of the first die will be greater than the outcome Y of the second, we can condition the event $A : X > Y$ on all possible values of X and obtain

$$\begin{aligned} P(A) &= \sum_{i=1}^{6} P(Y < X \mid X = i)P(X = i) \\ &= \sum_{i=1}^{6} P(Y < i)\frac{1}{6} = \sum_{i=1}^{6}\sum_{j=1}^{i-1} P(Y = j)\frac{1}{6} \\ &= \frac{1}{6}\sum_{i=2}^{6} \frac{i-1}{6} = \frac{5}{12}. \end{aligned}$$

It is worth reemphasizing that formulas like (1.10) are always understood to apply in some larger context K, which defines the assumptions taken as common knowledge (e.g., the fairness of dice rolling). Equation (1.10) is really a shorthand notation for the statement

$$P(A \mid K) = \sum_i P(A \mid B_i, K)P(B_i \mid K). \tag{1.11}$$

This equation follows from the fact that every conditional probability $P(A \mid K)$ is itself a genuine probability function; hence it satisfies (1.10).

Another useful generalization of the product rule (equation (1.9)) is the *chain rule* formula. It states that if we have a set of n events, $E_1, E_2, \ldots, E_n$, then the probability of the joint event $(E_1, E_2, \ldots, E_n)$ can be written as a product of n conditional probabilities:

$$P(E_1, E_2, \ldots, E_n) = P(E_n \mid E_{n-1}, \ldots, E_2, E_1) \cdots P(E_2 \mid E_1)P(E_1). \tag{1.12}$$

This product can be derived by repeated application of (1.9) in any convenient order.

The heart of Bayesian inference lies in the celebrated inversion formula,

$$P(H \mid e) = \frac{P(e \mid H)P(H)}{P(e)}, \tag{1.13}$$

which states that the belief we accord a hypothesis H upon obtaining evidence e can be computed by multiplying our previous belief $P(H)$ by the likelihood $P(e \mid H)$ that e will materialize if H is true. This $P(H \mid e)$ is sometimes called the posterior probability (or simply *posterior*), and $P(H)$ is called the prior probability (or *prior*). The denominator $P(e)$ of (1.13) hardly enters into consideration because it is merely a normalizing constant $P(e) = P(e \mid H)P(H) + P(e \mid \neg H)P(\neg H)$, which can be computed by requiring that $P(H \mid e)$ and $P(\neg H \mid e)$ sum to unity.

Whereas formally (1.13) might be dismissed as a tautology stemming from the definition of conditional probabilities,

$$P(A \mid B) = \frac{P(A, B)}{P(B)} \quad \text{and} \quad P(B \mid A) = \frac{P(A, B)}{P(A)}, \tag{1.14}$$

the Bayesian subjectivist regards (1.13) as a normative rule for updating beliefs in response to evidence. In other words, although conditional probabilities can be viewed as purely mathematical constructs (as in (1.14)), the Bayes adherent views them as primitives of the language and as faithful translations of the English expression "..., given that I know A." Accordingly, (1.14) is not a definition but rather an empirically verifiable relationship between English expressions. It asserts, among other things, that the belief a person attributes to B after discovering A is never lower than that attributed to $A \wedge B$ before discovering A. Also, the ratio between these two beliefs will increase proportionally with the degree of surprise $[P(A)]^{-1}$ one associates with the discovery of A.

The importance of (1.13) is that it expresses a quantity $P(H \mid e)$ – which people often find hard to assess – in terms of quantities that often can be drawn directly from our experiential knowledge. For example, if a person at the next gambling table declares the outcome "twelve," and we wish to know whether he was rolling a pair of dice or spinning a roulette wheel, our models of the gambling devices readily yield the quantities $P(\text{twelve} \mid \text{dice})$ and $P(\text{twelve} \mid \text{roulette})$: 1/36 for the former and 1/38 for the latter. Similarly, we can judge the prior probabilities $P(\text{dice})$ and $P(\text{roulette})$ by estimating the number of roulette wheels and dice tables at the casino. Issuing a direct judgment of

P(dice | twelve) would have been much more difficult; only a specialist in such judgments, trained at the very same casino, could do it reliably.

In order to complete this brief introduction, we must discuss the notion of *probabilistic model* (also called *probability space*). A probabilistic model is an encoding of information that permits us to compute the probability of every well-formed sentence S in accordance with the axioms of (1.1)–(1.3). Starting with a set of atomic propositions $A, B, C, \ldots$, the set of well-formed sentences consists of all Boolean formulas involving these propositions, for example, $S = (A \wedge B) \vee \neg C$. The traditional method of specifying probabilistic models employs a *joint distribution function,* which is a function that assigns nonnegative weights to every *elementary event* in the language (an elementary event being a conjunction in which every atomic proposition or its negation appears once) such that the sum of the weights adds up to 1. For example, if we have three atomic propositions, A, B, and C, then a joint distribution function should assign nonnegative weights to all eight combinations – $(A \wedge B \wedge C), (A \wedge B \wedge \neg C), \ldots, (\neg A \wedge \neg B \wedge \neg C)$ – such that the eight weights sum to 1.

The reader may recognize the set of elementary events as the *sample space* in probability textbooks. For example, if A, B, and C correspond to the propositions that coins 1, 2, and 3 will come up heads, then the sample space will consist of the set {HHH, HHT, HTH, ..., TTT}. Indeed, it is sometimes convenient to view the conjunctive formulas corresponding to elementary events as *points* (or *worlds* or *configurations*), and to regard other formulas as *sets* made up of these points. Since every Boolean formula can be expressed as a disjunction of elementary events, and since the elementary events are mutually exclusive, we can always compute $P(S)$ using the additivity axiom (equation (1.3)). Conditional probabilities can be computed the same way, using (1.14). Thus, any joint probability function represents a complete probabilistic model.

Joint distribution functions are mathematical constructs of great importance. They allow us to determine quickly whether we have sufficient information to specify a complete probabilistic model, whether the information we have is consistent, and at what point additional information is needed. The criteria are simply to check (i) whether the information available is sufficient for uniquely determining the probability of every elementary event in the domain and (ii) whether the probabilities add up to 1.

In practice, however, joint distribution functions are rarely specified explicitly. In the analysis of continuous random variables, the distribution functions are given by algebraic expressions such as those describing normal or exponential distributions; for discrete variables, indirect representation methods have been developed where the overall distribution is inferred from local relationships among small groups of variables. Graphical models, the most promising of these representations, provide the basis of discussion throughout this book. Their use and formal characterization will be discussed in the next few sections.

1.1.3 Combining Predictive and Diagnostic Supports

The essence of Bayes's rule (equation 1.13)) is conveniently portrayed using the *odds* and *likelihood ratio* parameters. Dividing (1.13) by the complementary form for $P(\neg H \mid e)$, we obtain

$$\frac{P(H \mid e)}{P(\neg H \mid e)} = \frac{P(e \mid H)}{P(e \mid \neg H)} \frac{P(H)}{P(\neg H)}. \tag{1.15}$$

Defining the *prior odds* on H as

$$O(H) = \frac{P(H)}{P(\neg H)} = \frac{P(H)}{1 - P(H)} \tag{1.16}$$

and the *likelihood ratio* as

$$L(e \mid H) = \frac{P(e \mid H)}{P(e \mid \neg H)}, \tag{1.17}$$

the *posterior odds*

$$O(H \mid e) = \frac{P(H \mid e)}{P(\neg H \mid e)} \tag{1.18}$$

are given by the product

$$O(H \mid e) = L(e \mid H) O(H). \tag{1.19}$$

Thus, Bayes's rule dictates that the overall strength of belief in a hypothesis H, based on both our previous knowledge K and the observed evidence e, should be the product of two factors: the prior odds $O(H)$ and the likelihood ratio $L(e \mid H)$. The first factor measures the *predictive* or *prospective* support accorded to H by the background knowledge alone, while the second represents the *diagnostic* or *retrospective* support given to H by the evidence actually observed.[2]

Strictly speaking, the likelihood ratio $L(e \mid H)$ might depend on the content of the tacit knowledge base K. However, the power of Bayesian techniques comes primarily from the fact that, in causal reasoning, the relationship $P(e \mid H)$ is fairly local: given that H is true, the probability of e can be estimated naturally since it is usually not dependent on many other propositions in the knowledge base. For example, once we establish that a patient suffers from a given disease H, it is natural to estimate the probability that she will develop a certain symptom e. The organization of medical knowledge rests on the paradigm that a symptom is a stable characteristic of the disease and should therefore be fairly independent of other factors, such as epidemic conditions, previous diseases, and faulty diagnostic equipment. For this reason the conditional probabilities $P(e \mid H)$, as opposed to $P(H \mid e)$, are the atomic relationships in Bayesian analysis. The former possess modularity features similar to logical rules. They convey a degree of confidence in rules such as "If H then e," a confidence that persists regardless of what other rules or facts reside in the knowledge base.

Example 1.1.1 Imagine being awakened one night by the shrill sound of your burglar alarm. What is your degree of belief that a burglary attempt has taken place? For

[2] In epidemiology, if H stands for exposure and e stands for disease, then the likelihood ratio L is called the "risk ratio" (Rothman and Greenland 1998, p. 50). Equation (1.18) would then give the odds that a person with disease e was exposed to H.

illustrative purposes we make the following judgments: (a) There is a 95% chance that an attempted burglary will trigger the alarm system – $P(\text{alarm} \mid \text{burglary}) = 0.95$; (b) based on previous false alarms, there is a slight (1%) chance that the alarm will be triggered by a mechanism other than an attempted burglary – $P(\text{alarm} \mid \text{no burglary}) = 0.01$; (c) previous crime patterns indicate that there is a one in ten thousand chance that a given house will be burglarized on a given night – $P(\text{burglary}) = 10^{-4}$.

Putting these assumptions together using (1.19), we obtain

$$O(\text{burglary} \mid \text{alarm}) = L(\text{alarm} \mid \text{burglary})O(\text{burglary})$$

$$= \frac{0.95}{0.01}\frac{10^{-4}}{1-10^{-4}} = 0.0095.$$

So, from

$$P(A) = \frac{O(A)}{1+O(A)} \tag{1.20}$$

we have

$$P(\text{burglary} \mid \text{alarm}) = \frac{0.0095}{1+0.0095} = 0.00941.$$

Thus, the retrospective support imparted to the burglary hypothesis by the alarm evidence has increased its degree of belief almost a hundredfold, from one in ten thousand to 94.1 in ten thousand. The fact that the belief in burglary is still below 1% should not be surprising, given that the system produces a false alarm almost once every three months. Notice that it was not necessary to estimate the absolute values of the probabilities $P(\text{alarm} \mid \text{burglary})$ and $P(\text{alarm} \mid \text{no burglary})$. Only their ratio enters the calculation, so a direct estimate of this ratio could have been used instead.

1.1.4 Random Variables and Expectations

By a *variable* we will mean an attribute, measurement or inquiry that may take on one of several possible outcomes, or *values*, from a specified domain. If we have beliefs (i.e., probabilities) attached to the possible values that a variable may attain, we will call that variable a *random variable*.[3] For example, the color of the shoes that I will wear tomorrow is a random variable named "color," and the values it may take come from the domain {yellow, green, red, ...}.

Most of our analysis will concern a finite set V of random variables (also called *partitions*) where each variable $X \in V$ may take on values from a finite domain D_X. We will use capital letters (e.g., X, Y, Z) for variable names and lowercase letters (x, y, z)

[3] This is a minor generalization of textbook definition, according to which a random variable is a mapping from the sample space (e.g., the set of elementary events) to the real line. In our definition, the mapping is from the sample space to any set of objects called "values," which may or may not be ordered.

as generic symbols for specific values taken by the corresponding variables. For example, if X stands for the color of an object, then x will designate any possible choice of an element from the set {yellow, green, red, ...}. Clearly, the proposition $X =$ yellow describes an *event*, namely, a subset of possible states of affair that satisfy the proposition "the color of the object is yellow." Likewise, each variable X can be viewed as a partition of the states of the world, since the statement $X = x$ defines a set of exhaustive and mutually exclusive sets of states, one for each value of x.

In most of our discussions, we will not make notational distinction between variables and sets of variables, because a set of variables essentially defines a compound variable whose domain is the Cartesian product of the domains of the individual constituents in the set. Thus, if Z stands for the set $\{X, Y\}$ then z stands for pairs (x, y) such that $x \in D_X$ and $y \in D_Y$. When the distinction between variables and sets of variables requires special emphasis, indexed letters (say, $X_1, X_2, \ldots, X_n$ or $V_1, V_2, \ldots, V_n$) will be used to represent individual variables.

We shall consistently use the abbreviation $P(x)$ for the probabilities $P(X = x)$, $x \in D_X$. Likewise, if Z stands for the set $\{X, Y\}$, then $P(z)$ will be defined as

$$P(z) \triangleq P(Z = z) = P(X = x, Y = y), \quad x \in D_X, \ y \in D_Y.$$

When the values of a random variable X are real numbers, X is called a *real* random variable; one can then define the *mean* or *expected value* of X as

$$E(X) \triangleq \sum_x xP(x) \tag{1.21}$$

and the *conditional mean* of X, given event $Y = y$, as

$$E(X \mid y) \triangleq \sum_x xP(x \mid y). \tag{1.22}$$

The expectation of any function g of X is defined as

$$E[g(X)] \triangleq \sum_x g(x)P(x). \tag{1.23}$$

In particular, the function $g(X) = (X - E(X))^2$ has received much attention; its expectation is called the *variance* of X, denoted σ_X^2;

$$\sigma_X^2 \triangleq E[(X - E(X))^2].$$

The conditional mean $E(X \mid Y = y)$ is the *best estimate* of X, given the observation $Y = y$, in the sense of minimizing the expected square error $\sum_x (x - \hat{x})^2 P(x \mid y)$ over all $\hat{x}$.

The expectation of a function $g(X, Y)$ of two variables, X and Y, requires the joint probability $P(x, y)$ and is defined as

$$E[g(X, Y)] \triangleq \sum_{x,y} g(x, y)P(x, y)$$

(cf. equation (1.23)). Of special importance is the expectation of the product ($g(X, Y) = (X - E(X))(Y - E(Y))$, which is known as the *covariance* of X and Y,

$$\sigma_{XY} \triangleq E[(X - E(X))(Y - E(Y))],$$

and which is often normalized to yield the *correlation coefficient*

$$\rho_{XY} = \frac{\sigma_{XY}}{\sigma_X \sigma_Y}$$

and the *regression coefficient*

$$r_{XY} \triangleq \rho_{XY} \frac{\sigma_X}{\sigma_Y} = \frac{\sigma_{XY}}{\sigma_Y^2}.$$

The *conditional* variance, covariance, and correlation coefficient, given $Z = z$, are defined in a similar manner, using the conditional distribution $P(x, y \mid z)$ in taking expectations. In particular, the *conditional correlation coefficient*, given $Z = z$, is defined as

$$\rho_{XY|z} = \frac{\sigma_{XY|z}}{\sigma_{X|z}\sigma_{Y|z}}. \tag{1.24}$$

Additional properties, specific to normal distributions, will be reviewed in Chapter 5 (Section 5.2.1).

The foregoing definitions apply to discrete random variables – that is, variables that take on finite or denumerable sets of values on the real line. The treatment of expectation and correlation is more often applied to continuous random variables, which are characterized by a *density function* $f(x)$ defined as follows:

$$P(a \leq X \leq b) = \int_a^b f(x)\, dx$$

for any two real numbers a and b with $a < b$. If X is discrete then $f(x)$ coincides with the probability function $P(x)$, once we interpret the integral through the translation

$$\int_{-\infty}^{\infty} f(x)\, dx \iff \sum_x P(x). \tag{1.25}$$

Readers accustomed to continuous analysis should bear this translation in mind whenever summation is used in this book. For example, the expected value of a continuous random variable X can be obtained from (1.21), to read

$$E(X) = \int_{-\infty}^{\infty} x f(x)\, dx,$$

with analogous translations for the variance, correlation, and so forth.

We now turn to define *conditional independence* relationships among variables, a central notion in the analysis of causal models.

1.1.5 Conditional Independence and Graphoids

Definition 1.1.2 (Conditional Independence)
Let $V = \{V_1, V_2, \ldots\}$ be a finite set of variables. Let $P(\cdot)$ be a joint probability function over the variables in V, and let X, Y, Z stand for any three subsets of variables in V. The sets X and Y are said to be conditionally independent given *Z if*

$$P(x \mid y, z) = P(x \mid z) \quad \textit{whenever} \quad P(y, z) > 0. \tag{1.26}$$

In words, learning the value of Y does not provide additional information about X, once we know Z. (Metaphorically, Z "screens off" X from Y.)

Equation (1.26) is a terse way of saying the following: For any configuration x of the variables in the set X and for any configurations y and z of the variables in Y and Z satisfying $P(Y = y,\ Z = z) > 0$, we have

$$P(X = x \mid Y = y,\ Z = z) = P(X = x \mid Z = z). \tag{1.27}$$

We will use Dawid's (1979) notation $(X \perp\!\!\!\perp Y \mid Z)_P$ or simply $(X \perp\!\!\!\perp Y \mid Z)$ to denote the conditional independence of X and Y given Z; thus,

$$(X \perp\!\!\!\perp Y \mid Z)_P \quad \text{iff} \quad P(x \mid y, z) = P(x \mid z) \tag{1.28}$$

for all values x, y, z such that $P(y, z) > 0$. Unconditional independence (also called *marginal independence*) will be denoted by $(X \perp\!\!\!\perp Y \mid \emptyset)$; that is,

$$(X \perp\!\!\!\perp Y \mid \emptyset) \text{ iff } P(x \mid y) = P(x) \quad \text{whenever } P(y) > 0 \tag{1.29}$$

("iff" is shorthand for "if and only if"). Note that $(X \perp\!\!\!\perp Y \mid Z)$ implies the conditional independence of all pairs of variables $V_i \in X$ and $V_j \in Y$, but the converse is not necessarily true.

The following is a (partial) list of properties satisfied by the conditional independence relation $(X \perp\!\!\!\perp Y \mid Z)$.

Symmetry: $(X \perp\!\!\!\perp Y \mid Z) \Longrightarrow (Y \perp\!\!\!\perp X \mid Z)$.

Decomposition: $(X \perp\!\!\!\perp YW \mid Z) \Longrightarrow (X \perp\!\!\!\perp Y \mid Z)$.

Weak union: $(X \perp\!\!\!\perp YW \mid Z) \Longrightarrow (X \perp\!\!\!\perp Y \mid ZW)$.

Contraction: $(X \perp\!\!\!\perp Y \mid Z)\ \&\ (X \perp\!\!\!\perp W \mid ZY) \Longrightarrow (X \perp\!\!\!\perp YW \mid Z)$.

Intersection: $(X \perp\!\!\!\perp W \mid ZY)\ \&\ (X \perp\!\!\!\perp Y \mid ZW) \Longrightarrow (X \perp\!\!\!\perp YW \mid Z)$.

(Intersection is valid in strictly positive probability distributions.)

The proof of these properties can be derived by elementary means from (1.28) and the basic axioms of probability theory.[4] These properties were called *graphoid axioms* by

[4] These properties were first introduced by Dawid (1979) and Spohn (1980) in a slightly different form, and were independently proposed by Pearl and Paz (1987) to characterize the relationships between graphs and informational relevance.

Pearl and Paz (1987) and Geiger et al. (1990) and have been shown to govern the concept of informational relevance in a wide variety of interpretations (Pearl 1988b). In graphs, for example, these properties are satisfied if we interpret $(X \perp\!\!\!\perp Y \mid Z)$ to mean "all paths from a subset X of nodes to a subset Y of nodes are intercepted by a subset Z of nodes."

The intuitive interpretation of the graphoid axioms is as follows (Pearl 1988b, p. 85). The *symmetry* axiom states that, in any state of knowledge Z, if Y tells us nothing new about X then X tells us nothing new about Y. The *decomposition* axiom asserts that if two combined items of information are judged irrelevant to X, then each separate item is irrelevant as well. The *weak union* axiom states that learning irrelevant information W cannot help the irrelevant information Y become relevant to X. The *contraction* axiom states that if we judge W irrelevant to X after learning some irrelevant information Y, then W must have been irrelevant before we learned Y. Together, the weak union and contraction properties mean that irrelevant information should not alter the relevance status of other propositions in the system; what was relevant remains relevant, and what was irrelevant remains irrelevant. The *intersection* axiom states that if Y is irrelevant to X when we know W and if W is irrelevant to X when we know Y, then neither W nor Y (nor their combination) is relevant to X.

1.2 GRAPHS AND PROBABILITIES

1.2.1 Graphical Notation and Terminology

A graph consists of a set V of *vertices* (or *nodes*) and a set E of *edges* (or *links*) that connect some pairs of vertices. The vertices in our graphs will correspond to variables (whence the common symbol V) and the edges will denote a certain relationship that holds in pairs of variables, the interpretation of which will vary with the application. Two variables connected by an edge are called *adjacent*.

Each edge in a graph can be either directed (marked by a single arrowhead on the edge), or undirected (unmarked links). In some applications we will also use "bidirected" edges to denote the existence of unobserved common causes (sometimes called *confounders*). These edges will be marked as dotted curved arcs with two arrowheads (see Figure 1.1(a)). If all edges are directed (see Figure 1.1(b)), we then have a *directed* graph. If we strip away all arrowheads from the edges in a graph G, the resultant undirected graph is called the *skeleton* of G. A *path* in a graph is a sequence of edges (e.g., $((W, Z), (Z, Y), (Y, X), (X, Z))$ in Figure 1.1(a)) such that each edge starts with the vertex ending the preceding edge. In other words, a path is any unbroken, nonintersecting route traced out along the edges in a graph, which may go either along or against the arrows. If every edge in a path is an arrow that points from the first to the second vertex of the pair, we have a *directed path*. In Figure 1.1(a), for example, the path $((W, Z), (Z, Y))$ is directed but the paths $((W, Z), (Z, Y), (Y, X))$ and $((W, Z), (Z, X))$ are not. If there exists a path between two vertices in a graph then the two vertices are said to be *connected;* else they are *disconnected.*

Directed graphs may include directed cycles (e.g., $X \rightarrow Y$, $Y \rightarrow X$), representing mutual causation or feedback processes, but not self-loops (e.g., $X \rightarrow X$). A graph (like the two in Figure 1.1) that contains no directed cycles is called *acyclic.* A graph that is

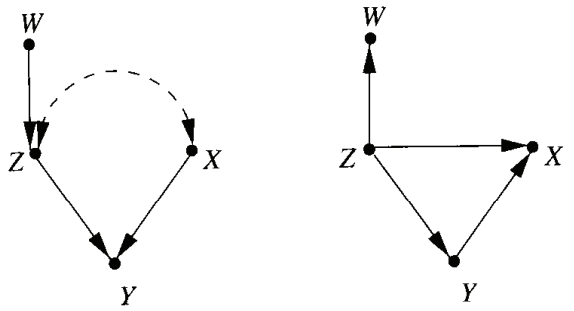

Figure 1.1 (a) A graph containing both directed and bidirected edges. (b) A directed acyclic graph (DAG) with the same skeleton as (a).

both directed and acyclic (Figure 1.1(b)) is called a *directed acyclic graph* (DAG), and such graphs will occupy much of our discussion of causality. We make free use of the terminology of kinship (e.g., *parents, children, descendants, ancestors, spouses*) to denote various relationships in a graph. These kinship relations are defined along the full arrows in the graph, including arrows that form directed cycles but ignoring bidirected and undirected edges. In Figure 1.1(a), for example, Y has two parents (X and Z), three ancestors (X, Z, and W), and no children, while X has no parents (hence, no ancestors), one spouse (Z), and one child (Y). A *family* in a graph is a set of nodes containing a node and all its parents. For example, $\{W\}$, $\{Z, W\}$, $\{X\}$, and $\{Y, Z, X\}$ are the families in the graph of Figure 1.1(a).

A node in a directed graph is called a *root* if it has no parents and a *sink* if it has no children. Every DAG has at least one root and at least one sink. A connected DAG in which every node has at most one parent is called a *tree,* and a tree in which every node has at most one child is called a *chain.* A graph in which every pair of nodes is connected by an edge is called *complete.* The graph in Figure 1.1(a), for instance, is connected but not complete, because the pairs (W, X) and (W, Y) are not adjacent.

1.2.2 Bayesian Networks

The role of graphs in probabilistic and statistical modeling is threefold:

1. to provide convenient means of expressing substantive assumptions;
2. to facilitate economical representation of joint probability functions; and
3. to facilitate efficient inferences from observations.

We will begin our discussion with item 2.

Consider the task of specifying an arbitrary joint distribution, $P(x_1, \ldots, x_n)$, for n dichotomous variables. To store $P(x_1, \ldots, x_n)$ explicitly would require a table with 2^n entries, an unthinkably large number by any standard. Substantial economy can be achieved when each variable depends on just a small subset of other variables. Such dependence information permits us to decompose large distribution functions into several small distributions – each involving a small subset of variables – and then to piece them together coherently to answer questions of global nature. Graphs play an essential role in such decomposition, for they provide a vivid representation of the sets of variables that are relevant to each other in any given state of knowledge.

Both directed and undirected graphs have been used by researchers to facilitate such decomposition. Undirected graphs, sometimes called *Markov networks* (Pearl 1988b), are used primarily to represent symmetrical spatial relationships (Isham 1981; Cox and Wermuth 1996; Lauritzen 1996). Directed graphs, especially DAGs, have been used to represent causal or temporal relationships (Lauritzen 1982; Wermuth and Lauritzen 1983; Kiiveri et al. 1984) and came to be known as *Bayesian networks,* a term coined in Pearl (1985) to emphasize three aspects: (1) the subjective nature of the input information; (2) the reliance on Bayes's conditioning as the basis for updating information; and (3) the distinction between causal and evidential modes of reasoning, a distinction that underscores Thomas Bayes's paper of 1763. Hybrid graphs (involving both directed and undirected edges) have also been proposed for statistical modeling (Wermuth and Lauritzen 1990), but in this book our main interest will focus on directed acyclic graphs, with occasional use of directed cyclic graphs to represent feedback cycles.

The basic decomposition scheme offered by directed acyclic graphs can be illustrated as follows. Suppose we have a distribution P defined on n discrete variables, which we may order arbitrarily as $X_1, X_2, \ldots, X_n$. The chain rule of probability calculus (equation (1.12)) always permits us to decompose P as a product of n conditional distributions:

$$P(x_1, \ldots, x_n) = \prod_j P(x_j \mid x_1, \ldots, x_{j-1}). \tag{1.30}$$

Now suppose that the conditional probability of some variable X_j is not sensitive to all the predecessors of X_j but only to a small subset of those predecessors. In other words, suppose that X_j is independent of all other predecessors, once we know the value of a select group of predecessors called PA_j. We can then write

$$P(x_j \mid x_1, \ldots, x_{j-1}) = P(x_j \mid pa_j) \tag{1.31}$$

in the product of (1.30), which will considerably simplify the input information required. Instead of specifying the probability of X_j conditional on all possible realizations of its predecessors $X_1, \ldots, X_{j-1}$, we need only concern ourselves with the possible realizations of the set PA_j. The set PA_j is called the *Markovian parents* of X_j, or *parents* for short. The reason for the name becomes clear when we build graphs around this concept.

Definition 1.2.1 (Markovian Parents)
Let $V = \{X_1, \ldots, X_n\}$ be an ordered set of variables, and let $P(v)$ be the joint probability distribution on these variables. A set of variables PA_j is said to be Markovian parents *of X_j if PA_j is a minimal set of predecessors of X_j that renders X_j independent of all its other predecessors. In other words, PA_j is any subset of $\{X_1, \ldots, X_{j-1}\}$ satisfying*

$$P(x_j \mid pa_j) = P(x_j \mid x_1, \ldots, x_{j-1}) \tag{1.32}$$

and such that no proper subset of PA_j satisfies (1.32).[5]

[5] Lowercase symbols (e.g., x_j, pa_j) denote particular realizations of the corresponding variables (e.g., X_j, PA_j).

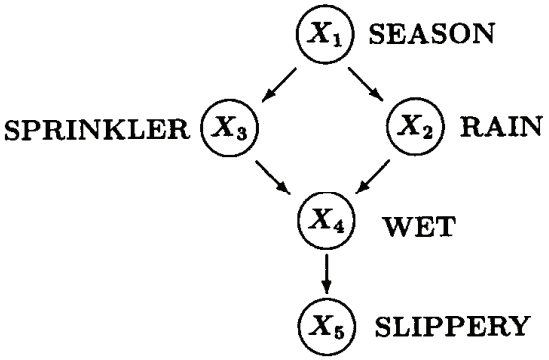

Figure 1.2 A Bayesian network representing dependencies among five variables.

Definition 1.2.1 assigns to each variable X_j a select set PA_j of preceding variables that are sufficient for determining the probability of X_j; knowing the values of other preceding variables is redundant once we know the values pa_j of the parent set PA_j. This assignment can be represented in the form of a DAG in which variables are represented by nodes and arrows are drawn from each node of the parent set PA_j toward the child node X_j. Definition 1.2.1 also suggests a simple recursive method for constructing such a DAG: Starting with the pair (X_1, X_2), we draw an arrow from X_1 to X_2 if and only if the two variables are dependent. Continuing to X_3, we draw no arrow in case X_3 is independent of $\{X_1, X_2\}$; otherwise, we examine whether X_2 screens off X_3 from X_1 or X_1 screens off X_3 from X_2. In the first case, we draw an arrow from X_2 to X_3; in the second, we draw an arrow from X_1 to X_3. If no screening condition is found, we draw arrows to X_3 from both X_1 and X_2. In general: at the jth stage of the construction, we select any minimal set of X_j's predecessors that screens off X_j from its other predecessors (as in equation (1.32)), call this set PA_j and draw an arrow from each member in PA_j to X_j. The result is a directed acyclic graph, called a Bayesian network, in which an arrow from X_i to X_j assigns X_i as a Markovian parent of X_j, consistent with Definition 1.2.1.

It can be shown (Pearl 1988b) that the set PA_j is unique whenever the distribution $P(v)$ is strictly positive (i.e., involving no logical or definitional constraints), so that every configuration v of variables, no matter how unlikely, has some finite probability of occurring. Under such conditions, the Bayesian network associated with $P(v)$ is unique, given the ordering of the variables.

Figure 1.2 illustrates a simple yet typical Bayesian network. It describes relationships among the season of the year (X_1), whether rain falls (X_2), whether the sprinkler is on (X_3), whether the pavement would get wet (X_4), and whether the pavement would be slippery (X_5). All variables in this figure are binary (taking a value of either true or false) except for the root variable X_1, which can take one of four values: spring, summer, fall, or winter. The network was constructed in accordance with Definition 1.2.1, using causal intuition as a guide. The absence of a direct link between X_1 and X_5, for example, captures our understanding that the influence of seasonal variations on the slipperiness of the pavement is mediated by other conditions (e.g., the wetness of the pavement). This intuition coincides with the independence condition of (1.32), since knowing X_4 renders X_5 independent of $\{X_1, X_2, X_3\}$.

The construction implied by Definition 1.2.1 defines a Bayesian network as a carrier of conditional independence relationships along the order of construction. Clearly, every distribution satisfying (1.32) must decompose (using the chain rule of (1.30)) into the product

$$P(x_1, \ldots, x_n) = \prod_i P(x_i \mid pa_i). \tag{1.33}$$

For example, the DAG in Figure 1.2 induces the decomposition

$$P(x_1, x_2, x_3, x_4, x_5) = P(x_1)P(x_2 \mid x_1)P(x_3 \mid x_1)P(x_4 \mid x_2, x_3)P(x_5 \mid x_4). \tag{1.34}$$

The product decomposition in (1.33) is no longer order-specific since, given P and G, we can test whether P decomposes into the product given by (1.33) without making any reference to variable ordering. We therefore conclude that a necessary condition for a DAG G to be a Bayesian network of probability distribution P is for P to admit the product decomposition dictated by G, as given in (1.33).

Definition 1.2.2 (Markov Compatibility)
If a probability function P admits the factorization of (1.33) *relative to DAG G, we say that G* represents *P, that G and P are* compatible, *or that P is* Markov relative *to G.*[6]

Ascertaining compatibility between DAGs and probabilities is important in statistical modeling primarily because compatibility is a necessary and sufficient condition for a DAG G to *explain* a body of empirical data represented by P, that is, to describe a stochastic process capable of *generating* P (e.g. Pearl 1988b, pp. 210–23). If the value of each variable X_i is chosen at random with some probability $P_i(x_i \mid pa_i)$, based solely on the values pa_i previously chosen for PA_i, then the overall distribution P of the generated instances $x_1, x_2, \ldots, x_n$ will be Markov relative to G. Conversely, if P is Markov relative to G then there exists a set of probabilities $P_i(x_i \mid pa_i)$ according to which we can choose the value of each variable X_i such that the distribution of the generated instances $x_1, x_2, \ldots, x_n$ will be equal to P. (In fact, the correct choice of $P_i(x_i \mid pa_i)$ would be simply $P(x_i \mid pa_i)$.)

A convenient way of characterizing the set of distributions compatible with a DAG G is to list the set of (conditional) independencies that each such distribution must satisfy. These independencies can be read off the DAG by using a graphical criterion called *d-separation* (Pearl 1988b; the *d* denotes *directional*), which will play a major role in many discussions in this book.

1.2.3 The *d*-Separation Criterion

Consider three disjoint sets of variables, X, Y, and Z, which are represented as nodes in a directed acyclic graph G. To test whether X is independent of Y given Z in any distribution compatible with G, we need to test whether the nodes corresponding to variables Z "block" all paths from nodes in X to nodes in Y. By *path* we mean a sequence of consecutive edges (of any directionality) in the graph, and blocking is to be interpreted as stopping the flow of information (or of dependency) between the variables that are connected by such paths, as defined next.

Definition 1.2.3 (*d*-Separation)
*A path p is said to be d-*separated (*or* blocked) *by a set of nodes Z if and only if*

[6] The latter expression seems to gain strength in recent literature (e.g. Spirtes et al. 1993; Lauritzen 1996). Pearl (1988b, p. 116) used "*G* is an *I-map* of *P*."

1. *p contains a chain* $i \rightarrow m \rightarrow j$ *or a fork* $i \leftarrow m \rightarrow j$ *such that the middle node m is in Z, or*
2. *p contains an inverted fork* (*or* collider) $i \rightarrow m \leftarrow j$ *such that the middle node m is not in Z and such that no descendant of m is in Z.*

A set Z is said to d-separate X from Y if and only if Z blocks every path from a node in X to a node in Y.

The intuition behind d-separation is simple and can best be recognized if we attribute causal meaning to the arrows in the graph. In causal chains $i \rightarrow m \rightarrow j$ and causal forks $i \leftarrow m \rightarrow j$, the two extreme variables are marginally dependent but become independent of each other (i.e., blocked) once we condition on (i.e., know the value of) the middle variable. Figuratively, conditioning on m appears to "block" the flow of information along the path, since learning about i has no effect on the probability of j, given m. Inverted forks $i \rightarrow m \leftarrow j$, representing two causes having a common effect, act the opposite way; if the two extreme variables are (marginally) independent, they will become dependent (i.e., connected through unblocked path) once we condition on the middle variable (i.e., the common effect) or any of its descendants. This can be confirmed in the context of Figure 1.2. Once we know the season, X_3 and X_2 are independent (assuming that sprinklers are set in advance, according to the season); whereas finding that the pavement is wet or slippery renders X_2 and X_3 dependent, because refuting one of these explanations increases the probability of the other.

In Figure 1.2, $X = \{X_2\}$ and $Y = \{X_3\}$ are d-separated by $Z = \{X_1\}$, because both paths connecting X_2 and X_3 are blocked by Z. The path $X_2 \leftarrow X_1 \rightarrow X_3$ is blocked because it is a fork in which the middle node X_1 is in Z, while the path $X_2 \rightarrow X_4 \leftarrow X_3$ is blocked because it is an inverted fork in which the middle node X_4 and all its descendants are outside Z. However, X and Y are not d-separated by the set $Z' = \{X_1, X_5\}$: the path $X_2 \rightarrow X_4 \leftarrow X_3$ (an inverted fork) is not blocked by Z', since X_5, a descendant of the middle node X_4, is in Z'. Metaphorically, learning the value of the consequence X_5 renders its causes X_2 and X_3 dependent, as if a pathway were opened along the arrows converging at X_4.

At first glance, readers might find it a bit odd that conditioning on a node not lying on a blocked path may unblock the path. However, this corresponds to a general pattern of causal relationships: observations on a common consequence of two independent causes tend to render those causes dependent, because information about one of the causes tends to make the other more or less likely, given that the consequence has occurred. This pattern is known as *selection bias* or *Berkson's paradox* in the statistical literature (Berkson 1946) and as the *explaining away effect* in artificial intelligence (Kim and Pearl 1983). For example, if the admission criteria to a certain graduate school call for either high grades as an undergraduate or special musical talents, then these two attributes will be found to be correlated (negatively) in the student population of that school, even if these attributes are uncorrelated in the population at large. Indeed, students with low grades are likely to be exceptionally gifted in music, which explains their admission to graduate school.

Figure 1.3 illustrates more elaborate examples of d-separation: example (a) contains a bidirected arc $Z_1 \dashleftarrow\dashrightarrow Z_3$ and (b) involves a directed cycle $X \rightarrow Z_2 \rightarrow Z_1 \rightarrow X$. In

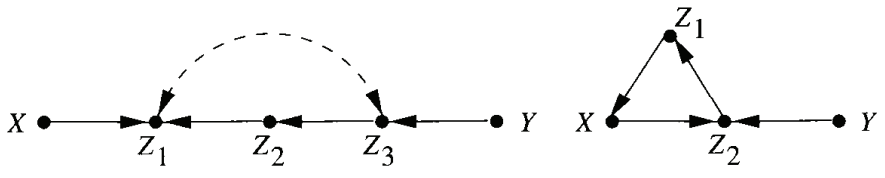

Figure 1.3 Graphs illustrating d-separation. In (a), X and Y are d-separated given Z_2 and d-connected given Z_1. In (b), X and Y cannot be d-separated by any set of nodes.

Figure 1.3(a), the two paths between X and Y are blocked when none of $\{Z_1, Z_2, Z_3\}$ is measured. However, the path $X \rightarrow Z_1 \dashleftarrow\dashrightarrow Z_3 \leftarrow Y$ becomes unblocked when Z_1 is measured. This is so because Z_1 unblocks the "colliders" at both Z_1 and Z_3; the first because Z_1 is the collision node of the collider, the second because Z_1 is a descendant of the collision node Z_3 through the path $Z_1 \leftarrow Z_2 \leftarrow Z_3$. In Figure 1.3(b), X and Y cannot be d-separated by any set of nodes, including the empty set. If we condition on Z_2, we block the path $X \leftarrow Z_1 \leftarrow Z_2 \leftarrow Y$ yet unblock the path $X \rightarrow Z_2 \leftarrow Y$. If we condition on Z_1, we again block the path $X \leftarrow Z_1 \leftarrow Z_2 \leftarrow Y$ and unblock the path $X \rightarrow Z_2 \leftarrow Y$, because Z_1 is a descendant of the collision node Z_2.

The connection between d-separation and conditional independence is established through the following theorem due to Verma and Pearl (1988; see also Geiger et al. 1990).

Theorem 1.2.4 (Probabilistic Implications of d-Separation)
If sets X and Y are d-separated by Z in a DAG G, then X is independent of Y conditional on Z in every distribution compatible with G. Conversely, if X and Y are not *d-separated by Z in a DAG G, then X and Y are dependent conditional on Z in at least one distribution compatible with G.*

The converse part of Theorem 1.2.4 is in fact much stronger – the absence of d-separation implies dependence in *almost all* distributions compatible with G. The reason is that a precise tuning of parameters is required to generate independency along an unblocked path in the diagram, and such tuning is unlikely to occur in practice (see Spirtes et al. 1993 and Sections 2.4 and 2.9.1).

In order to distinguish between the probabilistic notion of conditional independence $(X \perp\!\!\!\perp Y \mid Z)_P$ and the graphical notion of d-separation, for the latter we will use the notation $(X \perp\!\!\!\perp Y \mid Z)_G$. We can thereby express Theorem 1.2.4 more succinctly as follows.

Theorem 1.2.5
For any three disjoint subsets of nodes (X, Y, Z) in a DAG G and for all probability functions P, we have:

(i) $(X \perp\!\!\!\perp Y \mid Z)_G \Longrightarrow (X \perp\!\!\!\perp Y \mid Z)_P$ *whenever G and P are compatible; and*

(ii) *if $(X \perp\!\!\!\perp Y \mid Z)_P$ holds in all distributions compatible with G, it follows that $(X \perp\!\!\!\perp Y \mid Z)_G$.*

An alternative test for d-separation has been devised by Lauritzen et al. (1990), based on the notion of ancestral graphs. To test for $(X \perp\!\!\!\perp Y \mid Z)_G$, delete from G all nodes except those in $\{X, Y, Z\}$ and their ancestors, connect by an edge every pair of nodes that share

a common child, and remove all arrows from the arcs. Then $(X \perp\!\!\!\perp Y \mid Z)_G$ holds if and only if Z intercepts all paths between X and Y in the resulting undirected graph.

Note that the ordering with which the graph was constructed does not enter into the d-separation criterion; it is only the topology of the resulting graph that determines the set of independencies that the probability P must satisfy. Indeed, the following theorem can be proven (Pearl 1988b, p. 120).

Theorem 1.2.6 (Ordered Markov Condition)
A necessary and sufficient condition for a probability distribution P to be Markov relative a DAG G is that, conditional on its parents in G, each variable be independent of all its predecessors in some ordering of the variables that agrees with the arrows of G.

A consequence of this theorem is an order-independent criterion for determining whether a given probability P is Markov relative to a given DAG G.

Theorem 1.2.7 (Parental Markov Condition)
A necessary and sufficient condition for a probability distribution P to be Markov relative a DAG G is that every variable be independent of all its nondescendants (in G), conditional on its parents.

This condition, which Kiiveri et al. (1984) and Lauritzen (1996) called the "local" Markov condition, is sometimes taken as the definition of Bayesian networks (Howard and Matheson 1981). In practice, however, the ordered Markov condition is easier to use.

Another important property that follows from d-separation is a criterion for determining whether two given DAGs are observationally equivalent – that is, whether every probability distribution that is compatible with one of the DAGs is also compatible with the other.

Theorem 1.2.8 (Observational Equivalence)
Two DAGs are observationally equivalent if and only if they have the same skeletons and the same sets of v-structures, that is, two converging arrows whose tails are not connected by an arrow (Verma and Pearl 1990).[7]

Observational equivalence places a limit on our ability to infer directionality from probabilities alone. Two networks that are observationally equivalent cannot be distinguished without resorting to manipulative experimentation or temporal information. For example, reversing the direction of the arrow between X_1 and X_2 in Figure 1.2 would neither introduce nor destroy a v-structure. Therefore, this reversal yields an observationally equivalent network, and the directionality of the link $X_1 \rightarrow X_2$ cannot be determined from probabilistic information. The arrows $X_2 \rightarrow X_4$ and $X_4 \rightarrow X_5$, however, are of different nature; there is no way of reversing their directionality without creating a new v-structure. Thus, we see that some probability functions P (such as the one responsible for the construction of the Bayesian network in Figure 1.2), when unaccompanied

[7] An identical criterion was independently derived by Frydenberg (1990) in the context of chain graphs, where strict positivity is assumed.

by temporal information, can constrain the directionality of some arrows in the graph. The precise meaning of such directionality constraints – and the possibility of using these constraints for inferring causal relationships from data – will be formalized in Chapter 2.

1.2.4 Inference with Bayesian Networks

Bayesian networks were developed in the early 1980s to facilitate the tasks of prediction and "abduction" in artificial intelligence (AI) systems. In these tasks, it is necessary to find a coherent interpretation of incoming observations that is consistent with both the observations and the prior information at hand. Mathematically, the task boils down to the computation of $P(y \mid x)$, where X is a set of observations and Y is a set of variables that are deemed important for prediction or diagnosis.

Given a joint distribution P, the computation of $P(y \mid x)$ is conceptually trivial and invokes straightforward application of Bayes's rule to yield

$$P(y \mid x) = \frac{\sum_s P(y, x, s)}{\sum_{y,s} P(y, x, s)}, \tag{1.35}$$

where S stands for the set of all variables *excluding* X and Y. Because every Bayesian network defines a joint probability P (given by the product in (1.33)), it is clear that $P(y \mid x)$ can be computed from a DAG G and the conditional probabilities $P(x_i \mid pa_i)$ defined on the families of G.

The challenge, however, lies in performing these computations efficiently and within the representation level provided by the network topology. The latter is important in systems that generate explanations for their reasoning processes. Although such inference techniques are not essential to our discussion of causality, we will nevertheless survey them briefly, for they demonstrate (i) the effectiveness of organizing probabilistic knowledge in the form of graphs and (ii) the feasibility of performing coherent probabilistic calculations (and approximations thereof) on such organization. Details can be found in the references cited.

The first algorithms proposed for probabilistic calculations in Bayesian networks used message-passing architecture and were limited to trees (Pearl 1982; Kim and Pearl 1983). With this technique, each variable is assigned a simple processor and permitted to pass messages asynchronously with its neighbors until equilibrium is achieved (in a finite number of steps). Methods have since been developed that extend this tree propagation (and some of its synchronous variants) to general networks. Among the most popular are Lauritzen and Spiegelhalter's (1988) method of join-tree propagation and the method of cut-set conditioning (Pearl 1988b, pp. 204–10; Jensen 1996). In the join-tree method, we decompose the network into clusters (e.g. cliques) that form tree structures and then treat the set variables in each cluster as a compound variable that is capable of passing messages to its neighbors (which are also compound variables). For example, the network of Figure 1.2 can be structured as a Markov-compatible chain of three clusters:

$$\{X_1, X_2, X_3\} \rightarrow \{X_2, X_3, X_4\} \rightarrow \{X_4, X_5\}.$$

In the cut-set conditioning method, a set of variables is instantiated (given specific values) such that the remaining network forms a tree. The propagation is then performed on that tree, and a new instantiation chosen, until all instantiations have been exhausted; the results are then averaged. In Figure 1.2, for example, if we instantiate X_1 to any specific value (say, X_1 = summer), then we break the pathway between X_2 and X_3 and the remaining network becomes tree-structured. The main advantage of the cut-set conditioning method is that its storage-space requirement is minimal (linear in the size of the network), whereas that of the join-tree method might be exponential. Hybrid combinations of these two basic algorithms have also been proposed (Shachter et al. 1994; Dechter 1996) to allow flexible trade-off of storage versus time.

Whereas inference in general networks is "NP-hard" (Cooper 1990), the computational complexity for each of the methods cited here can be estimated prior to actual processing. When the estimates exceed reasonable bounds, an approximation method such as stochastic simulation (Pearl 1988b, pp. 210–23) can be used instead. This method exploits the topology of the network to perform Gibbs sampling on local subsets of variables, sequentially as well as concurrently.

Additional properties of DAGs and their applications to evidential reasoning in expert systems are discussed in Pearl (1988b), Lauritzen and Spiegelhalter (1988), Pearl (1993a), Spiegelhalter et al. (1993), Heckerman et al. (1995), and Shafer (1996b, 1997).

1.3 CAUSAL BAYESIAN NETWORKS

The interpretation of direct acyclic graphs as carriers of independence assumptions does not necessarily imply causation; in fact, it will be valid for any set of recursive independencies along any ordering of the variables, not necessarily causal or chronological. However, the ubiquity of DAG models in statistical and AI applications stems (often unwittingly) primarily from their causal interpretation – that is, as a system of processes, one per family, that could account for the generation of the observed data. It is this causal interpretation that explains why DAG models are rarely used in any variable ordering other than those which respect the direction of time and causation.

The advantages of building DAG models around causal rather than associational information are several. First, the judgments required in the construction of the model are more meaningful, more accessible and hence more reliable. The reader may appreciate this point by attempting to construct a DAG representation for the associations in Figure 1.2 along the ordering (X_5, X_1, X_3, X_2, X_4). Such exercises illustrate not only that some independencies are more vividly accessible to the mind than others but also that conditional independence judgments are accessible (hence reliable) only when they are anchored onto more fundamental building blocks of our knowledge, such as causal relationships. In the example of Figure 1.2, our willingness to assert that X_5 is independent of X_2 and X_3 once we know X_4 (i.e., whether the pavement is wet) is defensible because we can easily translate the assertion into one involving causal relationships: that the *influence* of rain and sprinkler on slipperiness is *mediated* by the wetness of the pavement. Dependencies that are not supported by causal links are considered odd or spurious and are even branded "paradoxical" (see the discussion of Berkson's paradox, Section 1.2.3).

We will have several opportunities throughout this book to demonstrate the primacy of causal over associational knowledge. In extreme cases, we will see that people tend to ignore probabilistic information altogether and attend to causal information instead (see Section 6.1.4).[8] This puts into question the ruling paradigm of graphical models in statistics (Wermuth and Lauritzen 1990; Cox and Wermuth 1996), according to which conditional independence assumptions are the primary vehicle for expressing substantive knowledge.[9] It seems that if conditional independence judgments are byproducts of stored causal relationships, then tapping and representing those relationships directly would be a more natural and more reliable way of expressing what we know or believe about the world. This is indeed the philosophy behind causal Bayesian networks.

The second advantage of building Bayesian networks on causal relationships – one that is basic to the understanding of causal organizations – is the ability to represent and respond to external or spontaneous *changes*. Any local reconfiguration of the mechanisms in the environment can be translated, with only minor modification, into an isomorphic reconfiguration of the network topology. For example, to represent a disabled sprinkler in the story of Figure 1.2, we simply delete from the network all links incident to the node Sprinkler. To represent the policy of turning the sprinkler off if it rains, we simply add a link between Rain and Sprinkler and revise $P(x_3 \mid x_1, x_2)$. Such changes would require much greater remodeling efforts if the network were not constructed along the causal direction but instead along (say) the order $(X_5, X_1, X_3, X_2, X_4)$. This remodeling flexibility may well be cited as the ingredient that marks the division between deliberative and reactive agents and that enables the former to manage novel situations instantaneously, without requiring training or adaptation.

1.3.1 Causal Networks as Oracles for Interventions

The source of this flexibility rests on the assumption that each parent–child relationship in the network represents a stable and autonomous physical mechanism – in other words, that it is conceivable to change one such relationship *without* changing the others. Organizing one's knowledge in such *modular* configurations permits one to predict the effect of external interventions with minimum of extra information. Indeed, causal models (assuming they are valid) are much more informative than probability models. A joint distribution tells us how probable events are and how probabilities would change with subsequent observations, but a causal model also tells us how these probabilities would change as a result of external interventions – such as those encountered in policy analysis, treatment management, or planning everyday activity. Such changes cannot be deduced from a join distribution, even if fully specified.

The connection between modularity and interventions is as follows. Instead of specifying a new probability function for each of the many possible interventions, we specify

---

[8] The Tversky and Kahneman (1980) experiments with causal biases in probability judgment constitute another body of evidence supporting this observation. For example, most people believe that it is more likely for a girl to have blue eyes, given that her mother has blue eyes, than the other way around; the two probabilities are in fact equal.

[9] The author was as guilty of advocating the centrality of conditional independence as were his colleagues in statistics; see Pearl (1988b, p. 79).

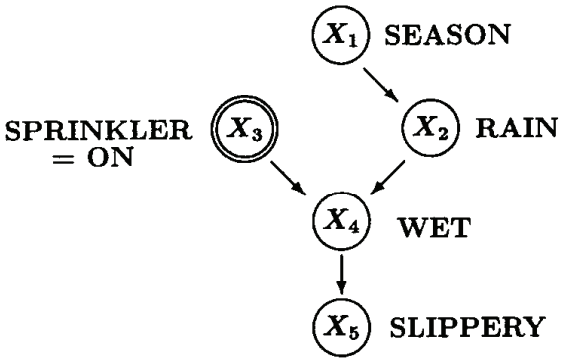

Figure 1.4 Network representation of the action "turning the sprinkler On."

merely the immediate change implied by the intervention and, by virtue of autonomy, we assume that the change is local, and does not spread over to mechanisms other than those specified. Once we know the identity of the mechanism altered by an intervention and the nature of the alteration, the overall effect of an intervention can be predicted by modifying the corresponding factors in (1.33) and using the modified product to compute a new probability function. For example, to represent the action "turning the sprinkler On" in the network of Figure 1.2, we delete the link $X_1 \rightarrow X_3$ and assign X_3 the value On. The graph resulting from this operation is shown in Figure 1.4, and the resulting joint distribution on the remaining variables will be

$$P_{X_3=\text{On}}(x_1, x_2, x_4, x_5) = P(x_1)P(x_2 \mid x_1)P(x_4 \mid x_2, X_3 = \text{On})P(x_5 \mid x_4), \quad (1.36)$$

in which all the factors on the right-hand side (r.h.s.), by virtue of autonomy, are the same as in (1.34).

The deletion of the factor $P(x_3 \mid x_1)$ represents the understanding that, whatever relationship existed between seasons and sprinklers prior to the action, that relationship is no longer in effect while we perform the action. Once we physically turn the sprinkler on and keep it on, a new mechanism (in which the season has no say) determines the state of the sprinkler.

Note the difference between the action $do(X_3 = \text{On})$ and the observation $X_3 = \text{On}$. The effect of the latter is obtained by ordinary Bayesian conditioning, that is, $P(x_1, x_2, x_4, x_5 \mid X_3 = \text{On})$, while that of the former by conditioning a mutilated graph, with the link $X_1 \rightarrow X_3$ removed. This mirrors indeed the difference between seeing and doing: after *observing* that the sprinkler is on, we wish to infer that the season is dry, that it probably did not rain, and so on; no such inferences should be drawn in evaluating the effects of a contemplated *action* "turning the sprinkler On."

The ability of causal networks to predict the effects of actions requires of course a stronger set of assumptions in the construction of those networks, assumptions that rest on causal (not merely associational) knowledge and that ensure the system would respond to interventions in accordance with the principle of autonomy. These assumptions are encapsulated in the following definition of causal Bayesian networks.

Definition 1.3.1 (Causal Bayesian Network)
Let $P(v)$ be a probability distribution on a set V of variables, and let $P_x(v)$ denote the distribution resulting from the intervention $do(X = x)$ that sets a subset X of variables

to constants x.[10] *Denote by* $\boldsymbol{P}_*$ *the set of all interventional distributions* $P_x(v)$, $X \subseteq V$, *including* $P(v)$, *which represents no intervention (i.e.,* $X = \emptyset$*). A DAG G is said to be a* causal Bayesian network *compatible with* $\boldsymbol{P}_*$ *if and only if the following three conditions hold for every* $P_x \in \boldsymbol{P}_*$:

(i) $P_x(v)$ *is Markov relative to G;*

(ii) $P_x(v_i) = 1$ *for all* $V_i \in X$ *whenever* v_i *is consistent with* $X = x$;

(iii) $P_x(v_i \mid pa_i) = P(v_i \mid pa_i)$ *for all* $V_i \notin X$ *whenever* pa_i *is consistent with* $X = x$.

Definition 1.3.1 imposes constraints on the interventional space $\boldsymbol{P}_*$ that permit us to encode this vast space economically, in the form of a single Bayesian network G. These constraints enable us to compute the distribution $P_x(v)$ resulting from any intervention $do(X = x)$ as a *truncated factorization*

$$P_x(v) = \prod_{\{i \mid V_i \notin X\}} P(v_i \mid pa_i) \quad \text{for all } v \text{ consistent with } x, \tag{1.37}$$

which follows from Definition 1.3.1 and justifies the family deletion procedure on G, as in (1.36). It is not hard to show that, whenever G is a causal Bayes network with respect to $\boldsymbol{P}_*$, the following two properties must hold.

Property 1
For all i,

$$P(v_i \mid pa_i) = P_{pa_i}(v_i). \tag{1.38}$$

Property 2
For all i and for every subset S of variables disjoint of $\{V_i, PA_i\}$, *we have*

$$P_{pa_i,s}(v_i) = P_{pa_i}(v_i). \tag{1.39}$$

Property 1 renders every parent set PA_i *exogenous* relative to its child V_i, ensuring that the conditional probability $P(v_i \mid pa_i)$ coincides with the effect (on V_i) of setting PA_i to pa_i by external control. Property 2 expresses the notion of invariance; once we control its direct causes PA_i, no other interventions will affect the probability of V_i.

1.3.2 Causal Relationships and Their Stability

This mechanism-based conception of interventions provides a semantical basis for notions such as "causal effects" or "causal influence," to be defined formally and analyzed in Chapters 3 and 4. For example, to test whether a variable X_i has a causal influence on another variable X_j, we compute (using the truncated factorization formula of (1.37)) the (marginal) distribution of X_j under the actions $do(X_i = x_i)$ – namely, $P_{x_i}(x_j)$ for all

[10] The notation $P_x(v)$ will be replaced in subsequent chapters with $P(v \mid do(x))$ and $P(v \mid \hat{x})$ to facilitate algebraic manipulations.

values x_i of X_i – and test whether that distribution is sensitive to x_i. It is easy to see from our previous examples that only variables that are descendants of X_i in the causal network can be influenced by X_i; deleting the factor $P(x_i \mid pa_i)$ from the joint distribution turns X_i into a root node in the mutilated graph, and root variables (as the d-separation criterion dictates) are independent of all other variables except their descendants.

This understanding of causal influence permits us to see precisely why, and in what way, causal relationships are more "stable" than probabilistic relationships. We expect such difference in stability because causal relationships are *ontological*, describing objective physical constraints in our world, whereas probabilistic relationships are *epistemic*, reflecting what we know or believe about the world. Therefore, causal relationships should remain unaltered as long as no change has taken place in the environment, even when our knowledge about the environment undergoes changes. To demonstrate, consider the causal relationship S_1, "Turning the sprinkler on would not affect the rain," and compare it to its probabilistic counterpart S_2, "The state of the sprinkler is independent of (or unassociated with) the state of the rain." Figure 1.2 illustrates two obvious ways in which S_2 will change while S_1 remains intact. First, S_2 changes from false to true when we learn what season it is (X_1). Second, given that we know the season, S_2 changes from true to false once we observe that the pavement is wet ($X_4 = \text{true}$). On the other hand, S_1 remains true regardless of what we learn or know about the season or about the pavement.

The example reveals a stronger sense in which causal relationships are more stable than the corresponding probabilistic relationships, a sense that goes beyond their basic ontological–epistemological difference. The relationship S_1 will remain invariant to changes in the mechanism that regulates how seasons affect sprinklers. In fact, it remains invariant to changes in *all* mechanisms shown in this causal graph. We thus see that causal relationships exhibit greater robustness to ontological changes as well; they are sensitive to a smaller set of mechanisms. More specifically, and in marked contrast to probabilistic relationships, causal relationships remain invariant to changes in the mechanism that governs the causal variables (X_3 in our example).

In view of this stability, it is no wonder that people prefer to encode knowledge in causal rather than probabilistic structures. Probabilistic relationships, such as marginal and conditional independencies, may be helpful in hypothesizing initial causal structures from uncontrolled observations. However, once knowledge is cast in causal structure, those probabilistic relationships tend to be forgotten; whatever judgments people express about conditional independencies in a given domain are derived from the causal structure acquired. This explains why people feel confident asserting certain conditional independencies (e.g., that the price of beans in China is independent on the traffic in Los Angeles) having no idea whatsoever about the numerical probabilities involved (e.g., whether the price of beans will exceed $10 per bushel).

The element of stability (of mechanisms) is also at the heart of the so-called explanatory accounts of causality, according to which causal models need not encode behavior under intervention but instead aim primarily to provide an "explanation" or "understanding" of how data are generated.[11] Regardless of what use is eventually made

[11] Elements of this explanatory account can be found in the writings of Dempster (1990), Cox (1992), and Shafer (1996a); see also King et al. (1994, p. 75).

of our "understanding" of things, we surely would prefer an understanding in terms of durable relationships, transportable across situations, over those based on transitory relationships. The sense of "comprehensibility" that accompanies an adequate explanation is a natural byproduct of the transportability of (and hence of our familiarity with) the causal relationships used in the explanation. It is for reasons of stability that we regard the falling barometer as predicting but not explaining the rain; those predictions are not transportable to situations where the pressure surrounding the barometer is controlled by artificial means. True understanding enables predictions in such novel situations, where some mechanisms change and others are added. It thus seems reasonable to suggest that, in the final analysis, the explanatory account of causation is merely a variant of the manipulative account, albeit one where interventions are dormant. Accordingly, we may as well view our unsatiated quest for understanding "how data is generated" or "how things work" as a quest for acquiring the ability to make predictions under wider range of circumstances, including circumstances in which things are taken apart, reconfigured, or undergo spontaneous change.

1.4 FUNCTIONAL CAUSAL MODELS

The way we have introduced the causal interpretation of Bayesian networks represents a fundamental departure from the way causal models (and causal graphs) were first introduced into genetics (Wright 1921), econometrics (Haavelmo 1943), and the social sciences (Duncan 1975), as well as from the way causal models are used routinely in physics and engineering. In those models, causal relationships are expressed in the form of deterministic, *functional* equations, and probabilities are introduced through the assumption that certain variables in the equations are unobserved. This reflects Laplace's (1814) conception of natural phenomena, according to which nature's laws are deterministic and randomness surfaces owing merely to our ignorance of the underlying boundary conditions. In contrast, all relationships in the definition of causal Bayesian networks were assumed to be inherently stochastic and thus appeal to the modern (i.e., quantum mechanical) conception of physics, according to which all nature's laws are inherently probabilistic and determinism is but a convenient approximation.

In this book, we shall express preference toward Laplace's quasi-deterministic conception of causality and will use it, often contrasted with the stochastic conception, to define and analyze most of the causal entities that we study. This preference is based on three considerations. First, the Laplacian conception is more general. Every stochastic model can be emulated by many functional relationships (with stochastic inputs), but not the other way around; functional relationships can only be approximated, as a limiting case, using stochastic models. Second, the Laplacian conception is more in tune with human intuition. The few esoteric quantum mechanical experiments that conflict with the predictions of the Laplacian conception evoke surprise and disbelief, and they demand that physicists give up deeply entrenched intuitions about locality and causality (Maudlin 1994). Our objective is to preserve, explicate, and satisfy – not destroy – those intuitions.[12]

[12] The often heard argument that human intuitions belong in psychology and not in science or philosophy is inapplicable when it comes to causal intuition – the original authors of causal thoughts

Finally, certain concepts that are ubiquitous in human discourse can be defined only in the Laplacian framework. We shall see, for example, that such simple concepts as "the probability that event B occured *because* of event A" and "the probability that event B would have been *different* if it were not for event A" cannot be defined in terms of purely stochastic models. These so-called *counterfactual* concepts will require a synthesis of the deterministic and probabilistic components embodied in the Laplacian model.

1.4.1 Structural Equations

In its general form, a functional causal model consists of a set of equations of the form

$$x_i = f_i(pa_i, u_i), \quad i = 1, \ldots, n, \tag{1.40}$$

where pa_i (connoting *parents*) stands for the set of variables judged to be immediate causes of X_i and where the U_i represent errors (or "disturbances") due to omitted factors. Equation (1.40) is a nonlinear, nonparametric generalization of the linear structural equation models (SEMs)

$$x_i = \sum_{k \neq i} \alpha_{ik} x_k + u_i, \quad i = 1, \ldots, n, \tag{1.41}$$

which have become a standard tool in economics and social science (see Chapter 5 for a detailed exposition of this enterprise). In linear models, pa_i corresponds to those variables on the r.h.s. of (1.41) that have nonzero coefficients.

A set of equations in the form of (1.40) and in which each equation represents an autonomous mechanism is called *structural model;* if each mechanism determines the value of just one distinct variable (called the *dependent* variable), then the model is called a *structural causal model* or a *causal model* for short.[13] Mathematically, the distinction between structural and algebraic equations is that the latter are characterized by the set of solutions to the entire system of equations, whereas the former are characterized by the solutions of each individual equation. The implication is that any subset of structural equations is, in itself, a valid model of reality – one that prevails under some set of interventions.

To illustrate, Figure 1.5 depicts a canonical econometric model relating price and demand through the equations

$$q = b_1 p + d_1 i + u_1, \tag{1.42}$$

$$p = b_2 q + d_2 w + u_2, \tag{1.43}$$

where Q is the quantity of household demand for a product A, P is the unit price of product A, I is household income, W is the wage rate for producing product A, and u_1 and

cannot be ignored when the meaning of the concept is in question. Indeed, compliance with human intuition has been the ultimate criterion of adequacy in every philosophical study of causation, and the proper incorporation of background information into statistical studies likewise relies on accurate interpretation of causal judgment.

[13] Formal treatment of causal models, structural equations, and error terms are given in Chapter 5 (Section 5.4.1) and Chapter 7 (Sections 7.1 and 7.2.5).

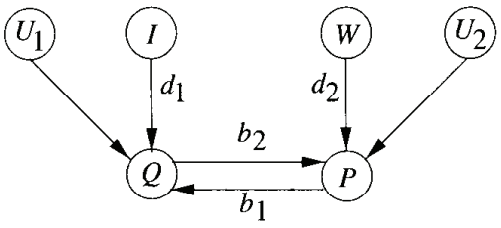

Figure 1.5 Causal diagram illustrating the relationship between price (P), demand (Q), income (Z), and wages (W).

u_2 represent error terms – unmodeled factors that affect quantity and price, respectively (Goldberger 1992). The graph associated with this model is cyclic, and the vertices associated with the variables U_1, U_2, I, and W are root nodes, conveying the assumption of mutual independence. The idea of *autonomy* (Aldrich 1989), in this context, means that the two equations represent two loosely coupled segments of the economy, consumers and producers. Equation (1.42) describes how consumers decide what quantity Q to buy, and (1.43) describes how manufacturers decide what price P to charge. Like all feedback systems, this too represents implicit dynamics; today's prices are determined on the basis of yesterday's demand, and these prices will determine the demand in the next period of transactions. The solution to such equations represents a long-term equilibrium under the assumption that the background quantities, U_1 and U_2, remain constant.

The two equations are considered to be "autonomous" relative to the dynamics of changes in the sense that external changes affecting one equation do not imply changes to the others. For example, if government decides on price control and sets the price P at p_0, then (1.43) will be modified to read $p = p_0$ but the relationships in (1.42) will remain intact, yielding $q = b_1 p_0 + d_1 i + u_1$. We thus see that b_1, the "demand elasticity," should be interpreted as the rate of change of Q per unit *controlled* change in P. This is different, of course, from the rate of change of Q per unit *observed* change in P (under uncontrolled conditions), which, besides b_1, is also affected by the parameters of (1.43) (see Section 7.2.1, equation (7.14)). The difference between controlled and observed changes is essential for the correct interpretation of structural equation models in social science and economics, and it will be discussed at length in Chapter 5. If we have reasons to believe that consumer behavior will also change under a price control policy, then this modified behavior would need to be modeled explicitly – for example, by treating the coefficients b_1 and d_1 as dependent variables in auxiliary equations involving P.[14] Section 7.2.1 will present an analysis of policy-related problems using this model.

To illustrate the workings of nonlinear functional models, consider again the causal relationships depicted in Figure 1.2. The causal model associated with these relationships will consist of five functions, each representing an autonomous mechanism governing one variable:

$$x_1 = u_1,$$

$$x_2 = f_2(x_1, u_2),$$

[14] Indeed, consumers normally react to price fixing by hoarding goods in anticipation of shortages (Lucas 1976). Such phenomena are not foreign to structural models, though; they simply call for more elaborate equations to capture consumers' expectations.

$$
\begin{aligned}
x_3 &= f_3(x_1, u_3), \\
x_4 &= f_4(x_3, x_2, u_4), \\
x_5 &= f_5(x_4, u_5).
\end{aligned}
\tag{1.44}
$$

The error variables $U_1, \ldots, U_5$ are not shown explicitly in the graph; by convention, this implies that they are assumed to be mutually independent. When some disturbances are judged to be dependent, it is customary to encode such dependencies by augmenting the graph with double-headed arrows, as shown in Figure 1.1(a).

A typical specification of the functions $\{f_1, \ldots, f_5\}$ and the disturbance terms is given by the following Boolean model:

$$
\begin{aligned}
x_2 &= [(X_1 = \text{winter}) \vee (X_1 = \text{fall}) \vee u_2] \wedge \neg u_2', \\
x_3 &= [(X_1 = \text{summer}) \vee (X_1 = \text{spring}) \vee u_3] \wedge \neg u_3', \\
x_4 &= (x_2 \vee x_3 \vee u_4) \wedge \neg u_4', \\
x_5 &= (x_4 \vee u_5) \wedge \neg u_5',
\end{aligned}
\tag{1.45}
$$

where x_i stands for X_i = true and where u_i and u_i' stand for triggering and inhibiting abnormalities, respectively. For example, u_4 stands for (unspecified) events that might cause the pavement to get wet (x_4) when the sprinkler is off ($\neg x_3$) and it does not rain ($\neg x_2$) (e.g., a broken water pipe), while u_4' stands for (unspecified) events that would keep the pavement dry in spite of the rain (x_2), the sprinkler (x_3), and u_4 (e.g., pavement covered with a plastic sheet).

It is important to emphasize that, in the two models just described, the variables placed on the left-hand side of the equality sign (the dependent or output variables) act distinctly from the other variables in each equation. The role of this distinction becomes clear when we discuss interventions, since it is only through this distinction that we can identify which equation ought to be modified under local interventions of the type "fix the price at p_0" ($do(P = p_0)$) or "turn the sprinkler On" ($do(X_3 = \text{true})$).[15]

We now compare the features of functional models as defined in (1.40) with those of causal Bayesian networks defined in Section 1.3. Toward this end, we will consider the processing of three types of queries:

predictions (e.g., would the pavement be slippery if we *find* the sprinkler off?);

interventions (e.g., would the pavement be slippery if we *make sure* that the sprinkler is off?); and

counterfactuals (e.g., would the pavement be slippery *had* the sprinkler been off, given that the pavement is in fact not slippery and the sprinkler is on?).

We shall see that these three types of queries represent a hierarchy of three fundamentally different types of problems, demanding knowledge with increasing level of details.

[15] Economists who write the supply–demand equations as $\{q = ap + u_1, q = bp + u_2\}$, with q appearing on the l.h.s. of both equations, are giving up the option of analyzing price control policies unless additional symbolic machinery is used to identify which equation will be modified by the $do(P = p_0)$ operator.

1.4.2 Probabilistic Predictions in Causal Models

Given a causal model (equation (1.40)), if we draw an arrow from each member of PA toward X_i then the resulting graph G will be called a *causal diagram*. If the causal dia gram is acyclic, then the corresponding model is called *semi-Markovian* and the value of the X variables will be uniquely determined by those of the U variables. Under sucl conditions, the joint distribution $P(x_1, \ldots, x_n)$ is determined uniquely by the distribu tion $P(u)$ of the error variables. If, in addition to acyclicity, the error terms are mutuall independent, the model is called *Markovian*.

A fundamental theorem about Markovian models establishes a connection betwee causation and probabilities via the parental Markov condition of Theorem 1.2.7.

Theorem 1.4.1 (Causal Markov Condition)
Every Markovian causal model M induces a distribution $P(x_1, \ldots, x_n)$ that satisfies th parental Markov condition relative the causal diagram G associated with M; that is, eacl variable X_i is independent on all its nondescendants, given its parents PA_i in G (Pear and Verma 1991).[16]

The proof is immediate. Considering that the set $\{PA_i, U_i\}$ determines one unique value o X_i, the distribution $P(x_1, \ldots, x_n, u_1, \ldots, u_n)$ is certainly Markov relative the augmente DAG $G(X, U)$, in which the U variables are represented explicitly. The required Markov condition of the marginal distribution $P(x_1, \ldots, x_n)$ follows by d-separation in $G(X, U)$.

Theorem 1.4.1 shows that the Markov condition of Theorem 1.2.7 follows from tw causal assumptions: (1) our commitment to include in the model (not in the background every variable that is a cause of two or more other variables; and (2) Reichenbach's (1956) common-cause assumption, also known as "no correlation without causation," stating that, if any two variables are dependent, then one is a cause of the other *or* there is a third variable causing both. These two assumptions imply that the background factors in U are mutually independent and hence that the causal model is Markovian. Theorem 1.4.1 explains both why Markovian models are so frequently assumed in causal analysis and why the parental Markov condition (Theorem 1.2.7) is so often regarded as an inherent feature of causal models (see e.g. Kiiveri et al. 1984; Spirtes et al. 1993).[17]

The causal Markov condition implies that characterizing each child–parent relationship as a deterministic function, instead of the usual conditional probability $P(x_i \mid pa_i)$, imposes equivalent independence constraints on the resulting distribution and leads to the same recursive decomposition that characterizes Bayesian networks (see equation (1.33)). More significantly, this holds regardless of the choice of functions $\{f_i\}$ and regardless

[16] Considering its generality and transparency, I would not be surprised if some version of this theorem has appeared earlier in the literature.

[17] Kiiveri et al.'s (1984) paper, entitled "Recursive Causal Models," provides the first proof (for strictly positive distributions) that the parental Markov condition of Theorem 1.2.7 follows from the factorization of (1.33). This implication, however, is purely probabilistic and invokes no aspect of causation. In order to establish a connection between causation and probability we must first devise a model for causation, either in terms of manipulations (as in Definition 1.3.1) or in terms of functional relationships in structural equations (as in Theorem 1.4.1).

of the error distributions $P(u_i)$. Thus, we need not specify in advance the functional form of $\{f_i\}$ or the distributions $P(u_i)$; once we measure (or estimate) $P(x_i \mid pa_i)$, all probabilistic properties of a Markovian causal model are determined, regardless of the mechanism that actually generates those conditional probabilities. Druzdzel and Simon (1993) showed that, for every Bayesian network G characterized by a distribution P (as in (1.33)), there exists a functional model (as in (1.40)) that generates a distribution identical to P.[18] It follows that in all probabilistic applications of Bayesian networks – including statistical estimation, prediction, and diagnosis – we can use an equivalent functional model as specified in (1.40), and we can regard functional models as just another way of encoding joint distribution functions.

Nonetheless, the causal–functional specification has several advantages over the probabilistic specification, even in purely predictive (i.e. nonmanipulative) tasks. First and foremost, all the conditional independencies that are displayed by the causal diagram G are guaranteed to be *stable* – that is, invariant to parametric changes in the mechanisms represented by the functions f_i and the distributions $P(u_i)$. This means that agents who choose to organize knowledge using Markovian causal models can make reliable assertions about conditional independence relations without assessing numerical probabilities – a common ability among humanoids[19] and a useful feature for inference. Second, the functional specification is often more meaningful and natural, and it yields a small number of parameters. Typical examples are the linear structural equations used in social science and economics (see Chapter 5) and the "noisy OR gate" that has become quite popular in modeling the effect of multiple dichotomous causes (Pearl 1988b, p. 184). Third (and perhaps hardest for an empiricist to accept), judgmental assumptions of conditional independence among observable quantities are simplified and made more reliable in functional models, because such assumptions are cast directly as judgments about the presence or absence of *unobserved* common causes (e.g., why is the price of beans in China judged to be independent of the traffic in Los Angeles?). In the construction of Bayesian networks, for example, instead of judging whether each variable is independent of all its nondescendants (given its parents), we need to judge whether the parent set contains *all* relevant immediate causes – in particular, whether no factor omitted from the parent set is a cause of another observed variable. Such judgments are more natural because they are discernible directly from a qualitative causal structure, the very structure that our mind has selected for storing stable aspects of experience.

Finally, there is an additional advantage to basing prediction models on causal mechanisms that stems from considerations of stability (Section 1.3.2). When some conditions in the environment undergo change, it is usually only a few causal mechanisms that are affected by the change; the rest remain unaltered. It is simpler then to reassess (judgmentally) or reestimate (statistically) the model parameters knowing that

[18] In Chapter 9 we will show that, except in some pathological cases, there actually exist an infinite number of functional models with such a property.

[19] Statisticians who are reluctant to discuss causality yet have no hesitation expressing background information in the form of conditional independence statements would probably be shocked to realize that such statements acquire their validity from none other but the *causal* Markov condition (Theorem 1.4.1). See note 9.

the corresponding symbolic change is also local, involving just a few parameters, than to reestimate the entire model from scratch.[20]

1.4.3 Interventions and Causal Effects in Functional Models

The functional characterization $x_i = f_i(pa_i, u_i)$, like its stochastic counterpart, provides a convenient language for specifying how the resulting distribution would change in response to external interventions. This is accomplished by encoding each intervention as an alteration on a select set of functions instead of a select set of conditional probabilities. The overall effect of the intervention can then be predicted by modifying the corresponding equations in the model and using the modified model to compute a new probability function. Thus, all features of causal Bayesian networks (Section 1.3) can be emulated in Markovian functional models.

For example, to represent the action "turning the sprinkler On" in the model of (1.44), we delete the equation $x_3 = f_3(x_1, u_3)$ and replace it with $x_3 = \text{On}$. The modified model will contain all the information needed for computing the effect of the action on other variables. For example, the probability function induced by the modified model will be equal to that given by (1.36), and the modified diagram will coincide with that of Figure 1.4.

More generally, when an intervention forces a subset X of variables to attain fixed values x, then a subset of equations is to be pruned from the model in (1.40), one for each member of X, thus defining a new distribution over the remaining variables that characterizes the effect of the intervention and coincides with the truncated factorization obtained by pruning families from a causal Bayesian network (equation (1.37)).[21]

The functional model's representation of interventions offers greater flexibility and generality than that of a stochastic model. First, the analysis of interventions can be extended to cyclic models, like the one in Figure 1.5, so as to answer policy-related questions[22] (e.g.: What would the demand quantity be if we control the price at p_0?). Second, interventions involving the modification of equational parameters (like b_1 and d_1 in (1.42)) are more readily comprehended than those described as modifiers of conditional probabilities, perhaps because stable physical mechanisms are normally associated with equations and not with conditional probabilities. Conditional probabilities are perceived to be derivable from, not generators of, joint distributions. Third, the analysis of causal effects in non-Markovian models will be greatly simplified using functional models. The reason is: there are infinitely many conditional probabilities $P(x_i \mid pa_i)$ but only a finite number of functions $x_i = f_i(pa_i, u_i)$ among discrete variables X_i and PA_i. This fact will enable us in Chapter 8 (Section 8.2.2) to use linear programming techniques to obtain sharp bounds on causal effects in studies involving noncompliance.

---

[20] To the best of my knowledge, this aspect of causal models has not been studied formally; it is suggested here as a research topic for students of adaptive systems.

[21] An explicit translation of interventions to "wiping out" equations from the model was first proposed by Strotz and Wold (1960) and later used in Fisher (1970) and Sobel (1990). More elaborate types of interventions, involving conditional actions and stochastic strategies, will be formulated in Chapter 4.

[22] Such questions, especially those involving the control of endogenous variables, are conspicuously absent from econometric textbooks (see Chapter 5).

Finally, functional models permit the analysis of context-specific actions and policies. The notion of causal effect as defined so far is of only minor use in practical policy making. The reason is that causal effects tell us the general tendency of an action to bring about a response (as with the tendency of a drug to enhance recovery in the overall population) but are not specific to actions in a given situation characterized by a set of particular observations that may themselves be affected by the action. A physician is usually concerned with the effect of a treatment on a patient who has already been examined and found to have certain symptoms. Some of those symptoms will themselves be affected by the treatment. Likewise, an economist is concerned with the effect of taxation in a given economical context characterized by various economical indicators, which (again) will be affected by taxation if applied. Such context-specific causal effects cannot be computed by simulating an intervention in a static Bayesian network, because the context itself varies with the intervention and so the conditional probabilities $P(x_i \mid pa_i)$ are altered in the process. However, the functional relationships $x_i = f_i(pa_i, u_i)$ remain invariant, which enables us to compute context-specific causal effects as outlined in the next section (see Sections 7.2.1, 8.3, and 9.3.4 for full details).

1.4.4 Counterfactuals in Functional Models

We now turn to the most distinctive characteristic of functional models – the analysis of *counterfactuals*. Certain counterfactual sentences, as we remarked before, cannot be defined in the framework of stochastic causal networks. To see the difficulties, let us consider the simplest possible causal Bayesian network consisting of a pair of independent (hence unconnected) binary variables X and Y. Such a network ensues, for example, in a controlled (i.e. randomized) clinical trial when we find that a treatment X has no effect on the distribution of subjects' response Y, which may stand for either recovery ($Y = 0$) or death ($Y = 1$). Assume that a given subject, Joe, has taken the treatment and died; we ask whether Joe's death occurred *because of* the treatment, *despite* the treatment, or *regardless of* the treatment. In other words, we ask for the probability Q that Joe would have died had he not been treated.

To highlight the difficulty in answering such counterfactual questions, let us take an extreme case where 50% of the patients recover and 50% die in both the treatment and the control groups; assume further that the sample size approaches infinity, thus yielding

$$P(y \mid x) = 1/2 \quad \text{for all } x \text{ and } y. \tag{1.46}$$

Readers versed in statistical testing will recognize immediately the impossibility of answering the counterfactual question from the available data, noting that Joe, who took the treatment and died, was never tested under the no-treatment condition. Moreover, the difficulty does not stem from addressing the question to a particular individual, Joe, for which we have only one data point. Rephrasing the question in terms of population frequencies – asking what percentage Q of subjects who died under treatment would have recovered had they not taken the treatment – will encounter the same difficulties because none of those subjects was tested under the no-treatment condition. Such difficulties have prompted some statisticians to dismiss counterfactual questions as metaphysical and to

advocate the restriction of statistical analysis to only those questions that can be answered by direct tests (Dawid 1997).

However, that our scientific, legal, and ordinary languages are loaded with counterfactual utterances indicates clearly that counterfactuals are far from being metaphysical; they must have definite testable implications and must carry valuable substantive information. The analysis of counterfactuals therefore represents an opportunity to anyone who shares the aims of this book: integrating substantive knowledge with statistical data so as to refine the former and interpret the latter. Within this framework, the counterfactual issue demands answers to tough, yet manageable technical questions: What is the empirical content of counterfactual queries? What knowledge is required to answer those queries? How can this knowledge be represented mathematically? Given such representation, what mathematical machinery is needed for deriving the answers?

Chapter 7 (Section 7.2.2) presents an empirical explication of counterfactuals as claims about the temporal persistence of certain mechanisms. In our example, the response to treatment of each (surviving) patient is assumed to be persistent. If the outcome Y were a reversible condition, rather than death, then the counterfactual claim would translate directly into predictions about response to future treatments. But even in the case of death, the counterfactual quantity Q implies not merely a speculation about the hypothetical behavior of subjects who died but also a testable claim about surviving untreated subjects under subsequent treatment. We leave it as an exercise for the reader to prove that, based on (1.46) and barring sampling variations, the percentage Q of deceased subjects from the treatment group who would have recovered had they not taken the treatment precisely equals the percentage Q' of surviving subjects in the nontreatment group who will die if given treatment.[23] Whereas Q is hypothetical, Q' is unquestionably testable.

Having sketched the empirical interpretation of counterfactuals, our next step in this introductory chapter is the question of representation: What knowledge is required to answer questions about counterfactuals? And how should this knowledge be formulated so that counterfactual queries be answered quickly and reliably? That such representation exists is evident by the swiftness and consistency with which people distinguish plausible from implausible counterfactual statements. Most people would agree that President Clinton's place in history would be different had he not met Monica Lewinsky, but only a few would assert that his place in history would change had he not eaten breakfast yesterday. In the cognitive sciences, such consistency of opinion is as close as one can get to a proof that an effective machinery for representing and manipulating counterfactuals resides someplace in the human mind. What then are the building blocks of that machinery?

A straightforward representational scheme would (i) store counterfactual knowledge in the form of counterfactual premises and (ii) derive answers to counterfactual queries using some logical rules of inference capable of taking us from premises to conclusions. This approach has indeed been taken by the philosophers Robert Stalnaker (1968) and David Lewis (1973a,b), who constructed logics of counterfactuals using closest-world

[23] For example, if Q equals 100% (i.e., all those who took the treatment and died would have recovered had they not taken the treatment) then all surviving subjects from the nontreatment group will die if given treatment (again, barring sampling variations). Such exercises will become routine when we develop the mathematical machinery for analyzing probabilities of causes (see Chapter 9, Theorem 9.2.12, equations (9.11)–(9.12)).

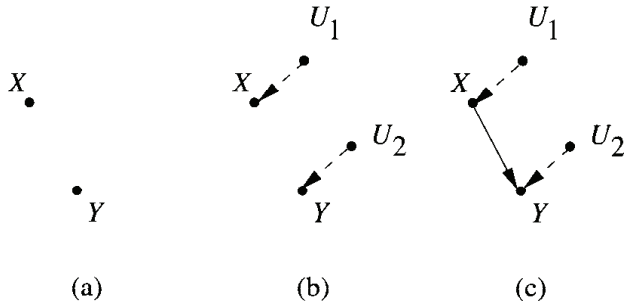

Figure 1.6 (a) A causal Bayesian network that represents the distribution of (1.47). (b) A causal diagram representing the process generating the distribution in (a), according to model 1. (c) Same, according to model 2. (Both U_1 and U_2 are unobserved.)

semantics (i.e., "B would be true if it were A" just in case B is true in the closest possible world (to ours) in which A is true). However, the closest-world semantics still leaves two questions unanswered. (1) What choice of distance measure would make counterfactual reasoning compatible with ordinary conception of cause and effect? (2) What mental representation of interworld distances would render the computation of counterfactuals manageable and practical (for both humans and machines)? These two questions are answered by the structural model approach expanded in Chapter 7.

An approach similar to Lewis's (though somewhat less formal) has been pursued by statisticians in the potential-outcome framework (Rubin 1974; Robins 1986; Holland 1988). Here, substantive knowledge is expressed in terms of probabilistic relationships (e.g. independence) among counterfactual variables and then used in the estimation of causal effects. The question of representation shifts from the closest-world to the potential-outcome approach: How are probabilistic relationships among counterfactuals stored or inferred in the investigator's mind? In Chapter 7 (see also Section 3.6.3) we provide an analysis of the closest-world and potential-outcome approaches and compare them to the structural model approach, to be outlined next, in which counterfactuals are *derived* from (and in fact defined by) a functional causal model (equation (1.40)).

In order to see the connection between counterfactuals and structural equations, we should first examine why the information encoded in a Bayesian network, even in its causal interpretation, is insufficient to answer counterfactual queries. Consider again our example of the controlled randomized experiment (equation (1.46)), which corresponds to an edgeless Bayesian network (Figure 1.6(a)) with two independent binary variables and a joint probability:

$$P(y, x) = 0.25 \quad \text{for all } x \text{ and } y. \tag{1.47}$$

We now present two functional models, each generating the joint probability of (1.47) yet each giving a different value to the quantity of interest, Q = the probability that a subject who died under treatment ($x = 1$, $y = 1$) would have recovered ($y = 0$) had he or she not been treated ($x = 0$).

Model 1 (Figure 1.6(b))
Let

$$x = u_1,$$

$$y = u_2,$$

where U_1 and U_2 are two independent binary variables with $P(u_1 = 1) = P(u_2 = 1) = \frac{1}{2}$ (e.g., random coins).

Model 1	$u_2 = 0$		$u_2 = 1$		Marginal	
	$x = 1$	$x = 0$	$x = 1$	$x = 0$	$x = 1$	$x = 0$
$y = 1$ (death)	0	0	0.25	0.25	0.25	0.25
$y = 0$ (recovery)	0.25	0.25	0	0	0.25	0.25

Model 2	$u_2 = 0$		$u_2 = 1$		Marginal	
	$x = 1$	$x = 0$	$x = 1$	$x = 0$	$x = 1$	$x = 0$
$y = 1$ (death)	0	0.25	0.25	0	0.25	0.25
$y = 0$ (recovery)	0.25	0	0	0.25	0.25	0.25

Figure 1.7 Contingency tables showing the distributions $P(x, y, u_2)$ and $P(x, y)$ for the two models discussed in the text.

Model 2 (Figure 1.6(c))
Let

$$\begin{aligned} x &= u_1, \\ y &= xu_2 + (1 - x)(1 - u_2), \end{aligned} \tag{1.48}$$

where, as before, U_1 and U_2 are two independent binary variables.

Model 1 corresponds to treatment (X) that has no effect on any of the subjects; in model 2, every subject is affected by treatment. The reason that the two models yield the same distribution is that model 2 describes a mixture of two subpopulations. In one ($u_2 = 1$), each subject dies ($y = 1$) if and only if treated; in the other ($u_2 = 0$), each subject recovers ($y = 0$) if and only if treated. The distributions $P(x, y, u_2)$ and $P(x, y)$ corresponding to these two models are shown in the tables of Figure 1.7.

The value of Q differs in these two models. In model 1, Q evaluates to zero, because subjects who died correspond to $u_2 = 1$ and, since the treatment has no effect on y, changing X from 1 to 0 would still yield $y = 1$. In model 2, however, Q evaluates to unity, because subjects who died under treatment must correspond to $u_2 = 1$ (i.e., those who die if treated), meaning they would recover if and only if not treated.

The first lesson of this example is that stochastic causal models are insufficient for computing probabilities of counterfactuals; knowledge of the actual process behind $P(y \mid x)$ is needed for the computation.[24] A second lesson is that a functional causal model constitutes a mathematical object sufficient for the computation (and definition) of such probabilities. Consider, for example, model 2 of (1.48). The way we concluded that a deceased treated subject ($y = 1, x = 1$) would have recovered if not treated involved three mental steps. First, we applied the evidence at hand, $e : \{y = 1, x = 1\}$, to the model and concluded that e is compatible with only one realization of U_1 and U_2 – namely, $\{u_1 = 1,$

[24] In the potential-outcome framework (Sections 3.6.3 and 7.4.4), such knowledge obtains stochastic appearance by defining distributions over *counterfactual variables* Y_1 and Y_0, which stand for the potential response of an individual to treatment and no treatment, respectively. These hypothetical variables play a role similar to the functions $f_i(pa_i, u_i)$ in our model; they represent the deterministic assumption that every individual possesses a definite response to treatment, regardless of whether that treatment was realized.

$u_2 = 1\}$. Second, to simulate the hypothetical condition "had he or she not been treated," we substituted $x = 0$ into (1.48) while ignoring the first equation $x = u_1$. Finally, we solved (1.48) for y (assuming $x = 0$ and $u_2 = 1$) and obtained $y = 0$, from which we concluded that the probability of recovery ($y = 0$) is unity under the hypothetical condition considered.

These three steps can be generalized to any causal model M as follows. Given evidence e, to compute the probability of $Y = y$ under the hypothetical condition $X = x$ (where X is a subset of variables), apply the following three steps to M.

***Step 1 (abduction)*:** Update the probability $P(u)$ to obtain $P(u \mid e)$.

***Step 2 (action)*:** Replace the equations corresponding to variables in set X by the equations $X = x$.

***Step 3 (prediction)*:** Use the modified model to compute the probability of $Y = y$.

In temporal metaphors, this three-step procedure can be interpreted as follows. Step 1 explains the past (U) in light of the current evidence e; step 2 bends the course of history (minimally) to comply with the hypothetical condition $X = x$; finally, step 3 predicts the future (Y) based on our new understanding of the past and our newly established condition, $X = x$.

Recalling that for each value u of U there is a unique solution for Y, it is clear that step 3 always gives a unique solution for the needed probability; we simply sum up the probabilities $P(u \mid e)$ assigned to all those u that yield $Y = y$ as a solution. Chapter 7 develops effective procedures for computing probabilities of counterfactuals, procedures that are based on probability propagation in "twin" networks (Balke and Pearl 1995): one network represents the actual world; the other, the counterfactual world.

Note that the hypothetical condition $X = x$ always stands in contradiction to the prevailing values u of U in the model considered (else $X = x$ would actually be realized and thus would not be considered hypothetical). It is for this reason that we invoke (in step 2) an external intervention (alternatively, a "theory change" or a "miracle"; Lewis 1973b), which modifies the model and thus explains the contradiction away. In Chapter 7 we extend this structural–interventional model to give a full semantical and axiomatic account both for counterfactuals and the probability of counterfactuals. In contrast with Lewis's theory, this account is not based on abstract notion of similarity among hypothetical worlds; rather, it rests on the actual mechanisms involved in the production of the hypothetical worlds considered. Likewise, in contrast with the potential-outcome framework, counterfactuals in the structural account are not treated as undefined primitives but rather as quantities to be derived from the more fundamental concepts of causal mechanisms and their structure.

The three-step model of counterfactual reasoning also uncovers the real reason why stochastic causal models are insufficient for computing probabilities of counterfactuals. Because the U variables do not appear explicitly in stochastic models, we cannot apply step 1 so as to update $P(u)$ with the evidence e at hand. This implies that several ubiquitous notions based on counterfactuals – including probabilities of causes (given the effects), probabilities of explanations, and context-dependent causal effect – cannot be defined in such models. For these, we must make some assumptions about the form of the functions f_i and the probabilities of the error terms. For example, the assumptions of

linearity, normality, and error independence are sufficient for computing all counterfactual queries in the model of Figure 1.5 (see Section 7.2.1). In Chapter 9, we will present conditions under which counterfactual queries concerning probability of causation can be inferred from data when f_i and $P(u)$ are unknown, and only general features (e.g. monotonicity) of these entities are assumed. Likewise, Chapter 8 (Section 8.3) will present methods of *bounding* probabilities of counterfactuals when only stochastic models are available.

The preceding considerations further imply that the three tasks listed in the beginning of this section – prediction, intervention, and counterfactuals – form a natural hierarchy of causal reasoning tasks, with increasing levels of refinement and increasing demands on the knowledge required for accomplishing these tasks. Prediction is the simplest of the three, requiring only a specification of a joint distribution function. The analysis of interventions requires a causal structure in addition to a joint distribution. Finally, processing counterfactuals is the hardest task because it requires some information about the functional relationships and/or the distribution of the omitted factors.

This hierarchy also defines a natural partitioning of the chapters in this book. Chapter 2 will deal primarily with the probabilistic aspects of causal Bayesian networks (though the underlying causal structure will serve as a conceptual guide). Chapters 3–6 will deal exclusively with the interventional aspects of causal models, including the identification of causal effects, the clarification of structural equation models, and the relationships between confounding and collapsibility. Chapters 7–10 will deal with counterfactual analysis, including axiomatic foundation, applications to policy analysis, the bounding of counterfactual queries, the identification of probabilities of causes, and the explication of single-event causation.

I wish the reader a smooth and rewarding journey through these chapters. But first, an important stop for terminological distinctions.

1.5 CAUSAL VERSUS STATISTICAL TERMINOLOGY

This section defines fundamental terms and concepts that will be used throughout this book. These definitions may not agree with those given in standard sources, so it is important to refer to this section in case of doubts regarding the interpretation of these terms.

A ***probabilistic parameter*** is any quantity that is defined in terms[25] of a joint probability function. Examples are the quantities defined in Sections 1.1 and 1.2.

A ***statistical parameter*** is any quantity that is defined in terms of a joint probability distribution of observed variables, making no assumption whatsoever regarding the existence or nonexistence of unobserved variables.

Examples: the conditional expectation $E(Y \mid x)$,
the regression coefficient r_{YX},
the value of the density function at $y = 0$, $x = 1$.

[25] A quantity Q is said to be *defined in terms of* an object of class C if Q can be computed uniquely from the description of any object in class C (i.e., if Q is defined by a functional mapping from C to the domain of Q).

A ***causal parameter*** is any quantity that is defined in terms of a causal model (as in (1.40)) and is not a statistical parameter.

Examples: the coefficients α_{ik} in (1.41),
whether X_9 has influence on X_3 for some u,
the expected value of Y under the intervention $do(X = 0)$,
the number of parents of variable X_7.

Remark: The distinction between probabilistic and statistical parameters is devised to exclude the construction of joint distributions that invoke hypothetical variables (e.g., counterfactual or theological). Such constructions, if permitted, would qualify any quantity as statistical and would obscure the distinction between causal and noncausal assumptions.

A ***statistical assumption*** is any constraint on a joint distribution of observed variable; for example, that f is multivariate normal or that P is Markov relative to a given DAG D.

A ***causal assumption*** is any constraint on a causal model that cannot be realized by imposing statistical assumptions; for example, that f_i is linear, that U_i and U_j (unobserved) are uncorrelated, or that x_3 does not appear in $f_4(pa_4, u_4)$. Causal assumptions may or may not have statistical implications. In the former case we say that the assumption is "testable" or "falsifiable."

Remark: The distinction between causal and statistical parameters is crisp and fundamental. Causal parameters can be discerned from joint distributions only when special assumptions are made, and such assumptions *must* have causal components to them. The formulation and simplification of these assumptions will occupy a major part of this book.

Remark: Temporal precedence among variables may furnish some information about (the absence of) causal relationships – a later event cannot be the cause of an earlier event. Temporally indexed distributions such as $P(y_t \mid y_{t-1}, x_t)$, $t = 1, \ldots$, which are used routinely in economic analysis, may therefore be regarded as borderline cases between statistical and causal models. We shall nevertheless classify those models as statistical because the great majority of policy-related questions *cannot* be discerned from such distributions, given our commitment to making no assumption regarding the presence or absence of unmeasured variables. Consequently, econometric concepts such as "Granger causality" (Granger 1969) and "strong exogeneity" (Engle et al. 1983) will be classified as statistical rather than causal.[26]

Remark: The terms "theoretical" and "structural" are often used interchangeably with "causal"; we will use the latter two, keeping in mind that some structural models may not be causal (see Section 7.2.5).

[26] Caution must also be exercised in labeling as "data-generating model" the probabilistic sequence $P(y_t \mid y_{t-1}, x_t)$, $t = 1, \ldots$ (e.g. Davidson and MacKinnon 1993, p. 53; Hendry 1995). Causal assumptions of the type developed in Chapter 2 (see Definitions 2.4.1 and 2.7.4) must be invoked before applying such sequences in policy-related tasks.

Causal versus Statistical Concepts

The demarcation line between causal and statistical parameters extends as well to general concepts and will be supported by terminological distinction. Examples of *statistical* concepts are: correlation, regression, conditional independence, association, likelihood, collapsibility, risk ratio, odds ratio, and so on. Examples of *causal* concepts are: randomization, influence, effect, confounding, exogeneity, ignorability, disturbance (e.g. (1.40)), spurious correlation, path coefficients, instrumental variables, intervention, explanation, and so on. The purpose of this demarcation line is not to exclude causal concepts from the province of statistical analysis but, rather, to encourage investigators to treat nonstatistical concepts with the proper set of tools.

Some readers may be surprised by the idea that textbook concepts such as randomization, confounding, spurious correlation, or effects are nonstatistical. Others may be shocked at the idea that controversial concepts such as exogeneity, confounding, and counterfactuals *can* be defined in terms of causal models. This book is written with these readers in mind, and the coming pages will demonstrate that the distinctions just made between causal and statistical concepts are essential for clarifying both.

CHAPTER TWO

A Theory of Inferred Causation

I would rather discover one causal law
than be King of Persia.
Democritus (460–370 B.C.)

Preface

The possibility of learning causal relationships from raw data has been on philosophers' dream lists since the time of Hume (1711–1776). That possibility entered the realm of formal treatment and feasible computation in the mid-1980s, when the mathematical relationships between graphs and probabilistic dependencies came into light. The approach described herein is an outgrowth of Pearl (1988b, chap. 8), which describes how causal relationships can be inferred from nontemporal statistical data if one makes certain assumptions about the underlying process of data generation (e.g., that it has a tree structure). The prospect of inferring causal relationships from weaker structural assumptions (e.g., general directed acyclic graphs) has motivated parallel research efforts at three universities: UCLA, Carnegie Mellon University (CMU), and Stanford. The UCLA and CMU teams pursued an approach based on searching the data for patterns of conditional independencies that reveal fragments of the underlying structure and then piecing those fragments together to form a coherent causal model (or a set of such models). On the other hand, the Stanford group pursued a Bayesian approach, where data are used to update the posterior probabilities assigned to candidate causal structures (Cooper and Herskovits 1991). The UCLA and CMU efforts have led to similar theories and almost identical discovery algorithms, which were implemented in the TETRAD II program (Spirtes et al. 1993). The Bayesian approach has since been pursued by a number of research teams (Singh and Valtorta 1995; Heckerman et al. 1994) and now serves as the basis for several graph-based learning methods (Jordan 1998). This chapter describes the approach pursued by Tom Verma and me in the period 1988–1992, and it briefly summarizes related extensions, refinements, and improvements that have been advanced by the CMU team and others. Some of the philosophical rationale behind this development, primarily the assumption of minimality, are implicit in the Bayesian approach as well (Section 2.9.1).

The basic idea of automating the discovery of causes – and the specific implementation of this idea in computer programs – came under fierce debate in a number of forums (Cartwright 1995a; Humphreys and Freedman 1996; Cartwright 1999; Korb and Wallace 1997; McKim and Turner 1997; Robins and Wasserman 1999). Selected aspects of this debate will be addressed in the discussion section at the end of this chapter (Section 2.9.1).

Acknowledging that statistical associations do not *logically* imply causation, this chapter asks whether weaker relationships exist between the two. In particular, we ask:

1. What clues prompt people to perceive causal relationships in uncontrolled observations?
2. Is it feasible to infer causal models from these clues?
3. Would the models inferred tell us anything useful about the causal mechanisms that underly the observations?

In Section 2.2 we define the notions of causal models and causal structures and then describe the task of causal modeling as an inductive game that scientists play against Nature. In Section 2.3 we formalize the inductive game by introducing "minimal model" semantics – the semantical version of Occam's razor – and exemplify how, contrary to common folklore, causal relationships can be distinguished from spurious covariations following this standard norm of inductive reasoning. Section 2.4 identifies a condition, called *stability* (or *faithfulness*), under which effective algorithms exist that uncover structures of casual influences as defined here. One such algorithm (called IC), introduced in Section 2.5, uncovers the set of all causal models compatible with the data, assuming all variables are observed. Another algorithm (IC*), described in Section 2.6, is shown to uncover many (though not all) valid causal relationships when some variables are *not* observable. In Section 2.7 we extract from the IC* algorithm the essential conditions under which causal influences are identified, and we offer these as independent definitions of genuine influences and spurious associations, with and without temporal information. Section 2.8 offers an explanation for the puzzling yet universal agreement between the temporal and statistical aspects of causation. Finally, Section 2.9 summarizes the claims made in this chapter, re-explicates the assumptions that lead to these claims, and offers new justifications of these assumption in light of ongoing debates.

2.1 INTRODUCTION

An autonomous intelligent system attempting to build a workable model of its environment cannot rely exclusively on preprogrammed causal knowledge; rather, it must be able to translate direct observations to cause-and-effect relationships. However, given that statistical analysis is driven by covariation, not causation, and assuming that the bulk of human knowledge derives from uncontrolled observations, we must still identify the clues that prompt people to perceive causal relationships in the data. We must also find a computational model that emulates this perception.

Temporal precedence is normally assumed to be essential for defining causation, and it is undoubtedly one of the most important clues that people use to distinguish causal from other types of associations. Accordingly, most theories of causation invoke an explicit requirement that a cause precedes its effect in time (Reichenbach 1956; Good 1961; Suppes 1970; Shoham 1988). Yet temporal information alone cannot distinguish genuine causation from spurious associations caused by unknown factors – the barometer falls before it rains yet does not cause the rain. In fact, the statistical and philosophical literature has adamantly warned analysts that, unless one knows in advance all causally

relevant factors or unless one can carefully manipulate some variables, no genuine causal inferences are possible (Fisher 1951; Skyrms 1980; Cliff 1983; Eells and Sober 1983; Holland 1986; Gardenfors 1988; Cartwright 1989).[1] Neither condition is realizable in normal learning environments, and the question remains how causal knowledge is ever acquired from experience.

The clues that we explore in this chapter come from certain patterns of statistical associations that are characteristic of causal organizations – patterns that, in fact, can be given meaningful interpretation only in terms of causal directionality. Consider, for example, the following *intransitive* pattern of dependencies among three events: A and B are dependent, B and C are dependent, yet A and C are independent. If you ask a person to supply an example of three such events, the example would invariably portray A and C as two independent causes and B as their common effect, namely, $A \rightarrow B \leftarrow C$. (In my favorite example, A and C are the outcomes of two fair coins, and B represents a bell that rings whenever either coin comes up heads.) Fitting this dependence pattern with a scenario in which B is the cause and A and C are the effects is mathematically feasible but very unnatural (the reader is encouraged to try this exercise).

Such thought experiments tell us that certain patterns of dependency, which are totally void of temporal information, are conceptually characteristic of certain causal directionalities and not others. Reichenbach (1956) suggested that this directionality is a characteristic of Nature, reflective of the temporal asymmetries associated with the second law of thermodynamics. In Section 2.8 we offer a more subjective explanation, attributing the directionality to choice of language and to certain assumptions (e.g., Occam's razor and stability) prevalent in scientific induction. The focus of our investigation in this chapter is to explore whether this directionality provides a significant source of causal information and whether this information can be given formal characterization and an algorithmic implementation.

We start by introducing a model-theoretic semantics that gives a plausible account for how causal models could coherently be inferred from observations. Using this semantics we show that, subject to certain plausible assumptions, genuine causal influences can in many cases be distinguished from spurious covariations and, moreover, the direction of causal influences can often be determined without resorting to chronological information. (Although, when available, chronological information can significantly simplify the modeling task.)

2.2 THE CAUSAL MODELING FRAMEWORK

We view the task of causal modeling as an induction game that scientists play against Nature. Nature possesses stable causal mechanisms that, on a detailed level of descriptions, are deterministic functional relationships between variables, some of which are unobservable. These mechanisms are organized in the form of an acyclic structure, which the scientist attempts to identify from the available observations.

[1] Some of the popular quotes are: "No causation without manipulation" (Holland 1986), "No causes in, no causes out" (Cartwright 1989), "No computer program can take account of variables that are not in the analysis" (Cliff 1983).

Definition 2.2.1. (Causal Structure)
A causal structure *of a set of variables V is a directed acyclic graph (DAG) in which each node corresponds to a distinct element of V, and each link represents direct functional relationship among the corresponding variables.*

A causal structure serves as a blueprint for forming a "causal model" – a precise specification of *how* each variable is influenced by its parents in the DAG, as in the structural equation model of (1.40). Here we assume that Nature is at liberty to impose arbitrary functional relationships between each effect and its causes and then to perturb these relationships by introducing arbitrary (yet mutually independent) disturbances. These disturbances reflect "hidden" or unmeasurable conditions and exceptions that Nature chooses to govern by some undisclosed probability function.

Definition 2.2.2 (Causal Model)
A causal model *is a pair* $M = \langle D, \Theta_D \rangle$ *consisting of a causal structure D and a set of parameters* Θ_D *compatible with D. The parameters* Θ_D *assign a function* $x_i = f_i(pa_i, u_i)$ *to each* $X_i \in V$ *and a probability measure* $P(u_i)$ *to each* u_i, *where* PA_i *are the parents of* X_i *in D and where each* U_i *is a random disturbance distributed according to* $P(u_i)$, *independently of all other u.*

As we have seen in Chapter 1 (Theorem 1.4.1), the assumption of independent disturbances renders the model *Markovian* in the sense that each variable is independent of all its nondescendants, conditional on its parents in D. This Markov assumption is more a convention than an assumption, for it merely defines the granularity of the models we wish to consider as candidates before we begin the search. We can start in the deterministic extreme, where all variables are explicated in microscopic details and where the Markov condition certainly holds. As we move up to macroscopic abstractions by aggregating variables and introducing probabilities to summarize omitted variables, we need to decide at what stage the abstraction has gone too far and where useful properties of causation are lost. Evidently, the Markov condition has been recognized by our ancestors (the authors of our causal thoughts) as a property worth protecting in this abstraction; correlations that are not explained by common causes are considered spurious, and models containing such correlations are considered incomplete. The Markov condition guides us in deciding when a set of parents PA_i is considered complete in the sense that it include *all* the relevant immediate causes of variable X_i. It permits us to leave some of these causes out of PA_i (and be summarized by probabilities), but not if they also affect other variables modeled in the system. If a set PA_i in a model is too narrow, there will be disturbance terms that influence several variables simultaneously and the Markov property will be lost. Such disturbances will be treated explicitly as "latent" variables (see Definition 2.3.2). Once we acknowledge the existence of latent variables and represent their existence explicitly as nodes in a graph, the Markov property is restored.

Once a causal model M is formed, it defines a joint probability distribution $P(M)$ over the variables in the system. This distribution reflects some features of the causal structure (e.g., each variable must be independent of its grandparents, given the values of its parents). Nature then permits the scientist to inspect a select subset $O \subseteq V$ of "observed" variables and to ask questions about $P_{[O]}$, the probability distribution over

the observables, but it hides the underlying causal model as well as the causal structure. We investigate the feasibility of recovering the topology D of the DAG from features of the probability distribution $P_{[O]}$.[2]

2.3 MODEL PREFERENCE (OCCAM'S RAZOR)

In principle, since V is unknown, there is an unbounded number of models that would fit a given distribution, each invoking a different set of "hidden" variables and each connecting the observed variables through different causal relationships. Therefore, with no restriction on the type of models considered, the scientist is unable to make any meaningful assertions about the structure underlying the phenomena. For example, every probability distribution $P_{[O]}$ can be generated by a structure in which no observed variable is a cause of another but instead all variables are consequences of one latent common cause, U.[3] Likewise, assuming $V = O$ but lacking temporal information, the scientist can never rule out the possibility that the underlying structure is a complete, acyclic, and arbitrarily ordered graph – a structure that (with the right choice of parameters) can *mimic* the behavior of any model, regardless of the variable ordering. However, following standard norms of scientific induction, it is reasonable to rule out any theory for which we find a simpler, less elaborate theory that is equally consistent with the data (see Definition 2.3.5). Theories that survive this selection process are called *minimal*. With this notion, we can construct our (preliminary) definition of inferred causation as follows.

Definition 2.3.1 (Inferred Causation (Preliminary))
A variable X is said to have a causal influence *on a variable Y if a directed path from X to Y exists in every minimal structure consistent with the data.*

Here we equate a causal structure with a scientific theory, since both contain a set of free parameters that can be adjusted to fit the data. We regard Definition 2.3.1 as preliminary because it assumes that all variables are observed. The next few definitions generalize the concept of minimality to structures with unobserved variables.

Definition 2.3.2 (Latent Structure)
A latent structure *is a pair* $L = \langle D, O \rangle$, *where D is a causal structure over V and where* $O \subseteq V$ *is a set of observed variables.*

Definition 2.3.3 (Structure Preference)
One latent structure $L = \langle D, O \rangle$ *is* preferred *to another* $L' = \langle D', O \rangle$ *(written* $L \preceq L'$*) if and only if* D' *can mimic D over O – that is, if and only if for every* Θ_D *there exists a*

[2] This formulation invokes several idealizations of the actual task of scientific discovery. It assumes, for example, that the scientist obtains the distribution directly, rather than events sampled from the distribution. Additionally, we assume that the observed variables actually appear in the original causal model and are not some aggregate thereof. Aggregation might result in feedback loops, which we do not discuss in this chapter.

[3] This can be realized by letting U have as many states as O, assigning to U the prior distribution $P(u) = P(o(u))$ (where $o(u)$ is the cell of O corresponding to state u), and letting each observed variable O_i take on its corresponding value in $o(u)$.

$\Theta'_{D'}$ *such that* $P_{[O]}(\langle D', \Theta'_{D'}\rangle) = P_{[O]}(\langle D, \Theta_D\rangle)$. *Two latent structures are* equivalent, *written* $L' \equiv L$, *if and only if* $L \preceq L'$ *and* $L \succeq L'$.[4]

Note that the preference for simplicity imposed by Definition 2.3.3 is gauged by the expressive power of a structure, not by its syntactic description. For example, one latent structure L_1 may invoke many more parameters than L_2 and still be preferred if L_2 can accommodate a richer set of probability distributions over the observables. One reason scientists prefer simpler theories is that such theories are more constraining and thus more falsifiable; they provide the scientist with less opportunities to overfit the data "hindsightedly" and therefore command greater credibility if a fit is found (Popper 1959; Pearl 1978; Blumer et al. 1987).

We also note that the set of independencies entailed by a causal structure imposes limits on its expressive power, that is, its power to mimic other structures. Indeed, L_1 cannot be preferred to L_2 if there is even one observable dependency that is permitted by L_1 and forbidden by L_2. Thus, tests for preference and equivalence can sometimes be reduced to tests of induced dependencies, which in turn can be determined directly from the topology of the DAGs without ever concerning ourselves with the set of parameters. This is the case in the absence of hidden variables (see Theorem 1.2.8) but does not hold generally in all latent structures. Verma and Pearl (1990) showed that some latent structures impose numerical rather than independence constraints on the observed distribution (see e.g. Section 8.4, equations (8.21)–(8.23)); this makes the task of verifying model preference complicated but does still permit us to extend the semantical definition of inferred causation (Definition 2.3.1) to latent structures.

Definition 2.3.4 (Minimality)
A latent structure L *is* minimal *with respect to a class* $\mathcal{L}$ *of latent structures if and only if there is no member of* $\mathcal{L}$ *that is strictly preferred to* L *– that is, if and only if for every* $L' \in \mathcal{L}$ *we have* $L \equiv L'$ *whenever* $L' \preceq L$.

Definition 2.3.5 (Consistency)
A latent structure $L = \langle D, O\rangle$ *is* consistent *with a distribution* $\hat{P}$ *over* O *if* D *can accommodate some model that generates* $\hat{P}$ *– that is, if there exists a parameterization* Θ_D *such that* $P_{[O]}(\langle D, \Theta_D\rangle) = \hat{P}$.

Clearly, a necessary (and sometimes sufficient) condition for L to be consistent with $\hat{P}$ is that L can account for all the dependencies embodied in $\hat{P}$.

Definition 2.3.6 (Inferred Causation)
Given $\hat{P}$, *a variable* C *has a* causal influence *on variable* E *if and only if there exists a directed path from* C *to* E *in every minimal latent structure consistent with* $\hat{P}$.

We view this definition as normative because it is based on one of the least disputed norms of scientific investigation: Occam's razor in its semantical casting. However, as with any

[4] We use the succinct term "preferred to" to mean "preferred or equivalent to," a relation that has also been named "a submodel of."

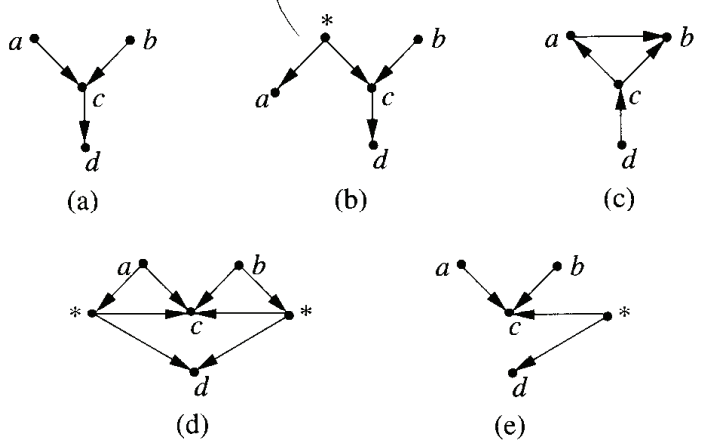

Figure 2.1 Causal structures illustrating the minimality of (a) and (b) and the justification for inferring the relationship $c \rightarrow d$. The node $(*)$ represents a hidden variable with any number of states.

scientific inquiry, we make no claims that this definition is guaranteed to always identify stable physical mechanisms in nature. It identifies the mechanisms we can plausibly infer from nonexperimental data; moreover, it guarantees that any alternative mechanism will be less trustworthy than the one inferred because the alternative would require more contrived, hindsighted adjustment of parameters (i.e. functions) to fit the data.

As an example of a causal relation that is identified by Definition 2.3.6, imagine that observations taken over four variables $\{a, b, c, d\}$ reveal two independencies: "a is independent of b" and "d is independent of $\{a, b\}$ given c." Assume further that the data reveals *no other* independence besides those that logically follow from these two. This dependence pattern would be typical, for example, of the following variables: a = having a cold, b = having hay fever, c = having to sneeze, d = having to wipe one's nose. It is not hard to see that structures (a) and (b) in Figure 2.1 are minimal, for they entail the observed independencies and none other.[5] Furthermore, any structure that explains the observed dependence between c and d by an arrow from d to c, or by a hidden common cause $(*)$ between the two, cannot be minimal, because any such structure would be able to "out-mimic" the one shown in Figure 2.1(a) (or the one in Figure 2.1(b)), which reflects all observed independencies. For example, the structure of Figure 2.1(c), unlike that of Figure 2.1(a), accommodates distributions with arbitrary relations between a and b. Similarly, Figure 2.1(d) is not minimal because it fails to impose the conditional independence between d and $\{a, b\}$ given c and will therefore accommodate distributions in which d and $\{a, b\}$ are dependent given c. In contrast, Figure 2.1(e) is not consistent with the data since it imposes an unobserved marginal independence between $\{a, b\}$ and d.

This example (taken from Pearl and Verma 1991) illustrates a remarkable connection between causality and probability: certain patterns of probabilistic dependencies (in our case, all dependencies except $(a \perp\!\!\!\perp b)$ and $(d \perp\!\!\!\perp \{a, b\} \mid c)$) imply unambiguous *causal* dependencies (in our case, $c \rightarrow d$) without making any assumption about the presence

[5] To verify that (a) and (b) are equivalent, we note that (b) can mimic (a) if we let the link $a \leftarrow *$ impose equality between the two variables. Conversely, (a) can mimic (b), since it is capable of generating every distribution that possesses the independencies entailed by (b). (For theory and methods of "reading off" conditional independencies from graphs, see Section 1.2.3 or Pearl 1988b.)

or absence of latent variables.[6] The only assumption invoked in this implication is minimality – models that overfit the data are ruled out.

2.4 STABLE DISTRIBUTIONS

Although the minimality principle is sufficient for forming a normative theory of inferred causation, it does not guarantee that the structure of the actual data-generating model would be minimal, or that the search through the vast space of minimal structures would be computationally practical. Some structures may admit peculiar parameterizations that would render them indistinguishable from many other minimal models that have totally disparate structures. For example, consider a binary variable C that takes the value 1 whenever the outcomes of two fair coins (A and B) are the same and takes the value 0 otherwise. In the trivariate distribution generated by this parameterization, each pair of variables is marginally independent yet is dependent conditional on the third variable. Such a dependence pattern may in fact be generated by three minimal causal structures, each depicting one of the variables as causally dependent on the other two, but there is no way to decide among the three. In order to rule out such "pathological" parameterizations, we impose a restriction on the distribution called *stability,* also known as DAG-isomorphism (Pearl 1988b, p. 128) and faithfulness (Spirtes et al. 1993). This restriction conveys the assumption that all the independencies embedded in P are stable; that is, they are entailed by the structure of the model D and hence remain invariant to any change in the parameters Θ_D. In our example, only the correct structure (namely, $A \rightarrow C \leftarrow B$) will retain its independence pattern in the face of changing parameterizations – say, when one of the coins becomes slightly biased.

Definition 2.4.1 (Stability)
Let $I(P)$ denote the set of all conditional independence relationships embodied in P. A causal model $M = \langle D, \Theta_D \rangle$ generates a stable *distribution if and only if $P(\langle D, \Theta_D \rangle)$ contains no extraneous independences – that is, if and only if $I(P(\langle D, \Theta_D \rangle)) \subseteq I(P(\langle D, \Theta'_D \rangle))$ for any set of parameters Θ'_D.*

The stability condition states that, as we vary the parameters from Θ to Θ', no independence in P can be destroyed; hence the name "stability." Succinctly, P is a stable distribution if there exists a DAG D such that $(X \perp\!\!\!\perp Y \mid Z)_P \iff (X \perp\!\!\!\perp Y \mid Z)_D$ for any three sets of variables X, Y, and Z (see Theorem 1.2.5).

The relationship between minimality and stability can be illustrated using the following analogy. Suppose we see a picture of a chair and that we need to decide between two theories as follows.

T_1: The object in the picture is a chair.

T_2: The object in the picture is either a chair or two chairs positioned such that one hides the other.

[6] Standard probabilistic definitions of causality (e.g. Suppes 1970; Eells 1991) invariably require knowledge of all relevant factors that may influence the observed variables (see Section 7.5.3).

Our preference for T_1 over T_2 can be justified on two principles, one based on minimality and the other on stability. The minimality principle argues that T_1 is preferred to T_2 because the set of scenes composed of single objects is a proper subset of scenes composed of two or fewer objects and, unless we have evidence to the contrary, we should prefer the more specific theory. The stability principle rules out T_2 a priori, arguing that it would be rather unlikely for two objects to align themselves so as to have one perfectly hide the other. Such an alignment would be *unstable* relative to slight changes in environmental conditions or viewing angle.

The analogy with independencies is clear. Some independencies are *structural,* that is, they would persist for every functional–distributional parameterization of the graph. Others are sensitive to the precise numerical values of the functions and distributions. For example, in the structure $Z \leftarrow X \rightarrow Y$, which stands for the relations

$$z = f_1(x, u_1), \qquad y = f_2(x, u_2), \tag{2.1}$$

the variables Z and Y will be independent, conditional on X, for all functions f_1 and f_2. In contrast, if we add an arrow $Z \rightarrow Y$ to the structure and use a linear model

$$z = \gamma x + u_1, \qquad y = \alpha x + \beta z + u_2, \tag{2.2}$$

with $\alpha = -\beta\gamma$, then Y and X will be independent. However, the independence between Y and X is unstable because it disappears as soon as the equality $\alpha = -\beta\gamma$ is violated. The stability assumption presumes that this type of independence is unlikely to occur in the data, that all independencies are structural.

To further illustrate the relations between stability and minimality, consider the causal structure depicted in Figure 2.1(c). The minimality principle rejects this structure on the ground that it fits a broader set of distributions than those fitted by structure (a). The stability principle rejects this structure on the ground that, in order to fit the data (specifically, the independence $(a \perp\!\!\!\perp b)$), the association produced by the arrow $a \rightarrow b$ must cancel precisely the one produced by the path $a \leftarrow c \rightarrow b$. Such precise cancelation cannot be stable, for it cannot be sustained for all functions connecting variables a, b, and c. In structure (a), by contrast, the independence $(a \perp\!\!\!\perp b)$ is stable.

2.5 RECOVERING DAG STRUCTURES

With the added assumption of stability, every distribution has a unique minimal causal structure (up to d-separation equivalence), as long as there are no hidden variables. This uniqueness follows from Theorem 1.2.8, which states that two causal structures are equivalent (i.e., they can mimic each other) if and only if they relay the same dependency information – namely, they have the same skeleton and same set of v-structures.

In the absence of unmeasured variables, the search for the minimal model then boils down to reconstructing the structure of a DAG D from queries about conditional independencies, assuming that those independencies reflect d-separation conditions in some undisclosed underlying DAG D_0. Naturally, since D_0 may have equivalent structures, the reconstructed DAG will not be unique, and the best we can do is to find a graphical representation for the equivalence class of D_0. Such graphical representation was introduced in Verma and Pearl (1990) under the name *pattern.* A pattern is a partially directed

DAG, in particular, a graph in which some edges are directed and some are nondirected. The directed edges represent arrows that are common to every member in the equivalence class of D_0, while the undirected edges represent ambivalence; they are directed one way in some equivalent structures and another way in others.

The following algorithm, introduced in Verma and Pearl (1990), takes as input a stable probability distribution $\hat{P}$ generated by some underlying DAG D_0 and outputs a pattern that represents the equivalence class of D_0.[7]

IC Algorithm (Inductive Causation)

Input: $\hat{P}$, a stable distribution on a set V of variables.

Output: a pattern $H(\hat{P})$ compatible with $\hat{P}$.

1. For each pair of variables a and b in V, search for a set S_{ab} such that $(a \perp\!\!\!\perp b \mid S_{ab})$ holds in $\hat{P}$ – in other words, a and b should be independent in $\hat{P}$, conditioned on S_{ab}. Construct an undirected graph G such that vertices a and b are connected with an edge if and only if no set S_{ab} can be found.
2. For each pair of nonadjacent variables a and b with a common neighbor c, check if $c \in S_{ab}$.
   If it is, then continue.
   If it is not, then add arrowheads pointing at c (i.e., $a \rightarrow c \leftarrow b$).
3. In the partially directed graph that results, orient as many of the undirected edges as possible subject to two conditions: (i) the orientation should not create a new v-structure; and (ii) the orientation should not create a directed cycle.

The IC algorithm leaves the details of steps 1 and 3 unspecified, and several refinements have been proposed for optimizing these two steps. Verma and Pearl (1990) noted that, in sparse graphs, the search can be trimmed substantially if commenced with the Markov network of $\hat{P}$, namely, the undirected graph formed by linking only pairs that are dependent conditionally on all other variables. In linear Gaussian models, the Markov network can be found in polynomial time, through matrix inversion, by assigning edges to pairs that correspond to the nonzero entries of the inverse covariance matrix. Spirtes and Glymour (1991) proposed a general systematic way of searching for the sets S_{ab} in step 1. Starting with sets S_{ab} of cardinality 0, then cardinality 1, and so on, edges are recursively removed from a complete graph as soon as separation is found. This refinement, called the PC algorithm (after its authors, Peter and Clark), enjoys polynomial time in graphs of finite degree because, at every stage, the search for a separating set S_{ab} can be limited to nodes that are adjacent to a and b.

Step 3 of the IC algorithm can be systematized in several ways. Verma and Pearl (1992) showed that, starting with any pattern, the following four rules are required for obtaining a maximally oriented pattern.

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[7] The IC algorithm, as introduced in Verma and Pearl (1990), was designed to operate on latent structures. For clarity, we here present the algorithm in two separate parts, IC and IC*, with IC restricted to DAGs and IC* operating on latent structures.

R_1: Orient $b - c$ into $b \rightarrow c$ whenever there is an arrow $a \rightarrow b$ such that a and c are nonadjacent.

R_2: Orient $a - b$ into $a \rightarrow b$ whenever there is chain $a \rightarrow c \rightarrow b$.

R_3: Orient $a - b$ into $a \rightarrow b$ whenever there are two chains $a - c \rightarrow b$ and $a - d \rightarrow b$ such that c and d are nonadjacent.

R_4: Orient $a - b$ into $a \rightarrow b$ whenever there are two chains $a - c \rightarrow d$ and $c \rightarrow d \rightarrow b$ such that c and b are nonadjacent.

Meek (1995) showed that these four rules are also sufficient, so that repeated application will eventually orient *all* arrows that are common to the equivalence class of D_0. Moreover, R_4 is not required if the starting orientation is limited to v-structures.

Another systematization is offered by an algorithm due to Dor and Tarsi (1992) that tests (in polynomial time) if a given partially oriented acyclic graph can be fully oriented without creating a new v-structure or a directed cycle. The test is based on recursively removing any vertex v that has the following two properties:

1. no edge is directed outward from v;
2. every neighbor of v that is connected to v through an undirected edge is also adjacent to all the other neighbors of v.

A partially oriented acyclic graph has an admissible extension in a DAG if and only if all its vertices can be removed in this fashion. Thus, to find the maximally oriented pattern, we can (i) separately try the two orientations, $a \rightarrow b$ and $a \leftarrow b$, for every undirected edge $a - b$, and (ii) test whether both orientations, or just one, have extensions. The set of uniquely orientable arrows constitutes the desired maximally oriented pattern. Additional refinements can be found in Chickering (1995), Andersson et al. (1997), and Moole (1997).

Latent structures, however, require special treatment, because the constraints that a latent structure imposes upon the distribution cannot be completely characterized by any set of conditional independence statements. Fortunately, certain sets of those independence constraints can be identified (Verma and Pearl 1990); this permits us to recover valid fragments of latent structures.

2.6 RECOVERING LATENT STRUCTURES

When Nature decides to "hide" some variables, the observed distribution $\hat{P}$ need no longer be stable relative to the observable set O. That is, we are no longer guaranteed that, among the minimal latent structures compatible with $\hat{P}$, there exists one that has a DAG structure. Fortunately, rather then having to search through this unbounded space of latent structures, the search can be confined to graphs with finite and well-defined structures. For every latent structure L, there is a dependency-equivalent latent structure (the projection) of L on O in which every unobserved node is a root node with exactly two observed children. We characterize this notion explicitly as follows.

Definition 2.6.1 (Projection)
A latent structure $L_{[O]} = \langle D_{[O]}, O \rangle$ *is a* projection *of another latent structure* L *if and only if:*

1. *every unobservable variable of* $D_{[O]}$ *is a parentless common cause of exactly two nonadjacent observable variables; and*
2. *for every stable distribution* P *generated by* L*, there exists a stable distribution* P' *generated by* $L_{[O]}$ *such that* $I(P_{[O]}) = I(P'_{[O]})$.

Theorem 2.6.2 (Verma 1993)
Any latent structure has at least one projection.

It is convenient to represent projections using a bidirectional graph with only the observed variables as vertices (i.e., leaving the hidden variables implicit). Each bidirected link in such a graph represents a common hidden cause of the variables corresponding to the link's endpoints.

Theorem 2.6.2 renders our definition of inferred causation (Definition 2.3.6) operational; it can be shown (Verma 1993) that the existence of a certain link in a distinguished projection of any minimal model of $\hat{P}$ must indicate the existence of a causal path in every minimal model of $\hat{P}$. Thus, our search reduces to finding the distinguished protection of any minimal model of $\hat{P}$ and identifying the appropriate links. Remarkably, these links can be identified by a simple variant of the IC algorithm, here called IC*, that takes a distribution $\hat{P}$ and returns a *marked* pattern, which is a partially directed acyclic graph that contains four types of edges:

1. a marked arrow $a \xrightarrow{*} b$, signifying a directed path from a to b in the underlying model;
2. an unmarked arrow $a \rightarrow b$, signifying either a directed path from a to b or a latent common cause $a \leftarrow L \rightarrow b$ in the underlying model;
3. a bidirected edge $a \leftrightarrow b$, signifying a latent common cause $a \leftarrow L \rightarrow b$ in the underlying model; and
4. an undirected edge $a - b$, standing for either $a \leftarrow b$ or $a \rightarrow b$ or $a \leftarrow L \rightarrow b$ in the underlying model.[8]

***IC* Algorithm* (Inductive Causation with Latent Variables)**

Input: $\hat{P}$, a sampled distribution.

Output: core($\hat{P}$), a marked pattern.

1. For each pair of variables a and b, search for a set S_{ab} such that a and b are independent in $\hat{P}$, conditioned on S_{ab}.

[8] Spirtes et al. (1993) used $a \circ\!\!\rightarrow b$ to represent uncertainty about the arrowhead at node a. Several errors in the original proof of IC* were pointed out to us by Peter Spirtes and were corrected in Verma (1993). Alternative proofs of correctness, as well as refinements in the algorithm, are given in Spirtes et al. (1993).

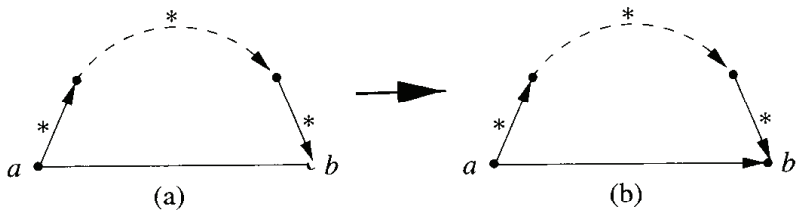


Figure 2.2 Illustration of R_2 in step 3 of the IC* algorithm.

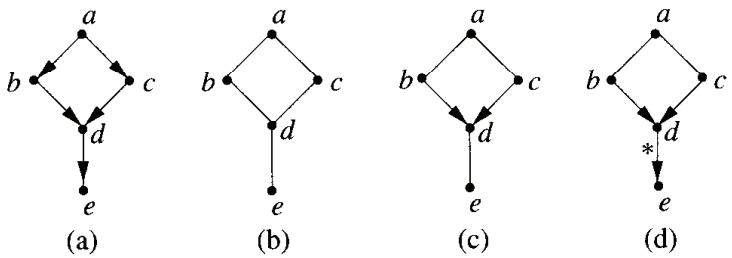


Figure 2.3 Graphs constructed by the IC* algorithm. (a) Underlying structure. (b) After step 1. (c) After step 2. (d) Output of IC*.

If there is no such S_{ab}, place an undirected link between the two variables, a — b.

2. For each pair of nonadjacent variables a and b with a common neighbor c, check if $c \in S_{ab}$.
   If it is, then continue.
   If it is not, then add arrowheads pointing at c (i.e., $a \rightarrow c \leftarrow b$).
3. In the partially directed graph that results, add (recursively) as many arrowheads as possible, and mark as many edges as possible, according to the following two rules:
   R_1: For each pair of nonadjacent nodes a and b with a common neighbor c, if the link between a and c has an arrowhead into c and if the link between c and b has no arrowhead into c, then add an arrowhead on the link between c and b pointing at b and mark that link to obtain $c \overset{*}{\rightarrow} b$.
   R_2: If a and b are adjacent and there is a directed path (composed strictly of marked links) from a to b (as in Figure 2.2), then add an arrowhead pointing toward b on the link between a and b.

Steps 1 and 2 of IC* are identical to those of IC, but the rules in step 3 are different; they do not orient edges but rather add arrowheads to the individual endpoints of the edges, thus accommodating bidirectional edges.

Figure 2.3 illustrates the operation of the IC* algorithm on the sprinkler example of Figure 1.2 (shown schematically in Figure 2.3(a)).

1. The conditional independencies entailed by this structure can be read off using the d-separation criterion (Definition 1.2.3), and the smallest conditioning sets corresponding to these independencies are given by $S_{ad} = \{b, c\}$, $S_{ae} = \{d\}$,

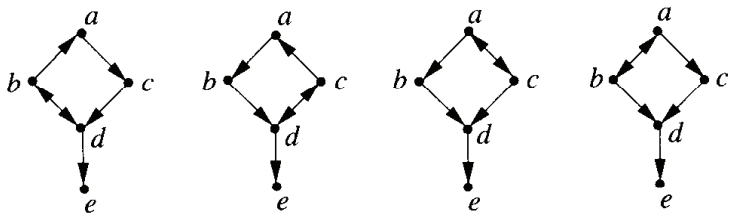


Figure 2.4 Latent structures equivalent to those of Figure 2.3(a).

$S_{bc} = \{a\}$, $S_{be} = \{d\}$, and $S_{ce} = \{d\}$. Thus, step 1 of IC* yields the undirected graph of Figure 2.3(b).

2. The triplet (b, d, c) is the only one that satisfies the condition of step 2, since d is not in S_{bc}. Accordingly, we obtain the partially directed graph of Figure 2.3(c).
3. Rule R_1 of step 3 is applicable to the triplet (b, d, e) (and to (c, d, e)), since b and e are nonadjacent and there is an arrowhead at d from b but not from e. We therefore add an arrowhead at e, and mark the link, to obtain Figure 2.3(d). This is also the final output of IC*, because R_1 and R_2 are no longer applicable.

The absence of arrowheads on a — b and a — c, and the absence of markings on $b \rightarrow d$ and $c \rightarrow d$, correctly represent the ambiguities presented by $\hat{P}$. Indeed, each of the latent structures shown in Figure 2.4 is observationally equivalent to that of Figure 2.3(a). Marking the link $d \rightarrow e$ in Figure 2.3(d) advertises the existence of a directed link $d \rightarrow e$ in each and every latent structure that is independence-equivalent to the one in Figure 2.3(a).

2.7 LOCAL CRITERIA FOR CAUSAL RELATIONS

The IC* algorithm takes a distribution $\hat{P}$ and outputs a partially directed graph. Some of the links are marked unidirectional (denoting genuine causation), some are *un*marked unidirectional (denoting potential causation), some are bidirectional (denoting spurious association), and some are undirected (denoting relationships that remain undetermined). The conditions that give rise to these labelings can be taken as definitions for the various kinds of causal relationships. In this section we present explicit definitions of potential and genuine causation as they emerge from the IC* algorithm. Note that, in all these definitions, the criterion for causation between two variables (X and Y) will require that a third variable Z exhibit a specific pattern of dependency with X and Y. This is not surprising, since the essence of causal claims is to stipulate the behavior of X and Y under the influence of a third variable, one that corresponds to an external control of X (or Y) – as echoed in the paradigm of "no causation without manipulation" (Holland 1986). The difference is only that the variable Z, acting as a virtual control, must be identified within the data itself, as if Nature had performed the experiment. The IC* algorithm can be regarded as offering a systematic way of searching for variables Z that qualify as virtual controls, given the assumption of stability.

Definition 2.7.1 (Potential Cause)
A variable X has a potential causal influence *on another variable Y (that is inferable from* $\hat{P}$*) if the following conditions hold.*

1. *X and Y are dependent in every context.*
2. *There exists a variable Z and a context S such that*
   (i) *X and Z are independent given S (i.e.,* $X \perp\!\!\!\perp Z \mid S$*) and*
   (ii) *Z and Y are dependent given S (i.e.,* $Z \not\perp\!\!\!\perp Y \mid S$*).*

By "context" we mean a set of variables tied to specific values. In Figure 2.3(a), for example, variable b qualifies as a potential cause of d by virtue of variable $Z = c$ being dependent on d and independent of b in context $S = a$. Likewise, c qualifies as potential cause of d (with $Z = b$ and $S = a$). Neither b nor c qualifies as genuine cause of d, because this pattern of dependencies is also compatible with a latent common cause, shown as bidirected arcs in Figures 2.4(a)–(b). However, Definition 2.7.1 disqualifies d as a cause of b (or c), and this leads to the classification of d as a *genuine* cause of e, as formulated in Definition 2.7.2.[9] Note that Definition 2.7.1 precludes a variable X from being a potential cause of itself or of any other variable that functionally determines X.

Definition 2.7.2 (Genuine Cause)
A variable X has a genuine causal influence *on another variable Y if there exists a variable Z such that either:*

1. *X and Y are dependent in any context and there exists a context S satisfying*
   (i) *Z is a potential cause of X (per Definition 2.7.1),*
   (ii) *Z and Y are dependent given S (i.e.,* $Z \not\perp\!\!\!\perp Y \mid S$*), and*
   (iii) *Z and Y are independent given S* $\cup$ *X (i.e.,* $Z \perp\!\!\!\perp Y \mid S \cup X$*);*
   or
2. *X and Y are in the transitive closure of the relation defined in criterion 1.*

Conditions (i)–(iii) are illustrated in Figure 2.3(a) with $X = d$, $Y = e$, $Z = b$, and $S = \emptyset$. The destruction of the dependence between b and e through conditioning on d cannot be attributed to spurious association between d and e; genuine causal influence is the only explanation, as shown in the structures of Figure 2.4.

Definition 2.7.3 (Spurious Association)
Two variables X and Y are spuriously associated *if they are dependent in some context and there exist two other variables* (Z_1 *and* Z_2) *and two contexts* (S_1 *and* S_2) *such that:*

[9] Definition 2.7.1 was formulated in Pearl (1990) as a relation between events (rather than variables) with the added condition $P(Y \mid X) > P(Y)$ (in the spirit of Reichenbach 1956, Good 1961, and Suppes 1970). This refinement is applicable to any of the definitions in this section, but it will not be formulated explicitly.

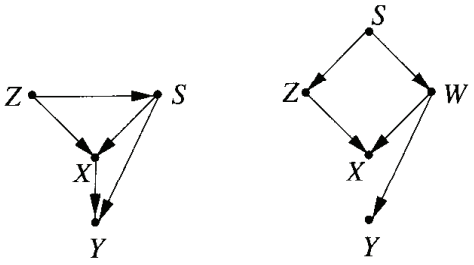


Figure 2.5 Illustration of how temporal information permits the inference of genuine causation and spurious associations (between X and Y) from the conditional independencies displayed in (a) and (b), respectively.

1. *Z_1 and X are dependent given S_1 (i.e., $Z_1 \not\perp\!\!\!\perp X \mid S_1$);*
2. *Z_1 and Y are independent given S_1 (i.e., $Z_1 \perp\!\!\!\perp Y \mid S_1$);*
3. *Z_2 and Y are dependent given S_2 (i.e., $Z_2 \not\perp\!\!\!\perp Y \mid S_2$); and*
4. *Z_2 and X are independent given S_2 (i.e., $Z_2 \perp\!\!\!\perp X \mid S_2$).*

Conditions 1 and 2 use Z_1 and S_1 to disqualify Y as a cause of X, paralleling conditions (i)–(ii) of Definition 2.7.1; conditions 3 and 4 use Z_2 and S_2 to disqualify X as a cause of Y. This leaves the existence of a latent common cause as the only explanation for the observed dependence between X and Y, as exemplified in the structure $Z_1 \rightarrow X \leftarrow\!\rightarrow Y \leftarrow Z_2$.

When temporal information is available (as is assumed in the most probabilistic theories of causality – Suppes 1970; Spohn 1983; Granger 1988), Definitions 2.7.2 and 2.7.3 simplify considerably because every variable preceding and adjacent to X now qualifies as a "potential cause" of X. Moreover, adjacency (i.e., condition 1 of Definition 2.7.1) is not required as long as the context S is confined to be earlier than X. These considerations lead to simpler conditions distinguishing genuine from spurious causes, as shown next.

Definition 2.7.4 (Genuine Causation with Temporal Information)
A variable X has a causal influence on Y if there is a third variable Z and a context S, both occurring before X, such that:

1. $(Z \not\perp\!\!\!\perp Y \mid S)$;
2. $(Z \perp\!\!\!\perp Y \mid S \cup X)$.

The intuition behind Definition 2.7.4 is the same as for Definition 2.7.2, except that temporal precedence is now used to establish Z as a potential cause of X. This is illustrated in Figure 2.5(a): If conditioning on X can turn Z and Y from dependent to independent (in context S), it must be that the dependence between Z and Y was mediated by X; given that Z precedes X, such mediation implies that X has a causal influence on Y.

Definition 2.7.5 (Spurious Association with Temporal Information)
Two variables X and Y are spuriously associated *if they are dependent in some context S, if X precedes Y, and if there exists a variable Z satisfying:*

1. $(Z \perp\!\!\!\perp Y \mid S)$;
2. $(Z \not\perp\!\!\!\perp X \mid S)$.

Figure 2.5(b) illustrates the intuition behind Definition 2.7.5. Here the dependence between X and Y cannot be attributed to causal connection between the two because such a connection would imply dependence between Z and Y, which is ruled out by condition 1.[10]

Examining the definitions just presented, we see that all causal relations are inferred from at least three variables. Specifically, the information that permits us to conclude that one variable is not a causal consequence of another comes in the form of an "intransitive triplet" – for example, the variables a, b, c in Figure 2.1(a) satisfying $(a \perp\!\!\!\perp b \mid \emptyset)$, $(a \not\perp\!\!\!\perp c \mid \emptyset)$, and $(b \not\perp\!\!\!\perp c \mid \emptyset)$. The argument goes as follows. If we find conditions (S_{ab}) where the variables a and b are each correlated with a third variable c but are independent of each other, then the third variable cannot act as a cause of a or b (recall that, in stable distributions, the presence of a common cause implies dependence among the effects); rather, c must either be their common effect $(a \rightarrow c \leftarrow b)$ or be associated with a and b via common causes, forming a pattern such as $a \leftarrow\!\!\rightarrow c \leftarrow\!\!\rightarrow b$. This is indeed the condition that permits the IC* algorithm to begin orienting edges in the graph (step 2) and to assign arrowheads pointing at c. It is also this intransitive pattern that is used to ensure that X is not a consequence of Y in Definition 2.7.1 and that Z is not a consequence of X in Definition 2.7.2. In Definition 2.7.3 we have two intransitive triplets, (Z_1, X, Y) and (X, Y, Z_2), thus ruling out direct causal influence between X and Y and so implying that spurious associations are the only explanation for their dependence.

This interpretation of intransitive triples involves a virtual control of the effect variable, rather than of the putative cause; this is analogous to testing the null hypothesis in the manipulative view of causation (Section 1.3). For example, one of the reasons people insist that the rain causes the grass to become wet, and not the other way around, is that they can easily find other means of getting the grass wet that are totally independent of the rain. Transferred to our chain $a - c - b$, we preclude c from being a cause of a if we find another means (b) of potentially controlling c without affecting a (Pearl 1988a, p. 396). The analogy is merely heuristic, of course, because in observational studies we must wait for Nature to provide the appropriate control and refrain from contaminating that control with spurious associations (with a).

2.8 NONTEMPORAL CAUSATION AND STATISTICAL TIME

Determining the direction of causal influences from nontemporal data raises some interesting philosophical questions about the relationships between time and causal explanations. For example, can the orientation assigned to the arrow $X \rightarrow Y$ in Definitions 2.7.2

[10] Recall that transitivity of causal dependencies is implied by stability. Although it is possible to construct causal chains $Z \rightarrow X \rightarrow Y$ in which Z and Y are independent, such independence will not be sustained for *all* parameterizations of the chain.

or 2.7.4 ever clash with the available temporal information (say, by a subsequent discovery that Y precedes X)? Since the rationale behind Definition 2.7.4 is based on strong intuitions about the statistical aspects of causal relationships (e.g., no correlation without some causation), it is apparent that such clashes, if they occur, are rather rare. The question then arises: Why should orientations determined solely by statistical dependencies have anything to do with the flow of time?

In human discourse, causal explanations satisfy two expectations: temporal and statistical. The temporal aspect is represented by the understanding that a cause should precede its effect. The statistical aspect expects a complete causal explanation to screen off its various effects (i.e., render the effects conditionally independent);[11] explanations that do not screen off their effects are considered "incomplete," and the residual dependencies are considered "spurious" or "unexplained." The clashless coexistence of these two expectations through centuries of scientific observations implies that the statistics of natural phenomena must exhibit some basic temporal bias. Indeed, we often encounter phenomenon where knowledge of a present state renders the variables of the future state conditionally independent (e.g., multivariate economic time series as in (2.3)). However, we rarely find the converse phenomenon, where knowledge of the present state would render the components of the past state conditionally independent. Is there any compelling reason for this temporal bias?

A convenient way to formulate this bias is through the notion of statistical time.

Definition 2.8.1 (Statistical Time)
Given an empirical distribution P, a statistical time *of P is any ordering of the variables that agrees with at least one minimal causal structure consistent with P.*

We see, for example, that a scalar Markov chain process has many statistical times; one coinciding with the physical time, one opposite to it, and others that correspond to orderings that agree with any orientation of the Markov chain away from one of the nodes (arbitrarily chosen as a root). On the other hand, a process governed by two coupled Markov chains, such as

$$\begin{aligned} X_t &= \alpha X_{t-1} + \beta Y_{t-1} + \xi_t, \\ Y_t &= \gamma X_{t-1} + \delta Y_{t-1} + \eta_t, \end{aligned} \tag{2.3}$$

has only one statistical time – the one coinciding with the physical time.[12] Indeed, running the IC algorithm on samples taken from such a process – while suppressing all temporal information – quickly identifies the components of X_{t-1} and Y_{t-1} as genuine

[11] This expectation, known as Reichenbach's "conjunctive fork" or "common-cause" criterion (Reichenbach 1956; Suppes and Zaniotti 1981; Sober and Barrett 1992) has been criticized by Salmon (1984a), who showed that some events qualify as causal explanations though they fail to meet Reichenbach's criterion. However, Salmon's examples involve incomplete explanations, as they leave out variables that mediate between the cause and its various effects (see Section 2.9.1).

[12] Here ξ_t and η_t are assumed to be two independent, white-noise time series. Also, $\alpha \neq \delta$ and $\gamma \neq \beta$.

causes of X_t and Y_t. This can be seen from Definition 2.7.1 (where X_{t-2} qualifies as a potential cause of X_{t-1} using $Z = Y_{t-2}$ and $S = \{X_{t-3}, Y_{t-3}\}$) and Definition 2.7.2 (where X_{t-1} qualifies as a genuine cause of X_t using $Z = X_{t-2}$ and $S = \{Y_{t-1}\}$).

The temporal bias postulated earlier can be expressed as follows.

Conjecture 2.8.2 (Temporal Bias)
In most natural phenomenon, the physical time coincides with at least one statistical time.

Reichenbach (1956) attributed the asymmetry associated with his conjunctive fork to the second law of thermodynamics. It is doubtful that the second law can provide a full account of the temporal bias just described, since the influence of the external noise ξ_t and η_t renders the process in (2.3) nonconservative.[13] Moreover, the temporal bias is language-dependent. For example, expressing (2.3) in a different coordinate system – say, using a linear transformation

$$X'_t = aX_t + bY_t,$$
$$Y'_t = cX_t + dY_t$$

– it is possible to make the statistical time in the (X', Y') representation run contrary to the physical time; that is, X'_t and Y'_t will be independent of each other conditional on their future values (X'_{t+1} and Y'_{t+1}) rather than their past values. This suggests that the consistent agreement between physical and statistical times is a byproduct of the human choice of linguistic primitives and not a feature of physical reality. For example, if X_t and Y_t stand for the positions of two interacting particles at time t, with X'_t the position of their center of gravity and Y'_t their relative distance, then describing the particles' motion in the (X, Y) versus (X', Y') coordinate system is (in principle) a matter of choice. Evidently, however, this choice is not entirely whimsical; it reflects a preference toward coordinate systems in which the *forward* disturbances (ξ_t and η_t in (2.3)) are orthogonal to each other, rather than the corresponding backward disturbances (ξ'_t and η'_t). Pearl and Verma (1991) speculated that this preference represents survival pressure to facilitate predictions of future events, and that evolution has evidently ranked this facility more urgent than that of finding hindsighted explanations for current events. Whether this or some other force has shaped our choice of language remains to be investigated (see discussions in Price 1996), which makes the statistical–temporal agreement that much more interesting.

2.9 CONCLUSIONS

The theory presented in this chapter shows that, although statistical analysis cannot distinguish genuine causation from spurious covariation in every conceivable case, in many cases it can. Under the assumptions of model minimality (and/or stability), there are

[13] I am grateful to Seth Lloyd for this observation.

patterns of dependencies that should be sufficient to uncover genuine causal relationships. These relationships cannot be attributed to hidden causes lest we violate one of the basic maxims of scientific methodology: the semantical version of Occam's razor. Adherence to this maxim may explain why humans reach consensus regarding the directionality and nonspuriousness of causal relationships in the face of opposing alternatives that are perfectly consistent with observation. Echoing Cartwright (1989), we summarize our claim with the slogan "No causes in – No causes out; Occam's razor in – Some causes out."

How safe are the causal relationships inferred by the IC algorithm – or by the TETRAD program of Spirtes et al. (1993) or the Bayesian methods of Cooper and Herskovits (1991) or Heckerman et al. (1994)?

Recasting this question in the context of visual perception, we may equally well ask: How safe are our predictions when we recognize three-dimensional objects from their two-dimensional shadows, or from the two-dimensional pictures that objects reflect on our retinas? The answer is: Not absolutely safe, but good enough to tell a tree from a house and good enough to make useful inferences without having to touch every physical object that we see. Returning to causal inference, our question then amounts to assessing whether there are enough discriminating clues in a typical learning environment (say, in skill acquisition tasks or in epidemiological studies) to allow us to make reliable discriminations between cause and effect. This can only be determined by experiments – once we understand the logic behind the available clues and once we learn to piece these clues together coherently in large programs that tackle real-life problems.

The model-theoretic semantics presented in this chapter provides a conceptual and theoretical basis for such experiments. The IC* algorithm and the algorithms developed by the TETRAD group (Spirtes et al. 1993) demonstrate the computational feasibility of the approach. Waldmann et al. (1995) described psychological experiments on how humans use the causal clues discussed in this chapter.

On the practical side, we have shown that the assumption of model minimality, together with that of "stability" (no accidental independencies) lead to an effective algorithm for structuring candidate causal models capable of generating the data, transparent as well as latent. Simulation studies conducted at our laboratory in 1990 showed that networks containing tens of variables require fewer than 5,000 samples to have their structure recovered by the algorithm. For example, 1,000 samples taken from (a binary version of) the process shown in (2.3), each containing ten successive X, Y pairs, were sufficient to recover its double-chain structure (and the correct direction of time). The greater the noise, the quicker the recovery (up to a point). In testing this modeling scheme on real-life data, we have examined the observations reported in Sewal Wright's seminal paper "Corn and Hog Correlations" (Wright 1925). As expected, corn price (X) can clearly be identified as a cause of hog price (Y) but not the other way around. The reason lies in the existence of the variable corn crop (Z), which satisfies the conditions of Definition 2.7.2 (with $S = \emptyset$). Several applications of the principles and algorithms discussed in this chapter are described in Glymour and Cooper (1999, pp. 441–541).

It should be interesting to explore how the new criteria for causation could benefit current research in machine learning and data mining. In some sense, our method resembles a standard, machine-learning search through a space of hypotheses (Mitchell 1982)

where each hypothesis stands for a causal model. Unfortunately, this is where the resemblance ends. The prevailing paradigm in the machine-learning literature has been to define each hypothesis (or theory, or concept) as a subset of observable instances; once we observe the entire extension of this subset, the hypothesis is defined unambiguously. This is not the case in causal modeling. Even if the training sample exhausts the hypothesis subset (in our case, this corresponds to observing P precisely), we are still left with a vast number of equivalent causal theories, each stipulating a drastically different set of causal claims. Therefore, *fitness to data is an insufficient criterion for validating causal theories.* Whereas in traditional learning tasks we attempt to generalize from one set of instances to another, the causal modeling task is to generalize from behavior under one set of conditions to behavior under another set. Causal models should therefore be chosen by a criterion that challenges their stability against changing conditions, and these show up in the data in the form of virtual control variables. Thus, the dependence patterns identified by Definitions 2.7.1–2.7.4 constitute islands of stability as well as virtual validation tests for causal models. It would be interesting to examine whether these criteria, when incorporated into existing machine-learning and data-mining programs, would improve the stability of relationships discovered by such programs.

2.9.1 On Minimality, Markov, and Stability

The idea of inferring causation from association cannot be expected to go unchallenged by scientists trained along the lines of traditional doctrines. Naturally, the assumptions underlying the theory described in this chapter – minimality and stability – come under attack from statisticians and philosophers. This section contains additional thoughts in defense of these assumptions.

Although few have challenged the principle of minimality (to do so would amount to challenging scientific induction), objections have been voiced against the way we defined the objects of minimization – namely, causal models. Definition 2.2.2 assumes that the stochastic terms u_i are mutually independent, an assumption that endows each model with the Markov property: conditioned on its parents (direct causes), each variable is independent of its nondescendants. This implies, among the other ramifications of d-separation, several familiar relationships between causation and association that are usually associated with Reichenbach's (1956) principle of common cause – for example, "no correlation without causation," "causes screen off their effects," "no action at a distance."

The Markovian assumption, as explained in our discussion of Definition 2.2.2, is a matter of convention, and it has been adopted here as a useful abstraction of the underlying physical processes because such processes are too detailed to be of practical use. After all, investigators are free to decide what level of abstraction is useful for a given purpose, and Markovian models have been selected as targets of pursuit because of their usefulness in both prediction and decision making.[14] By building the Markovian assumption into the definition of complete causal models (Definition 2.2.2) and then relaxing

[14] Discovery algorithms for certain non-Markovian models, involving cycles and selection bias, have been reported in Spirtes et al. (1995) and Richardson (1996).

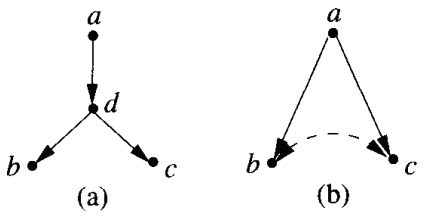


Figure 2.6 (a) Interactive fork. (b) Latent structure equivalent to (a).

the assumption through latent structures (Definition 2.3.2), we confess our preparedness to miss the discovery of non-Markovian causal models that cannot be described as latent structures. I do not consider this loss to be very serious, because such models – even if any exist in the macroscopic world – would have limited utility as guides to decisions. For example, it is not clear how one would predict the effects of interventions from such a model, save for explicitly listing the effect of every conceivable intervention in advance.

It is not surprising, therefore, that criticisms of the Markov assumption, most notably those of Cartwright (1995a, 1997) and Lemmer (1993), have two characteristics in common:

1. they present macroscopic non-Markovian counterexamples that are reducible to Markovian latent structures of the type considered by Salmon (1984), that is, interactive forks; and
2. they propose no alternative, non-Markovian models from which one could predict the effects of actions and action combinations.

The interactive fork model is shown in Figure 2.6(a). If the intermediate node d is unobserved (or unnamed), then one is tempted to conclude that the Markov assumption is violated, since the observed cause (a) does not screen off its effects (b and c). The latent structure of Figure 2.6(b) can emulate the one of Figure 2.6(a) in all respects; the two can be indistinguishable both observationally and experimentally.

Only quantum-mechanical phenomena exhibit associations that cannot be attributed to latent variables, and it would be considered a scientific miracle if anyone were to discover such peculiar associations in the macroscopic world. Still, critics of the Markov condition insist that certain alleged counterexamples must be modeled as $P(bc \mid a)$ and not as $\sum_d P(b \mid d, a)P(c \mid d, a)$ – assuming, perhaps, that some insight or generality would be gained by leaving the dependency between b and c unexplained. The former model, in addition to being observationally indistinguishable from the latter, also leaves the causal effect $P_{ac}(b)$ unspecified. In contrast, the latent model predicts $P_{ac}(b) = P_a(b)$ and thus fulfills its role as a predictor of (experimentally testable) causal effects.

Ironically, perhaps the strongest evidence for the ubiquity of the Markov condition can be found in the philosophical program known as "probabilistic causality" (see Section 7.5), of which Cartwright is a leading proponent. In this program, causal dependence is defined as a probabilistic dependence that persists after conditioning on some set of relevant factors (Good 1961; Suppes 1970; Skyrms 1980; Cartwright 1983; Eells 1991). This definition rests on the assumption that conditioning on the right set of factors enables one to suppress all spurious associations – an assumption equivalent to the Markov condition. The intellectual survival of probabilistic causality as an active philosophical

program for the past 30 years attests to the fact that counterexamples to the Markov condition are relatively rare and can be explained away through latent variables.

I now address the assumption of stability. The argument usually advanced to justify stability (Spirtes et al. 1993) appeals to the fact that strict equalities among products of parameters have zero Lebesgue measure in any probability space in which parameters can vary independently of one another. For example, the equality $\alpha = -\beta\gamma$ in the model of (2.2) has zero probability if we consider any continuous joint density over the parameters α, β, and γ, unless that density somehow embodies the constraint $\alpha = -\beta\gamma$ on a priori grounds. Freedman (1997), in contrast, claimed that there is no reason to assume that parameters are not in fact tied together by constraints of this sort, which would render the resulting distribution unstable (using Definition 2.4.1).

Freedman's critique receives unexpected support from the practice of structural modeling itself, where equality constraints are commonplace. Indeed, the conditional independencies that a causal model advertises amount to none other than equality constraints on the joint distribution. The chain model $Y \rightarrow X \rightarrow Z$, for example, entails the equality

$$\rho_{YZ} = \rho_{XZ} \cdot \rho_{YX},$$

where ρ_{XY} is the correlation coefficient between X and Y; this equality constraint ties the three correlation coefficients in a permanent bond. What, then, gives equalities among correlation coefficients a privileged status over equalities among another set of parameters – say, α, β, and γ? Why do we consider the equality $\rho_{YZ} = \rho_{XZ} \cdot \rho_{YX}$ "substantive" and the equality $\alpha = -\beta\gamma$ "accidental," and why do we tie the notion of stability to the absence of the latter, not the former?

The answer, I believe, rests again on the notion of *autonomy* (Aldrich 1989), a notion at the heart of all causal concepts (see Sections 1.3 and 1.4). A causal model is not just another scheme of encoding probability distribution through a set of parameters. When we come to define mathematical objects such as causal models, we must ensure that the definition captures the distinct ways in which these objects are being used and conceptualized. The distinctive feature of causal models is that each variable is determined by a set of other variables through a relationship (called "mechanism") that remains *invariant* when those other variables are subjected to external influences. Only by virtue of this invariance do causal models allow us to predict the effect of changes and interventions, capitalizing on the locality of such changes. This invariance means that mechanisms *can* vary independently of one another, which in turns implies that the set of structural coefficients (e.g., α, β, γ in our example of (2.2)) – rather than other types of parameters (e.g., ρ_{YZ}, ρ_{XZ}, ρ_{YX}) – can and will vary independently when experimental conditions change. Consequently, equality constraints of the form $\alpha = -\beta\gamma$ are contrary to the idea of autonomy and thus should not be considered part of the model.

For this reason, it has been suggested that causal modeling methods based solely on associations, like those embodied in the IC* algorithm or the TETRAD-II program, will find their greatest potential in longitudinal studies conducted under slightly varying conditions, where accidental independencies are destroyed and only structural independencies are preserved. This assumes that, under such varying conditions, the parameters of the model will be perturbed while its structure remains intact – a delicate balance that

might be hard to verify. Still, considering the alternative of depending only on controlled, randomized experiments, such longitudinal studies are an exciting opportunity.

Relation to the Bayesian Approach

It is important to stress that elements of the principles of minimality and stability also underlie causal discovery in the Bayesian approach. In this approach, one assigns prior probabilities to a set of candidate causal networks, based on their structures and parameters, and then uses Bayes's rule to score the degree to which a given network fits the data (Cooper and Herskovits 1991; Heckerman et al. 1999). A search is then conducted over the space of possible structures to seek the one(s) with the highest posterior score. Methods based on this approach have the advantage of operating well under small-sample conditions, but they encounter difficulties in coping with hidden variables. The assumption of parameter independence, which is made in all practical implementations of the Bayesian approach, induces preferences toward models with fewer parameters and hence toward minimality. Likewise, parameter independence can be justified only when the parameters represent mechanisms that are free to change independently of one another – that is, when the system is autonomous and hence stable.

CHAPTER THREE

Causal Diagrams and the Identification of Causal Effects

The eye obeys exactly
the action of the mind.
Emerson (1860)

Preface

In the previous chapter we dealt with ways of learning causal relationships from raw data. In this chapter we explore the ways of learning such relationships from a combination of data and qualitative causal assumptions that are deemed plausible in a given domain. More broadly, this chapter aims to help researchers communicate qualitative assumptions about cause–effect relationships, elucidate the ramifications of such assumptions, and derive causal inferences from a combination of assumptions, experiments, and data. Our major task will be to decide whether the assumptions given are sufficient for assessing the strength of causal effects from nonexperimental data.

Causal effects permit us to predict how systems would respond to hypothetical interventions – for example, policy decisions or actions performed in everyday activity. As we have seen in Chapter 1 (Section 1.3), such predictions are the hallmark of causal modeling, since they are not discernible from probabilistic information alone; they rest on – and, in fact, define – causal relationships. This chapter uses causal diagrams to give formal semantics to the notion of *intervention,* and it provides explicit formulas for postintervention probabilities in terms of preintervention probabilities. The implication is that the effects of every intervention can be estimated from nonexperimental data, provided the data is supplemented with a causal diagram that is both acyclic and contains no latent variables.

If some variables are not measured then the question of identifiability arises, and this chapter develops a nonparametric framework for analyzing the identification of causal relationships in general and causal effects in particular. We will see that causal diagrams provide a powerful mathematical tool in this analysis; they can be queried, using extremely simple tests, to determine if the assumptions available are sufficient for identifying causal effects. If so, the diagrams produce mathematical expressions for causal effects in terms of observed distributions; otherwise, the diagrams can be queried to suggest additional observations or auxiliary experiments from which the desired inferences can be obtained.

Another tool that emerges from the graphical analysis of causal effects is a *calculus of interventions* – a set of inference rules by which sentences involving interventions and observations can be transformed into other such sentences, thus providing a syntactic method of deriving (or verifying) claims about interventions and the way they interact

with observations. With the help of this calculus the reader will be able to (i) determine mathematically whether a given set of covariates is appropriate for control of confounding, (ii) deal with measurements that lie on the causal pathways, and (iii) trade one set of measurements for another.

Finally, we will show how the new calculus disambiguates concepts that have triggered controversy and miscommunication among philosophers, statisticians, economists, and psychologists. These include distinctions between structural and regression equations, definitions of direct and indirect effects, and relationships between structural equations and Neyman-Rubin models.

3.1 INTRODUCTION

The problems addressed in this chapter can best be illustrated through a classical example due to Cochran (see Wainer 1989). Consider an experiment in which soil fumigants (X) are used to increase oat crop yields (Y) by controlling the eelworm population (Z); the fumigants may also have direct effects (both beneficial and adverse) on yields beside the control of eelworms. We wish to assess the total effect of the fumigants on yields when this typical study is complicated by several factors. First, controlled randomized experiments are unfeasible – farmers insist on deciding for themselves which plots are to be fumigated. Second, farmers' choice of treatment depends on last year's eelworm population (Z_0), an unknown quantity that is strongly correlated with this year's population. Thus we have a classical case of confounding bias that interferes with the assessment of treatment effects regardless of sample size. Fortunately, through laboratory analysis of soil samples, we can determine the eelworm populations before and after the treatment; furthermore, because the fumigants are known to be active for a short period only, we can safely assume that they do not affect the growth of eelworms surviving the treatment. Instead, eelworms' growth depends on the population of birds (and other predators), which is correlated with last year's eelworm population and hence with the treatment itself.

The method developed in this chapter permits the investigator to translate complex considerations of this sort into a formal language and thereby facilitate the following tasks:

1. explicating the assumptions that underlie the model;
2. deciding whether the assumptions are sufficient to obtain consistent estimates of the target quantity – the total effect of the fumigants on yields;
3. providing (if the answer to item 2 is affirmative) a closed-form expression for the target quantity in terms of distributions of observed quantities; and
4. suggesting (if the answer to item 2 is negative) a set of observations and experiments that, if performed, would render a consistent estimate feasible.

The first step in this analysis is to construct a causal diagram like the one given in Figure 3.1, which represents the investigator's understanding of the major causal influences among measurable quantities in the domain. For example, the quantities Z_1, Z_2, Z_3 represent the eelworm population before treatment, after treatment, and at the end of the season, respectively. The Z_0 term represents last year's eelworm population; because it is an unknown quantity, it is denoted by a hollow circle, as is the quantity B, the

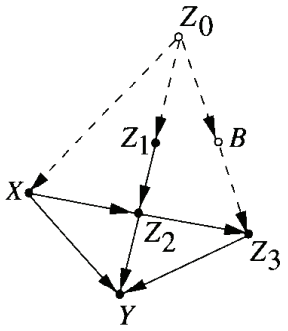


Figure 3.1 A causal diagram representing the effect of fumigants (X) on yields (Y).

population of birds and other predators. Links in the diagram are of two kinds: those that connect unmeasured quantities are designated by dashed arrows, those connecting measured quantities by solid arrows. The substantive assumptions embodied in the diagram are negative causal assertions which are conveyed through the links *missing* from the diagram. For example, the missing arrow between Z_1 and Y signifies the investigator's understanding that pretreatment eelworms can not affect oat plants directly; their entire influence on oat yields is mediated by the posttreatment conditions, Z_2 and Z_3. Our purpose is not to validate or repudiate such domain-specific assumptions but rather to test whether a given set of assumptions is sufficient for quantifying causal effects from nonexperimental data – here, estimating the total effect of fumigants on yields.

The causal diagram in Figure 3.1 is similar in many respects to the path diagrams devised by Wright (1921); both reflect the investigator's subjective and qualitative knowledge of causal influences in the domain, both employ directed acyclic graphs, and both allow for the incorporation of latent or unmeasured quantities. The major differences lie in the method of analysis. First, whereas path diagrams have been analyzed mostly in the context of linear models with Gaussian noise, causal diagrams permit arbitrary nonlinear interactions. In fact, our analysis of causal effects will be entirely nonparametric, entailing no commitment to a particular functional form for equations and distributions. Second, causal diagrams will be used not only as a passive language to communicate assumptions but also as an active computational device through which the desired quantities are derived. For example, the method to be described allows an investigator to inspect the diagram of Figure 3.1 and make the following immediate conclusions.

1. The total effect of X on Y can be estimated consistently from the observed distribution of X, Z_1, Z_2, Z_3, and Y.
2. The total effect of X on Y (assuming discrete variables throughout) is given by the formula[1]

$$P(y \mid \hat{x}) = \sum_{z_1} \sum_{z_2} \sum_{z_3} P(y \mid z_2, z_3, x) P(z_2 \mid z_1, x) \times \sum_{x'} P(z_3 \mid z_1, z_2, x') P(z_1, x'), \tag{3.1}$$

[1] The notation $P_x(y)$ was used in Chapter 1; it is changed henceforth to $P(y \mid \hat{x})$ or $P(y \mid do(x))$ because of the inconvenience in handling subscripts. The reader need not be intimidated if, at this point, (3.1) appears unfamiliar. After reading Section 3.4, the reader should be able to derive such formulas with greater ease than solving algebraic equations. Note that x' is merely an index of summation that ranges over the values of X.

where $P(y \mid \hat{x})$ stands for the probability of achieving a yield level of $Y = y$, given that the treatment is set to level $X = x$ by external intervention.

3. A consistent estimation of the total effect of X on Y would not be feasible if Y were confounded with Z_3; however, confounding Z_2 and Y will not invalidate the formula for $P(y \mid \hat{x})$.

These conclusions will be obtained either by analyzing the graphical properties of the diagram or by performing a sequence of symbolic derivations (governed by the diagram) that gives rise to causal effect formulas such as (3.1).

3.2 INTERVENTION IN MARKOVIAN MODELS

3.2.1 Graphs as Models of Interventions

In Chapter 1 (Section 1.3) we saw how causal models, unlike probabilistic models, can serve to predict the effect of interventions. This added feature requires that the joint distribution P be supplemented with a causal diagram – that is, a directed acyclic graph G that identifies the causal connections among the variables of interest. In this section we elaborate on the nature of interventions and give explicit formulas for their effects.

The connection between the causal and associational readings of DAGs is formed through the mechanism-based account of causation, which owes its roots to early works in econometrics (Frisch 1938; Haavelmo 1943; Simon 1953). In this account, assertions about causal influences, such as those specified by the links in Figure 3.1, stand for autonomous physical mechanisms among the corresponding quantities; these mechanisms are represented as functional relationships perturbed by random disturbances. Echoing this tradition, Pearl and Verma (1991) interpreted the causal reading of a DAG in terms of functional, rather than probabilistic, relationships (see (1.40) and Definition 2.2.2); in other words, each child–parent family in a DAG G represents a deterministic function

$$x_i = f_i(pa_i, \varepsilon_i), \quad i = 1, \ldots, n, \tag{3.2}$$

where pa_i are the parents of variable X_i in G; the ε_i $(1 \leq i \leq n)$ are mutually independent, arbitrarily distributed random disturbances. These disturbance terms represent independent background factors that the investigator chooses not to include in the analysis. If any of these factors is judged to be influencing two or more variables (thus violating the independence assumption), then that factor must enter the analysis as an unmeasured (or latent) variable and be represented in the graph by a hollow node, such as Z_0 and B in Figure 3.1. For example, the causal assumptions conveyed by the model in Figure 3.1 correspond to the following set of equations:

$$\begin{aligned} Z_0 &= f_0(\varepsilon_0), & B &= f_B(Z_0, \varepsilon_B), \\ Z_1 &= f_1(Z_0, \varepsilon_1), & X &= f_X(Z_0, \varepsilon_X), \\ Z_2 &= f_2(X, Z_1, \varepsilon_2), & Y &= f_Y(X, Z_2, Z_3, \varepsilon_Y), \\ Z_3 &= f_3(B, Z_2, \varepsilon_3). \end{aligned} \tag{3.3}$$

More generally, we may lump together all unobserved factors (including the ε_i) into a set U of background variables and then summarize their characteristics by a distribution function $P(u)$ – or by some aspects (e.g. independencies) of $P(u)$. Thus, a full specification of a causal model would entail two components: a set of functional relationships

$$x_i = f_i(pa_i, u_i), \quad i = 1, \ldots, n, \tag{3.4}$$

and a joint distribution function $P(u)$ on the background factors. If the diagram $G(M)$ associated with a causal model M is acyclic, then M is called *semi-Markovian*. If, in addition, the background variables are independent, M is called *Markovian*, since the resulting distribution of the observed variables would then be Markov relative to $G(M)$ (see Theorem 1.4.1). Thus, the model described in Figure 3.1 is semi-Markovian if the observed variables are $\{X, Y, Z_1, Z_2, Z_3\}$; it would turn Markovian if Z_0 and B were observed as well. In Chapter 7 we will pursue the analysis of general non-Markovian models, but in this chapter all models are assumed to be either Markovian or Markovian with unobserved variables (i.e. semi-Markovian).

Needless to state, we would seldom be in possession of $P(u)$ or even f_i. It is important nevertheless to explicate the mathematical content of a fully specified model in order to draw valid inferences from partially specified models, such as the one described in Figure 3.1.

The equational model (3.2) is the nonparametric analog of the so-called structural equations model (Wright 1921; Goldberger 1973), except that: the functional form of the equations (as well as the distribution of the disturbance terms) will remain unspecified. The equality signs in structural equations convey the asymmetrical counterfactual relation of "is determined by," and each equation represents a stable autonomous mechanism. For example, the equation for Y states that, regardless of what we currently observe about Y and regardless of any changes that might occur in other equations, if variables $(X, Z_2, Z_3, \varepsilon_Y)$ were to assume the values $(x, z_2, z_3, \varepsilon_Y)$, respectively, then Y would take on the value dictated by the function f_Y.

Recalling our discussion in Section 1.4, the functional characterization of each child–parent relationship leads to the same recursive decomposition of the joint distribution that characterizes Bayesian networks:

$$P(x_1, \ldots, x_n) = \prod_i P(x_i \mid pa_i), \tag{3.5}$$

which, in our example of Figure 3.1, yields

$$\begin{aligned} P(z_0, x, z_1, b, z_2, z_3, y) = {} & P(z_0)P(x \mid z_0)P(z_1 \mid z_0)P(b \mid z_0) \\ & \times P(z_2 \mid x, z_1)P(z_3 \mid z_2, b)P(y \mid x, z_2, z_3). \end{aligned} \tag{3.6}$$

Moreover, the functional characterization provides a convenient language for specifying how the resulting distribution would change in response to external interventions. This is accomplished by encoding each intervention as an alteration on a select subset of functions while keeping the other functions intact. Once we know the identity of the mechanisms altered by the intervention and the nature of the alteration, the overall effect

of the intervention can be predicted by modifying the corresponding equations in the model and using the modified model to compute a new probability function.

The simplest type of external intervention is one in which a single variable, say X_i, is forced to take on some fixed value x_i. Such an intervention, which we call "atomic," amounts to lifting X_i from the influence of the old functional mechanism $x_i = f_i(pa_i, u_i)$ and placing it under the influence of a new mechanism that sets the value x_i while keeping all other mechanisms unperturbed. Formally, this atomic intervention, which we denote by $do(X_i = x_i)$, or $do(x_i)$ for short,[2] amounts to removing the equation $x_i = f_i(pa_i, u_i)$ from the model and substituting $X_i = x_i$ in the remaining equations. The new model thus created represents the system's behavior under the intervention $do(X_i = x_i)$ and, when solved for the distribution of X_j, yields the causal effect of X_i on X_j, which is denoted $P(x_j \mid \hat{x}_i)$. More generally, when an intervention forces a subset X of variables to attain fixed values x, then a subset of equations is to be pruned from the model given in (3.4), one for each member of X, thus defining a new distribution over the remaining variables that completely characterizes the effect of the intervention.[3]

Definition 3.2.1 (Causal Effect)
Given two disjoint sets of variables, X and Y, the causal effect *of X on Y, denoted either as* $P(y \mid \hat{x})$ *or as* $P(y \mid do(x))$, *is a function from X to the space of probability distributions on Y. For each realization x of X,* $P(y \mid \hat{x})$ *gives the probability of* $Y = y$ *induced by deleting from the model of* (3.4) *all equations corresponding to variables in X and substituting* $X = x$ *in the remaining equations.*

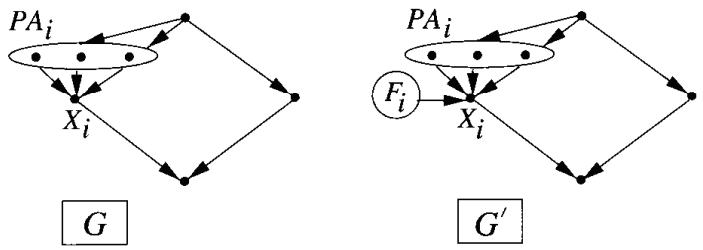
Clearly, the graph corresponding to the reduced set of equations is a subgraph of G from which all arrows entering X have been pruned (Spirtes et al. 1993). The difference $E(Y \mid do(x')) - E(Y \mid do(x''))$ is sometimes taken as the definition of "causal effect" (Rosenbaum and Rubin 1983), where x' and x'' are two distinct realizations of X. This difference can always be computed from the general function $P(y \mid do(x))$, which is defined for every level x of X and provides a more refined characterization of the effect of interventions.

3.2.2 Interventions as Variables

An alternative (but sometimes more appealing) account of intervention treats the force responsible for the intervention as a variable within the system (Pearl 1993b). This is

[2] An equivalent notation, using $set(x)$ instead of $do(x)$, was used in Pearl (1995a). The $do(x)$ notation was first used in Goldszmidt and Pearl (1992) and is gaining in popular support. The expression $P(y \mid do(x))$ is equivalent in intent to $P(Y_x = y)$ in the potential-outcome model introduced by Neyman (1923) and Rubin (1974) and to the expression $P[(X = x) \square\!\!\rightarrow (Y = y)]$ in the counterfactual theory of Lewis (1973b). The semantical differences among these notions are discussed in Section 3.6.3 and in Chapter 7.

[3] The basic view of interventions as equation modifiers originates with Marschak (1950) and Simon (1953). An explicit translation of interventions to "wiping out" equations from the model was first proposed by Strotz and Wold (1960) and later used in Fisher (1970) and Sobel (1990). Graphical ramifications of this translation were explicated first in Spirtes et al. (1993) and later in Pearl (1993b).



G G'

Figure 3.2 Representing external intervention F_i by an augmented network $G' = G \cup \{F_i \rightarrow X_i\}$.

facilitated by representing the function f_i itself as a value of a variable F_i and then writing (3.2) as

$$x_i = I(pa_i, f_i, u_i), \tag{3.7}$$

where I is a three-argument function satisfying

$$I(a, b, c) = f_i(a, c) \quad \text{whenever } b = f_i.$$

This amounts to conceptualizing the intervention as an external force F_i that alters the function f_i between X_i and its parents. Graphically, we can represent F_i as an added parent node of X_i, and the effect of such an intervention can be analyzed by standard conditionalization – that is, by conditioning our probability on the event that variable F_i attains the value f_i.

The effect of an atomic intervention $do(X_i = x_i')$ is encoded by adding to G a link $F_i \rightarrow X_i$ (see Figure 3.2), where F_i is a new variable taking values in $\{do(x_i'), \text{idle}\}$, x_i' ranges over the domain of X_i, and "idle" represents no intervention. Thus, the new parent set of X_i in the augmented network is $PA_i' = PA_i \cup \{F_i\}$, and it is related to X_i by the conditional probability

$$P(x_i \mid pa_i') = \begin{cases} P(x_i \mid pa_i) & \text{if } F_i = \text{idle}, \\ 0 & \text{if } F_i = do(x_i') \text{ and } x_i \neq x_i', \\ 1 & \text{if } F_i = do(x_i') \text{ and } x_i = x_i'. \end{cases} \tag{3.8}$$

The effect of the intervention $do(x_i')$ is to transform the original probability function $P(x_1, \ldots, x_n)$ into a new probability function $P(x_1, \ldots, x_n \mid \hat{x}_i')$, given by

$$P(x_1, \ldots, x_n \mid \hat{x}_i') = P'(x_1, \ldots, x_n \mid F_i = do(x_i')), \tag{3.9}$$

where P' is the distribution specified by the augmented network $G' = G \cup \{F_i \rightarrow X_i\}$ and (3.8), with an arbitrary prior distribution on F_i. In general, by adding a hypothetical intervention link $F_i \rightarrow X_i$ to each node in G, we can construct an augmented probability function $P'(x_1, \ldots, x_n; F_1, \ldots, F_n)$ that contains information about richer types of interventions. Multiple interventions would be represented by conditioning P' on a subset of the F_i (taking values in their respective $do(x_i')$ domains), and the preintervention probability function P would be viewed as the posterior distribution induced by conditioning each F_i in P' on the value "idle."

One advantage of the augmented network representation is that it is applicable to *any* change in the functional relationship f_i and not merely to the replacement of f_i by a

constant. It also displays clearly the ramifications of spontaneous changes in f_i, unmediated by external control. Figure 3.2 predicts, for example, that only descendants of X_i would be effected by changes in f_i and hence the marginal probability $P(z)$ will remain unaltered for every set Z of nondescendants of X_i. Likewise, Figure 3.2 dictates that the conditional probability $P(y \mid x_i)$ remains invariant to changes in f_i for any set Y of descendants of X_i, provided X_i d-separates F_i from Y. Kevin Hoover (1990, 1999) used such invariant features to determine the direction of causal influences among economic variables (e.g., employment and money supply) by observing the changes induced by sudden modifications in the processes that govern these variables (e.g., tax reform, labor dispute). Indeed, whenever we obtain reliable information (e.g., from historical or institutional knowledge) that an abrupt local change has taken place in a specific mechanism f_i that constrains a given family (X_i, PA_i) of variables, we can use the observed changes in the marginal and conditional probabilities surrounding those variables to determine whether X_i is indeed the child (or dependent variable) of that family, thus determining the direction of causal influences in the domain. The statistical features that remain invariant under such changes, as well as the causal assumptions underlying this invariance, are displayed in the augmented network G'.

3.2.3 Computing the Effect of Interventions

Regardless of whether we represent interventions as a modification of an existing model (Definition 3.2.1) or as a conditionalization in an augmented model (equation (3.9)), the result is a well-defined transformation between the preintervention and postintervention distributions. In the case of an atomic intervention $do(X_i = x_i')$, this transformation can be expressed in a simple *truncated factorization* formula that follows immediately from (3.2) and Definition 3.2.1:[4]

$$P(x_1, \ldots, x_n \mid \hat{x}_i') = \begin{cases} \prod_{j \neq i} P(x_j \mid pa_j) & \text{if } x_i = x_i', \\ 0 & \text{if } x_i \neq x_i'. \end{cases} \tag{3.10}$$

Equation (3.10) reflects the removal of the term $P(x_i \mid pa_i)$ from the product of (3.5), since pa_i no longer influence X_i. For example, the intervention $do(X = x')$ will transform the preintervention distribution given in (3.6) into the product

$$\begin{aligned} P(z_0, z_1, b, z_2, z_3, y \mid \hat{x}') = {} & P(z_0)P(z_1 \mid z_0)P(b \mid z_0) \\ & \times P(z_2 \mid x', z_1)P(z_3 \mid z_2, b)P(y \mid x', z_2, z_3). \end{aligned}$$

Graphically, the removal of the term $P(x_i \mid pa_i)$ is equivalent to removing the links between PA_i and X_i while keeping the rest of the network intact. Clearly, the transformation defined in (3.10) satisfies the condition of Definition 1.3.1 as well as the properties of (1.38)–(1.39).

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[4] Equation (3.10) can also be obtained from the G-computation formula of Robins (1986, p. 1423; see also Section 3.6.4) and the manipulation theorem of Spirtes et al. (1993) (according to this source, said formula was "independently conjectured by Fienberg in a seminar in 1991"). Additional properties of the transformation defined in (3.10) and (3.11) are given in Goldszmidt and Pearl (1992) and Pearl (1993b).

Multiplying and dividing (3.10) by $P(x_i' \mid pa_i)$, the relationship to the preintervention distribution becomes more transparent:

$$P(x_1, \ldots, x_n \mid \hat{x}_i') = \begin{cases} \dfrac{P(x_1, \ldots, x_n)}{P(x_i' \mid pa_i)} & \text{if } x_i = x_i', \\ 0 & \text{if } x_i \neq x_i'. \end{cases} \tag{3.11}$$

If we regard a joint distribution as an assignment of mass to a collection of abstract points $(x_1, \ldots, x_n)$, each representing a possible state of the world, then the transformation described in (3.11) reveals some interesting properties of the change in mass distribution that take place as a result of an intervention $do(X_i = x_i')$ (Goldszmidt and Pearl 1992). Each point $(x_1, \ldots, x_n)$ is seen to increase its mass by a factor equal to the inverse of the conditional probability $P(x_i' \mid pa_i)$ corresponding to that point. Points for which this conditional probability is low would boost their mass value substantially, while those possessing a pa_i value that anticipates a natural (noninterventional) realization of x_i' (i.e., $P(x_i' \mid pa_i) \approx 1$) will keep their mass unaltered. In standard Bayes conditionalization, each excluded point $(x_i \neq x_i')$ transfers its mass to the entire set of preserved points through a renormalization constant. However, (3.11) describes a different transformation: each excluded point $(x_i \neq x_i')$ transfers its mass to a select set of points that share the same value of pa_i. This can be seen from the constancy of both the total mass assigned to each stratum pa_i and the relative masses of points within each such stratum:

$$P(pa_i \mid do(x_i')) = P(pa_i);$$

$$\frac{P(s_i, pa_i \mid do(x_i'))}{P(s_i', pa_i \mid do(x_i'))} = \frac{P(s_i, pa_i)}{P(s_i', pa_i)}.$$

Here S_i denotes the set of all variables excluding $\{PA_i \cup X_i\}$. This select set of mass-receiving points can be regarded as "closest" to the point excluded by virtue of sharing the same history, as summarized by pa_i (see Sections 4.1.3 and 7.4.3).

Another interesting form of (3.11) obtains when we interpret the division by $P(x_i' \mid pa_i)$ as conditionalization on x_i' and pa_i:

$$P(x_1, \ldots, x_n \mid \hat{x}_i') = \begin{cases} P(x_1, \ldots, x_n \mid x_i', pa_i)P(pa_i) & \text{if } x_i = x_i', \\ 0 & \text{if } x_i \neq x_i'. \end{cases} \tag{3.12}$$

This formula becomes familiar when used to compute the effect of an intervention $do(X_i = x_i')$ on a set of variables Y disjoint of $(X_i \cup PA_i)$. Summing (3.12) over all variables except $Y \cup X_i$ yields the following theorem.

Theorem 3.2.2 (Adjustment for Direct Causes)
Let PA_i denote the set of direct causes of variable X_i, and let Y be any set of variables disjoint of $\{X_i \cup PA_i\}$. The effect of the intervention $do(X_i = x_i')$ on Y is given by

$$P(y \mid \hat{x}_i') = \sum_{pa_i} P(y \mid x_i', pa_i)P(pa_i), \tag{3.13}$$

where $P(y \mid x_i', pa_i)$ and $P(pa_i)$ represent preintervention probabilities.

Equation (3.13) calls for conditioning $P(y \mid x_i')$ on the parents of X_i and then averaging the result, weighted by the prior probability of $PA_i = pa_i$. The operation defined by this conditioning and averaging is known as "adjusting for PA_i."

Variations of this adjustment have been advanced by many philosophers as probabilistic definitions of causality and causal effect (see Section 7.5). Good (1961), for example, calls for conditioning on "the state of the universe just before" the occurrence of the cause. Suppes (1970) calls for conditioning on the entire past, up to the occurrence of the cause. Skyrms (1980, p. 133) calls for conditioning on "maximally specific specifications of the factors outside of our influence at the time of the decision which are causally relevant to the outcome of our actions ...". The aim of conditioning in these proposals is, of course, to eliminate spurious correlations between the cause (in our case, $X_i = x_i'$) and the effect ($Y = y$); clearly, the set of parents PA_i can accomplish this aim with great economy. In the structural account that we pursue in this book, causal effects are defined in a radically different way. The conditioning operator is not introduced into (3.13) as a remedial "adjustment" aimed at eradicating spurious correlations. Rather, it emerges formally from the deeper principle represented in (3.10) – that of preserving all the invariant information that the preintervention distribution can provide.

The transformation of (3.10) can easily be extended to more elaborate interventions in which several variables are manipulated simultaneously. For example, if we consider the compound intervention $do(S = s)$ where S is a subset of variables, then (echoing (1.37)) we should delete from the product of (3.5) all factors $P(x_i \mid pa_i)$ corresponding to variables in S and obtain the more general truncated factorization

$$P(x_1, \ldots, x_n \mid \hat{s}) = \begin{cases} \prod_{i \mid X_i \notin S} P(x_i \mid pa_i) & \text{for } x_1, \ldots, x_n \text{ consistent with } s, \\ 0 & \text{otherwise.} \end{cases} \tag{3.14}$$

Likewise, we need not limit ourselves to simple interventions that set variables to constants. Instead, we may consider a more general modification of the causal model whereby some mechanisms are *replaced*. For example, if we replace the mechanism that determines the value of X_i by another equation, one that involves perhaps a new set PA_i^* of variables, then the resultant distribution would obtain by replacing the factor $P(x_i \mid pa_i)$ with the conditional probability $P^*(x_i \mid pa_i^*)$ induced by the new equation. The modified joint distribution would then be given by $P^*(x_1, \ldots, x_n) = P(x_1, \ldots, x_n)P^*(x_i \mid pa_i^*)/P(x_i \mid pa_i)$.

An Example: Process Control

To illustrate these operations, let us consider an example involving process control; analogous applications in the areas of health management, economic policy making, product marketing, or robot motion planning should follow in a straightforward way. Let the variable Z_k stand for the state of a production process at time t_k, and let X_k stand for a set of variables (at time t_k) that is used to control that process (see Figure 3.3). For example, Z_k could stand for such measurements as temperature and pressure at various location in the plant, and X_k could stand for the rate at which various chemicals are permitted to flow in strategic conduits. Assume that data are gathered while the process is controlled by a strategy S in which each X_k is determined by (i) monitoring three previous variables (X_{k-1}, Z_k, and Z_{k-1}) and (ii) choosing $X_k = x_k$ with probability $P(x_k \mid x_{k-1}, z_k, z_{k-1})$.

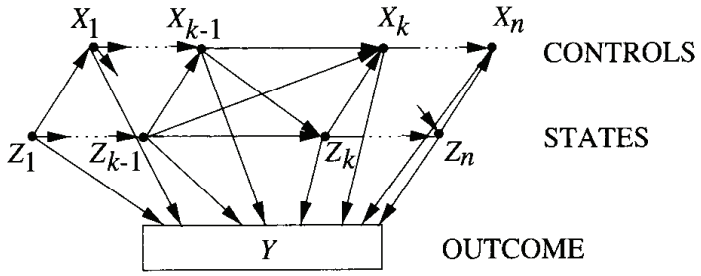

Figure 3.3 Dynamic causal diagram illustrating typical dependencies among the control variables $X_1, \ldots, X_n$, the state variables $Z_1, \ldots, Z_n$, and the outcome variable Y of a sequential process.

The performance of S is monitored and summarized in the form of a joint probability function $P(y, z_1, z_2, \ldots, z_n, x_1, x_2, \ldots, x_n)$, where Y is an outcome variable (e.g., the quality of the final product). Finally, let us assume (for simplicity) that the state Z_k of the process depends only on the previous state Z_{k-1} and on the previous control X_{k-1}. We wish to evaluate the merit of replacing S with a new strategy, S^*, in which X_k is chosen according to a new conditional probability $P^*(x_k \mid x_{k-1}, z_k, z_{k-1})$.

Based on our previous analysis (equation (3.14)), the performance $P^*(y)$ of the new strategy S^* will be governed by the distribution

$$\begin{aligned} &P^*(y, z_1, z_2, \ldots, z_n, x_1, x_2, \ldots, x_n) \\ &\quad = P^*(y \mid z_1, z_2, \ldots, z_n, x_1, x_2, \ldots, x_n) \\ &\qquad \times \prod_k P^*(z_k \mid z_{k-1}, x_{k-1}) \prod_k P^*(x_k \mid x_{k-1}, z_k, z_{k-1}). \end{aligned} \tag{3.15}$$

Because the first two terms remain invariant and the third one is known, we have

$$\begin{aligned} P^*(y) &= \sum_{z_1, \ldots, z_n, x_1, \ldots, x_n} P^*(y, z_1, z_2, \ldots, z_n, x_1, x_2, \ldots, x_n) \\ &= \sum_{z_1, \ldots, z_n, x_1, \ldots, x_n} P(y \mid z_1, z_2, \ldots, z_n, x_1, x_2, \ldots, x_n) \\ &\qquad \times \prod_k P(z_k \mid z_{k-1}, x_{k-1}) \prod_k P^*(x_k \mid x_{k-1}, z_k, z_{k-1}). \end{aligned} \tag{3.16}$$

In the special case where S^* is deterministic and time-invariant, X_k becomes a function of X_{k-1}, Z_k, and Z_{k-1}:

$$x_k = g(x_{k-1}, z_k, z_{k-1}).$$

Then the summation over $x_1, \ldots, x_n$ can be performed, yielding

$$P^*(y) = \sum_{z_1, \ldots, z_n} P(y \mid z_1, z_2, \ldots, z_n, g_1, g_2, \ldots, g_n) \prod_k P(z_k \mid z_{k-1}, g_{k-1}), \tag{3.17}$$

where g_k is defined recursively as

$$g_1 = g(z_1) \quad \text{and} \quad g_k = g(g_{k-1}, z_k, z_{k-1}).$$

In the special case of a strategy S^* composed of elementary actions $do(X_k = x_k)$ the function g degenerates into a constant, x_k, and we obtain

$$P^*(y) = P(y \mid \hat{x}_1, \hat{x}_2, \ldots, \hat{x}_n)$$
$$= \sum_{z_1, \ldots, z_n} P(y \mid z_1, z_2, \ldots, z_n, x_1, x_2, \ldots, x_n) \prod_k P(z_k \mid z_{k-1}, x_{k-1}), \quad (3.18$$

which can also be obtained from (3.14).

The planning problem illustrated by this example is typical of Markov decision pro cesses (MDPs) (Howard 1960; Dean and Wellman 1991; Bertsekas and Tsitsiklis 1996 where the target of analysis is finding the best next action $do(X_k = x_k)$, given the cu rent state Z_k and past actions. In MDPs, we are normally given the transition functior $P(z_{k+1} \mid z_k, \hat{x}_k)$ and the cost function to be minimized. In the problem we have just ana lyzed, neither function is given; instead, they must be learned from data gathered unde past (presumably suboptimal) strategies. Fortunately, because all variables in the mode were measured, both functions were identifiable and could be estimated directly from th corresponding conditional probabilities as follows:

$$P(z_{k+1} \mid z_k, \hat{x}_k) = P(z_{k+1} \mid z_k, x_k);$$
$$P(y \mid z_1, z_2, \ldots, z_n, \hat{x}_1, \hat{x}_2, \ldots, \hat{x}_n) = P(y \mid z_1, z_2, \ldots, z_n, x_1, x_2, \ldots, x_n).$$

In Chapter 4 (Section 4.4) we will deal with partially observable Markov decision pro cesses (POMDPs), where some states Z_k are unobserved; learning the transition and co functions in those problems will require a more intricate method of identification.

It is worth noting that, in this example, to predict the effect of a new strategy it is nec essary first to measure variables (Z_k) that are affected by some control variables (X_{k-1} Such measurements are generally shunned in the classical literature on experimental de sign (Cox 1958, p. 48), because they lie on the causal pathways between treatment an outcome and thus tend to confound the desired effect estimate. However, our analy sis shows that, when properly processed, such measurements may be indispensable i predicting the effect of certain control programs. This will be especially true in sem Markovian models (i.e., DAGs involving unmeasured variables), which are analyzed i Section 3.3.2.

Summary

The immediate implication of the analysis provided in this section is that – given a caus diagram in which all direct causes (i.e. parents) of intervened variables are observable one can infer postintervention distributions from preintervention distributions; henc under such assumptions we can estimate the effects of interventions from passive (i. nonexperimental) observations, using the truncated factorization formula of (3.14). Y the more challenging problem is to derive causal effects in situations like Figure 3. where some members of PA_i are unobservable and so prevent estimation of $P(x_i' \mid pa_i$ In Sections 3.3 and 3.4 we provide simple graphical tests for deciding when $P(x_j \mid \hat{x}_i)$ estimable in such models. But first we need to define more formally what it means for causal quantity Q to be estimable from passive observations, a question that falls und the technical term *identification*.

3.2.4 Identification of Causal Quantities

Causal quantities, unlike statistical parameters, are defined relative to a causal model M and not relative to a joint distribution $P_M(v)$ over the set V of observed variables. Since nonexperimental data provides information about $P_M(v)$ alone, and since several models can generate the same distribution, the danger exists that the desired quantity will not be discernible unambiguously from the data – even when infinitely many samples are taken. Identifiability ensures that the added assumptions we make about M (e.g., the causal graph or the zero coefficients in structural equations) will supply the missing information without explicating M in full detail.

Definition 3.2.3 (Identifiability)
Let $Q(M)$ be any computable quantity of a model M. We say that Q is identifiable *in a class $\boldsymbol{M}$ of models if, for any pairs of models M_1 and M_2 from $\boldsymbol{M}$, $Q(M_1) = Q(M_2)$ whenever $P_{M_1}(v) = P_{M_2}(v)$. If our observations are limited and permit only a partial set F_M of features (of $P_M(v)$) to be estimated, we define Q to be* identifiable from F_M *if $Q(M_1) = Q(M_2)$ whenever $F_{M_1} = F_{M_2}$.*

Identifiability is essential for integrating statistical data (summarized by $P(v)$) with incomplete causal knowledge of $\{f_i\}$, as it enables us to estimate quantities Q consistently from large samples of P without specifying the details of M; the general characteristics of the class $\boldsymbol{M}$ suffice. For the purpose of our analysis, the quantity Q of interest is the causal effect $P_M(y \mid \hat{x})$, which is certainly computable from a given model M (using Definition 3.2.1) but which we often need to compute from an incomplete specification of M – in the form of general characteristics portrayed in the graph G associated with M. We will therefore consider a class $\boldsymbol{M}$ of models that have the following characteristics in common:

(i) they share the same parent–child families (i.e., the same causal graph G); and

(ii) they induce positive distributions on the observed variables (i.e., $P(v) > 0$).

Relative to such classes, we now have the following.

Definition 3.2.4 (Causal Effect Identifiability)
The causal effect *of X on Y is* identifiable *from a graph G if the quantity $P(y \mid \hat{x})$ can be computed uniquely from any positive probability of the observed variables – that is, if $P_{M_1}(y \mid \hat{x}) = P_{M_2}(y \mid \hat{x})$ for every pair of models M_1 and M_2 with $P_{M_1}(v) = P_{M_2}(v) > 0$ and $G(M_1) = G(M_2) = G$.*

The identifiability of $P(y \mid \hat{x})$ ensures that it is possible to infer the effect of action $do(X = x)$ on Y from two sources of information:

(i) passive observations, as summarized by the probability function $P(v)$; and

(ii) the causal graph G, which specifies (qualitatively) which variables make up the stable mechanisms in the domain or, alternatively, which variables participate in the determination of each variable in the domain.

Restricting identifiability to positive distributions assures us that the condition $X = x$ is represented in the data in the appropriate context, thus avoiding a zero denominator in (3.10). It would be impossible to infer the effect of action $do(X = x)$ from data in which X never attains the value x in the context wherein the action is applied. Extensions to some nonpositive distributions are feasible but will not be treated here. Note that, to prove nonidentifiability, it is sufficient to present two sets of structural equations that induce identical distributions over observed variables but have different causal effects.

Using the concept of identifiability, we can now summarize the results of Section 3.2.3 in the following theorem.

Theorem 3.2.5

Given a causal diagram G of any Markovian model in which a subset V of variables are measured, the causal effect $P(y \mid \hat{x})$ is identifiable whenever $\{X \cup Y \cup PA_X\} \subseteq V$, that is, whenever X, Y, and all parents of variables in X are measured. The expression for $P(y \mid \hat{x})$ is then obtained by adjusting for PA_x, as in (3.13).

A special case of Theorem 3.2.5 holds when *all* variables are assumed to be observed.

Corollary 3.2.6

Given the causal diagram G of any Markovian model in which all variables are measured, the causal effect $P(y \mid \hat{x})$ is identifiable for every two subsets of variables X and Y and is obtained from the truncated factorization of (3.14).

We now turn our attention to identification problems in semi-Markovian models.

3.3 CONTROLLING CONFOUNDING BIAS

Whenever we undertake to evaluate the effect of one factor (X) on another (Y), the question arises as to whether we should adjust our measurements for possible variations in some other factors (Z), otherwise known as "covariates," "concomitants," or "confounders" (Cox 1958, p. 48) Adjustment amounts to partitioning the population into groups that are homogeneous relative to Z, assessing the effect of X on Y in each homogeneous group, and then averaging the results (as in (3.13)). The illusive nature of such adjustment was recognized as early as 1899, when Karl Pearson discovered what is now called *Simpson's paradox* (see Section 6.1): Any statistical relationship between two variables may be reversed by including additional factors in the analysis. For example, we may find that students who smoke obtain higher grades than those who do not smoke but, adjusting for age, smokers obtain lower grades in every age group and, further adjusting for family income, smokers again obtain higher grades than nonsmokers in every income–age group, and so on.

Despite a century of analysis, Simpson's reversal continues to "trap the unwary" (Dawid 1979), and the practical question that it poses – whether an adjustment for a given covariate is appropriate – has resisted mathematical treatment. Epidemiologists, for example, are still debating the meaning of "confounding" (Grayson 1987; Shapiro 1997) and often adjust for wrong sets of covariates (Weinberg 1993; see also Chapter 6). The potential-outcome analyses of Rosenbaum and Rubin (1983) and Pratt and Schlaifer

(1988) have led to a concept named "ignorability," which recasts the covariate selection problem in counterfactual vocabulary but falls short of providing a workable solution. Ignorability reads: "Z is an admissible set of covariates if, given Z, the value that Y would obtain had X been x is independent of X." Since counterfactuals are not observable, and since judgments about conditional independence of counterfactuals are not readily assertable from ordinary understanding of causal processes, the question has remained open: What criterion should one use to decide which variables are appropriate for adjustment?

Section 3.3.1 presents a general and formal solution of the adjustment problem using the language of causal graphs. In Section 3.3.2 we extend this result to nonstandard covariates that are affected by X and hence require several steps of adjustment. Finally, Section 3.3.3 illustrates the use of these criteria in an example.

3.3.1 The Back-Door Criterion

Assume we are given a causal diagram G, together with nonexperimental data on a subset V of observed variables in G, and suppose we wish to estimate what effect the interventions $do(X = x)$ would have on a set of response variables Y, where X and Y are two subsets of V. In other words, we seek to estimate $P(y \mid \hat{x})$ from a sample estimate of $P(v)$.

We show that there exists a simple graphical test, named the "back-door criterion" in Pearl (1993b), that can be applied directly to the causal diagram in order to test if a set $Z \subseteq V$ of variables is sufficient for identifying $P(y \mid \hat{x})$.[5]

Definition 3.3.1 (Back-Door)
A set of variables Z satisfies the back-door *criterion relative to an ordered pair of variables (X_i, X_j) in a DAG G if:*

(i) *no node in Z is a descendant of X_i; and*

(ii) *Z blocks every path between X_i and X_j that contains an arrow into X_i.*

Similarly, if X and Y are two disjoint subsets of nodes in G, then Z is said to satisfy the back-door criterion relative to (X, Y) if it satisfies the criterion relative to any pair (X_i, X_j) such that $X_i \in X$ and $X_j \in Y$.

The name "back-door" echoes condition (ii), which requires that only paths with arrows pointing at X_i be blocked; these paths can be viewed as entering X_i through the back door. In Figure 3.4, for example, the sets $Z_1 = \{X_3, X_4\}$ and $Z_2 = \{X_4, X_5\}$ meet the back-door criterion, but $Z_3 = \{X_4\}$ does not because X_4 does not block the path $(X_i, X_3, X_1, X_4, X_2, X_5, X_j)$.

Theorem 3.3.2 (Back-Door Adjustment)
If a set of variables Z satisfies the back-door criterion relative to (X, Y), then the causal effect of X on Y is identifiable and is given by the formula

[5] This criterion may also be obtained from Theorem 7.1 of Spirtes et al. (1993). An alternative criterion, using a single d-separation test, is established in Section 3.4 (see (3.37)).

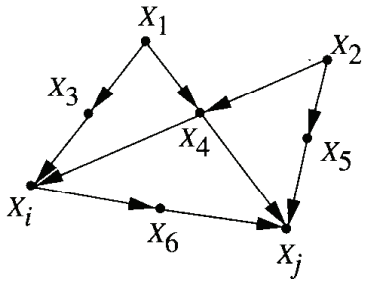

Figure 3.4 A diagram representing the back-door criterion; adjusting for variables $\{X_3, X_4\}$ (or $\{X_4, X_5\}$) yields a consistent estimate of $P(x_j \mid \hat{x}_i)$.

$$P(y \mid \hat{x}) = \sum_z P(y \mid x, z)P(z). \tag{3.19}$$

The summation in (3.19) represents the standard formula obtained under adjustment for Z; variables X for which the equality in (3.19) is valid were named "conditionally ignorable given Z" in Rosenbaum and Rubin (1983). Reducing ignorability conditions to the graphical criterion of Definition 3.3.1 replaces judgments about counterfactual dependencies with judgments about the structure of causal processes, as represented in the diagram. The graphical criterion can be tested by systematic procedures that are applicable to diagrams of any size and shape. The criterion also enables the analyst to search for an optimal set of covariate – namely, a set Z that minimizes measurement cost or sampling variability (Tian et al. 1998). The use of a similar graphical criterion for identifying path coefficients in linear structural equations is demonstrated in Chapter 5. Applications to epidemiological research are given in Greenland et al. (1999a), where the set Z is called "sufficient set" for control of confounding.

Proof of Theorem 3.3.2

The proof originally offered in Pearl (1993b) was based on the observation that, when Z blocks all back-door paths from X to Y, setting $(X = x)$ or conditioning on $X = x$ has the same effect on Y. This can best be seen from the augmented diagram G' of Figure 3.2, to which the intervention arcs $F_X \rightarrow X$ were added. If all back-door paths from X to Y are blocked, then all paths from F_X to Y must go through the children of X, and those would be blocked if we condition on X. The implication is that Y is independent of F_X given X,

$$P(y \mid x, F_X = do(x)) = P(y \mid x, F_X = \text{idle}) = P(y \mid x), \tag{3.20}$$

which means that the observation $X = x$ cannot be distinguished from the intervention $F_X = do(x)$.

Formally, we can prove this observation by writing $P(y \mid \hat{x})$ in terms of the augmented probability function P' in accordance with (3.9) and conditioning on Z to obtain

$$\begin{aligned} P(y \mid \hat{x}) = P'(y \mid F_x) &= \sum_z P'(y \mid z, F_x)P'(z \mid F_x) \\ &= \sum_z P'(y \mid z, x, F_x)P'(z \mid F_x). \end{aligned} \tag{3.21}$$

The addition of x to the last expression is licensed by the implication $F_x \implies X = x$. To eliminate F_x from the two terms on the right-hand side of (3.21), we invoke the two

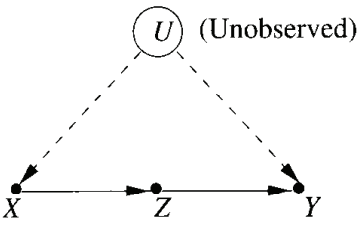

Figure 3.5 A diagram representing the front-door criterion. A two-step adjustment for Z yields a consistent estimate of $P(y \mid \hat{x})$.

conditions of Definition 3.3.1. Since F_x consists of root nodes with children restricted to X, it must be independent of all nondescendants of X, including Z. Thus, condition (i) yields

$$P'(z \mid F_x) = P'(z) = P(z).$$

Invoking now the back-door condition (ii), together with (3.20), permits us to eliminate F_x from (3.21), thus proving (3.19). □

3.3.2 The Front-Door Criterion

Condition (i) of Definition 3.3.1 reflects the prevailing practice that "the concomitant observations should be quite unaffected by the treatment" (Cox 1958, p. 48). This section demonstrates how concomitants that *are* affected by the treatment can be used to facilitate causal inference. The emerging criterion, named the front-door criterion in Pearl (1995a), will constitute the second building block of the general test for identifying causal effects (Section 3.4).

Consider the diagram in Figure 3.5, which represents the model of Figure 3.4 when the variables $X_1, \ldots, X_5$ are unobserved and $\{X_i, X_6, X_j\}$ are relabeled $\{X, Z, Y\}$, respectively. Although Z does not satisfy any of the back-door conditions, measurements of Z can nevertheless enable consistent estimation of $P(y \mid \hat{x})$. This will be shown by reducing the expression for $P(y \mid \hat{x})$ to formulas that are computable from the observed distribution function $P(x, y, z)$.

The joint distribution associated with Figure 3.5 can be decomposed (equation (3.5)) into

$$P(x, y, z, u) = P(u)P(x \mid u)P(z \mid x)P(y \mid z, u). \tag{3.22}$$

From (3.10), the intervention $do(x)$ removes the factor $P(x \mid u)$ and induces the post-intervention distribution

$$P(y, z, u \mid \hat{x}) = P(y \mid z, u)P(z \mid x)P(u). \tag{3.23}$$

Summing over z and u then gives

$$P(y \mid \hat{x}) = \sum_z P(z \mid x) \sum_u P(y \mid z, u)P(u). \tag{3.24}$$

In order to eliminate u from the r.h.s. of (3.24), we use the two conditional independence assumptions encoded in the graph of Figure 3.5:

$$P(u \mid z, x) = P(u \mid x), \tag{3.25}$$

$$P(y \mid x, z, u) = P(y \mid z, u). \tag{3.26}$$

This yields the equalities

$$\begin{aligned}\sum_u P(y \mid z, u)P(u) &= \sum_x \sum_u P(y \mid z, u)P(u \mid x)P(x) \\ &= \sum_x \sum_u P(y \mid x, z, u)P(u \mid x, z)P(x) \\ &= \sum_x P(y \mid x, z)P(x) \end{aligned} \tag{3.27}$$

and allows the reduction of (3.24) to a form involving only observed quantities:

$$P(y \mid \hat{x}) = \sum_z P(z \mid x) \sum_{x'} P(y \mid x', z)P(x'). \tag{3.28}$$

All factors on the r.h.s. of (3.28) are consistently estimable from nonexperimental data, so it follows that $P(y \mid \hat{x})$ is estimable as well. Thus, we are in possession of an identifiable nonparametric estimand for the causal effect of X on Y whenever we can find a mediating variable Z that meets the conditions of (3.25) and (3.26).

Equation (3.28) can be interpreted as a two-step application of the back-door formula. In the first step, we find the causal effect of X on Z; since there is no back-door path from X to Z, we simply have

$$P(z \mid \hat{x}) = P(z \mid x).$$

Next, we compute the causal effect of Z on Y, which we can no longer equate with the conditional probability $P(y \mid z)$ because there is a back-door path $Z \leftarrow X \leftarrow U \rightarrow Y$ from Z to Y. However, since X blocks (d-separates) this path, X can play the role of a concomitant in the back-door criterion, which allows us to compute the causal effect of Z on Y in accordance with (3.19), giving $P(y \mid \hat{z}) = \sum_{x'} P(y \mid x', z)P(x')$. Finally, we combine the two causal effects via

$$P(y \mid \hat{x}) = \sum_z P(y \mid \hat{z})P(z \mid \hat{x}),$$

which reduces to (3.28).

We summarize this result by a theorem after formally defining the assumptions.

Definition 3.3.3 (Front-Door)
A set of variables Z is said to satisfy the front-door *criterion relative to an ordered pair of variables (X, Y) if:*

(i) *Z intercepts all directed paths from X to Y;*

(ii) *there is no back-door path from X to Z; and*

(iii) *all back-door paths from Z to Y are blocked by X.*

Theorem 3.3.4 (Front-Door Adjustment)
If Z satisfies the front-door criterion relative to (X, Y) and if $P(x, z) > 0$, then the causal effect of X on Y is identifiable and is given by the formula

$$P(y \mid \hat{x}) = \sum_z P(z \mid x) \sum_{x'} P(y \mid x', z) P(x'). \tag{3.29}$$

The conditions stated in Definition 3.3.3 are overly restrictive; some of the back-door paths excluded by conditions (ii) and (iii) can actually be allowed provided they are blocked by some concomitants. For example, the variable Z_2 in Figure 3.1 satisfies a front-door-like criterion relative to (X, Z_3) by virtue of Z_1 blocking all back-door paths from X to Z_2 as well as those from Z_2 to Z_3. To allow the analysis of such intricate structures, including nested combinations of back-door and front-door conditions, a more powerful symbolic machinery will be introduced in Section 3.4, one that will sidestep algebraic manipulations such as those used in the derivation of (3.28). But first let us look at an example illustrating possible applications of the front-door condition.

3.3.3 Example: Smoking and the Genotype Theory

Consider the century-old debate on the relation between smoking (X) and lung cancer (Y) (Sprites et al. 1993, pp. 291–302). According to many, the tobacco industry has managed to forestall antismoking legislation by arguing that the observed correlation between smoking and lung cancer could be explained by some sort of carcinogenic genotype (U) that involves inborn craving for nicotine.

The amount of tar (Z) deposited in a person's lungs is a variable that promises to meet the conditions listed in Definition 3.3.3, thus fitting the structure of Figure 3.5. To meet condition (i), we must assume that smoking cigarettes has no effect on the production of lung cancer except as mediated through tar deposits. To meet conditions (ii) and (iii), we must assume that, even if a genotype is aggravating the production of lung cancer, it nevertheless has no effect on the amount of tar in the lungs except indirectly (through cigarette smoking). Likewise, we must assume that no other factor that affects tar deposit has any influence on smoking. Finally, condition $P(x, z) > 0$ of Theorem 3.3.4 requires that high levels of tar in the lungs be the result not only of cigarette smoking but also of other factors (e.g., exposure to environmental pollutants) and that tar may be absent in some smokers (owing perhaps to an extremely efficient tar-rejecting mechanism). Satisfaction of this last condition can be tested in the data.

To demonstrate how we can assess the degree to which cigarette smoking increases (or decreases) lung-cancer risk, we will assume a hypothetical study in which the three variables X, Y, Z were measured simultaneously on a large, randomly selected sample of the population. To simplify the exposition, we will further assume that all three variables are binary, taking on true (1) or false (0) values. A hypothetical data set from a study on the relations among tar, cancer, and cigarette smoking is presented in Table 3.1. It shows that 95% of smokers and 5% of nonsmokers have developed high levels of tar in their lungs. Moreover, 81% of subjects with tar deposits have developed lung cancer, compared to only 14% among those with no tar deposits. Finally, within each of these two groups (tar and no-tar), smokers show a much higher percentage of cancer than nonsmokers.

Table 3.1

	Group Type	$P(x, z)$ Group Size (% of Population)	$P(Y = 1 \mid x, z)$ % of Cancer Cases in Group
$X = 0, Z = 0$	Nonsmokers, No tar	47.5	10
$X = 1, Z = 0$	Smokers, No tar	2.5	90
$X = 0, Z = 1$	Nonsmokers, Tar	2.5	5
$X = 1, Z = 1$	Smokers, Tar	47.5	85

These results seem to prove that smoking is a major contributor to lung cancer. However, the tobacco industry might argue that the table tells a different story – that smoking actually decreases one's risk of lung cancer. Their argument goes as follows. If you decide to smoke, then your chances of building up tar deposits are 95%, compared to 5% if you decide not to smoke. In order to evaluate the effect of tar deposits, we look separately at two groups, smokers and nonsmokers. The table shows that tar deposits have a protective effect in both groups: in smokers, tar deposits lower cancer rates from 90% to 85%; in nonsmokers, they lower cancer rates from 10% to 5%. Thus, regardless of whether I have a natural craving for nicotine, I should be seeking the protective effect of tar deposits in my lungs, and smoking offers a very effective means of acquiring those deposits.

To settle the dispute between the two interpretations, we now apply the front-door formula (equation (3.29)) to the data in Table 3.1. We wish to calculate the probability that a randomly selected person will develop cancer under each of the following two actions: smoking (setting $X = 1$) or not smoking (setting $X = 0$).

Substituting the appropriate values of $P(z \mid x)$, $P(y \mid x, z)$, and $P(x)$, we have

$$\begin{aligned} P(Y = 1 \mid do(X = 1)) &= .05(.10 \times .50 + .90 \times .50) \\ &\quad +.95(.05 \times .50 + .85 \times .50) \\ &= .05 \times .50 + .95 \times .45 = .4525, \\ P(Y = 1 \mid do(X = 0)) &= .95(.10 \times .50 + .90 \times .50) \\ &\quad +.05(.05 \times .50 + .85 \times .50) \\ &= .95 \times .50 + .05 \times .45 = .4975. \end{aligned} \tag{3.30}$$

Thus, contrary to expectation, the data prove smoking to be somewhat beneficial to one's health.

The data in Table 3.1 are obviously unrealistic and were deliberately crafted so as to support the genotype theory. However, the purpose of this exercise was to demonstrate how reasonable qualitative assumptions about the workings of mechanisms, coupled with nonexperimental data, can produce precise quantitative assessments of causal effects. In reality, we would expect observational studies involving mediating variables to refute the genotype theory by showing, for example, that the mediating consequences of smoking

(such as tar deposits) tend to increase, not decrease, the risk of cancer in smokers and nonsmokers alike. The estimand of (3.29) could then be used for quantifying the causal effect of smoking on cancer.

3.4 A CALCULUS OF INTERVENTION

This section establishes a set of inference rules by which probabilistic sentences involving interventions and observations can be transformed into other such sentences, thus providing a syntactic method of deriving (or verifying) claims about interventions. Each inference rule will respect the interpretation of the $do(\cdot)$ operator as an intervention that modifies a select set of functions in the underlying model. The set of inference rules that emerge from this interpretation will be called *do calculus.*

We will assume that we are given the structure of a causal diagram G in which some of the nodes are observable while others remain unobserved. Our objective will be to facilitate the syntactic derivation of causal effect expressions of the form $P(y \mid \hat{x})$, where X and Y stand for any subsets of observed variables. By "derivation" we mean stepwise reduction of the expression $P(y \mid \hat{x})$ to an equivalent expression involving standard probabilities of observed quantities. Whenever such reduction is feasible, the causal effect of X on Y is identifiable (see Definition 3.2.4).

3.4.1 Preliminary Notation

Let X, Y, and Z be arbitrary disjoint sets of nodes in a causal DAG G. We denote by $G_{\overline{X}}$ the graph obtained by deleting from G all arrows pointing to nodes in X. Likewise, we denote by $G_{\underline{X}}$ the graph obtained by deleting from G all arrows emerging from nodes in X. To represent the deletion of both incoming and outgoing arrows, we use the notation $G_{\overline{X}\underline{Z}}$ (see Figure 3.6 for an illustration). Finally, the expression $P(y \mid \hat{x}, z) \triangleq P(y, z \mid \hat{x})/P(z \mid \hat{x})$ stands for the probability of $Y = y$ given that X is held constant at x and that (under this condition) $Z = z$ is observed.

3.4.2 Inference Rules

The following theorem states the three basic inference rules of the proposed calculus. Proofs are provided in Pearl (1995a).

Theorem 3.4.1 (Rules of *do* Calculus)
Let G be the directed acyclic graph associated with a causal model as defined in (3.2), *and let $P(\cdot)$ stand for the probability distribution induced by that model. For any disjoint subsets of variables X, Y, Z, and W, we have the following rules.*

Rule 1 *(Insertion/deletion of observations)*:

$$P(y \mid \hat{x}, z, w) = P(y \mid \hat{x}, w) \quad \text{if } (Y \perp\!\!\!\perp Z \mid X, W)_{G_{\overline{X}}}. \tag{3.31}$$

Rule 2 *(Action/observation exchange)*:

$$P(y \mid \hat{x}, \hat{z}, w) = P(y \mid \hat{x}, z, w) \quad \text{if } (Y \perp\!\!\!\perp Z \mid X, W)_{G_{\overline{X}\underline{Z}}}. \tag{3.32}$$

Rule 3 *(Insertion/deletion of actions)*:

$$P(y \mid \hat{x}, \hat{z}, w) = P(y \mid \hat{x}, w) \quad \text{if } (Y \perp\!\!\!\perp Z \mid X, W)_{G_{\overline{X},\overline{Z(W)}}}, \tag{3.33}$$

where $Z(W)$ is the set of Z-nodes that are not ancestors of any W-node in $G_{\overline{X}}$.

Each of these inference rules follows from the basic interpretation of the "hat" $\hat{x}$ operator as a replacement of the causal mechanism that connects X to its preaction parents by a new mechanism $X = x$ introduced by the intervening force. The result is a submodel characterized by the subgraph $G_{\overline{X}}$ (named "manipulated graph" in Spirtes et al. 1993).

Rule 1 reaffirms d-separation as a valid test for conditional independence in the distribution resulting from the intervention $do(X = x)$, hence the graph $G_{\overline{X}}$. This rule follows from the fact that deleting equations from the system does not introduce any dependencies among the remaining disturbance terms (see (3.2)).

Rule 2 provides a condition for an external intervention $do(Z = z)$ to have the same effect on Y as the passive observation $Z = z$. The condition amounts to $\{X \cup W\}$ blocking all back-door paths from Z to Y (in $G_{\overline{X}}$), since $G_{\overline{X}\underline{Z}}$ retains all (and only) such paths.

Rule 3 provides conditions for introducing (or deleting) an external intervention $do(Z = z)$ without affecting the probability of $Y = y$. The validity of this rule stems, again, from simulating the intervention $do(Z = z)$ by the deletion of all equations corresponding to the variables in Z (hence the graph $G_{\overline{XZ}}$). The reason for limiting the deletion to nonancestors of W-nodes is provided with the proofs of Rules 1–3 in Pearl (1995a).

Corollary 3.4.2

A causal effect $q = P(y_1, \ldots, y_k \mid \hat{x}_1, \ldots, \hat{x}_m)$ is identifiable in a model characterized by a graph G if there exists a finite sequence of transformations, each conforming to one of the inference rules in Theorem 3.4.1, that reduces q into a standard (i.e., "hat"-free) probability expression involving observed quantities.

Whether Rules 1–3 are sufficient for deriving all identifiable causal effects remains an open question. However, the task of finding a sequence of transformations (if such exists) for reducing an arbitrary causal effect expression can be systematized and executed by efficient algorithms (Galles and Pearl 1995; Pearl and Robins 1995), to be discussed in Chapter 4. As we illustrate in Section 3.4.3, symbolic derivations using the hat notation are much more convenient than algebraic derivations that aim at eliminating latent variables from standard probability expressions (as in Section 3.3.2, equation (3.24)).

3.4.3 Symbolic Derivation of Causal Effects: An Example

We will now demonstrate how Rules 1–3 can be used to derive all causal effect estimands in the structure of Figure 3.5. Figure 3.6 displays the subgraphs that will be needed for the derivations that follow.

Task 1: Compute $P(z \mid \hat{x})$

This task can be accomplished in one step, since G satisfies the applicability condition for Rule 2. That is, $X \perp\!\!\!\perp Z$ in $G_{\underline{X}}$ (because the path $X \leftarrow U \rightarrow Y \leftarrow Z$ is blocked by the converging arrows at Y) and we can write

$$P(z \mid \hat{x}) = P(z \mid x). \tag{3.34}$$

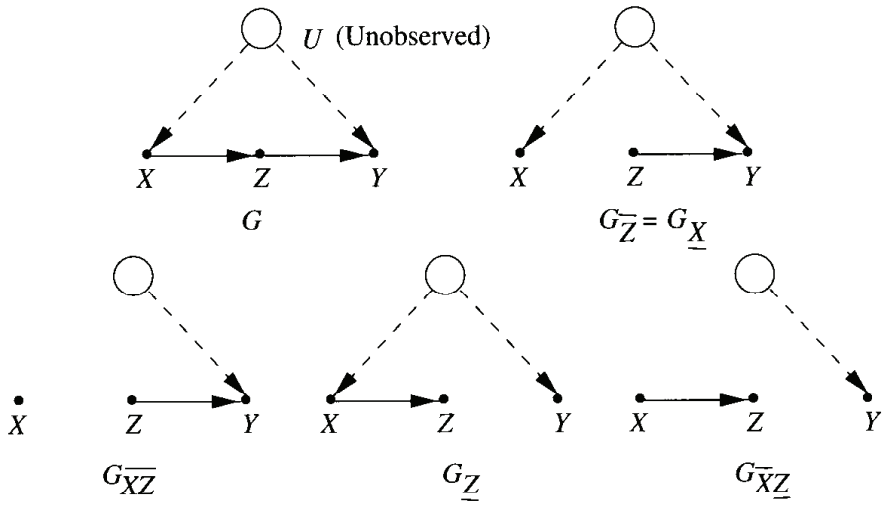

Figure 3.6 Subgraphs of G used in the derivation of causal effects.

Task 2: Compute $P(y \mid \hat{z})$

Here we cannot apply Rule 2 to exchange $\hat{z}$ with z because $G_{\underline{Z}}$ contains a back-door path from Z to Y: $Z \leftarrow X \leftarrow U \rightarrow Y$. Naturally, we would like to block this path by measuring variables (such as X) that reside on that path. This involves conditioning and summing over all values of X:

$$P(y \mid \hat{z}) = \sum_x P(y \mid x, \hat{z}) P(x \mid \hat{z}). \tag{3.35}$$

We now have to deal with two terms involving $\hat{z}$, $P(y \mid x, \hat{z})$ and $P(x \mid \hat{z})$. The latter can be readily computed by applying Rule 3 for action deletion:

$$P(x \mid \hat{z}) = P(x) \quad \text{if } (Z \perp\!\!\!\perp X)_{G_{\overline{Z}}}, \tag{3.36}$$

since X and Z are d-separated in $G_{\overline{Z}}$. (Intuitively, manipulating Z should have no effect on X, because Z is a descendant of X in G.) To reduce the former term, $P(y \mid x, \hat{z})$, we consult Rule 2:

$$P(y \mid x, \hat{z}) = P(y \mid x, z) \quad \text{if } (Z \perp\!\!\!\perp Y \mid X)_{G_{\underline{Z}}}, \tag{3.37}$$

noting that X d-separates Z from Y in $G_{\underline{Z}}$. This allows us to write (3.35) as

$$P(y \mid \hat{z}) = \sum_x P(y \mid x, z) P(x) = E_x P(y \mid x, z), \tag{3.38}$$

which is a special case of the back-door formula (equation (3.19)). The legitimizing condition, $(Z \perp\!\!\!\perp Y \mid X)_{G_{\underline{Z}}}$, offers yet another graphical test for a set X to be sufficient for control of confounding (between Y and Z) that is equivalent to the ignorability condition of Rosenbaum and Rubin (1983).

Task 3: Compute $P(y \mid \hat{x})$

Writing

$$P(y \mid \hat{x}) = \sum_z P(y \mid z, \hat{x}) P(z \mid \hat{x}), \tag{3.39}$$

we see that the term $P(z \mid \hat{x})$ was reduced in (3.34) but that no rule can be applied to eliminate the hat symbol $\hat{}$ from the term $P(y \mid z, \hat{x})$. However, we can legitimately add this symbol via Rule 2:

$$P(y \mid z, \hat{x}) = P(y \mid \hat{z}, \hat{x}), \tag{3.40}$$

since the applicability condition $(Y \perp\!\!\!\perp Z \mid X)_{G_{\overline{X}\underline{Z}}}$ holds (see Figure 3.6). We can now delete the action $\hat{x}$ from $P(y \mid \hat{z}, \hat{x})$ using Rule 3, since $Y \perp\!\!\!\perp X \mid Z$ holds in $G_{\overline{X}\overline{Z}}$. Thus, we have

$$P(y \mid z, \hat{x}) = P(y \mid \hat{z}), \tag{3.41}$$

which was calculated in (3.38). Substituting (3.38), (3.41), and (3.34) back into (3.39) finally yields

$$P(y \mid \hat{x}) = \sum_{z} P(z \mid x) \sum_{x'} P(y \mid x', z) P(x'), \tag{3.42}$$

which is identical to the front-door formula of (3.28).

Task 4: Compute $P(y, z \mid \hat{x})$
We have

$$P(y, z \mid \hat{x}) = P(y \mid z, \hat{x}) P(z \mid \hat{x}).$$

The two terms on the r.h.s. were derived before in (3.34) and (3.41), from which we obtain

$$\begin{aligned} P(y, z \mid \hat{x}) &= P(y \mid \hat{z}) P(z \mid x) \\ &= P(z \mid x) \sum_{x'} P(y \mid x', z) P(x'). \end{aligned} \tag{3.43}$$

Task 5: Compute $P(x, y \mid \hat{z})$
We have

$$\begin{aligned} P(x, y \mid \hat{z}) &= P(y \mid x, \hat{z}) P(x \mid \hat{z}) \\ &= P(y \mid x, z) P(x). \end{aligned} \tag{3.44}$$

The first term on the r.h.s. is obtained by Rule 2 (licensed by $G_{\underline{Z}}$) and the second term by Rule 3 (as in (3.36)).

Note that, in all the derivations, the graph G has provided both the license for applying the inference rules and the guidance for choosing the right rule to apply.

3.4.4 Causal Inference by Surrogate Experiments

Suppose we wish to learn the causal effect of X on Y when $P(y \mid \hat{x})$ is not identifiable and, for practical reasons of cost or ethics, we cannot control X by randomized experiment. The question arises of whether $P(y \mid \hat{x})$ can be identified by randomizing

a surrogate variable Z that is easier to control than X. For example, if we are interested in assessing the effect of cholesterol levels (X) on heart disease (Y), a reasonable experiment to conduct would be to control subjects' diet (Z), rather than exercising direct control over cholesterol levels in subjects' blood.

Formally, this problem amounts to transforming $P(y \mid \hat{x})$ into expressions in which only members of Z obtain the hat symbol. Using Theorem 3.4.1, it can be shown that the following conditions are sufficient for admitting a surrogate variable Z:

(i) X intercepts all directed paths from Z to Y; and

(ii) $P(y \mid \hat{x})$ is identifiable in $G_{\overline{Z}}$.

Indeed, if condition (i) holds then we can write $P(y \mid \hat{x}) = P(y \mid \hat{x}, \hat{z})$, because $(Y \perp\!\!\!\perp Z \mid X)_{G_{\overline{X}\underline{Z}}}$. But $P(y \mid \hat{x}, \hat{z})$ stands for the causal effect of X on Y in a model governed by $G_{\overline{Z}}$, which – by condition (ii) – is identifiable. Translated to our cholesterol example, these condition require that there be no direct effect of diet on heart conditions and no confounding of cholesterol levels and heart disease, unless we can neutralize such confounding by additional measurements.

Figures 3.9(e) and 3.9(h) (in Section 3.5.2) illustrate models in which both conditions hold. With Figure 3.9(e), for example, we obtain this estimand

$$P(y \mid \hat{x}) = P(y \mid x, \hat{z}) = \frac{P(y, x \mid \hat{z})}{P(x \mid \hat{z})}. \tag{3.45}$$

This can be established directly by first applying Rule 3 to add $\hat{z}$,

$$P(y \mid \hat{x}) = P(y \mid \hat{x}, \hat{z}) \quad \text{because } (Y \perp\!\!\!\perp Z \mid X)_{G_{\overline{X}\overline{Z}}},$$

and then applying Rule 2 to exchange $\hat{x}$ with x:

$$P(y \mid \hat{x}, \hat{z}) = P(y \mid x, \hat{z}) \quad \text{because } (Y \perp\!\!\!\perp X \mid Z)_{G_{\underline{X}\overline{Z}}}.$$

According to (3.45), only one level of Z suffices for the identification of $P(y \mid \hat{x})$ for any values of y and x. In other words, Z need not be varied at all; it can simply be held constant by external means and, if the assumptions embodied in G are valid, the r.h.s. of (3.45) should attain the same value regardless of the (constant) level at which Z is being held. In practice, however, several levels of Z will be needed to ensure that enough samples are obtained for each desired value of X. For example, if we are interested in the difference $E(Y \mid \hat{x}) - E(Y \mid \hat{x}')$, where x and x' are two treatment levels, then we should choose two values z and z' of Z that maximize the number of samples in x and x' (respectively) and then estimate

$$E(Y \mid \hat{x}) - E(Y \mid \hat{x}') = E(Y \mid x, \hat{z}) - E(Y \mid x', \hat{z}').$$

3.5 GRAPHICAL TESTS OF IDENTIFIABILITY

Figure 3.7 shows simple diagrams in which $P(y \mid \hat{x})$ cannot be identified owing to the presence of a "bow" pattern – a confounding arc (dashed) embracing a causal link between X and Y. A confounding arc represents the existence in the diagram of a back-door

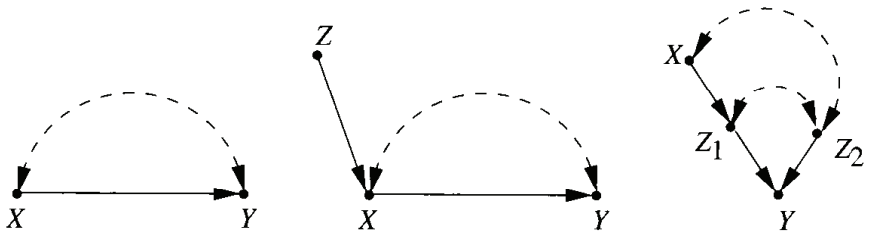

Figure 3.7 (a) A bow pattern: a confounding arc embracing a causal link $X \rightarrow Y$, thus preventing the identification of $P(y \mid \hat{x})$ even in the presence of an instrumental variable Z, as in (b). (c) A bowless graph that still prohibits the identification of $P(y \mid \hat{x})$.

path that contains only unobserved variables and has no converging arrows. For example, the path X, Z_0, B, Z_3 in Figure 3.1 can be represented as a confounding arc between X and Z_3. A bow pattern represents an equation $y = f_Y(x, u, \varepsilon_Y)$, where U is unobserved and dependent on X. Such an equation does not permit the identification of causal effects, since any portion of the observed dependence between X and Y may always be attributed to spurious dependencies mediated by U.

The presence of a bow pattern prevents the identification of $P(y \mid \hat{x})$ even when it is found in the context of a larger graph, as in Figure 3.7(b). This is in contrast to linear models, where the addition of an arc to a bow pattern can render $P(y \mid \hat{x})$ identifiable (see Chapter 5, Figure 5.9). For example, if Y is related to X via a linear relation $y = bx + u$, where U is an unobserved disturbance possibly correlated with X, then $b = \frac{\partial}{\partial x} E(Y \mid \hat{x})$ is not identifiable. However, adding an arc $Z \rightarrow X$ to the structure (i.e., finding a variable Z that is correlated with X but not with U) would facilitate the computation of $E(Y \mid \hat{x})$ via the instrumental variable formula (Bowden and Turkington 1984; see also Chapter 5):

$$b \triangleq \frac{\partial}{\partial x} E(Y \mid \hat{x}) = \frac{E(Y \mid z)}{E(X \mid z)} = \frac{r_{YZ}}{r_{XZ}}. \tag{3.46}$$

In nonparametric models, adding an instrumental variable Z to a bow pattern (Figure 3.7(b)) does not permit the identification of $P(y \mid \hat{x})$. This is a familiar problem in the analysis of clinical trials in which treatment assignment (Z) is randomized (hence, no link enters Z) but compliance is imperfect (see Chapter 8). The confounding arc between X and Y in Figure 3.7(b) represents unmeasurable factors that influence subjects' choice of treatment (X) as well as subjects' response to treatment (Y). In such trials, it is not possible to obtain an unbiased estimate of the treatment effect $P(y \mid \hat{x})$ without making additional assumptions on the nature of the interactions between compliance and response (as is done, for example, in the potential-outcome approach to instrumental variables developed in Imbens and Angrist 1994 and Angrist et al. 1996). Although the added arc $Z \rightarrow X$ permits us to calculate bounds on $P(y \mid \hat{x})$ (Robins 1989, sec. 1g; Manski 1990; Balke and Pearl 1997) and the upper and lower bounds may even coincide for certain types of distributions $P(x, y, z)$ (Section 8.2.4), there is no way of computing $P(y \mid \hat{x})$ for *every* positive distribution $P(x, y, z)$, as required by Definition 3.2.4.

In general, the addition of arcs to a causal diagram can impede, but never assist, the identification of causal effects in nonparametric models. This is because such addition

reduces the set of d-separation conditions carried by the diagram; hence, if a causal effect derivation fails in the original diagram, it is bound to fail in the augmented diagram as well. Conversely, any causal effect derivation that succeeds in the augmented diagram (by a sequence of symbolic transformations, as in Corollary 3.4.2) would succeed in the original diagram.

Our ability to compute $P(y_1 \mid \hat{x})$ and $P(y_2 \mid \hat{x})$ for pairs (Y_1, Y_2) of singleton variables does not ensure our ability to compute joint distributions, such as $P(y_1, y_2 \mid \hat{x})$. Figure 3.7(c), for example, shows a causal diagram where both $P(z_1 \mid \hat{x})$ and $P(z_2 \mid \hat{x})$ are computable yet $P(z_1, z_2 \mid \hat{x})$ is not. Consequently, we cannot compute $P(y \mid \hat{x})$. It is interesting to note that this diagram is the smallest graph that does not contain a bow pattern and still presents an uncomputable causal effect.

Another interesting feature demonstrated by Figure 3.7(c) is that computing the effect of a joint intervention is often easier than computing the effects of its constituent singleton interventions.[6] Here, it is possible to compute $P(y \mid \hat{x}, \hat{z}_2)$ and $P(y \mid \hat{x}, \hat{z}_1)$, yet there is no way of computing $P(y \mid \hat{x})$. For example, the former can be evaluated by invoking Rule 2 in $G_{\overline{X}\underline{Z}_2}$, giving

$$\begin{aligned} P(y \mid \hat{x}, \hat{z}_2) &= \sum_{z_1} P(y \mid z_1, \hat{x}, \hat{z}_2) P(z_1 \mid \hat{x}, \hat{z}_2) \\ &= \sum_{z_1} P(y \mid z_1, x, z_2) P(z_1 \mid x). \end{aligned} \tag{3.47}$$

However, Rule 2 cannot be used to convert $P(z_1 \mid \hat{x}, z_2)$ into $P(z_1 \mid x, z_2)$ because, when conditioned on Z_2, X and Z_1 are d-connected in $G_{\underline{X}}$ (through the dashed lines). A general approach to computing the effect of joint interventions is developed in Pearl and Robins (1995); this is described in Chapter 4 (Section 4.4).

3.5.1 Identifying Models

Figure 3.8 shows simple diagrams in which the causal effect of X on Y is identifiable (where X and Y are single variables). Such models are called "identifying" because their structures communicate a sufficient number of assumptions (missing links) to permit the identification of the target quantity $P(y \mid \hat{x})$. Latent variables are not shown explicitly in these diagrams; rather, such variables are implicit in the confounding arcs (dashed). Every causal diagram with latent variables can be converted to an equivalent diagram involving measured variables interconnected by arrows and confounding arcs. This conversion corresponds to substituting out all latent variables from the structural equations of (3.2) and then constructing a new diagram by connecting any two variables X_i and X_j by (i) an arrow from X_j to X_i whenever X_j appears in the equation for X_i and (ii) a confounding arc whenever the same ε term appears in both f_i and f_j. The result is a diagram in which all unmeasured variables are exogenous and mutually independent.

Several features should be noted from examining the diagrams in Figure 3.8.

[6] This was brought to my attention by James Robins, who has worked out many of these computations in the context of sequential treatment management (Robins 1986, p. 1423).

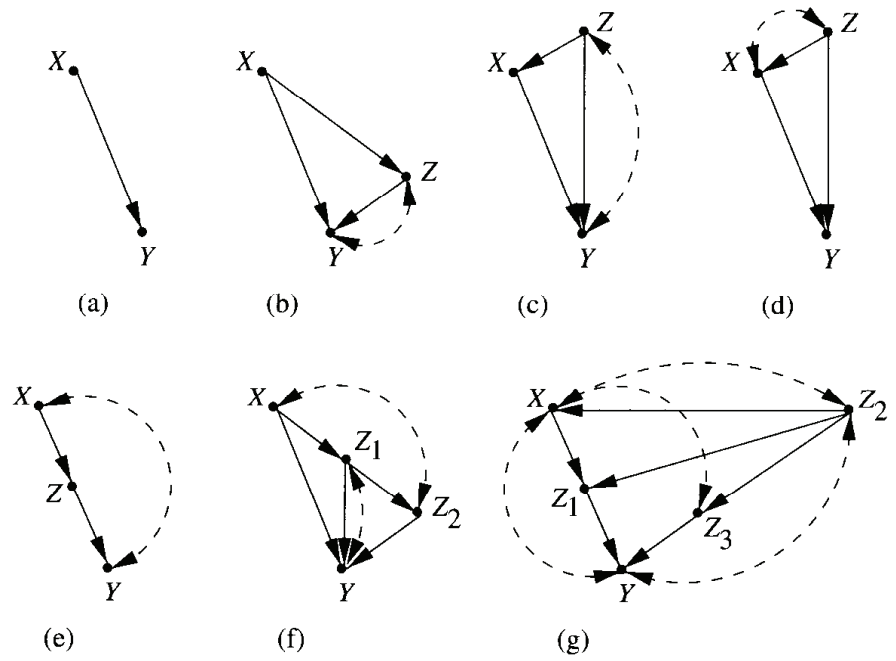

Figure 3.8 Typical models in which the effect of X on Y is identifiable. Dashed arcs represent confounding paths, and Z represents observed covariates.

1. Since the removal of any arc or arrow from a causal diagram can only assist the identifiability of causal effects, $P(y \mid \hat{x})$ will still be identified in any edge subgraph of the diagrams shown in Figure 3.8. Likewise, the introduction of mediating observed variables onto any edge in a causal graph can assist, but never impede, the identifiability of any causal effect. Therefore, $P(y \mid \hat{x})$ will still be identified from any graph obtained by adding mediating nodes to the diagrams shown in Figure 3.8.
2. The diagrams in Figure 3.8 are maximal in the sense that the introduction of any additional arc or arrow onto an existing pair of nodes would render $P(y \mid \hat{x})$ no longer identifiable.
3. Although most of the diagrams in Figure 3.8 contain bow patterns, none of these patterns emanates from X (as is the case in Figures 3.9(a) and (b) to follow). In general, a necessary condition for the identifiability of $P(y \mid \hat{x})$ is the absence of a confounding arc between X and any child of X that is an ancestor of Y.
4. Diagrams (a) and (b) in Figure 3.8 contain no back-door paths between X and Y and thus represent experimental designs in which there is no confounding bias between the treatment (X) and the response (Y); hence, $P(y \mid \hat{x}) = P(y \mid x)$. Likewise, diagrams (c) and (d) in Figure 3.8 represent designs in which observed covariates Z block every back-door path between X and Y (i.e., X is "conditionally ignorable" given Z, in the language of Rosenbaum and Rubin 1983); hence, $P(y \mid \hat{x})$ is obtained by standard adjustment for Z (as in (3.19)):

$$P(y \mid \hat{x}) = \sum_z P(y \mid x, z)P(z).$$

5. For each of the diagrams in Figure 3.8, we readily obtain a formula for $P(y \mid \hat{x})$ by using symbolic derivations patterned after those in Section 3.4.3. The derivation

is often guided by the graph topology. For example, diagram (f) in Figure 3.8 dictates the following derivation. Writing

$$P(y \mid \hat{x}) = \sum_{z_1, z_2} P(y \mid z_1, z_2, \hat{x}) P(z_1, z_2 \mid \hat{x}),$$

we see that the subgraph containing $\{X, Z_1, Z_2\}$ is identical in structure to that of diagram (e), with (Z_1, Z_2) replacing (Z, Y), respectively. Thus, $P(z_1, z_2 \mid \hat{x})$ can be obtained from (3.43). Likewise, the term $P(y \mid z_1, z_2, \hat{x})$ can be reduced to $P(y \mid z_1, z_2, x)$ by Rule 2, since $(Y \perp\!\!\!\perp X \mid Z_1, Z_2)_{G_{\underline{X}}}$. We therefore have

$$P(y \mid \hat{x}) = \sum_{z_1, z_2} P(y \mid z_1, z_2, x) P(z_1 \mid x) \sum_{x'} P(z_2 \mid z_1, x')\, P(x'). \quad (3.48)$$

Applying a similar derivation to diagram (g) of Figure 3.8 yields

$$P(y \mid \hat{x}) = \sum_{z_1} \sum_{z_2} \sum_{x'} P(y \mid z_1, z_2, x') P(x' \mid z_2) \times P(z_1 \mid z_2, x) P(z_2). \quad (3.49)$$

Note that the variable Z_3 does not appear in (3.49), which means that Z_3 need not be measured if all one wants to learn is the causal effect of X on Y.

6. In diagrams (e), (f), and (g) of Figure 3.8, the identifiability of $P(y \mid \hat{x})$ is rendered feasible through observed covariates Z that are affected by the treatment X (since members of Z are descendants of X). This stands contrary to the warning – repeated in most of the literature on statistical experimentation – to refrain from adjusting for concomitant observations that are affected by the treatment (Cox 1958; Rosenbaum 1984; Pratt and Schlaifer 1988; Wainer 1989). It is commonly believed that a concomitant Z that is affected by the treatment must be excluded from the analysis of the total effect of the treatment (Pratt and Schlaifer 1988). The reason given for the exclusion is that the calculation of total effects amounts to integrating out Z, which is functionally equivalent to omitting Z to begin with. Diagrams (e), (f), and (g) show cases where the total effects of X are indeed the target of investigation and, even so, the measurement of concomitants that are affected by X (e.g., Z or Z_1) is still necessary. However, the adjustment needed for such concomitants is nonstandard, involving two or more stages of the standard adjustment of (3.19) (see (3.28), (3.48), and (3.49)).

7. In diagrams (b), (c), and (f) of Figure 3.8, Y has a parent whose effect on Y is not identifiable; even so, the effect of X on Y is identifiable. This demonstrates that local identifiability is not a necessary condition for global identifiability. In other words, to identify the effect of X on Y we need not insist on identifying each and every link along the paths from X to Y.

3.5.2 Nonidentifying Models

Figure 3.9 presents typical diagrams in which the total effect of X on Y, $P(y \mid \hat{x})$, is not identifiable. Noteworthy features of these diagrams are as follows.

1. All graphs in Figure 3.9 contain unblockable back-door paths between X and Y, that is, paths ending with arrows pointing to X that cannot be blocked by observed nondescendants of X. The presence of such a path in a graph is, indeed,

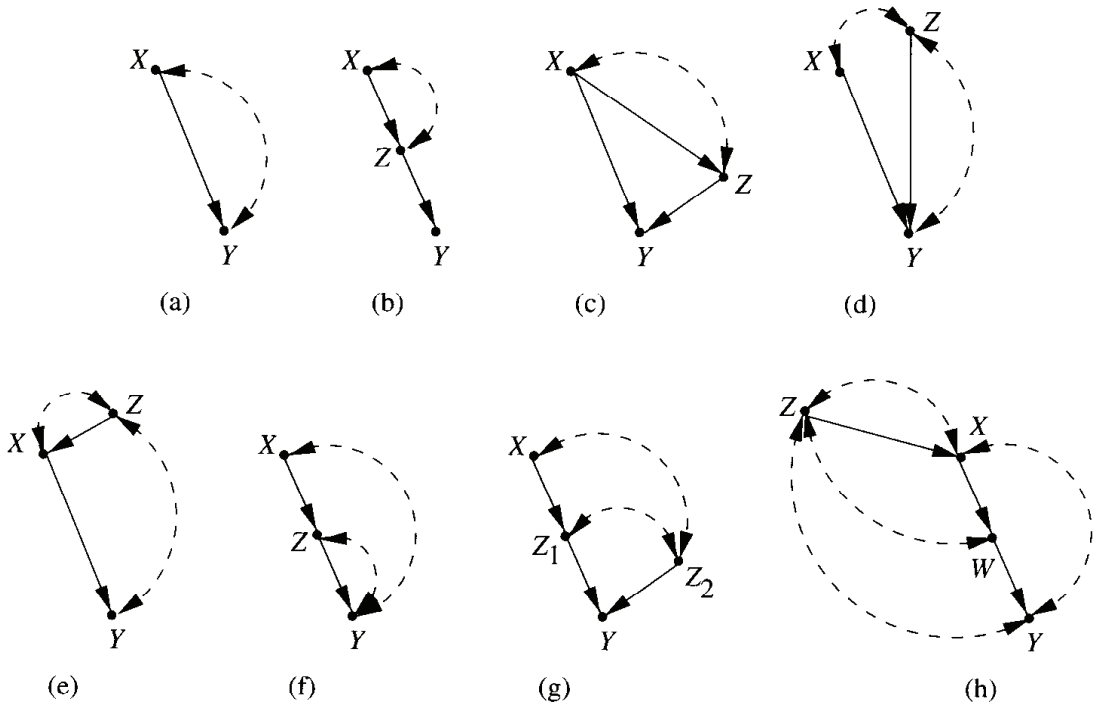

Figure 3.9 Typical models in which $P(y \mid \hat{x})$ is not identifiable.

a necessary test for nonidentifiability (see Theorem 3.3.2). That it is not a sufficient test is demonstrated by Figure 3.8(e), in which the back-door path (dashed) is unblockable and yet $P(y \mid \hat{x})$ is identifiable.

2. A sufficient condition for the nonidentifiability of $P(y \mid \hat{x})$ is the existence of a confounding path between X and any of its children on a path from X to Y, as shown in Figures 3.9(b) and (c). A stronger sufficient condition is that the graph contain any of the patterns shown in Figure 3.9 as an edge subgraph.
3. Graph (g) in Figure 3.9 (same as Figure 3.7(c)) demonstrates that local identifiability is not sufficient for global identifiability. For example, we can identify $P(z_1 \mid \hat{x})$, $P(z_2 \mid \hat{x})$, $P(y \mid \hat{z}_1)$, and $P(y \mid \hat{z}_2)$ but not $P(y \mid \hat{x})$. This is one of the main differences between nonparametric and linear models; in the latter, all causal effects can be determined from the structural coefficients and each coefficient represents the causal effect of one variable on its immediate successor.

3.6 DISCUSSION

3.6.1 Qualifications and Extensions

The methods developed in this chapter facilitate the drawing of quantitative causal inferences from a combination of qualitative causal assumptions (encoded in the diagram) and nonexperimental observations. The causal assumptions in themselves cannot generally be tested in nonexperimental studies, unless they impose constraints on the observed distributions. The most common type of constraints appears in the form of conditional independencies, as communicated through the d-separation conditions in the diagrams. Another type of constraints takes the form of numerical inequalities. In Chapter 8, for example, we show that the assumptions associated with instrumental variables (Figure 3.7(b)) are

subject to falsification tests in the form of inequalities on conditional probabilities (Pearl 1995b). Still, such constraints permit the testing of merely a small fraction of the causal assumptions embodied in the diagrams; the bulk of those assumptions must be substantiated from domain knowledge as obtained from either theoretical considerations (e.g., that falling barometers do not cause rain) or related experimental studies. For example, the experimental study of Moertel et al. (1985), which refuted the hypothesis that vitamin C is effective against cancer, can be used as a substantive assumption in observational studies involving vitamin C and cancer patients; it would be represented as a missing link (between vitamin C and cancer) in the associated diagram. In summary, the primary use of the methods described in this chapter lies not in testing causal assumptions but in providing an effective language for making those assumptions precise and explicit. Assumptions can thereby be isolated for deliberation or experimentation and then (once validated) be integrated with statistical data to yield quantitative estimates of causal effects.

An important issue that will be considered only briefly in this book (see Section 8.5) is sampling variability. The mathematical derivation of causal effect estimands should be considered a first step toward supplementing these estimands with confidence intervals and significance levels, as in traditional analysis of controlled experiments. We should remark, though, that having obtained nonparametric estimands for causal effects does not imply that one should refrain from using parametric forms in the estimation phase of the study. For example, if the assumptions of Gaussian, zero-mean disturbances and additive interactions are deemed reasonable, then the estimand given in (3.28) can be converted to the product $E(Y \mid \hat{x}) = r_{ZX} r_{YZ \cdot X} x$, where $r_{YZ \cdot X}$ is the standardized regression coefficient (Section 5.3.1); the estimation problem then reduces to that of estimating regression coefficients (e.g., by least squares). More sophisticated estimation techniques can be found in Rosenbaum and Rubin (1983), Robins (1989, sec. 17), and Robins et al. (1992, pp. 331–3). For example, the "propensity score" method of Rosenbaum and Rubin (1983) was found to be quite useful when the dimensionality of the adjusted covariates is high. In a more recent scheme called "marginal models," Robins (1999) shows that, rather than estimating individual factors in the adjustment formula of (3.19), it is often more advantageous to use $P(y \mid \hat{x}) = \sum_z \frac{P(x,y,z)}{P(x|z)}$, where the preintervention distribution remains unfactorized. One can then separately estimate the denominator $P(x \mid z)$, weigh individual samples by the inverse of this estimate, and treat the weighted samples as if they were drawn at random from the postintervention distribution $P(y \mid \hat{x})$. Postintervention parameters, such as $\frac{\partial}{\partial x} E(Y \mid \hat{x})$, can then be estimated by ordinary least squares. This method is especially advantageous in longitudinal studies with time-varying covariates, as in the process control problem discussed in Section 3.2.3 (see (3.18)).

Several extensions of the methods proposed in this chapter are noteworthy. First, the identification analysis for atomic interventions can be generalized to complex policies in which a set X of controlled variables is made to respond in a specified way to some set Z of covariates via functional or stochastic strategies, as in Section 3.2.3. In Chapter 4 (Section 4.2) it is shown that identifying the effect of such policies is equivalent to computing the expression $P(y \mid \hat{x}, z)$.

A second extension concerns the use of the intervention calculus (Theorem 3.4.1) in nonrecursive models, that is, in causal diagrams involving directed cycles or feedback loops. The basic definition of causal effects in term of "wiping out" equations from the model (Definition 3.2.1) still carries over to nonrecursive systems (Strotz and Wold

1960; Sobel 1990), but then two issues must be addressed. First, the analysis of identification must ensure the stability of the remaining submodels (Fisher 1970). Second, the d-separation criterion for DAGs must be extended to cover cyclic graphs as well. The validity of d-separation has been established for nonrecursive linear models (Spirtes 1995) as well as for nonlinear systems involving discrete variables (Pearl and Dechter 1996). However, the computation of causal effect estimands will be harder in cyclic nonlinear systems, because symbolic reduction of $P(y \mid \hat{x})$ to hat-free expressions may require the solution of nonlinear equations. In Chapter 7 (Section 7.2.1) we demonstrate the evaluation of policies and counterfactuals in nonrecursive linear systems (see also Balke and Pearl 1995).

A third extension concerns generalizations of intervention calculus (Theorem 3.4.1) to situations where the data available is not obtained under i.i.d. (independent and identically distributed) sampling. One can imagine, for instance, a physician who prescribes a certain treatment to patients only when the fraction of survivors among previous patients drops below some threshold. In such cases, it is required to estimate the causal effect $P(y \mid \hat{x})$ from nonindependent samples. Vladimir Vovk (1996) gave conditions under which the rules of Theorem 3.4.1 will be applicable when sampling is not i.i.d., and he went on to cast the three inference rules as a logical production system.

3.6.2 Diagrams as a Mathematical Language

The benefit of incorporating substantive background knowledge into probabilistic inference was recognized as far back as Thomas Bayes (1763) and Pierre Laplace (1814), and its crucial role in the analysis and interpretation of complex statistical studies is generally acknowledged by most modern statisticians. However, the mathematical language available for expressing background knowledge has remained in a rather pitiful state of development.

Traditionally, statisticians have approved of only one way of combining substantive knowledge with statistical data: the Bayesian method of assigning subjective priors to distributional parameters. To incorporate causal information within this framework, plain causal statements such as "Y is not affected by X" must be converted into sentences or events capable of receiving probability values (e.g. counterfactuals). For instance, to communicate the innocent assumption that mud does not cause rain, we would have to use a rather unnatural expression and say that the probability of the counterfactual event "rain if it were not muddy" is the same as the probability of "rain if it were muddy." Indeed, this is how the potential-outcome approach of Neyman and Rubin has achieved statistical legitimacy: causal judgments are expressed as constraints on probability functions involving counterfactual variables (see Section 3.6.3).

Causal diagrams offer an alternative language for combining data with causal information. This language simplifies the Bayesian route by accepting plain causal statements as its basic primitives. Such statements, which merely indicate whether a causal connection between two variables of interest exists, are commonly used in ordinary discourse and provide a natural way for scientists to communicate experience and organize knowledge.[7]

[7] Remarkably, many readers of this chapter (including two referees of this book) classified the methods presented here as belonging to the "Bayesian camp" and as depending on a "good prior." This

It can be anticipated, therefore, that the language of causal graphs will find applications in problems requiring substantial domain knowledge.

The language is not new. The use of diagrams and structural equations models to convey causal information has been quite popular in the social sciences and econometrics. Statisticians, however, have generally found these models suspect, perhaps because social scientists and econometricians have failed to provide an unambiguous definition of the empirical content of their models – that is, to specify the experimental conditions, however hypothetical, whose outcomes would be constrained by a given structural equation. (Chapter 5 discusses the bizarre history of structural equations in the social sciences and economics). As a result, even such basic notions as "structural coefficients" or "missing links" become the object of serious controversy (Freedman 1987; Goldberger 1992) and misinterpretations (Whittaker 1990, p. 302; Wermuth 1992; Cox & Wermuth 1993).

To a large extent, this history of controversy and miscommunication stems from the absence of an adequate mathematical notation for defining basic notions of causal modeling. For example, standard probabilistic notation cannot express the empirical content of the coefficient b in the structural equation $y = bx + \varepsilon_Y$, even if one is prepared to assume that ε_Y (an unobserved quantity) is uncorrelated with X.[8] Nor can any probabilistic meaning be attached to the analyst's excluding from the equation variables that are highly correlated with X or Y but do not "directly affect" Y.[9]

The notation developed in this chapter gives these (causal) notions a clear empirical interpretation, because it permits one to specify precisely what is being held constant and what is merely measured in a given experiment. (The need for this distinction was recognized by many researchers, most notably Pratt and Schlaifer 1988 and Cox 1992). The meaning of b is simply $\frac{\partial}{\partial x}E(Y \mid \hat{x})$, that is, the rate of change (in x) of the expectation of Y in an experiment where X is held at x by external control. This interpretation holds regardless of whether ε_Y and X are correlated (e.g., via another equation $x = ay + \varepsilon_x$). Likewise, the analyst's decision as to which variables should be included in a given equation can be based on a hypothetical controlled experiment: A variable Z is excluded from the equation for Y if (for every level of ε_Y) Z has no influence on Y when all other variables (S_{YZ}) are held constant; this implies $P(y \mid \hat{z}, \hat{s}_{YZ}) = P(y \mid \hat{s}_{YZ})$. Specifically, variables that are excluded from the equation $y = bx + \varepsilon_Y$ are not conditionally independent of Y given measurements of X but instead are *causally* irrelevant to Y given settings of X. The operational meaning of the "disturbance term" ε_Y is likewise demystified: ε_Y is defined as the difference $Y - E(Y \mid \hat{s}_Y)$. Two disturbance terms, ε_X and ε_Y, are correlated if $P(y \mid \hat{x}, \hat{s}_{XY}) \neq P(y \mid x, \hat{s}_{XY})$, and so on (see Chapter 5, Section 5.4, for further elaboration).

The distinctions provided by the hat notation clarify the empirical basis of structural equations and should make causal models more acceptable to empirical researchers.

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classification is misleading. The method does depend on subjective assumptions (e.g., mud does not cause rain), but such assumptions are causal, not statistical, and cannot be expressed as prior probabilities on parameters of joint distributions.

[8] Voluminous literature on the subject of "exogeneity" (e.g. Richard 1980; Engle et al. 1983; Hendry 1995) has emerged from economists' struggle to give statistical interpretation to the causal assertion "X and ε_Y are uncorrelated" (Aldrich 1993; see Section 5.4.3).

[9] The bitter controversy between Goldberger (1992) and Wermuth (1992) revolves around Wermuth's insistence on giving a statistical interpretation to the zero coefficients in structural equations (see Section 5.4.1).

Moreover, since most scientific knowledge is organized around the operation of "holding X fixed" rather than "conditioning on X," the notation and calculus developed in this chapter should provide an effective means for scientists to communicate substantive information and to infer its logical consequences.

3.6.3 Translation from Graphs to Potential Outcomes

This chapter uses two representations of causal information: graphs and structural equations, where the former is an abstraction of the latter. Both representations have been controversial for almost a century. On the one hand, economists and social scientists have embraced these modeling tools, but they continue to question and debate the causal content of the parameters they estimate (see Sections 5.1 and 5.4 for details); as a result, the use of structural models in policy-making contexts is often viewed with suspicion. Statisticians, on the other hand, reject both representations as problematic (Freedman 1987) if not meaningless (Wermuth 1992; Holland 1995), and they sometimes resort to the Neyman–Rubin potential-outcome notation when pressed to communicate causal information (Rubin 1990).[10] A detailed formal analysis of the relationships between the structural and potential-outcome approaches is offered in Chapter 7 (Section 7.4.4) and proves their mathematical equivalence. In this section we highlight commonalities and differences between the two approaches as they pertain to the elicitation of causal assumptions.

The primitive object of analysis in the potential-outcome framework is the unit-based response variable, denoted $Y(x, u)$ or $Y_x(u)$, read: "the value that Y would obtain in unit u, had X been x." This counterfactual entity has natural interpretation in structural equations models. Consider a general structural model M that contains a set of equations

$$x_i = f_i(pa_i, u_i), \quad i = 1, \ldots, n, \tag{3.50}$$

as in (3.4). Let U stand for the vector $(U_1, \ldots, U_n)$ of background variables, let X and Y be two disjoint subsets of observed variables, and let M_x be the submodel created by replacing the equations corresponding to variables in X with $X = x$, as in Definition 3.2.1. The structural interpretation of $Y(x, u)$ is given by

$$Y(x, u) \triangleq Y_{M_x}(u). \tag{3.51}$$

That is, $Y(x, u)$ is the (unique) solution of Y under the realization $U = u$ in the submodel M_x of M. Although the term *unit* in the potential-outcome literature normally stands for the identity of a specific individual in a population, a unit may also be thought of as the set of attributes that characterize that individual, the experimental conditions under study, the time of day, and so on – all of which are represented as components of the vector u in structural modeling. In fact, the only requirements on U are (i) that it represent as many background factors as needed to render the relations among endogenous variables deterministic and (ii) that the data consist of independent samples drawn from $P(u)$. The

[10] A parallel framework was developed in the econometrics literature under the rubric "switching regression" Manski (1995, p. 38), which Heckman (1996) attributed to Roy (1951) and Quandt (1958).

identity of an individual person in an experiment is often sufficient for this purpose because it represents the anatomical and genetic makings of that individual, which are often sufficient for determining that individual's response to treatments or other programs of interest.

Equation (3.51) forms a connection between the opaque English phrase "the value that Y would obtain in unit u, had X been x" and the physical processes that transfer changes in X into changes in Y. The formation of the submodel M_x explicates precisely how the hypothetical phrase "had X been x" could be realized, as well as what process must give in to make $X = x$ a reality.

Given this interpretation of $Y(x, u)$, it is instructive to contrast the methodologies of causal inference in the counterfactual versus structural frameworks. If U is treated as a random variable then the value of the counterfactual $Y(x, u)$ becomes a random variable as well, denoted as $Y(x)$ or Y_x. The potential-outcome analysis proceeds by imagining the observed distribution $P(x_1, \ldots, x_n)$ as the marginal distribution of an augmented probability function P^* defined over both observed and counterfactual variables. Queries about causal effects (written $P(y \mid \hat{x})$ in our structural analysis) are phrased as queries about the marginal distribution of the counterfactual variable of interest, written $P^*(Y(x) = y)$. The new hypothetical entities $Y(x)$ are treated as ordinary random variables; for example, they are assumed to obey the axioms of probability calculus, the laws of conditioning, and the axioms of conditional independence. Moreover, these hypothetical entities are assumed to be connected to observed variables via consistency constraints (Robins 1986) such as[11]

$$X = x \implies Y(x) = Y, \tag{3.52}$$

which states that, for every u, if the actual value of X turns out to be x, then the value that Y would take on if X were x is equal to the actual value of Y. Thus, whereas the structural approach views the intervention $do(x)$ as an operation that changes the model (and the distribution) but keeps all variables the same, the potential-outcome approach views the variable Y under $do(x)$ to be a different variable, $Y(x)$, loosely connected to Y through relations such as (3.52). In Chapter 7 we show, using the structural interpretation of $Y(x, u)$, that it is indeed legitimate to treat counterfactuals as random variables in all respects and, moreover, that consistency constraints like (3.52) follow as theorems from the structural interpretation.

To communicate substantive causal knowledge, the potential-outcome analyst must express causal assumptions as constraints on P^*, usually in the form of conditional independence assertions involving counterfactual variables. For example, to communicate the understanding that – in a randomized clinical trial with imperfect compliance (see Figure 3.7(b)) – the way subjects react (Y) to treatments (X) is statistically independent of the treatment assignment (Z), the potential-outcome analyst would write $Y(x) \perp\!\!\!\perp Z$. Likewise, to convey the understanding that the assignment is randomized and hence independent of how subjects comply with the assignment, the potential-outcome analyst would use the independence constraint $Z \perp\!\!\!\perp X(z)$.

[11] Gibbard and Harper (1976, p. 156) expressed this constraint as $A \supset [(A \mathrel{\Box\!\!\rightarrow} S) \equiv S]$.

A collection of constraints of this type might sometimes be sufficient to permit a unique solution to the query of interest; in other cases, only bounds on the solution can be obtained. For example, if one can plausibly assume that a set Z of covariates satisfies the conditional independence

$$Y(x) \perp\!\!\!\perp X \mid Z \tag{3.53}$$

(an assumption that was termed "conditional ignorability" by Rosenbaum and Rubin 1983), then the causal effect $P^*(Y(x) = y)$ can readily be evaluated, using (3.52), to yield[12]

$$\begin{aligned} P^*(Y(x) = y) &= \sum_z P^*(Y(x) = y \mid z) P(z) \\ &= \sum_z P^*(Y(x) = y \mid x, z) P(z) \\ &= \sum_z P^*(Y = y \mid x, z) P(z) \\ &= \sum_z P(y \mid x, z) P(z). \end{aligned} \tag{3.54}$$

The last expression contains no counterfactual quantities (thus permitting us to drop the asterisk from P^*) and coincides precisely with the adjustment formula of (3.19), which obtains from the back-door criterion. However, the assumption of conditional ignorability (equation (3.53)) – the key to the derivation of (3.54) – is not straightforward to comprehend or ascertain. Paraphrased in experimental metaphors, this assumption reads: The way an individual with attributes Z would react to treatment $X = x$ is independent of the treatment actually received by that individual.

Section 3.6.2 explains why this approach may appeal to some statisticians, even though the process of eliciting judgments about counterfactual dependencies has been extremely difficult and error-prone; instead of constructing new vocabulary and new logic for causal expressions, all mathematical operations in the potential-outcome framework are conducted within the safe confines of probability calculus. The drawback lies in the requirement of using independencies among counterfactual variables to express plain causal knowledge. When counterfactual variables are not viewed as byproducts of a deeper, process-based model, it is hard to ascertain whether *all* relevant counterfactual independence judgments have been articulated,[13] whether the judgments articulated are redundant, or whether those judgments are self-consistent. The elicitation of such counterfactual judgments can be systematized by using the following translation from graphs (see Section 7.1.4 for additional relationships).

Graphs encode substantive information in both the equations and the probability function $P(u)$; the former is encoded as missing arrows, the latter as missing dashed arcs.

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[12] Gibbard and Harper (1976, p. 157) used the "ignorability assumption" $Y(x) \perp\!\!\!\perp X$ to derive the equality $P(Y(x) = y) = P(y \mid x)$.

[13] A typical oversight in the example of Figure 3.7(b) has been to write $Z \perp\!\!\!\perp Y(x)$ and $Z \perp\!\!\!\perp X(z)$ instead of $Z \perp\!\!\!\perp \{Y(x), X(z)\}$, as dictated by (3.56).

Each parent–child family (PA_i, X_i) in a causal diagram G corresponds to an equation in the model M of (3.50). Hence, missing arrows encode exclusion assumptions, that is, claims that adding excluded variables to an equation will not change the outcome of the hypothetical experiment described by that equation. Missing dashed arcs encode independencies among disturbance terms in two or more equations. For example, the absence of dashed arcs between a node Y and a set of nodes $\{Z_1, \ldots, Z_k\}$ implies that the corresponding background variables, U_Y and $\{U_{Z_1}, \ldots, U_{Z_k}\}$, are independent in $P(u)$.

These assumptions can be translated into the potential-outcome notation using two simple rules (Pearl 1995a, p. 704); the first interprets the missing arrows in the graph, the second, the missing dashed arcs.

1. *Exclusion restrictions:* For every variable Y having parents PA_Y and for every set of variables S disjoint of PA_Y, we have
$$Y(pa_Y) = Y(pa_Y, s). \tag{3.55}$$
2. *Independence restrictions:* If $Z_1, \ldots, Z_k$ is any set of nodes not connected to Y via dashed arcs, we have[14]
$$Y(pa_Y) \perp\!\!\!\perp \{Z_1(pa_{Z_1}), \ldots, Z_k(pa_{Z_k})\}. \tag{3.56}$$

The independence restriction translates the independence between U_Y and $\{U_{Z_1}, \ldots, U_{Z_k}\}$ into independence between the corresponding potential-outcome variables. This follows from the observation that, once we set their parents, the variables in $\{Y, Z_1, \ldots, Z_k\}$ stand in functional relationships to the U terms in their corresponding equations.

As an example, the model shown in Figure 3.5 displays the following parent sets:

$$PA_X = \{\emptyset\}, \quad PA_Z = \{X\}, \quad PA_Y = \{Z\}. \tag{3.57}$$

Consequently, the exclusion restrictions translate into:

$$Z(x) = Z(y, x), \tag{3.58}$$

$$X(y) = X(z, y) = X(z) = X, \tag{3.59}$$

$$Y(z) = Y(z, x); \tag{3.60}$$

the absence of a dashed arc between Z and $\{Y, X\}$ translates into the independence restriction

$$Z(x) \perp\!\!\!\perp \{Y(z), X\}. \tag{3.61}$$

Given a sufficient number of such restrictions on P^*, the analyst attempts to compute causal effects $P^*(Y(x) = y)$ using standard probability calculus together with the logical constraints (e.g. (3.52)) that couple counterfactual variables with their measurable counterparts. These constraints can be used as axioms, or rules of inference, in attempting to

[14] The restriction is in fact stronger, jointly applying to all instantiations of the PA variables. For example, $X \perp\!\!\!\perp Y(pa_Z)$ should be interpreted as $X \perp\!\!\!\perp \{Y(pa'_Z), Y(pa''_Z), Y(pa'''_Z), \ldots\}$, where $pa'_Z, pa''_Z, pa'''_Z, \ldots$ are the values that the set PA_Z may take on.

transform causal effect expressions of the form $P^*(Y(x) = y)$ into expressions involving only measurable variables. When such a transformation is found, the corresponding causal effect is identifiable, since P^* then reduces to P.

The question naturally arises of whether the constraints used by potential-outcome analysts are *complete* – that is, whether they are sufficient for deriving every valid statement about causal processes, interventions, and counterfactuals. To answer this question, the validity of counterfactual statements need be defined relative to more basic mathematical objects, such as possible worlds (Section 1.4.4) or structural equations (equation (3.51)). In the standard potential-outcome framework, however, the question of completeness remains open, because $Y(x, u)$ is taken as a primitive notion and because consistency constraints such as (3.52) – although they appear plausible for the English expression "had X been x," – are not derived from a deeper mathematical object. This question of completeness is settled in Chapter 7, where a necessary and sufficient set of axioms is derived from the structural semantics given to $Y(x, u)$ by (3.51).

In assessing the historical development of structural equations and potential-outcome models, one cannot overemphasize the importance of the conceptual clarity that structural equations offer vis-à-vis the potential-outcome model. The reader may appreciate this importance by attempting to judge whether the condition of (3.61) holds in a given familiar situation. This condition reads: "the value that Z would obtain had X been x is jointly independent of both X and the value that Y would obtain had Z been z." (In the structural representation, the sentence reads: "Z shares no cause with either X or Y, except for X itself, as shown in Figure 3.5.") The thought of having to express, defend, and manage formidable counterfactual relationships of this type may explain why the enterprise of causal inference is currently viewed with such awe and despair among rank-and-file epidemiologists and statisticians – and why economists and social scientists continue to use structural equations instead of the potential-outcome alternatives advocated in Holland (1988), Angrist et al. (1996), and Sobel (1998). On the other hand, the algebraic machinery offered by the potential-outcome notation, once a problem is properly formalized, can be quite powerful in refining assumptions, deriving probabilities of counterfactuals, and verifying whether conclusions follow from premises – as we demonstrate in Chapter 9. The translation given in (3.51)–(3.56) should help researchers combine the best features of the two approaches.

3.6.4 Relations to Robins's *G*-Estimation

Among the investigations conducted in the potential-outcome framework, the one closest in spirit to the structural analysis described in this chapter is Robins's work on "causally interpreted structured tree graphs" (Robins 1986, 1987). Robins was the first to realize the potential of Neyman's counterfactual notation $Y(x)$ as a general mathematical language for causal inference, and he used it to extend Rubin's (1978) "time-independent treatment" model to studies with direct and indirect effects and time-varying treatments, concomitants, and outcomes.

Robins considered a set $V = \{V_1, \ldots, V_M\}$ of temporally ordered discrete random variables (as in Figure 3.3) and asked under what conditions one can identify the effect of control policy $g : X = x$ on outcomes $Y \subseteq V \backslash X$, where $X = \{X_1, \ldots, X_K\} \subseteq V$ are

the temporally ordered and potentially manipulable treatment variables of interest. The causal effect of $X = x$ on Y was expressed as the probability

$$P(y \mid g = x) \triangleq P\{Y(x) = y\},$$

where the counterfactual variable $Y(x)$ stands for the value that outcome variables Y would take had the treatment variables X been x.

Robins showed that $P(y \mid g = x)$ is identified from the distribution $P(v)$ if each component X_k of X is "assigned at random, given the past," a notion explicated as follows. Let L_k be the variables occurring between X_{k-1} and X_k, with L_1 being the variables preceding X_1. Write $\bar{L}_k = (L_1, \ldots, L_k)$, $L = \bar{L}_K$, and $\bar{X}_k = (X_1, \ldots, X_k)$, and define $\bar{X}_0, \bar{L}_0, \bar{V}_0$ to be identically zero. The treatment $X_k = x_k$ is said to be *assigned at random, given the past,* if the following relation holds:

$$(Y(x) \perp\!\!\!\perp X_k \mid \bar{L}_k, \bar{X}_{k-1} = \bar{x}_{k-1}). \tag{3.62}$$

Robins further proved that, if (3.62) holds for every k, then the causal effect is given by

$$P(y \mid g = x) = \sum_{\bar{l}_K} P(y \mid \bar{l}_K, \bar{x}_K) \prod_{k=1}^{K} P(l_k \mid \bar{l}_{k-1}, \bar{x}_{k-1}), \tag{3.63}$$

an expression he called the "G-computation algorithm formula." This expression can be derived by applying condition (3.62) iteratively, as in the derivation of (3.54). If X is univariate, then (3.63) reduces to the standard adjustment formula

$$P(y \mid g = x) = \sum_{l_1} P(y \mid x, l_1) P(l_1),$$

paralleling (3.54). Likewise, in the special structure of Figure 3.3, (3.63) reduces to (3.18).

To place this result in the context of our analysis in this chapter, we note that the class of semi-Markovian models satisfying assumption (3.62) corresponds to complete DAGs in which all arrowheads pointing to X_k originate from observed variables. Indeed, in such models, the parents $PA_k = \bar{L}_k, \bar{X}_{k-1}$ of variable X_k satisfy the back-door condition of Definition 3.3.1,

$$(X_k \perp\!\!\!\perp Y \mid PA_k)_{G_{\underline{X}_k}},$$

which implies (3.62).[15] This class of models falls under Theorem 3.2.5, which states that all causal effects in this class are identifiable and are given by the truncated factorization formula of (3.14); the formula coincides with (3.63) after marginalizing over the uncontrolled covariates.

[15] Alternatively, (3.62) can be obtained by applying the translation rule of (3.56) to graphs with no confounding arcs between X_k and $\{Y, PA_k\}$. Note, however, that the implication goes only one way; Robins's condition is the weakest assumption needed for identifying the causal effect.

The structural analysis introduced in this chapter supports and generalizes Robins's result from a new theoretical perspective. First, on the technical front, this analysis offers systematic ways of managing models with unmeasured confounders (i.e., unobserved parents of control variables, as in Figures 3.8(d)–(g)), where Robins's starting assumption (3.62) is inapplicable. Second, on the conceptual front, the structural framework represents a fundamental shift from the vocabulary of counterfactual independencies (e.g. (3.62)) to the vocabulary of processes and mechanisms, from which human judgment of counterfactuals originates. Although expressions of counterfactual independencies can be engineered to facilitate algebraic derivations of causal effects (as in (3.54)), articulating the right independencies for a problem or assessing the assumptions behind such independencies may often be the hardest part of the problem. In the structural framework, the counterfactual expressions themselves are derived (if needed) from a mathematical theory (as in (3.56) and (3.61)). Still, Robins's pioneering research has proven (i) that algebraic methods can handle causal analysis in complex multistage problems and (ii) that causal effects in such problems can be reduced to estimable quantities (see also Sections 3.6.1 and 4.4).

Postscript

The work recounted in this chapter sprang from two simple ideas that totally changed my attitude toward causality. The first idea arose in the summer of 1990, while I was working with Tom Verma on "A Theory of Inferred Causation" (Pearl and Verma 1991; see also Chapter 2). We played around with the possibility of replacing the parents–child relationship $P(x_i \mid pa_i)$ with its functional counterpart $x_i = f_i(pa_i, u_i)$ and, suddenly, everything began to fall into place: we finally had a mathematical object to which we could attribute familiar properties of physical mechanisms instead of those slippery epistemic probabilities $P(x_i \mid pa_i)$ with which we had been working so long in the study of Bayesian networks. Danny Geiger, who was writing his dissertation at that time, asked with astonishment: "Deterministic equations? Truly deterministic?" Although we knew that deterministic structural equations have a long history in econometrics, we viewed this representation as a relic of the past. For us at UCLA in the early 1990s, the idea of putting the semantics of Bayesian networks on a deterministic foundation seemed a heresy of the worst kind.

The second simple idea came from Peter Spirtes's lecture at the International Congress of Philosophy of Science (Uppsala, Sweden, 1991). In one of his slides, Peter illustrated how a causal diagram would change when a variable is manipulated. To me, that slide of Spirtes's – when combined with the deterministic structural equations – was the key to unfolding the manipulative account of causation and led to most of the explorations described in this chapter.

I should really mention another incident that contributed to this chapter. In early 1993 I read the fierce debate between Arthur Goldberger and Nanny Wermuth on the meaning of structural equations (Goldberger 1992; Wermuth 1992). It suddenly hit me that the century-old tension between economists and statisticians stems from simple semantic confusion: Statisticians read structural equations as statements about $E(Y \mid x)$,

while economists read them as $E(Y \mid do(x))$. This would explain why statisticians claim that structural equations have no meaning and why economists retort that statistics has no substance. I wrote a technical report, "On the Statistical Interpretation of Structural Equations" (Pearl 1993c), hoping to see the two camps embrace in reconciliation. Nothing of the sort happened. The statisticians in the dispute continued to insist that anything that is not interpreted as $E(Y \mid x)$ simply lacks meaning. The economists, in contrast, are still trying to decide if it was $do(x)$ that they have been meaning to say all along.

Encouraging colleagues receive far too little credit in official channels, considering the immense impact they have on the encouraged. I must take this opportunity to acknowledge four colleagues who saw clarity shining through the $do(x)$ operator before it gained popularity: Steffen Lauritzen, David Freedman, James Robins, and Philip Dawid. Phil showed special courage in printing my paper in *Biometrika* (Pearl 1995a), the journal founded by causality's worst adversary – Karl Pearson.

CHAPTER FOUR

Actions, Plans, and Direct Effects

He whose actions exceed his wisdom,
his wisdom shall endure.
Rabbi Hanina ben Dosa
(1st century A.D.)

Preface

So far, our analysis of causal effects has focused on primitive interventions of the form $do(x)$, which stood for setting the value of variable X to a fixed constant, x, and asking for the effect of this action on the probabilities of some response variables Y. In this chapter we introduce several extensions of this analysis.

First (Section 4.1), we discuss the status of actions vis-à-vis observations in probability theory, decision analysis, and causal modeling, and we advance the thesis that the main role of causal models is to facilitate the evaluation of the effect of *novel* actions and policies that were unanticipated during the construction of the model.

In Section 4.2 we extend the identification analysis of Chapter 3 to conditional actions of the form "do x if you see z" and stochastic policies of the form "do x with probability p if you see z." We shall see that the evaluation and identification of these more elaborate interventions can be obtained from the analysis of primitive interventions. In Section 4.3, we use the intervention calculus developed in Chapter 3 to give a graphical characterization of the set of semi-Markovian models for which the causal effect of one variable on another can be identified.

We address in Section 4.4 the problem of evaluating the effect of sequential plans – namely, sequences of time-varying actions (some taken concurrently) designed to produce a certain outcome. We provide a graphical method of estimating the effect of such plans from nonexperimental observations in which some of the actions are influenced by their predecessors, some observations are influenced by the actions, and some confounding variables are unmeasured. We show that there is substantial advantage to analyzing a plan into its constituent actions rather than treating the set of actions as a single entity.

Finally, in Section 4.5 we address the question of distinguishing direct from indirect effects. We show that direct effects can be identified by the graphical method developed in Section 4.4. An example using alleged sex discrimination in college admission will serve to demonstrate the assumptions needed for proper analysis of direct effects.

4.1 INTRODUCTION

4.1.1 Actions, Acts, and Probabilities

Actions admit two interpretations: reactive and deliberative. The reactive interpretation sees action as a consequence of an agent's beliefs, disposition, and environmental inputs, as in "Adam ate the apple because Eve handed it to him." The deliberative interpretation sees action as an option of choice in contemplated decision making, usually involving comparison of consequences, as in "Adam was wondering what God would do if he ate the apple." We shall distinguish the two views by calling the first "act" and the second "action." An act is viewed from the outside, an action from the inside. Therefore, an act can be predicted and can serve as evidence for the actor's stimuli and motivations (provided the actor is part of our model). Actions, in contrast, can neither be predicted nor provide evidence since (by definition) they are pending deliberation and turn into *acts* once executed.

The confusion between actions and acts has led to Newcomb's paradox (Nozick 1969) and other oddities in the so-called evidential decision theory, which encourages decision makers to take into consideration the evidence that an action would provide, if enacted. This bizarre theory seems to have loomed from Jeffrey's influential book *The Logic of Decision* (Jeffrey 1965), in which actions are treated as ordinary events (rather than interventions) and, accordingly, the effects of actions are obtained through conditionalization rather than through a mechanism-modifying operation like $do(x)$. (See Stalnaker 1972; Gibbard and Harper 1976; Skyrms 1980; Meek and Glymour 1994; Hitchcock 1996.)

Traditional decision theory[1] instructs rational agents to choose the option x that maximizes expected utility,[2]

$$U(x) = \sum_y P(y \mid do(x))u(y),$$

where $u(y)$ is the utility of outcome y; in contrast, "evidential decision" theory calls for maximizing the conditional expectation

$$U_{\text{ev}}(x) = \sum_y P(y \mid x)u(y),$$

in which x is (improperly) treated as an observed proposition.

The paradoxes that emerge from this fallacy are obvious: patients should avoid going to the doctor "to reduce the probability that one is seriously ill" (Skyrms 1980, p. 130); workers should never hurry to work, to reduce the probability of having overslept; students

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[1] I purposely avoid the common title "causal decision theory" in order to suppress even the slightest hint that any alternative, noncausal theory can be used to guide decisions.

[2] Following a suggestion of Stalnaker (1972), Gibbard and Harper (1976) used $P(x \mathrel{\square\!\!\rightarrow} y)$ in $U(x)$, rather than $P(y \mid do(x))$, where $x \mathrel{\square\!\!\rightarrow} y$ stands for the subjunctive conditional "y if it were x." The semantics of the two operators are closely related (see Section 7.4), but the equation-removal interpretation of the $do(x)$ operator is less ambiguous and clearly suppresses inference from effect to cause.

should not prepare for exams, lest this would prove them behind in their studies; and so on. In short, all remedial actions should be banished lest they increase the probability that a remedy is indeed needed.

The oddity in this kind of logic stems from treating actions as acts that are governed by past associations instead of as objects of free choice, as dictated by the semantics of the $do(x)$ operator. This "evidential" decision theory preaches that one should never ignore genuine statistical evidence (in our case, the evidence that an act normally provides regarding whether the act is needed), but decision theory proper reminds us that actions – by their very definition – render such evidence irrelevant to the decision at hand, for actions *change* the probabilities that acts normally obey.[3]

The moral of this story can be summarized in the following mnemonic rhymes:

Whatever evidence an act might provide
On facts that preceded the act,
Should never be used to help one decide
On whether to choose that same act.

Evidential decision theory was a passing episode in the philosophical literature, and no philosopher today takes the original version of this theory seriously. Still, some recent attempts have been made to revive interest in Jeffrey's expected utility by replacing $P(y \mid x)$ with $P(y \mid x, K)$, where K stands for various background contexts, chosen to suppress spurious associations (as in (3.13)) (Price 1991; Hitchcock 1996). Such attempts echo an overly restrictive empiricist tradition, according to which rational agents live and die by one source of information – statistical associations – and hence expected utilities should admit no other operation but Bayes's conditionalization. This tradition is rapidly giving way to a more accommodating conception: rational agents should act according to theories of actions; naturally, such theories demand action-specific conditionalization (e.g. $do(x)$) while reserving Bayes's conditionalization for representing passive observations (see Goldszmidt and Pearl 1992; Meek and Glymour 1994; Woodward 1995).

In principle, actions are not part of probability theory, and understandably so: probabilities capture normal relationships in the world, whereas actions represent interventions that perturb those relationships. It is no wonder, then, that actions are treated as foreign entities throughout the literature on probability and statistics; they serve neither as arguments of probability expressions nor as events for conditioning such expressions.

Even in the statistical decision-theoretic literature (e.g. Savage 1954), where actions are the main target of analysis, the symbols given to actions serve merely as indices for distinguishing one probability function from another, not as entities that stand in logical relationships to the variables on which probabilities are defined. Savage (1954, p. 14) defined "act" as a "function attaching a consequence to each state of the world," and he treated a chain of decisions, one leading to other, as a single decision. However, the

[3] Such evidence is rendered irrelevant within the actor's own probability space; in multiagent decision situations, however, each agent should definitely be cognizant of how other agents might interpret each of his pending "would-be" acts.

logic that leads us to infer the consequences of actions and strategies from more elementary considerations is left out of the formalism. For example, consider the actions: "raise taxes," "lower taxes," and "raise interest rates." The consequences of all three actions must be specified separately, prior to analysis; none can be inferred from the others. As a result, if we are given two probabilities, P_A and P_B, denoting the probabilities prevailing under actions A or B, respectively, there is no way we can deduce from this input the probability $P_{A \wedge B}$ corresponding to the joint action $A \wedge B$ or indeed any Boolean combination of the propositions A and B. This means that, in principle, the impact of all anticipated joint actions would need to be specified in advance – an insurmountable task.

The peculiar status of actions in probability theory can be seen most clearly in comparison to the status of observations. By specifying a probability function $P(s)$ on the possible states of the world, we automatically specify how probabilities should change with every conceivable observation e, since $P(s)$ permits us to compute (by conditioning on e) the posterior probabilities $P(E \mid e)$ for every pair of events E and e. However, specifying $P(s)$ tells us nothing about how probabilities should change in response to an external action $do(A)$. In general, if an action $do(A)$ is to be described as a function that takes $P(s)$ and transforms it to $P_A(s)$, then $P(s)$ tells us nothing about the nature of $P_A(s)$, even when A is an elementary event for which $P(A)$ is well-defined (e.g., "raise the temperature by 1 degree" or "turn the sprinkler on"). With the exception of the trivial requirement that $P_A(s)$ be zero if s implies $\neg A$, a requirement that applies uniformly to every $P(s)$, probability theory does not tell us how $P_A(s)$ should differ from $P'_A(s)$, where $P'(s)$ is some other preaction probability function. Conditioning on A is clearly inadequate for capturing this transformation, as we have seen in many examples in Chapters 1 and 3 (see e.g. Section 1.3.1), because conditioning represents passive observations in an unchanging world whereas actions change the world.

Drawing analogy to visual perception, we may say that the information contained in $P(s)$ is analogous to a precise description of a three-dimensional object; it is sufficient for predicting how that object will be viewed from any angle outside the object, but it is insufficient for predicting how the object will be viewed if manipulated and squeezed by external forces. Additional information about the physical properties of the object must be supplied for making such predictions. By analogy, the additional information required for describing the transformation from $P(s)$ to $P_A(s)$ should identify those elements of the world that remain invariant under the action $do(A)$. This extra information is provided by causal knowledge, and the $do(\cdot)$ operator enables us to capture the invariant elements (thus defining $P_A(s)$) by locally modifying the graph or the structural equations. The next section will compare this device to the way actions are handled in standard decision theory.

4.1.2 Actions in Decision Analysis

Instead of introducing new operators into probability calculus, the traditional approach has been to attribute the differences between seeing and doing to differences in the total evidence available. Consider the statements: "the barometer reading was observed to be x" and "the barometer reading was set to level x." The former helps us predict the weather, the latter does not. While the evidence described in the first statement is limited

to the reading of the barometer, the second statement also tells us that the barometer was manipulated by some agent, and conditioning on this additional evidence should render the barometer reading irrelevant to predicting the rain.

The practical aspects of this approach amount to embracing the acting agents as variables in the analysis, constructing an augmented distribution function including the decisions of those agents, and inferring the effect of actions by conditioning those decision variables to particular values. Thus, for example, the agent manipulating the barometer might enter the system as a decision variable "squeezing the barometer"; after incorporating this variable into the probability distribution, we could infer the impact of manipulating the barometer simply by conditioning the augmented distribution on the event "the barometer was squeezed by force y and has reached level x."

For this conditioning method to work properly in evaluating the effect of future actions, the manipulating agent must be treated as an ideal experimenter acting out of free will, and the associated decision variables must be treated as exogenous – causally unaffected by other variables in the system. For example, if the augmented probability function encodes the fact that the current owner of the barometer tends to squeeze the barometer each time she feels arthritis pain, we will be unable to use that function for evaluating the effects of deliberate squeezing of the barometer, even by the same owner. Recalling the difference between acts and actions, whenever we set out to calculate the effect of a pending action, we must ignore all mechanisms that constrained or triggered the execution of that action in the past. Accordingly, the event "The barometer was squeezed" must enter the augmented probability function as independent of all events that occurred prior to the time of manipulation, similar to the way action variable F entered the augmented network in Figure 3.2.

This solution corresponds precisely to the way actions are treated in decision analysis, as depicted in the literature on influence diagrams (IDs) (Howard and Matheson 1981; Shachter 1986; Pearl 1988b, chap. 6). Each decision variable is represented as exogenous variable (a parentless node in the diagram), and its impact on other variables is assessed and encoded in terms of conditional probabilities, similar to the impact of any other parent node in the diagram.[4]

The difficulty with this approach is that we need to anticipate in advance, and represent explicitly, all actions whose effects we might wish to evaluate in the future. This renders the modeling process unduly cumbersome, if not totally unmanageable. In circuit diagnosis, for example, it would be awkward to represent every conceivable act of component replacement (similarly, every conceivable connection to a voltage source, current source, etc.) as a node in the diagram. Instead, the effects of such replacements are implicit in the circuit diagram itself and can be deduced from the diagram, given its causal interpretation. In econometric modeling likewise, it would be awkward to represent every conceivable variant of policy intervention as a new variable in the economic equations. Instead, the effects of such interventions can be deduced from the structural

[4] The ID literature's insistence on divorcing the links in the ID from any causal interpretation (Howard and Matheson 1981; Howard 1990) is at odds with prevailing practice. The causal interpretation is what allows us to treat decision variables as root nodes, unassociated with all other nodes (except their descendants).

interpretation of those equations, if only we can tie the immediate effects of each policy to the corresponding variables and parameters in the equations. The compound action "raise taxes and lower interest rates," for example, need not be introduced as a new variable in the equations, because the effect of that action can be deduced if we have the quantities "taxation level" and "interest rates" already represented as (either exogenous or endogenous) variables in the equations.

The ability to predict the effect of interventions without enumerating those interventions in advance is one of the main advantages we draw from causal modeling and one of the main functions served by the notion of causation. Since the number of actions or action combinations is enormous, they cannot be represented explicitly in the model but rather must be indexed by the propositions that each action enforces directly. Indirect consequences of enforcing those propositions are then inferred from the causal relationships among the variables represented in the model. We will return to this theme in Chapter 7 (Section 7.2.4), where we further explore the invariance assumptions that must be met for this encoding scheme to work.

4.1.3 Actions and Counterfactuals

As an alternative to Bayesian conditioning, philosophers (Lewis 1976; Gardenfors 1988) have studied another probability transformation called "imaging," which was deemed useful in the analysis of subjunctive conditionals and which more adequately represents the transformations associated with actions. Whereas Bayes conditioning of $P(s \mid e)$ transfers the entire probability mass from states excluded by e to the remaining states (in proportion to their current probabilities, $P(s)$), imaging works differently: each excluded state s transfers its mass individually to a select set of states $S^*(s)$ that are considered to be "closest" to s (see Section 7.4.3). Although providing a more adequate and general framework for actions (Gibbard and Harper 1976), imaging leaves the precise specification of the selection function $S^*(s)$ almost unconstrained. Consequently, the problem of enumerating future actions is replaced by the problem of encoding distances among states in a way that would be both economical and respectful of common understanding of the causal laws that operate in the domain. The second requirement is not trivial, considering that indirect ramifications of actions often result in worlds that are quite dissimilar to the one from which we start (Fine 1975).

The difficulties associated with making the closest-world approach conform to causal laws will be further elaborated in Chapter 7 (Section 7.4). The structural approach pursued in this book escapes these difficulties by basing the notion of interventions directly on causal mechanisms and by capitalizing on the properties of invariance and autonomy that accompany these mechanisms. This mechanism-modification approach can be viewed as a special instance of the closest-world approach, where the closeness measure is crafted so as to respect the causal mechanisms in the domain; the selection function $S^*(s)$ that ensues is represented in (3.11) (see discussion that follows).

The operationality of this mechanism-modification semantics was demonstrated in Chapter 3 and led to the quantitative predictions of the effects of actions, including actions that were not contemplated during the model's construction. The *do* calculus that

emerged (Theorem 3.4.1) extends this prediction facility to cases where some of the variables are unobserved. In Chapter 7 we further use the mechanism-modification interpretation to provide semantics for counterfactual statements, as outlined in Section 1.1.4. In this chapter, we will extend the applications of the *do* calculus in several directions, as outlined in the Preface.

4.2 CONDITIONAL ACTIONS AND STOCHASTIC POLICIES

The interventions considered in our analysis of identification (Sections 3.3–3.4) were limited to actions that merely force a variable or a group of variables X to take on some specified value x. In general (see the process control example in Section 3.2.3), interventions may involve complex policies in which a variable X is made to respond in a specified way to some set Z of other variables – say, through a functional relationship $x = g(z)$ or through a stochastic relationship whereby X is set to x with probability $P^*(x \mid z)$. We will show, based on Pearl (1994b), that identifying the effect of such policies is equivalent to identifying the expression $P(y \mid \hat{x}, z)$.

Let $P(y \mid do(X = g(z)))$ stand for the distribution (of Y) prevailing under the policy $do(X = g(z))$. To compute $P(y \mid do(X = g(z)))$, we condition on Z and write

$$\begin{aligned} P(y \mid do(X = g(z))) &= \sum_z P(y \mid do(X = g(z)), z) P(z \mid do(X = g(z))) \\ &= \sum_z P(y \mid \hat{x}, z)|_{x=g(z)} P(z) \\ &= E_z[P(y \mid \hat{x}, z)|_{x=g(z)}]. \end{aligned}$$

The equality

$$P(z \mid do(X = g(z))) = P(z)$$

stems, of course, from the fact that Z cannot be a descendant of X; hence, any control exerted on X can have no effect on the distribution of Z. Thus, we see that the causal effect of a policy $do(X = g(z))$ can be evaluated directly from the expression of $P(y \mid \hat{x}, z)$ simply by substituting $g(z)$ for x and taking the expectation over Z (using the observed distribution $P(z)$).

This identifiability criterion for conditional policy is somewhat stricter than that for unconditional intervention. Clearly, if a policy $do(X = g(z))$ is identifiable then the simple intervention $do(X = x)$ is identifiable as well, since we can always obtain the latter by setting $g(z) = x$. The converse does not hold, however, because conditioning on Z might create dependencies that will prevent the successful reduction of $P(y \mid \hat{x}, z)$ to a hat-free expression.

A stochastic policy, which imposes a new conditional distribution $P^*(x \mid z)$ for x, can be handled in a similar manner. We regard the stochastic intervention as a random process in which the unconditional intervention $do(X = x)$ is enforced with probability $P^*(x \mid z)$. Thus, given $Z = z$, the intervention $do(X = x)$ will occur with probability

$P^*(x \mid z)$ and will produce a causal effect given by $P(y \mid \hat{x}, z)$. Averaging over x and z gives the effect (on Y) of the stochastic policy $P^*(x \mid z)$:

$$P(y)|_{P^*(x|z)} = \sum_x \sum_z P(y \mid \hat{x}, z) P^*(x \mid z) P(z).$$

Because $P^*(x \mid z)$ is specified externally, we see again that the identifiability of $P(y \mid \hat{x}, z)$ is a necessary and sufficient condition for the identifiability of any stochastic policy that shapes the distribution of X by the outcome of Z.

Of special importance in planning is a STRIPS-like action (Fikes and Nilsson 1971) whose immediate effects $X = x$ depend on the satisfaction of some enabling precondition $C(w)$ on a set W of variables. To represent such actions, we let $Z = W \cup PA_X$ and set

$$P^*(x \mid z) = \begin{cases} P(x \mid pa_X) & \text{if } C(w) = \text{false}, \\ 1 & \text{if } C(w) = \text{true and } X = x, \\ 0 & \text{if } C(w) = \text{true and } X \neq x. \end{cases}$$

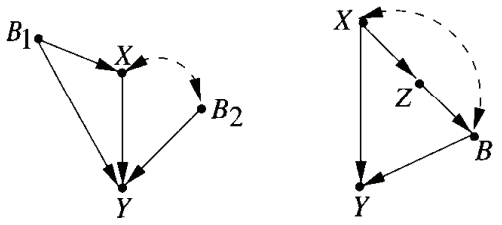
4.3 WHEN IS THE EFFECT OF AN ACTION IDENTIFIABLE?

In Chapter 3 we developed several graphical criteria for recognizing when the effect of one variable on another, $P(y \mid do(x))$, is identifiable in the presence of unmeasured variables. These criteria, like the back-door (Theorem 3.3.2) and front-door (Theorem 3.3.4), are special cases of a more general class of semi-Markovian models for which repeated application of the inference rules of *do* calculus (Theorem 3.4.1) will reduce $P(y \mid \hat{x})$ to a hat-free expression, thus rendering it identifiable. The semi-Markovian model of Figure 3.1 (or Figure 3.8(f)) is an example where direct application of either the back-door or front-door criterion would not be sufficient for identifying $P(y \mid \hat{x})$ and yet the expression is reducible (hence identifiable) by a sequence of inference rules of Theorem 3.4.1. In this section we establish a complete characterization of the class of models in which the causal effect $P(y \mid \hat{x})$ is identifiable in *do* calculus.

4.3.1 Graphical Conditions for Identification

Theorem 4.3.1 characterizes the class of "*do*-identifiable" models in the form of four graphical conditions, any one of which is sufficient for the identification of $P(y \mid \hat{x})$ when X and Y are singleton nodes in the graph. Theorem 4.3.2 then asserts the completeness (or necessity) of these four conditions; one of which must hold in the model for $P(y \mid \hat{x})$ to be identifiable in *do* calculus. Whether these four conditions are necessary in general (in accordance with the semantics of Definition 3.2.4) depends on whether the inference rules of *do* calculus are complete. This question, to the best of my knowledge, is still open.

Theorem 4.3.1 (Galles and Pearl 1995)
Let X and Y denote two singleton variables in a semi-Markovian model characterized by graph G. A sufficient condition for the identifiability of $P(y \mid \hat{x})$ is that G satisfy one of the following four conditions.



(a) (b)

Figure 4.1 Condition 3 of Theorem 4.3.1. In (a), the set $\{B_1, B_2\}$ blocks all back-door paths from X to Y, and $P(b_1, b_2 \mid \hat{x}) = P(b_1, b_2)$. In (b), the node B blocks all back-door paths from X to Y, and $P(b \mid \hat{x})$ is identifiable using Condition 4.

1. *There is no back-door path from X to Y in G; that is,* $(X \perp\!\!\!\perp Y)_{G_{\underline{X}}}$.
2. *There is no directed path from X to Y in G.*
3. *There exists a set of nodes B that blocks all back-door paths from X to Y so that $P(b \mid \hat{x})$ is identifiable.* (A special case of this condition occurs when B consists entirely of nondescendants of X, in which case $P(b \mid \hat{x})$ reduces immediately to $P(b)$.)
4. *There exist sets of nodes Z_1 and Z_2 such that:*
   (i) *Z_1 blocks every directed path from X to Y (i.e.,* $(Y \perp\!\!\!\perp X \mid Z_1)_{G_{\overline{Z_1}\underline{X}}}$*);*
   (ii) *Z_2 blocks all back-door paths between Z_1 and Y (i.e.,* $(Y \perp\!\!\!\perp Z_1 \mid Z_2)_{G_{\overline{X}\underline{Z_1}}}$*);*
   (iii) *Z_2 blocks all back-door paths between X and Z_1 (i.e.,* $(X \perp\!\!\!\perp Z_1 \mid Z_2)_{G_{\underline{X}}}$*; and*
   (iv) *Z_2 does not activate any back-door paths from X to Y (i.e.,* $(X \perp\!\!\!\perp Y \mid Z_1, Z_2)_{G_{\overline{Z_1 X(Z_2)}}}$*).* (This condition holds if (i)–(iii) are met and no member of Z_2 is a descendant of X.)

   (A special case of condition 4 occurs when $Z_2 = \emptyset$ and there is no back-door path from X to Z_1 or from Z_1 to Y.)

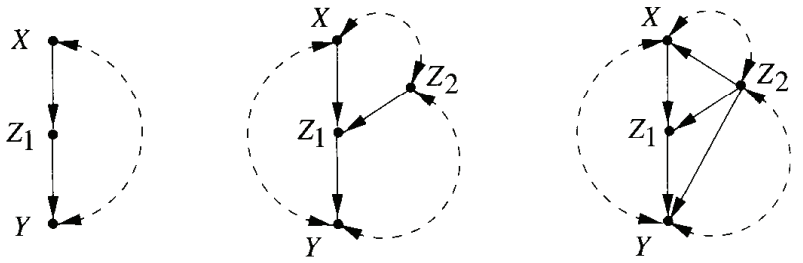
Proof

Condition 1. This condition follows directly from Rule 2 (see Theorem 3.4.1). If $(Y \perp\!\!\!\perp X)_{G_{\underline{X}}}$ then we can immediately change $P(y \mid \hat{x})$ to $P(y \mid x)$, so the query is identifiable.

Condition 2. If there is no directed path from X to Y in G, then $(Y \perp\!\!\!\perp X)_{G_{\overline{X}}}$. Hence, by Rule 3, $P(y \mid \hat{x}) = P(y)$ and so the query is identifiable.

Condition 3. If there is a set of nodes B that blocks all back-door paths from X to Y (i.e., $(Y \perp\!\!\!\perp X \mid B)_{G_{\underline{X}}}$), then we can expand $P(y \mid \hat{x})$ as $\sum_b P(y \mid \hat{x}, b)P(b \mid \hat{x})$ and, by Rule 2, rewrite $P(y \mid \hat{x}, b)$ as $P(y \mid x, b)$. If the query $(b \mid \hat{x})$ is identifiable, then the original query must also be identifiable. See examples in Figure 4.1.

Condition 4. If there is a set of nodes Z_1 that block all directed paths from X to Y and a set of nodes Z_2 that block all back-door paths between Y and Z_1 in $G_{\overline{X}}$, then we expand $P(y \mid \hat{x}) = \sum_{z_1, z_2} P(y \mid \hat{x}, z_1, z_2)P(z_1, z_2 \mid \hat{x})$ and rewrite $P(y \mid \hat{x}, z_1, z_2)$ as $P(y \mid \hat{x}, \hat{z}_1, z_2)$ using Rule 2, since all back-door paths between Z_1 and Y are blocked by Z_2 in $G_{\overline{X}}$. We can reduce $P(y \mid \hat{x}, \hat{z}_1, z_2)$ to $P(y \mid \hat{z}_1, z_2)$ using Rule 3, since $(Y \perp\!\!\!\perp X \mid Z_1, Z_2)_{G_{\overline{Z_1 X(Z_2)}}}$. We can rewrite $P(y \mid \hat{z}_1, z_2)$ as $P(y \mid z_1, z_2)$ if $(Y \perp\!\!\!\perp Z_1 \mid Z_2)_{G_{\underline{Z_1}}}$. The only way that this independence cannot hold is if there is a path from Y to Z_1 through X, since $(Y \perp\!\!\!\perp Z_1 \mid Z_2)_{G_{\overline{X}\underline{Z_1}}}$. However, we can block this path by conditioning and



(a) (b) (c)

Figure 4.2 Condition 4 of Theorem 4.3.1. In (a), Z_1 blocks all directed paths from X to Y, and empty set blocks all back-door paths from Z_1 to Y in $G_{\overline{X}}$ and all back-door paths from X to Z_1 in In (b) and (c), Z_1 blocks all directed paths from X to Y, and Z_2 blocks all back-door paths from to Y in $G_{\overline{X}}$ and all back-door paths from X to Z_1 in G.

summing over X and so derive $\sum_{x'} P(y \mid \hat{z}_1, z_2, x')P(x' \mid \hat{z}_1, z_2)$. Now we can rewr $P(y \mid \hat{z}_1, z_2, x')$ as $P(y \mid z_1, z_2, x')$ using Rule 2. The $P(x' \mid \hat{z}_1, z_2)$ term can be rew ten as $P(x' \mid z_2)$ using Rule 3, since Z_1 is a child of X and the graph is acyclic. The qu can therefore be rewritten as $\sum_{z_1,z_2} \sum_{x'} P(y \mid z_1, z_2, x')P(x' \mid z_2)P(z_1, z_2 \mid \hat{x})$, we have $P(z_1, z_2 \mid \hat{x}) = P(z_2 \mid \hat{x})P(z_1 \mid \hat{x}, z_2)$. Since Z_2 consists of nondescendants X, we can rewrite $P(z_2 \mid \hat{x})$ as $P(z_2)$ using Rule 3. Since Z_2 blocks all back-door pa from X to Z_1, we can rewrite $P(z_1 \mid \hat{x}, z_2)$ as $P(z_1 \mid x, z_2)$ using Rule 2. The ent query can thus be rewritten as $\sum_{z_1,z_2} \sum_{x'} P(y \mid z_1, z_2, x')P(x' \mid z_2)P(z_1 \mid x, z_2)P(z$ See examples in Figure 4.2.

Theorem 4.3.2

The four conditions of Theorem 4.3.1 are necessary for identifiability in do *calculus. Th is, if all four conditions of Theorem 4.3.1 fail in a graph G, then there exists no fin sequence of inference rules that reduces* $P(y \mid \hat{x})$ *to a hat-free expression.*

A proof of Theorem 4.3.2 is given in Galles and Pearl (1995).

4.3.2 Remarks on Efficiency

In implementing Theorem 4.3.1 as a systematic method for determining identifiabili Conditions 3 and 4 would seem to require exhaustive search. In order to prove that Co dition 3 does not hold, for instance, we need to prove that no such blocking set B c exist. Fortunately, the following theorems allow us to significantly prune the search spa so as to render the test tractable.

Theorem 4.3.3

If $P(b_i \mid \hat{x})$ *is identifiable for one minimal set* B_i*, then* $P(b_j \mid \hat{x})$ *is identifiable for a other minimal set* B_j*.*

Theorem 4.3.3 allows us to test Condition 3 with a single minimal blocking set B. If meets the requirements of Condition 3 then the query is identifiable; otherwise, Con tion 3 cannot be satisfied. In proving this theorem, we use the following lemma.

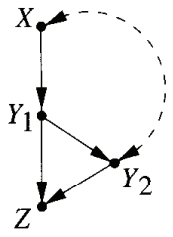


Figure 4.3 Theorem 4.3.1 ensures a reducing sequence for $P(y_2 \mid \hat{x}, y_1)$ and $P(y_1 \mid \hat{x})$, although none exists for $P(y_1 \mid \hat{x}, y_2)$.

Lemma 4.3.4

If the query $P(y \mid \hat{x})$ is identifiable and if a set of nodes Z lies on a directed path from X to Y, then the query $P(z \mid \hat{x})$ is identifiable.

Theorem 4.3.5

Let Y_1 and Y_2 be two subsets of nodes such that either (i) *no nodes Y_1 are descendants of X or* (ii) *all nodes Y_1 and Y_2 are descendants of X and all nodes Y_1 are nondescendants of Y_2. A reducing sequence for $P(y_1, y_2 \mid \hat{x})$ exists (per Corollary 3.4.2) if and only if there are reducing sequences for both $P(y_1 \mid \hat{x})$ and $P(y_2 \mid \hat{x}, y_1)$.*

The probability $P(y_1, y_2 \mid \hat{x})$ might pass the test in Theorem 4.3.1 if we apply the procedure to both $P(y_2 \mid \hat{x}, y_1)$ and $P(y_1 \mid \hat{x})$, but if we try to apply the test to $P(y_1 \mid \hat{x}, y_2)$ then we will not find a reducing sequence of rules. Figure 4.3 shows just such an example. Theorem 4.3.5 guarantees that, if there is a reducing sequence for $P(y_1, y_2 \mid \hat{x})$, then we should always be able to find such a sequence for both $P(y_1 \mid \hat{x})$ and $P(y_2 \mid \hat{x}, y_1)$ by proper choice of Y_1.

Theorem 4.3.6

If there exists a set Z_1 that meets all of the requirements for Z_1 in Condition 4, then the set consisting of the children of X intersected with the ancestors of Y will also meet all of the requirements for Z_1 in Condition 4.

Theorem 4.3.6 removes the need to search for Z_1 in Condition 4 of Theorem 4.3.1. Proofs of Theorems 4.3.3–4.3.6 are given in Galles and Pearl (1995).

4.3.3 Deriving a Closed-Form Expression for Control Queries

The algorithm defined by Theorem 4.3.1 not only determines the identifiability of a control query but also provides a closed-form expression for $P(y \mid \hat{x})$ in terms of the observed probability distribution (when such a closed form exists) as follows.

Function: ClosedForm$(P(y \mid \hat{x}))$.

Input: Control query of the form $P(y \mid \hat{x})$.

Output: Either a closed-form expression for $P(y \mid \hat{x})$, in terms of observed variables only, or FAIL when the query is not identifiable.

1. If $(X \perp\!\!\!\perp Y)_{G_{\overline{X}}}$ then return $P(y)$.
2. Otherwise, if $(X \perp\!\!\!\perp Y)_{G_{\underline{X}}}$ then return $P(y \mid x)$.

3. Otherwise, let $B = \text{BlockingSet}(X, Y)$ and $Pb = \text{ClosedForm}(b \mid \hat{x})$; if $Pb \neq$ FAIL then return $\sum_b P(y \mid b, x) * Pb$.
4. Otherwise, let $Z_1 = \text{Children}(X) \cap (Y \cup \text{Ancestors}(Y))$, $Z_3 = \text{BlockingSet}(X, Z_1)$, $Z_4 = \text{BlockingSet}(Z_1, Y)$, and $Z_2 = Z_3 \cup Z_4$; if $Y \notin Z_1$ and $X \notin Z_2$ then return $\sum_{z_1, z_2} \sum_{x'} P(y \mid z_1, z_2, x')P(x' \mid z_2)P(z_1 \mid x, z_2)P(z_2)$.
5. Otherwise, return FAIL.

Steps 3 and 4 invoke the function $\text{BlockingSet}(X, Y)$, which selects a set of nodes Z that d-separate X from Y. Such sets can be found in polynomial time (Tian et al. 1998). Step 3 contains a recursive call to the algorithm $\text{ClosedForm}(b \mid \hat{x})$ itself, in order to obtain an expression for causal effect $P(b \mid \hat{x})$.

4.3.4 Summary

The conditions of Theorem 4.3.1 sharply delineate the boundary between the class of identifying models (such as those depicted in Figure 3.8) and nonidentifying models (Figure 3.9). These conditions lead to an effective algorithm for determining the identifiability of control queries of the type $P(y \mid \hat{x})$, where X is a single variable. Such queries are identifiable in *do* calculus if and only if they meet the conditions of Theorem 4.3.1. The algorithm further gives a closed-form expression for the causal effect $P(y \mid \hat{x})$ in terms of estimable probabilities.

Applications to causal analysis of nonexperimental data in the social and medical sciences are discussed in Chapter 3 and further elaborated in Chapters 5 and 6. In Chapter 9 (Corollary 9.2.17) we will apply these results to problems of *causal attribution,* that is, to estimate the probability that a specific observation (e.g., a disease case) is causally attributable to a given event (e.g., exposure).

4.4 THE IDENTIFICATION OF PLANS

This section, based on Pearl and Robins (1995), concerns the probabilistic evaluation of plans in the presence of unmeasured variables, where each plan consists of several concurrent or sequential actions and each action may be influenced by its predecessors in the plan. We establish a graphical criterion for recognizing when the effects of a given plan can be predicted from passive observations on measured variables only. When the criterion is satisfied, a closed-form expression is provided for the probability that the plan will achieve a specified goal.

4.4.1 Motivation

To motivate the discussion, consider an example discussed in Robins (1993, apx. 2), as depicted in Figure 4.4. The variables X_1 and X_2 stand for treatments that physicians prescribe to a patient at two different times, Z represents observations that the second physician consults to determine X_2, and Y represents the patient's survival. The hidden variables U_1 and U_2 represent, respectively, part of the patient's history and the patient's disposition

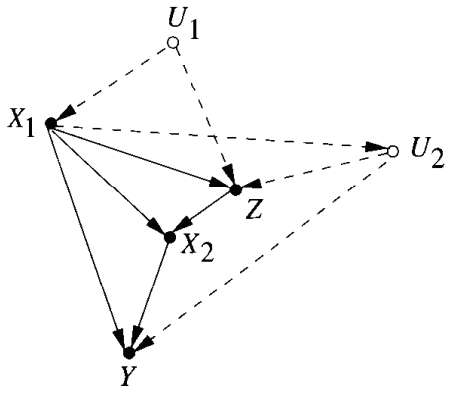


Figure 4.4 The problem of evaluating the effect of the plan $(do(x_1), do(x_2))$ on Y, from nonexperimental data taken on X_1, Z, X_2, and Y.

to recover. A simple realization of such structure could be found among AIDS patients, where Z represents episodes of PCP. This is a common opportunistic infection of AIDS patients that (as the diagram shows) does not have a direct effect on survival Y because it can be treated effectively, but it is an indicator of the patient's underlying immune status (U_2), which can cause death. The terms X_1 and X_2 stand for bactrim, a drug that prevents PCP (Z) and may also prevent death by other mechanisms. Doctors used the patient's earlier PCP history (U_1) to prescribe X_1, but its value was not recorded for data analysis.

The problem we face is as follows. Assume we have collected a large amount of data on the behavior of many patients and physicians, which is summarized in the form of (an estimated) joint distribution P of the observed four variables (X_1, Z, X_2, Y). A new patient comes in, and we wish to determine the impact of the (unconditional) plan $(do(x_1), do(x_2))$ on survival, where x_1 and x_2 are two predetermined dosages of bactrim to be administered at two prespecified times.

In general, our problem amounts to that of evaluating a new plan by watching the performance of other planners whose decision strategies are indiscernible. Physicians do not provide a description of all inputs that prompted them to prescribe a given treatment; all they communicate to us is that U_1 was consulted in determining X_1 and that Z and X_1 were consulted in determining X_2. But U_1, unfortunately, was not recorded. In epidemiology, the plan evaluation problem is known as "time-varying treatment with time-varying confounders" (Robins 1993). In artificial intelligence applications, the evaluation of such plans enables one agent to learn to act by observing the performance of another agent, even in cases where the actions of the other agent are predicated on factors that are not visible to the learner. If the learner is permitted to act as well as observe, then the task becomes much easier: the topology of the causal diagram could also be inferred (at least partially), and the effects of some previously unidentifiable actions could be determined.

As in the identification of actions (Section 4.3), the main problem in plan identification is the control of "confounders," that is, unobserved factors that trigger actions and simultaneously affect the response. However, unlike the problem treated in Section 4.3, plan identification is further complicated by the fact that some of the confounders (e.g. Z) are affected by control variables. As remarked in Chapter 3, one of the deadliest sins in the design of statistical experiments (Cox 1958, p. 48) is to adjust for such variables, because the adjustment would simulate holding a variable constant; holding constant a variable that stands between an action and its consequence interferes with the very quantity we wish estimate – the total effect of that action.

Two other features of Figure 4.4 are worth noting. First, the quantity $P(y \mid \hat{x}_1, \hat{x}_2)$ cannot be computed if we treat the control variables X_1 and X_2 as a single compound variable X. The graph corresponding to such compounding would depict X as connected to Y by both an arrow and a curved arc (through U) and thus would form a bow pattern (see Figure 3.9), which is indicative of nonidentifiability. Second, the causal effect $P(y \mid \hat{x}_1)$ in isolation is not identifiable because U_1 creates a bow pattern around the link $X \rightarrow Z$, which lies on a directed path from X to Y (see the discussion in Section 3.5).

The feature that facilitates the identifiability of $P(y \mid \hat{x}_1, \hat{x}_2)$ is the identifiability of $P(y \mid x_1, z, \hat{x}_2)$ – the causal effect of the action $do(X_2 = x_2)$ alone, conditioned on the observations available at the time of this action. This can be verified using the back-door criterion, observing that $\{X_1, Z\}$ blocks all back-door paths between X_2 and Y. Thus, the identifiability of $P(y \mid \hat{x}_1, \hat{x}_2)$ can be readily proven by writing

$$P(y \mid \hat{x}_1, \hat{x}_2) = P(y \mid x_1, \hat{x}_2) \tag{4.1}$$

$$= \sum_z P(y \mid z, x_1, \hat{x}_2) P(z \mid x_1) \tag{4.2}$$

$$= \sum_z P(y \mid z, x_1, x_2) P(z \mid x_1), \tag{4.3}$$

where (4.1) and (4.3) follow from Rule 2, and (4.2) follows from Rule 3. The subgraphs that permit the application of these rules are shown in Figure 4.5 (in Section 4.4.3).

This derivation also highlights how conditional plans can be evaluated. Assume we wish to evaluate the effect of the plan $\{do(X_1 = x_1), do(X_2 = g(x_1, z))\}$. Following the analysis of Section 4.2, we write

$$\begin{aligned} P(y \mid do(X_1 = x_1), do(X_2 = g(x_1, z))) &= P(y \mid x_1, do(X_2 = g(x_1, z))) \\ &= \sum_z P(y \mid z, x_1, do(X_2 = g(x_1, z))) P(z \mid x_1) \\ &= \sum_z P(y \mid z, x_1, x_2) P(z \mid x_1)|_{x_2 = g(x_1, z)}. \end{aligned}$$

Again, the identifiability of this conditional plan rests on the identifiability of the expression $P(y \mid z, x_1, \hat{x}_2)$, which reduces to $P(y \mid z, x_1, x_2)$ because $\{X_1, Z\}$ blocks all back-door paths between X_2 and Y.

The criterion developed in the next section will enable us to recognize in general, by graphical means, whether a proposed plan can be evaluated from the joint distribution on the observables and, if so, to identify which covariates should be measured and how they should be adjusted.

4.4.2 Plan Identification: Notation and Assumptions

Our starting point is a knowledge specification scheme in the form of a causal diagram, like the one shown in Figure 4.4, that provides a qualitative summary of the analyst's understanding of the relevant data-generating processes.[5]

[5] An alternative specification scheme using counterfactual statements was developed by Robins (1986, 1987), as described in Section 3.6.4.

Notation

A *control problem* consists of a directed acyclic graph (DAG) G with vertex set V, partitioned into four disjoint sets $V = \{X, Z, U, Y\}$, where

X = the set of control variables (exposures, interventions, treatments, etc.);

Z = the set of observed variables, often called *covariates*;

U = the set of unobserved (latent) variables; and

Y = an outcome variable.

We let the control variables be ordered $X = X_1, X_2, \ldots, X_n$ such that every X_k is a nondescendant of X_{k+j} ($j > 0$) in G, and we let the outcome Y be a descendant of X_n. Let N_k stand for the set of observed nodes that are nondescendants of any element in the set $\{X_k, X_{k+1}, \ldots, X_n\}$. A *plan* is an ordered sequence $(\hat{x}_1, \hat{x}_2, \ldots, \hat{x}_n)$ of value assignments to the control variables, where $\hat{x}_k$ means "X_k is set to x_k." A *conditional plan* is an ordered sequence $(\hat{g}_1(z_1), \hat{g}_2(z_2), \ldots, \hat{g}_n(z_n))$, where each g_k is a function from a set Z_k to X_k and where $\hat{g}_k(z_k)$ stands for the statement "set X_k to $g_k(z_k)$ whenever Z_k attains the value z_k." The support Z_k of each $g_k(z_k)$ function must not contain any variables that are descendants of X_k in G.

Our problem is to *evaluate* an unconditional plan[6] by computing $P(y \mid \hat{x}_1, \hat{x}_2, \ldots, \hat{x}_n)$, which represents the impact of the plan $(\hat{x}_1, \ldots, \hat{x}_n)$ on the outcome variable Y. The expression $P(y \mid \hat{x}_1, \hat{x}_2, \ldots, \hat{x}_n)$ is said to be *identifiable* in G if, for every assignment $(\hat{x}_1, \hat{x}_2, \ldots, \hat{x}_n)$, the expression can be determined uniquely from the joint distribution of the observables $\{X, Y, Z\}$. A control problem is identifiable whenever $P(y \mid \hat{x}_1, \hat{x}_2, \ldots, \hat{x}_n)$ is identifiable.

Our main identifiability criteria are presented in Theorems 4.41 and 4.4.6. These invoke d-separation tests on various subgraphs of G, defined in the same manner as in Section 4.3. We denote by $G_{\overline{X}}$ (and $G_{\underline{X}}$, respectively) the graphs obtained by deleting from G all arrows pointing to (emerging from) nodes in X. To represent the deletion of both incoming and outgoing arrows, we use the notation $G_{\overline{X}\underline{Z}}$. Finally, the expression $P(y \mid \hat{x}, z) \triangleq P(y, z \mid \hat{x})/P(z \mid \hat{x})$ stands for the probability of $Y = y$ given that $Z = z$ is observed and X is held constant at x.

4.4.3 Plan Identification: A General Criterion

Theorem 4.4.1 (Pearl and Robins 1995)
The probability $P(y \mid \hat{x}_1, \ldots, \hat{x}_n)$ is identifiable if, for every $1 \leq k \leq n$, there exists a set Z_k of covariates satisfying

$$Z_k \subseteq N_k, \tag{4.4}$$

(i.e., Z_k consists of nondescendants of $\{X_k, X_{k+1}, \ldots, X_n\}$) and

[6] Identification of conditional plans can be obtained from Theorem 4.4.1 using the method described in Section 4.2 and exemplified in Section 4.4.1.

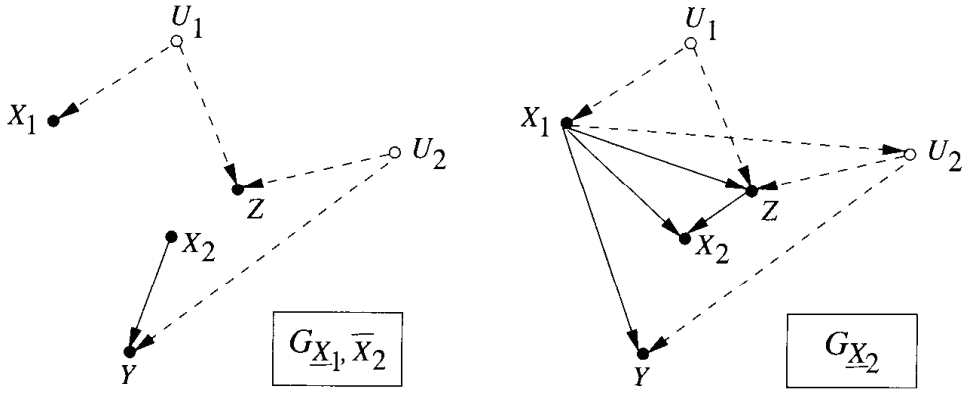


Figure 4.5 The two subgraphs of G used in testing the identifiability of the plan $(\hat{x}_1, \hat{x}_2)$ in Figure 4.4.

$$(Y \perp\!\!\!\perp X_k \mid X_1, \ldots, X_{k-1}, Z_1, Z_2, \ldots, Z_k)_{G_{\underline{X}_k, \overline{X}_{k+1}, \ldots, \overline{X}_n}}. \tag{4.5}$$

When these conditions are satisfied, the effect of the plan is given by

$$P(y \mid \hat{x}_1, \ldots, \hat{x}_n) = \sum_{z_1, \ldots, z_n} P(y \mid z_1, \ldots, z_n, x_1, \ldots, x_n) \times \prod_{k=1}^{n} P(z_k \mid z_1, \ldots, z_{k-1}, x_1, \ldots, x_{k-1}). \tag{4.6}$$

Before presenting its proof, let us demonstrate how Theorem 4.4.1 can be used to test the identifiability of the control problem shown in Figure 4.4. First, we will show that $P(y \mid \hat{x}_1, \hat{x}_2)$ cannot be identified without measuring Z; in other words, that the sequence $Z_1 = \emptyset$, $Z_2 = \emptyset$ would not satisfy conditions (4.4)–(4.5). The two d-separation tests encoded in (4.5) are

$$(Y \perp\!\!\!\perp X_1)_{G_{\underline{X}_1, \overline{X}_2}} \quad \text{and} \quad (Y \perp\!\!\!\perp X_2 \mid X_1)_{G_{\underline{X}_2}}.$$

The two subgraphs associated with these tests are shown in Figure 4.5. We see that $(Y \perp\!\!\!\perp X_1)$ holds in $G_{\underline{X}_1, \overline{X}_2}$ but that $(Y \perp\!\!\!\perp X_2 \mid X_1)$ fails to hold in $G_{\underline{X}_2}$. Thus, in order to pass the test, we must have either $Z_1 = \{Z\}$ or $Z_2 = \{Z\}$; since Z is a descendant of X_1, only the second alternative satisfies (4.4). The tests applicable to the sequence $Z_1 = \emptyset$, $Z_2 = \{Z\}$ are $(Y \perp\!\!\!\perp X_1)_{G_{\underline{X}_1, \overline{X}_2}}$ and $(Y \perp\!\!\!\perp X_2 \mid X_1, Z)_{G_{\underline{X}_2}}$. Figure 4.5 shows that both tests are now satisfied, because $\{X_1, Z\}$ d-separates Y from X_2 in $G_{\underline{X}_2}$. Having satisfied conditions (4.4)–(4.5), equation (4.6) provides a formula for the effect of plan $(\hat{x}_1, \hat{x}_2)$ on Y:

$$P(y \mid \hat{x}_1, \hat{x}_2) = \sum_{z} P(y \mid z, x_1, x_2) P(z \mid x_1), \tag{4.7}$$

which coincides with (4.3).

The question naturally arises of whether the sequence $Z_1 = \emptyset$, $Z_2 = \{Z\}$ can be identified without exhaustive search. This question will be answered in Corollary 4.4.5 and Theorem 4.4.6.

Proof of Theorem 4.4.1

The proof given here is based on the inference rules of *do* calculus (Theorem 3.4.1), which facilitate the reduction of causal effect formulas to hat-free expressions. An alternative proof, using latent variable elimination, is given in Pearl and Robins (1995).

Step 1. The condition $Z_k \subseteq N_k$ implies $Z_k \subseteq N_j$ for all $j \geq k$. Therefore, we have

$$P(z_k \mid z_1, \ldots, z_{k-1}, x_1, \ldots, x_{k-1}, \hat{x}_k, \hat{x}_{k+1}, \ldots, \hat{x}_n) = P(z_k \mid z_1, \ldots, z_{k-1}, x_1, \ldots, x_{k-1}).$$

This is so because no node in $\{Z_1, \ldots, Z_k, X_1, \ldots, X_{k-1}\}$ can be a descendant of any node in $\{X_k, \ldots, X_n\}$. Hence, Rule 3 allows us to delete the hat variables from the expression.

Step 2. The condition in (4.5) permits us to invoke Rule 2 and write:

$$P(y \mid z_1, \ldots, z_k, x_1, \ldots, x_{k-1}, \hat{x}_k, \hat{x}_{k+1}, \ldots, \hat{x}_n) = P(y \mid z_1, \ldots, z_k, x_1, \ldots, x_{k-1}, x_k, \hat{x}_{k+1}, \ldots, \hat{x}_n).$$

Thus, we have

$$\begin{aligned}
P(y \mid \hat{x}_1, \ldots, \hat{x}_n) &= \sum_{z_1} P(y \mid z_1, \hat{x}_1, \hat{x}_2, \ldots, \hat{x}_n) P(z_1 \mid \hat{x}_1, \ldots, \hat{x}_n) \\
&= \sum_{z_1} P(y \mid z_1, x_1, \hat{x}_2, \ldots, \hat{x}_n) P(z_1) \\
&= \sum_{z_2} \sum_{z_1} P(y \mid z_1, z_2, x_1, \hat{x}_2, \ldots, \hat{x}_n) P(z_1) P(z_2 \mid z_1, x_1, \hat{x}_2, \ldots, \hat{x}_n) \\
&= \sum_{z_2} \sum_{z_1} P(y \mid z_1, z_2, x_1, x_2, \hat{x}_3, \ldots, \hat{x}_n) P(z_1) P(z_2 \mid z_1, x_1) \\
&\vdots \\
&= \sum_{z_n} \cdots \sum_{z_2} \sum_{z_1} P(y \mid z_1, \ldots, z_n, x_1, \ldots, x_n) \\
&\quad \times P(z_1) P(z_2 \mid z_1, x_1) \cdots P(z_n \mid z_1, x_1, z_2, x_2, \ldots, z_{n-1}, x_{n-1}) \\
&= \sum_{z_1, \ldots, z_n} P(y \mid z_1, \ldots, z_n, x_1, \ldots, x_n) \prod_{k=1}^{n} P(z_k \mid z_1, \ldots, z_{k-1}, x_1, \ldots, x_{k-1}). \qquad \square
\end{aligned}$$

Definition 4.4.2 (Admissible Sequence and *G*-Identifiability)

Any sequence $Z_1, \ldots, Z_n$ of covariates satisfying the conditions in (4.4)–(4.5) *will be called* admissible, *and any expression $P(y \mid \hat{x}_1, \hat{x}_2, \ldots, \hat{x}_n)$ that is identifiable by the criterion of Theorem 4.4.1 will be called* G-identifiable.[7]

[7] The term "G-admissibility" was used in Pearl and Robins (1995) to evoke two associations: (1) Robins's *G-estimation* formula (equation (3.63)), which coincides with (4.6) when G is complete and contains no unobserved confounders; and (2) the *graphical* nature of the conditions in (4.4)–(4.5).

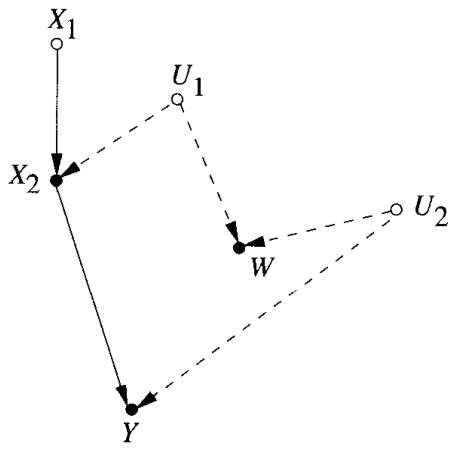


Figure 4.6 An admissible choice $Z_1 = W$ that rules out any admissible choice for Z_2. The choice $Z_1 = \emptyset$ would permit the construction of an admissible sequence ($Z_1 = \emptyset$, $Z_2 = \emptyset$).

The following corollary is immediate.

Corollary 4.4.3
A control problem is G-identifiable if and only if it has an admissible sequence.

The property of G-identifiability is sufficient but not necessary for general plan identifiability as defined in Section 4.4.2. The reasons are twofold. First, the completeness of the three inference rules of *do* calculus is still a pending conjecture. Second, the kth step in the reduction of (4.6) refrains from conditioning on variables Z_k that are descendants of X_k – namely, variables that may be affected by the action $do(X_k = x_k)$. In certain causal structures, the identifiability of causal effects requires that we condition on such variables, as demonstrated by the front-door criterion (Theorem 3.3.4).

4.4.4 Plan Identification: A Procedure

Theorem 4.4.1 provides a declarative condition for plan identifiability. It can be used to ratify that a proposed formula is valid for a given plan, but it does not provide an effective procedure for deriving such formulas because the choice of each Z_k is not spelled out procedurally. The possibility exists that some unfortunate choice of Z_k satisfying (4.4) and (4.5) might prevent us from continuing the reduction process even though another reduction sequence is feasible.

This is illustrated in Figure 4.6. Here W is an admissible choice for Z_1, but if we make this choice then we will not be able to complete the reduction, since no set Z_2 can be found that satisfies condition (4.5): $(Y \perp\!\!\!\perp X_2 \mid X_1, W, Z_2)_{G_{\underline{X}_2}}$. In this example it would be wiser to choose $Z_1 = Z_2 = \emptyset$, which satisfies both $(Y \perp\!\!\!\perp X_1 \mid \emptyset)_{G_{\underline{X}_1,\bar{X}_2}}$ and $(Y \perp\!\!\!\perp X_2 \mid X_1, \emptyset)_{G_{\underline{X}_2}}$.

The obvious way to avoid bad choices of covariates, like the one illustrated in Figure 4.6, is to insist on always choosing a "minimal" Z_k, namely, a set of covariates satisfying (4.5) that has no proper subset satisfying (4.5). However, since there are usually many such minimal sets (see Figure 4.7), the question remains of whether every choice of a minimal Z_k is "safe": Can we be sure that no choice of a minimal subsequence $Z_1, \ldots, Z_k$ will ever prevent us from finding an admissible Z_{k+1} when some admissible sequence $Z_1^*, \ldots, Z_n^*$ exists?

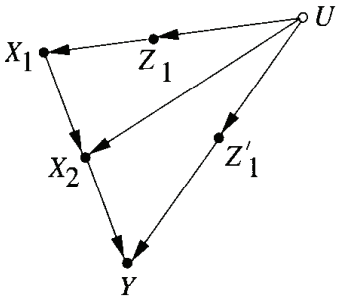


Figure 4.7 Nonuniqueness of minimal admissible sets: Z_1 and Z_1' are each minimal and admissible, since $(Y \perp\!\!\!\perp X_1 \mid Z_1)$ and $(Y \perp\!\!\!\perp X_1 \mid Z_1')$ both hold in $G_{\underline{X}_1, \overline{X}_2}$.

The next result guarantees the safety of every minimal subsequence $Z_1, \ldots, Z_k$ and hence provides an effective test for G-identifiability.

Theorem 4.4.4

If there exists an admissible sequence $Z_1^*, \ldots, Z_n^*$ then, for every minimally admissible subsequence $Z_1, \ldots, Z_{k-1}$ of covariates, there is an admissible set Z_k.

A proof is given in Pearl and Robins (1995).

Theorem 4.4.4 now yields an effective decision procedure for testing G-identifiability as follows.

Corollary 4.4.5

A control problem is G-identifiable if and only if the following algorithm exits with success.

1. *Set $k = 1$.*
2. *Choose any minimal $Z_k \subseteq N_k$ satisfying* (4.5).
3. *If no such Z_k exists then exit with failure; else set $k = k + 1$.*
4. *If $k = n + 1$ then exit with success; else return to step* 2.

A further variant of Theorem 4.4.4 can be stated that avoids the search for minimal sets Z_k. This follows from the realization that, if an admissible sequence exists, we can rewrite Theorem 4.4.1 in terms of an explicit sequence of covariates $W_1, W_2, \ldots, W_n$ that can easily be identified in G.

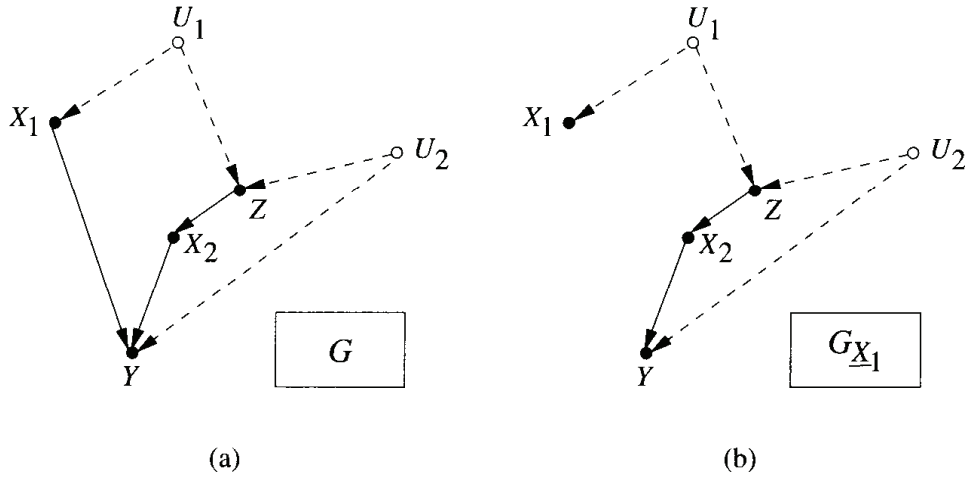
Theorem 4.4.6

The probability $P(y \mid \hat{x}_1, \ldots, \hat{x}_n)$ is G-identifiable if and only if the following condition holds for every $1 \leq k \leq n$:

$$(Y \perp\!\!\!\perp X_k \mid X_1, \ldots, X_{k-1}, W_1, W_2, \ldots, W_k)_{G_{\underline{X}_k, \overline{X}_{k+1}, \ldots, \overline{X}_n}},$$

where W_k is the set of all covariates in G that are both nondescendants of $\{X_k, X_{k+1}, \ldots, X_n\}$ and have either Y or X_k as descendant in $G_{\underline{X}_k, \overline{X}_{k+1}, \ldots, \overline{X}_n}$. Moreover, if this condition is satisfied then the plan evaluates as

$$P(y \mid \hat{x}_1, \ldots, \hat{x}_n) = \sum_{w_1, \ldots, w_n} P(y \mid w_1, \ldots, w_n, x_1, \ldots, x_n) \times \prod_{k=1}^{n} P(w_k \mid w_1, \ldots, w_{k-1}, x_1, \ldots, x_{k-1}). \tag{4.8}$$



(a) (b)

Figure 4.8 Causal diagram G in which proper ordering of the control variables X_1 and X_2 is important.

A proof of Theorem 4.4.6, together with several generalizations, can be found in Pearl and Robins (1995). Extensions to G-identifiability are reported in Kuroki and Miyakawa (1999).

The reader should note that, although Corollary 4.4.5 and Theorem 4.4.6 are procedural in the sense of offering systematic tests for plan identifiability, they are still *order-dependent*. It is quite possible that an admissible sequence exists in one ordering of the control variables and not in another when both orderings are consistent with the arrows in G. The graph G in Figure 4.8 illustrates such a case. It is obtained from Figure 4.4 by deleting the arrows $X_1 \rightarrow X_2$ and $X_1 \rightarrow Z$, so that the two control variables (X_1 and X_2) can be ordered arbitrarily. The ordering (X_1, X_2) would still admit the admissible sequence $(\emptyset, Z)$ as before, but no admissible sequence can be found for the ordering (X_2, X_1). This can be seen immediately from the graph $G_{\underline{X}_1}$, in which (according to (4.5) with $k = 1$) we need to find a set Z such that $\{X_2, Z\}$ d-separates Y from X_1. No such set exists.

The implication of this order sensitivity is that, whenever G permits several orderings of the control variables, all orderings need be examined before we can be sure that a plan is not G-identifiable. Whether an effective search exists through the space of such orderings remains an open question.

4.5 DIRECT EFFECTS AND THEIR IDENTIFICATION

4.5.1 Direct versus Total Effects

The causal effect we have analyzed so far, $P(y \mid \hat{x})$, measures the *total* effect of a variable (or a set of variables) X on a response variable Y. In many cases, this quantity does not adequately represent the target of investigation and attention is focused instead on the direct effect of X on Y. The term "direct effect" is meant to quantify an effect that is not mediated by other variables in the model or, more accurately, the sensitivity of Y to changes in X while all other factors in the analysis are held fixed. Naturally, holding those factors fixed would sever all causal paths from X to Y with the exception of the direct link $X \rightarrow Y$, which is not intercepted by any intermediaries.

A classical example of the ubiquity of direct effects (see Hesslow 1976; Cartwright 1989) tells the story of a birth-control pill that is suspect of producing thrombosis in women and, at the same time, has a negative indirect effect on thrombosis by reducing the rate of pregnancies (pregnancy is known to encourage thrombosis). In this example, interest is focused on the direct effect of the pill because it represents a stable biological relationship that, unlike the total effect, is invariant to marital status and other social factors that may affect women's chances of getting pregnant or of sustaining pregnancy.

Another class of examples involves legal disputes over race or sex discrimination in hiring. Here, neither the effect of sex or race on applicants' qualification nor the effect of qualification on hiring are targets of litigation. Rather, defendants must prove that sex and race do not *directly* influence hiring decisions, whatever indirect effects they might have on hiring by way of applicant qualification.

In all these examples, the requirement of holding the mediating variables fixed must be interpreted as (hypothetically) setting these variables to constants by physical intervention, not by analytical means such as selection, conditioning, or adjustment. For example, it will not be sufficient to measure the association between the birth-control pill and thrombosis separately among pregnant and nonpregnant women and then aggregate the results. Instead, we must perform the study among women who became pregnant before the use of the pill and among women who prevented pregnancy by means other than the drug. The reason is that, by conditioning on an intermediate variable (pregnancy in the example), we may create spurious associations between X and Y even when there is no direct effect of X on Y. This can easily be illustrated in the model $X \rightarrow Z \leftarrow U \rightarrow Y$, where X has no direct effect on Y. Physically holding Z constant would permit no association between X and Y, as can be seen by deleting all arrows entering Z. But if we were to condition on Z, a spurious association would be created through U (unobserved) that might be construed as a direct effect of X on Y.

4.5.2 Direct Effects, Definition, and Identification

Controlling all variables in a problem is obviously a major undertaking, if not an impossibility. The analysis of identification tells us under what conditions direct effects can be estimated from nonexperimental data even without such control. Using our $do(x)$ notation (or $\hat{x}$ for short), we can express the direct effect as follows.

Definition 4.5.1 (Direct Effect)
The direct effect of X on Y is given by $P(y \mid \hat{x}, \hat{s}_{XY})$, where S_{XY} is the set of all endogenous variables except X and Y in the system.

We see that the measurement of direct effects is ascribed to an ideal laboratory; the scientist controls for all possible conditions S_{XY} and need not be aware of the structure of the diagram or of which variables are truly intermediaries between X and Y. Much of the experimental control can be eliminated, however, if we know the structure of the diagram. For one thing, there is no need to actually hold *all* other variables constant; holding constant the direct parents of Y (excluding X) should suffice. Thus, we obtain the following equivalent definition of a direct effect.

Corollary 4.5.2

The direct effect of X on Y is given by $P(y \mid \hat{x}, \widehat{pa}_{Y \setminus X})$, *where* $pa_{Y \setminus X}$ *stands for any realization of the parents of Y, excluding X.*

Clearly, if X does not appear in the equation for Y (equivalently, if X is not a parent of Y), then $P(y \mid \hat{x}, \widehat{pa}_{Y \setminus X})$ defines a constant distribution on Y that is independent of x, thus matching our understanding of "having no direct effect." In general, assuming that X is a parent of Y, Corollary 4.5.2 implies that the direct effect of X on Y is identifiable whenever $P(y \mid \widehat{pa}_Y)$ is identifiable. Moreover, since the conditioning part of this expression corresponds to a plan in which the parents of Y are the control variables, we conclude that a direct effect is identifiable whenever the effect of the corresponding parents' plan is identifiable. We can now use the analysis of Section 4.4 and apply the graphical criteria of Theorems 4.4.1 and 4.4.6 to the analysis of direct effects. In particular, we can state our next theorem.

Theorem 4.5.3

Let $PA_Y = \{X_1, \ldots, X_k, \ldots, X_m\}$. *The direct effect of any* X_k *on Y is identifiable whenever the conditions of Corollary 4.4.5 hold for the plan* $(\hat{x}_1, \hat{x}_2, \ldots, \hat{x}_m)$ *in some admissible ordering of the variables. The direct effect is then given by* (4.8).

Theorem 4.5.3 implies that if the effect of one parent of Y is identifiable then the effect of every parent of Y is identifiable as well. Of course, the magnitude of the effect would differ from parent to parent, as seen in (4.8).

The following corollary is immediate.

Corollary 4.5.4

Let X_j *be a parent of Y. The direct effect of* X_j *on Y is, in general, nonidentifiable if there exists a confounding arc that embraces any link* $X_k \rightarrow Y$.

4.5.3 Example: Sex Discrimination in College Admission

To illustrate the use of this result, consider the study of Berkeley's alleged sex bias in graduate admission (Bickel et al. 1975), where data showed a higher rate of admission for male applicants overall but, when broken down by departments, a slight bias toward female applicants. The explanation was that female applicants tend to apply to the more competitive departments, where rejection rates are high; based on this finding, Berkeley was exonerated from charges of discrimination. The philosophical aspects of such reversals, known as Simpson's paradox, will be discussed more fully in Chapter 6. Here we focus on the question of whether adjustment for department is appropriate for assessing sex discrimination in college admission. Conventional wisdom has it that such adjustment is appropriate because "We know that applying to a popular department (one with considerably more applicants than positions) is just the kind of thing that causes rejection" (Cartwright 1983, p. 38), but we will soon see that additional factors should be considered.

Let us assume that the relevant factors in the Berkeley example are configured as in Figure 4.9, with the following interpretation of the variables:

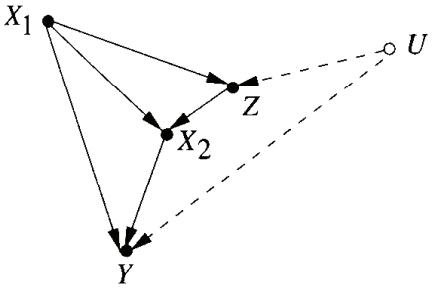


Figure 4.9 Causal relationships relevant to Berkeley's sex discrimination study. Adjusting for department choice (X_2) or career objective (Z) (or both) would be inappropriate in estimating the direct effect of gender on admission. The appropriate adjustment is given in (4.10).

X_1 = applicant's gender;

X_2 = applicant's choice of department;

Z = applicant's career objectives;

Y = admission outcome (accept/reject);

U = applicant's aptitude (unrecorded).

Note that U affects applicant's career objective and also the admission outcome Y (say, through verbal skills (unrecorded)).

Adjusting for department choice amounts to computing the following expression:

$$E_{x_2} P(y \mid \hat{x}_1, x_2) = \sum_{x_2} P(y \mid x_1, x_2) P(x_2). \tag{4.9}$$

In contrast, the direct effect of X_1 on Y, as given by (4.7), reads

$$P(y \mid \hat{x}_1, \hat{x}_2) = \sum_{z} P(y \mid z, x_1, x_2) P(z \mid x_1). \tag{4.10}$$

It is clear that the two expressions may differ substantially. The first measures the (average) effect of sex on admission among applicants to a given department, a quantity that is sensitive to the fact that some gender–department combinations may be associated with high admission rates merely because such combinations are indicative of certain aptitude (U) that was unrecorded. The second expression eliminates such spurious associations by separately adjusting for career objectives (Z) in each of the two genders.

To verify that (4.9) does not properly measure the direct effect of X_1 on Y, we note that the expression depends on the value of X_1 even in cases where the arrow between X_1 and Y is absent. Equation (4.10), on the other hand, becomes insensitive to x_1 in such cases – an exercise that we leave for the reader to verify.[8]

To cast this analysis in a concrete numerical setting, let us imagine a college consisting of two departments, A and B, both admitting students on the basis of qualification, Q, alone. Let us further assume (i) that the applicant pool consists of 100 males and 100 females and (ii) that 50 applicants in each gender have high qualifications (hence are admitted) and 50 have low qualifications (hence are rejected). Clearly, this college cannot be accused of sex discrimination.

[8] *Hint:* Factorize $P(y, u, z \mid \hat{x}_1, \hat{x}_2)$ using the independencies in the graph and eliminate u as in the derivation of (3.27).

Table 4.1. *Admission Rate among Males and Females in Each Department*

	Males		Females		Total	
	Admitted	Applied	Admitted	Applied	Admitted	Applied
Dept. A	50	50	0	0	50	50
Dept. B	0	50	50	100	50	150
Unadjusted	50%		50%		50%	
Adjusted	25%		37.5%			

A different result would surface, however, if we adjust for departments while ignoring qualifications, which amounts to using (4.9) to estimate the effect of gender on admission. Assume that the nature of the departments is such that *all and only* qualified male applicants apply to department A, while all females apply to department B (see Table 4.1).

We see from the table that adjusting for department would falsely indicate a bias of $37.5:25$ $(= 3:2)$ in favor of female applicants. An unadjusted (sometimes called "crude") analysis happens to give the correct result in this example – 50% admission rate for males and females alike – thus exonerating the school from charges of sex discrimination.

Our analysis is not meant to imply that the Berkeley study of Bickel et al. (1975) is defective, or that adjustment for department was not justified in that study. The purpose is to emphasize that no adjustment is guaranteed to give an unbiased estimate of causal effects, direct or indirect, absent a careful examination of the causal assumptions that ensure identification. Theorem 4.5.3 provides us with the understanding of those assumptions and with a mathematical means of expressing them. We note that if applicants' qualifications were not recorded in the data, then the direct effect of gender on admission will not be identifiable unless we can measure some proxy variable that stands in the same relation to Q as Z stands to U in Figure 4.9.

4.5.4 Average Direct Effects

Readers versed in structural equation models (SEMs) will note that, in linear systems, the direct effect $P(Y \mid \hat{x}, \widehat{pa}_{Y\setminus X})$ is fully specified by the path coefficient attached to the link from X to Y (see (5.24) for mathematical definition); therefore, the direct effect is independent of the values $pa_{Y\setminus X}$ at which we hold the other parents of Y. In nonlinear systems, those values would, in general, modify the effect of X on Y and thus should be chosen carefully to represent the target policy under analysis. For example, the direct effect of a pill on thrombosis would most likely be different for pregnant and nonpregnant women. Epidemiologists call such differences "effect modification" and insist on separately reporting the effect in each subpopulation.

Although the direct effect is sensitive to the levels at which we hold the parents of the outcome variable, it is sometimes meaningful to average the direct effect over those levels. For example, if we wish to assess the degree of discrimination in a given school without reference to specific departments, we can compute the difference

$$P(\text{admission} \mid \widehat{\text{male}}, \widehat{\text{dept}}) - P(\text{admission} \mid \widehat{\text{female}}, \widehat{\text{dept}})$$

and average this difference over all departments. This average measures the increase in admission rate in a hypothetical experiment in which we instruct all female candidates to retain their department preferences but change their gender identification (on the application form) from female to male.

In general, the average direct effect can be defined as a set of probabilities

$$\sum_{pa_{Y\setminus X}} P(y \mid \hat{x}, \widehat{pa}_{Y\setminus X}) P(pa_{Y\setminus X}),$$

one for each level of X. Several variants of this definition may be used when X affects other parents of Y. For example, we may wish to assess the average change in $E(Y)$ induced by changing X from x to x' while keeping the other parents of Y constant at whatever value they obtain under $do(x)$. The appropriate expression for this change is

$$\Delta_{x,x'}(Y) = \sum_{pa_{Y\setminus X}} [E(Y \mid do(x'), do(pa_{Y\setminus X})) - E(Y \mid do(x), do(pa_{Y\setminus X}))] \times P(pa_{Y\setminus X} \mid do(x)).$$

This expression represents what we actually wish to measure in race or sex discrimination cases, where we are instructed to assess the effect of one factor (X) while keeping "all other factors constant."

Acknowledgment

Sections 4.3 and 4.4 are based, respectively, on collaborative works with David Galles and James Robins.

CHAPTER FIVE

Causality and Structural Models in Social Science and Economics

Do two men travel together
unless they have agreed?
Amos 3:3

Preface

Structural equation modeling (SEM) has dominated causal analysis in economics and the social sciences since the 1950s, yet the prevailing interpretation of SEM differs substantially from the one intended by its originators and also from the one expounded in this book. Instead of carriers of substantive causal information, structural equations are often interpreted as carriers of probabilistic information; economists view them as convenient representations of density functions, and social scientists see them as summaries of covariance matrices. The result has been that many SEM researchers have difficulty articulating the causal content of SEM, and the most distinctive capabilities of SEM are currently ill understood and underutilized.

This chapter is written with the ambitious goal of reinstating the causal interpretation of SEM. We shall demonstrate how developments in the areas of graphical models and the logic of intervention can alleviate the current difficulties and thus revitalize structural equations as the primary language of causal modeling. Toward this end, we recast several of the results of Chapters 3 and 4 in parametric form (the form most familiar to SEM researchers) and demonstrate how practical and conceptual issues of model testing and parameter identification can be illuminated through graphical methods. We then move back to nonparametric analysis, from which an operational semantics will evolve that offers a coherent interpretation of what structural equations are all about (Section 5.4). In particular, we will provide answers to the following fundamental questions: What do structural equations claim about the world? What portion of those claims is testable? Under what conditions can we estimate structural parameters through regression analysis?

In Section 5.1 we survey the history of SEM and suggest an explanation for the current erosion of its causal interpretation. The testable implications of structural models are explicated in Section 5.2. For recursive models (herein termed *Markovian*), we find that the statistical content of a structural model can be fully characterized by a set of zero partial correlations that are entailed by the model. These zero partial correlations can be read off the graph using the *d-separation* criterion, which in linear models applies to graphs with cycles and correlated errors as well (Section 5.2). The application of this criterion to model testing is discussed in Section 5.2.2, which advocates local over global testing

strategies. Section 5.2.3 provides simple graphical tests of model equivalence and thus clarifies the *nontestable* part of structural models.

In Section 5.3 we deal with the issue of determining the identifiability of structural parameters prior to gathering any data. In Section 5.3.1, simple graphical tests of identifiability are developed for linear Markovian and semi-Markovian models (i.e., acyclic diagrams with correlated errors). These tests result in a simple procedure for determining when a path coefficient can be equated to a regression coefficient and, more generally, when structural parameters can be estimated through regression analysis. Section 5.3.2 discusses the connection between parameter identification in linear models and causal effect identification in nonparametric models, and Section 5.3.3 offers the latter as a semantical basis for the former.

Finally, in Section 5.4 we discuss the logical foundations of SEM and resolve a number of difficulties that were kept dormant in the past. These include operational definitions for structural equations, structural parameters, error terms, and total and direct effects, as well as a causal-theoretic explication of exogeneity in econometrics.

5.1 INTRODUCTION

5.1.1 Causality in Search of a Language

The word *cause* is not in the vocabulary of standard probability theory. It is an embarrassing yet inescapable fact that probability theory, the official mathematical language of many empirical sciences, does not permit us to express sentences such as "Mud does not cause rain"; all we can say is that the two events are mutually correlated, or dependent – meaning that if we find one, we can expect to encounter the other. Scientists seeking causal explanations for complex phenomena or rationales for policy decisions must therefore supplement the language of probability with a vocabulary for causality, one in which the symbolic representation for the causal relationship "Mud does not cause rain" is distinct from the symbolic representation for "Mud is independent of rain." Oddly, such distinctions have yet to be incorporated into standard scientific analysis.[1]

Two languages for causality have been proposed: path analysis or structural equation modeling (SEM) (Wright 1921; Haavelmo 1943); and the Neyman–Rubin potential-outcome model (Neyman 1923; Rubin 1974). The former has been adopted by economists and social scientists (Goldberger 1972; Duncan 1975), while a group of statisticians champion the latter (Rubin 1974; Robins 1986; Holland 1988). These two languages are mathematically equivalent (see Chapter 7, Section 7.4.4), yet neither has become standard in causal modeling – the structural equation framework because it has been greatly misused and inadequately formalized (Freedman 1987) and the potential-outcome framework because it has been only partially formalized and (more significantly) because it rests on an esoteric and seemingly metaphysical vocabulary of counterfactual variables that bears no apparent relation to ordinary understanding of cause–effect processes (see Section 3.6.3).

[1] A summary of attempts by philosophers to reduce causality to probabilities is given in Chapter 7 (Section 7.5).

Currently, potential-outcome models are understood by few and used by even fewer. Structural equation models are used by many, but their causal interpretation is generally questioned or avoided, even by their leading practitioners. In Chapters 3 and 4 we described how structural equation models, in nonparametric form, can provide the semantic basis for theories of interventions. In Section 1.4 we outlined how these models provide the semantical basis for a theory of counterfactuals as well. It is somewhat embarrassing that these distinctive features are hardly recognized and rarely utilized in the modern SEM literature. The current dominating philosophy treats SEM as just a convenient way to encode density functions (in economics) or covariance information (in social science). Ironically, we are witnessing one of the most bizarre circles in the history of science: causality in search of a language and, simultaneously, the language of causality in search of its meaning.

The purpose of this chapter is to formulate the causal interpretation and outline the proper use of structural equation models, thereby reinstating confidence in SEM as the primary formal language for causal analysis in the social and behavioral sciences. First, however, we present a brief analysis of the current crisis in SEM research in light of its historical development.

5.1.2 SEM: How its Meaning Became Obscured

Structural equation modeling was developed by geneticists (Wright 1921) and economists (Haavelmo 1943; Koopmans 1950, 1953) so that qualitative cause–effect information could be combined with statistical data to provide quantitative assessment of cause–effect relationships among variables of interest. Thus, to the often asked question, "Under what conditions can we give causal interpretation to structural coefficients?" Wright and Haavelmo would have answered, "Always!" According to the founding fathers of SEM, the conditions that make the equation $y = \beta x + \varepsilon$ *structural* are precisely those that make the causal connection between X and Y have no other value but β and ensure that nothing about the statistical relationship between x and ε can ever change this interpretation of β. Amazingly, this basic understanding of SEM has all but disappeared from the literature, leaving modern econometricians and social scientists in a quandary over β.

Most SEM researchers today are of the opinion that extra ingredients are necessary for structural equations to qualify as carriers of causal claims. Among social scientists, James, Mulaik, and Brett (1982, p. 45), for example, stated that a condition called *self-containment* is necessary for consecrating the equation $y = \beta x + \varepsilon$ with causal meaning, where self-containment stands for $\text{cov}(x, \varepsilon) = 0$. According to James et al. (1982), if self-containment does not hold then "neither the equation nor the functional relation represents a causal relation." Bollen (1989, p. 44) reiterated the necessity of self-containment (under the rubric *isolation* or *pseudo-isolation*) – contrary to the understanding that structural equations attain their causal interpretation prior to, and independently of, any statistical relationships among their constituents. Since the early 1980s, it has become exceedingly rare to find an open endorsement of the original SEM logic: that β defines the sensitivity of $E(Y)$ to experimental manipulations of X; that ε is defined in terms of β, not the other way around; and that the orthogonality condition $\text{cov}(x, \varepsilon) = 0$ is neither necessary nor

sufficient for the causal interpretation of β (see Sections 3.6.2 and 5.4.1).[2] It is therefore not surprising that many SEM textbooks have given up on causal interpretation altogether: "We often see the terms cause, effect, and causal modeling used in the research literature. We do not endorse this practice and therefore do not use these terms here" (Schumaker and Lomax 1996, p. 90).

Econometricians have just as much difficulty with the causal reading of structural parameters. Leamer (1985, p. 258) observed, "It is my surprising conclusion that economists know very well what they mean when they use the words 'exogenous,' 'structural,' and 'causal,' yet no textbook author has written adequate definitions." There has been little change since Leamer made these observations. Econometric textbooks invariably devote most of their analysis to estimating structural parameters, but they rarely discuss the role of these parameters in policy evaluation. The few books that deal with policy analysis (e.g. Goldberger 1991; Intriligator et al. 1996, p. 28) assume that policy variables satisfy the orthogonality condition by their very nature, thus rendering structural information superfluous. Hendry (1995, p. 62), for instance, explicitly tied the interpretation of β to the orthogonality condition, stating as follows:

> the status of β may be unclear until the conditions needed to estimate the postulated model are specified. For example, in the model:
>
> $$y_t = z_t\beta + u_t \quad \text{where } u_t \sim \text{IN}[0, \sigma_u^2],$$
>
> until the relationship between z_t and u_t is specified the meaning of β is uncertain since $E[z_t u_t]$ could be either zero or nonzero on the information provided.

LeRoy (1995, p. 211) goes even further: "It is a commonplace of elementary instruction in economics that endogenous variables are not generally causally ordered, implying that the question 'What is the effect of y_1 on y_2' where y_1 and y_2 are endogenous variables is generally meaningless." According to LeRoy, causal relationships cannot be attributed to any variable whose causes have separate influence on the effect variable, a position that denies any causal reading to most of the structural parameters that economists and social scientists labor to estimate.

Cartwright (1995b, p. 49), a renowned philosopher of science, addresses these difficulties by initiating a renewed attack on the tormenting question, "*Why* can we assume that we can read off causes, including causal order, from the parameters in equations whose exogenous variables are uncorrelated?" Cartwright, like SEM's founders, recognizes that causes cannot be derived from statistical or functional relationships alone and that causal assumptions are prerequisite for validating any causal conclusion. Unlike Wright and Haavelmo, however, she launches an all-out search for the assumptions that would endow the parameter β in the regression equation $y = \beta x + \varepsilon$ with a legitimate causal meaning and endeavors to prove that the assumptions she proposes are indeed sufficient. What is revealing in Cartwright's analysis is that she does not consider the answer Haavelmo would have provided – namely, that the assumptions needed for drawing

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[2] In fact, this condition is not necessary even for the *identification* of β, once β is interpreted (see the identification of α in Figures 5.7 and 5.9).

causal conclusions from parameters are communicated to us by the scientist who declared the equation "structural"; they are already encoded in the *syntax* of the equations and can be read off the associated graph as easily as a shopping list;[3] they need not be searched for elsewhere, nor do they require new proofs of sufficiency. Again, Haavelmo's answer applies to models of any size and shape, including models with correlated exogenous variables.

These examples bespeak an alarming tendency among economists and social scientists to view a structural equation as an algebraic object that carries functional and statistical assumptions but is void of causal content. This statement from one leading social scientist is typical: "It would be very healthy if more researchers abandoned thinking of and using terms such as cause and effect" (Muthen 1987, p. 180). Perhaps the boldest expression of this tendency was voiced by Holland (1995, p. 54): "I am speaking, of course, about the equation: $\{y = a + bx + \varepsilon\}$. What does it mean? The only meaning I have ever determined for such an equation is that it is a shorthand way of describing the conditional distribution of $\{y\}$ given $\{x\}$."[4]

The founders of SEM had an entirely different conception of structures and models. Wright (1923, p. 240) declared that "prior knowledge of the causal relations is assumed as prerequisite" in the theory of path coefficients, and Haavelmo (1943) explicitly interpreted each structural equation as a statement about a hypothetical controlled experiment. Likewise, Marschak (1950), Koopmans (1953), and Simon (1953) stated that the purpose of postulating a structure behind the probability distribution is to cope with the hypothetical changes that can be brought about by policy. One wonders, therefore, what has happened to SEM over the past 50 years, and why the basic (and still valid) teachings of Wright, Haavelmo, Marschak, Koopmans, and Simon have been forgotten.

Some economists attribute the decline in the understanding of structural equations to Lucas's (1976) critique, according to which economic agents anticipating policy interventions would tend to act contrary to SEM's predictions, which often ignore such anticipations. However, since this critique merely shifts the model's invariants and the burden of structural modeling – from the behavioral level to a deeper level that involves agents' motivations and expectations – it does not exonerate economists from defining and representing the causal content of structural equations at some level of discourse.

I believe that the causal content of SEM has gradually escaped the consciousness of SEM practitioners mainly for the following reasons.

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[3] These assumptions are explicated and operationalized in Section 5.4. Briefly, if G is the graph associated with a causal model that renders a certain parameter identifiable, then two assumptions are sufficient for authenticating the causal reading of that parameter: (1) every missing arrow, say between X and Y, represents the assumption that X has no effect on Y once we intervene and hold the parents of Y fixed; and (2) every missing bidirected arc $X \dashleftarrow\dashrightarrow Y$ represents the assumption that all omitted factors that affect Y are uncorrelated with those that affect X. Each of these assumptions is *testable* in experimental settings, where interventions are feasible (Section 5.4.1).

[4] All but forgotten, the structural interpretation of the equation (Haavelmo 1943) poses no restriction whatsoever on the conditional distribution of $\{y\}$ given $\{x\}$. Paraphrased in our vocabulary, it reads: "In an ideal experiment where we control X to x and any other set Z of variables (not containing X or Y) to z, Y will attain a value y given by $a + bx + \varepsilon$, where ε is a random variable that is (pointwise) independent of the settings x and z" (see Section 5.4.1). This statement implies that $E[Y \mid do(x), do(z)] = a + bx + c$ but says nothing about $E(Y \mid X = x)$.

1. SEM practitioners have sought to gain respectability for SEM by keeping causal assumptions implicit, since statisticians, the arbiters of respectability, abhor assumptions that are not directly testable.
2. The algebraic language that has dominated SEM lacks the notational facility needed to make causal assumptions, as distinct from statistical assumptions, explicit. By failing to equip causal relations with precise mathematical notation, the founding fathers in fact committed the causal foundations of SEM to oblivion. Their disciples today are seeking foundational answers elsewhere.

Let me elaborate on the latter point. The founders of SEM understood quite well that, in structural models, the equality sign conveys the asymmetrical relation "is determined by" and hence behaves more like an assignment symbol (:=) in programming languages than like an algebraic equality. However, perhaps for reasons of mathematical purity, they refrained from introducing a symbol to represent the asymmetry. According to Epstein (1987), in the 1940s Wright gave a seminar on path diagrams to the Cowles Commission (the breeding ground for SEM), but neither side saw particular merit in the other's methods. Why? After all, a diagram is nothing but a set of nonparametric structural equations in which, to avoid confusion, the equality signs are replaced with arrows.

My explanation is that the early econometricians were extremely careful mathematicians who thought they could keep the mathematics in purely equational–statistical form and just reason about structure in their heads. Indeed, they managed to do so surprisingly well, because they were truly remarkable individuals who *could* do it in their heads. The consequences surfaced in the early 1980s, when their disciples began to mistake the equality sign for an algebraic equality. The upshot was that suddenly the "so-called disturbance terms" did not make any sense at all (Richard 1980, p. 3). We are living with the sad end to this tale. By failing to express their insights in mathematical notation, the founders of SEM brought about the current difficulties surrounding the interpretation of structural equations, as summarized by Holland's "What does it mean?"

5.1.3 Graphs as a Mathematical Language

Recent developments in graphical methods promise to bring causality back into the mainstream of scientific modeling and analysis. These developments involve an improved understanding of the relationships between graphs and probabilities, on the one hand, and graphs and causality, on the other. But the crucial change has been the emergence of graphs as a mathematical language. This mathematical language is not simply a heuristic mnemonic device for displaying algebraic relationships, as in the writings of Blalock (1962) and Duncan (1975). Rather, graphs provide a fundamental notational system for concepts and relationships that are not easily expressed in the standard mathematical languages of algebraic equations and probability calculus. Moreover, graphical methods now provide a powerful symbolic machinery for deriving the consequences of causal assumptions when such assumptions are combined with statistical data.

A concrete example that illustrates the power of the graphical language – and that will set the stage for the discussions in Sections 5.2 and 5.3 – is Simpson's paradox, discussed

in Section 3.3 and further analyzed in Section 6.1. This paradox concerns the reversal of an association between two variables (e.g., gender and admission to school) that occurs when we partition a population into finer groups, (e.g., departments). Simpson's reversal has been the topic of much statistical research since its discovery in 1899. This research has focused on conditions for escaping the reversal instead of addressing the practical questions posed by the reversal: "Which association is more valid, before or after partitioning?" In linear analysis, the problem surfaces through the choice of regressors – for example, determining whether a variate Z can be added to a regression equation without biasing the result. Such an addition may easily reverse the sign of the coefficients of the other regressors, a phenomenon known as "suppressor effect" (Darlington 1990).

Despite a century of analysis, questions of regressor selection or adjustment for covariates continue to be decided informally, case-by-case, with the decision resting on folklore and intuition rather than on hard mathematics. The standard statistical literature is remarkably silent on this issue. Aside from noting that one should not adjust for a covariate that is affected by the putative cause (X),[5] the literature provides no guidelines as to what covariates might be admissible for adjustment and what assumptions would be needed for making such a determination formally. The reason for this silence is clear: the solution to Simpson's paradox and the covariate selection problem (as we have seen in Sections 3.3.1 and 4.5.3) rests on causal assumptions, and such assumptions cannot be expressed formally in the standard language of statistics.[6]

In contrast, formulating the covariate selection problem in the language of graphs immediately yields a general solution that is both natural and formal. The investigator expresses causal knowledge (or assumptions) in the familiar qualitative terminology of path diagrams, and once the diagram is complete, a simple procedure decides whether a proposed adjustment (or regression) is appropriate relative to the quantity under evaluation. This procedure, which we called the *back-door criterion* in Definition 3.3.1, was applicable when the quantity of interest is the total effect of X on Y. If instead the direct effect is to be evaluated, then the graphical criterion of Theorem 4.5.3 is applicable. A modified criterion for identifying direct effects (i.e., a path coefficient) in linear models will be given in Theorem 5.3.1.

This example is not an isolated instance of graphical methods affording clarity and understanding. In fact, the conceptual basis for SEM achieves a new level of precision through graphs. What makes a set of equations "structural," what assumptions are expressed by the authors of such equations, what the testable implications of those assumptions are, and what policy claims a given set of structural equations advertises are some of the questions that receive simple and mathematically precise answers via graphical methods. These and related issues in SEM will be discussed in the following sections.

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[5] This advice, which rests on the causal relationship "not affected by," is (to the best of my knowledge) the *only* causal notion that has found a place in statistics textbooks. The advice is neither necessary nor sufficient, as readers can verify from the discussion in Chapter 3.

[6] Simpson's reversal, as well as the supressor effect, are paradoxical only when we attach causal reading to the associations involved; see Section 6.1.

5.2 GRAPHS AND MODEL TESTING

In 1919, Wright developed his "method of path coefficients," which allows researchers to compute the magnitudes of cause–effect relationships from correlation measurements provided the path diagram represents correctly the causal processes underlying the data. Wright's method consists of writing a set of equations, one for each pair of variables (X_i, X_j), and equating the (standardized) correlation coefficient ρ_{ij} with a sum of products of path coefficients and residual correlations along the various paths connecting X_i and X_j. One can then attempt to solve these equations for the path coefficients in terms of the observed correlations. Whenever the resulting equations give a unique solution to some path coefficient p_{mn} that is independent of the (unobserved) residual correlations, that coefficient is said to be *identifiable.* If every set of correlation coefficients ρ_{ij} is compatible with some choice of path coefficients then the model is said to be *untestable* or *unfalsifiable* (also called *saturated, just identified,* etc.), because it is capable of perfectly fitting any data whatsoever.

Whereas Wright's method is partly graphical and partly algebraic, the theory of directed graphs permits us to analyze questions of testability and identifiability in purely graphical terms, prior to data collection, and it also enables us to extend these analyses from linear to nonlinear or nonparametric models. This section deals with issues of testability in linear and nonparametric models.

5.2.1 The Testable Implications of Structural Models

When we hypothesize a model of the data-generating process, that model often imposes restrictions on the statistics of the data collected. In observational studies, these restrictions provide the only view under which the hypothesized model can be tested or falsified. In many cases, such restrictions can be expressed in the form of zero partial correlations; more significantly, the restrictions are implied by the structure of the path diagram alone, independent of the numerical values of the parameters, as revealed by the d-separation criterion.

Preliminary Notation

Before addressing the testable implication of structural models, let us first review some definitions from Section 1.4 and relate them to the standard notation used in the SEM literature.

The graphs we discuss in this chapter represent sets of structural equations of the form

$$x_i = f_i(pa_i, \varepsilon_i), \quad i = 1, \ldots, n, \tag{5.1}$$

where pa_i (connoting *parents*) stands for (values of) the set of variables judged to be immediate causes of X_i and where the ε_i represent errors due to omitted factors. Equation (5.1) is a nonlinear, nonparametric generalization of the standard linear equations

$$x_i = \sum_{k \neq i} \alpha_{ik} x_k + \varepsilon_i, \quad i = 1, \ldots, n, \tag{5.2}$$

in which pa_i correspond to those variables on the r.h.s. of (5.2) that have nonzero coefficients. A set of equations in the form of (5.1) will be called a *causal model* if each equation represents the process by which the value (not merely the probability) of variable X_i is selected. The graph G obtained by drawing an arrow from every member of pa_i to X_i will be called a *causal diagram*. In addition to full arrows, a causal diagram should contain a bidirected (i.e. double-arrowed) arc between any pair of variables whose corresponding errors are dependent.

It is important to emphasize that causal diagrams (as well as traditional path diagrams) should be distinguished from the wide variety of graphical models in the statistical literature whose construction and interpretation rest solely on properties of the joint distribution (Kiiveri et al. 1984; Whittaker 1990; Cox and Wermuth 1996; Lauritzen 1996; Andersson et al. 1999). The missing links in those statistical models represent conditional independencies, whereas the missing links in causal diagrams represent absence of causal connections (see note 3 and Section 5.4), which may or may not imply conditional independencies in the distribution.

A causal model will be called *Markovian* if its graph contains no directed cycles and if its ε_i are mutually independent (i.e., if there are no bidirected arcs). A model is *semi-Markovian* if its graph is acyclic and if it contains dependent errors.

If the ε_i are multivariate normal (a common assumption in the SEM literature), then the X_i in (5.2) will also be multivariate normal and will be fully characterized by the correlation coefficients ρ_{ij}. A useful property of multivariate normal distributions is that the conditional variance $\sigma^2_{X|z}$, conditional covariance $\sigma_{XY|z}$, and conditional correlation coefficient $\rho_{XY|z}$ are all independent of the value z. These are known as *partial* variance, covariance, and correlation coefficient and are denoted by $\sigma_{X \cdot Z}$, $\sigma_{XY \cdot Z}$, and $\rho_{XY \cdot Z}$ (respectively), where X and Y are single variables and Z is a set of variables. Moreover, the partial correlation coefficient $\rho_{XY \cdot Z}$ is zero if and only if $(X \perp\!\!\!\perp Y \mid Z)$ holds in the distribution.

The *partial regression coefficient* is given by

$$r_{YX \cdot Z} = \rho_{YX \cdot Z} \frac{\sigma_{Y \cdot Z}}{\sigma_{X \cdot Z}};$$

it is equal to the coefficient of X in the linear regression of Y on X and Z (the order of the subscripts is essential). In other words, the coefficient of x in the regression equation

$$y = ax + b_1 z_1 + \cdots + b_k z_k$$

is given by

$$a = r_{YX \cdot Z_1 Z_2 \ldots Z_k}.$$

These coefficients can therefore be estimated by the method of least squares (Crámer 1946).

d-Separation and Partial Correlations

Markovian models (the parallel term in the SEM literature is *recursive models*;[7] Bollen 1989) satisfy the Markov property of Theorem 1.2.7; as a result, the statistical parameters

[7] The term *recursive* is ambiguous; some authors exclude correlated errors but others do not.

of Markovian models can be estimated by ordinary regression analysis. In particular, the d-separation criterion is valid in such models (here we restate Theorem 1.2.4).

Theorem 5.2.1 (Verma and Pearl 1988; Geiger et al. 1990)
If sets X and Y are d-separated by Z in a DAG G, then X is independent of Y conditional on Z in every Markovian model structured according to G. Conversely, if X and Y are not *d-separated by Z in a DAG G, then X and Y are dependent conditional on Z in* almost all *Markovian models structured according to G.*

Because conditional independence implies zero partial correlation, Theorem 5.2.1 translates into a graphical test for identifying those partial correlations that must vanish in the model.

Corollary 5.2.2
In any Markovian model structured according to a DAG G, the partial correlation $\rho_{XY \cdot Z}$ vanishes whenever the nodes corresponding to the variables in Z d-separate node X from node Y in G, regardless of the model's parameters. Moreover, no other partial correlation would vanish for all the model's parameters.

Unrestricted semi-Markovian models can always be emulated by Markovian models that include latent variables, with the latter accounting for all dependencies among error terms. Consequently, the d-separation criterion remains valid in such models if we interpret bidirected arcs as emanating from latent common parents. This may not be possible in some linear semi-Markovian models where each latent variable is restricted to influence at most two observed variables (Spirtes et al. 1996). However, it has been shown that the d-separation criterion remains valid in such restricted systems (Spirtes et al. 1996) and, moreover, that the validity is preserved when the network contains cycles (Spirtes et al. 1998; Koster 1999). These results are summarized in the next theorem.

Theorem 5.2.3 (*d*-Separation in General Linear Models)
For any linear model structured according to a diagram D, which may include cycles and bidirected arcs, the partial correlation $\rho_{XY \cdot Z}$ vanishes if the nodes corresponding to the set of variables Z d-separate node X from node Y in D. (Each bidirected arc $i \dashleftarrow\!\dashrightarrow j$ is interpreted as a latent common parent $i \leftarrow L \rightarrow j$.)

For linear structural equation models (see (5.2)), Theorem 5.2.3 implies that those (and only those) partial correlations identified by the d-separation test are guaranteed to vanish independent of the model parameters α_{ik} and independent of the error variances. This suggests a simple and direct method for testing models: rather than going through the standard exercise of finding a maximum likelihood estimate for the model's parameters and scoring those estimates for fit to the data, we can directly test for each zero partial correlation implied by the free model. The advantages of using such tests were noted by Shipley (1997), who also devised implementations of these tests.

However, the question arises of whether it is feasible to test for the vast number of zero partial correlations entailed by a given model. Fortunately, these partial correlations

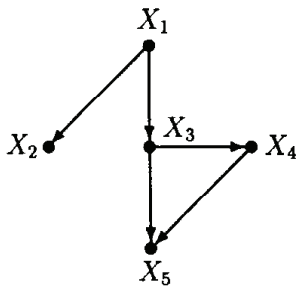

Figure 5.1 Model testable with two regressors for each missing link (equation (5.3)).

are not independent of each other; they can be derived from a relatively small number of partial correlations that constitutes a *basis* for the entire set (Pearl and Verma 1987).

Definition 5.2.4 (Basis)
Let S be a set of partial correlations. A basis *B for S is a set of zero partial correlations where* (i) *B implies (using the laws of probability) the zero of every element of S and* (ii) *no proper subset of B sustains such implication.*

An obvious choice of a basis for the zero partial correlations entailed by a DAG D is the set of equalities $B = \{\rho_{ij \cdot pa_i} = 0 \mid i > j\}$, where i ranges over all nodes in D and j ranges over all predecessors of i in any order that agrees with the arrows of D. In fact, this set of equalities reflects the "parent screening" property of Markovian models (Theorem 1.2.7), which is the source of all the probabilistic information encoded in a DAG. Testing for these equalities is therefore sufficient for testing all the statistical claims of a linear Markovian model. Moreover, when the parent sets PA_i are large, it may be possible to select a more economical basis, as shown in the next theorem.[8]

Theorem 5.2.5 (Graphical Basis)
Let (i, j) be a pair of nonadjacent nodes in a DAG D, and let Z_{ij} be any set of nodes that are closer to i than j is to i and such that Z_{ij} d-separates i from j. The set of zero partial correlations $B = \{\rho_{ij \cdot Z_{ij}} = 0 \mid i > j\}$, consisting of one element per nonadjacent pair, constitutes a basis for the set of all zero partial correlations entailed by D.

Theorem 5.2.5 states that the set of zero partial correlations corresponding to *any* separation between nonadjacent nodes in the diagram encapsulates all the statistical information conveyed by a linear Markovian model. A proof of Theorem 5.2.5 is given in Pearl and Meshkat (1998).

Examining Figure 5.1, we see that each of following two sets forms a basis for the model in the figure:

$$\begin{aligned} B_1 &= \{\rho_{32 \cdot 1} = 0,\ \rho_{41 \cdot 3} = 0,\ \rho_{42 \cdot 3} = 0,\ \rho_{51 \cdot 43} = 0,\ \rho_{52 \cdot 43} = 0\}, \\ B_2 &= \{\rho_{32 \cdot 1} = 0,\ \rho_{41 \cdot 3} = 0,\ \rho_{42 \cdot 1} = 0,\ \rho_{51 \cdot 3} = 0,\ \rho_{52 \cdot 1} = 0\}. \end{aligned} \tag{5.3}$$

The basis B_1 employs the parent set PA_i for separating i from j $(i > j)$. Basis B_2, on the other hand, employs smaller separating sets and thus leads to tests that involve fewer

[8] The possibility that linear models may possess more economical bases came to my awareness during a conversation with Rod McDonald.

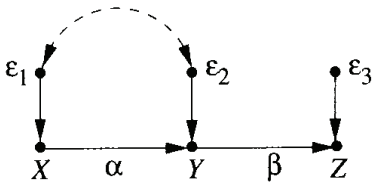

Figure 5.2 A testable model containing unidentified parameter (α).

regressors. Note that each member of a basis corresponds to a missing arrow in the DAG; therefore, the number of tests required to validate a DAG is equal to the number of missing arrows it contains. The sparser the graph, the more it constrains the covariance matrix and more tests are required to verify those constraints.

5.2.2 Testing the Testable

In linear structural equation models, the hypothesized causal relationships between variables can be expressed in the form of a directed graph annotated with coefficients, some fixed a priori (usually to zero) and some free to vary. The conventional method for testing such a model against the data involves two stages. First, the free parameters are estimated by iteratively maximizing a fitness measure such as the likelihood function. Second, the covariance matrix implied by the estimated parameters is compared to the sample covariances and a statistical test is applied to decide whether the latter could originate from the former (Bollen 1989; Chou and Bentler 1995).

There are two major weaknesses to this approach:

1. if some parameters are not identifiable, then the first phase may fail to reach stable estimates for the parameters and the investigator must simply abandon the test;
2. if the model fails to pass the data fitness test, the investigator receives very little guidance about which modeling assumptions are wrong.

For example, Figure 5.2 shows a path model in which the parameter α is not identifiable if $\text{cov}(\varepsilon_1, \varepsilon_2)$ is assumed to be unknown, which means that the maximum likelihood method may fail to find a suitable estimate for α, thus precluding the second phase of the test. Still, this model is no less testable than the one in which $\text{cov}(\varepsilon_1, \varepsilon_2) = 0$, α is identifiable, and the test can proceed. These models impose the same restrictions on the covariance matrix – namely, that the partial correlation $\rho_{XZ \cdot Y}$ should vanish (i.e., $\rho_{XZ} = \rho_{XY}\rho_{YZ}$) – yet the model with free $\text{cov}(\varepsilon_1, \varepsilon_2)$, by virtue of α being nonidentifiable, cannot be tested for this restriction.

Figure 5.3 illustrates the weakness associated with model diagnosis. Suppose the true data-generating model has a direct causal connection between X and W, as shown in Figure 5.3(a), while the hypothesized model (Figure 5.3(b)) has no such connection. Statistically, the two models differ in the term $\rho_{XW \cdot Z}$, which should vanish according to Figure 5.3(b) and is left free according to Figure 5.3(a). Once the nature of the discrepancy is clear, the investigator must decide whether substantive knowledge justifies alteration of the model by adding either a link or a curved arc between X and W. However, because the effect of the discrepancy will be spread over several covariance terms, global fitness tests will not be able to isolate the discrepancy easily. Even multiple fitness tests

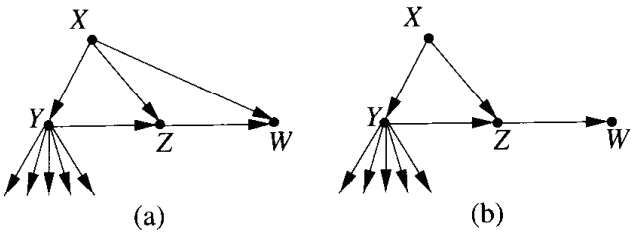

Figure 5.3 Models differing in one local test, $\rho_{XW \cdot Z} = 0$.

on various local modifications of the model (such tests are provided by LISREL) may not help much, because the results may be skewed by other discrepancies in different parts of the model, such as the subgraph rooted at Y. Thus, testing for global fitness is often of only minor use in model debugging.

An attractive alternative to global fitness testing is local fitness testing, which involves listing the restrictions implied by the model and testing them one by one. A restriction such as $\rho_{XW \cdot Z} = 0$, for example, can be tested locally without measuring Y or any of its descendants, thus keeping errors associated with those measurements from interfering with the test for $\rho_{XW \cdot Z} = 0$, which is the real source of the lack of fit. More generally, typical SEM models are often close to being "saturated," claiming but a few restrictions in the form of a few edges missing from large, otherwise unrestrictive diagrams. Local and direct tests for those restrictions are more reliable than global tests, since they involve fewer degrees of freedom and are not contaminated with irrelevant measurement errors. The missing edges approach described in Section 5.2.1 provides a systematic way of detecting and enumerating the local tests needed for testing a given model.

5.2.3 Model Equivalence

In Section 2.3 (Definition 2.3.3) we defined two structural equation models to be observationally equivalent if every probability distribution that is generated by one of the models can also be generated by the other. In standard SEM, models are assumed to be linear and data are characterized by covariance matrices. Thus, two such models are observationally indistinguishable if they are *covariance equivalent,* that is, if every covariance matrix generated by one model (through some choice of parameters) can also be generated by the other. It can be easily verified that the equivalence criterion of Theorem 1.2.8 extends to covariance equivalence.

Theorem 5.2.6

Two Markovian linear-normal models are covariance equivalent if and only if they entail the same sets of zero partial correlations. Moreover, two such models are covariance equivalent if and only if their corresponding graphs have the same sets of edges and the same sets of v-structures.

The first part of Theorem 5.2.6 defines the testable implications of Markovian models. It states that, in nonmanipulative studies, Markovian structural equation models cannot be tested for any feature other than those zero partial correlations that the d-separation test reveals. It also provides a simple test for equivalence that requires, instead of checking all the d-separation conditions, merely a comparison of corresponding edges and their directionalities.

In semi-Markovian models (DAGs with correlated errors), the d-separation criterion is still valid for testing independencies (see Theorem 5.2.3), but independence equivalence no longer implies observational equivalence.[9] Two models that entail the same set of zero partial correlations among the observed variables may yet impose different inequality constraints on the covariance matrix. Nevertheless, Theorems 5.2.3 and 5.2.6 still provide necessary conditions for testing equivalence.

Generating Equivalent Models

By permitting arrows to be reversed as long as no v-structures are destroyed or created, we can use Theorem 5.2.6 to generate equivalent alternatives to any Markovian model. Meek (1995) and Chickering (1995) showed that $X \rightarrow Y$ can be replaced by $X \leftarrow Y$ if and only if all parents of X are also parents of Y. They also showed that, for any two equivalent models, there is always some sequence of such edge reversals that takes one model into the other. This simple rule for edge reversal coincides with those proposed by Stelzl (1986) and Lee and Hershberger (1990).

In semi-Markovian models, the rules for generating equivalent models are more complicated. Nevertheless, Theorem 5.2.6 yields convenient graphical principles for testing the correctness of edge-replacement rules. The basic principle is that if we regard each bidirected arc $X \dashleftarrow\dashrightarrow Y$ as representing a latent common cause $X \leftarrow L \rightarrow Y$, then the "if" part of Theorem 5.2.6 remains valid; that is, any edge-replacement transformation that does not destroy or create a v-structure is allowed. Thus, for example, an edge $X \rightarrow Y$ can be replaced by a bidirected arc $X \dashleftarrow\dashrightarrow Y$ whenever X and Y have no other parents, latent or observed. Likewise, an edge $X \rightarrow Y$ can be replaced by a bidirected arc $X \dashleftarrow\dashrightarrow Y$ whenever (1) X and Y have no latent parents and (2) every parent of X or Y is a parent of both. Such replacements do not introduce new v-structures. However, since v-structures may now involve latent variables, we can tolerate the creation or destruction of some v-structures as long as this does not affect partial correlations among the observed variables. Figure 5.4(a) demonstrates that the creation of certain v-structures can be tolerated. By reversing the arrow $X \rightarrow Y$ we create two converging arrows $Z \rightarrow X \leftarrow Y$ whose tails are connected, not directly, but through a latent common cause. This is tolerated because, although the new convergence at X blocks the path (Z, X, Y), the connection between Z and Y (through the arc $Z \dashleftarrow\dashrightarrow Y$) remains unblocked and, in fact, cannot be blocked by any set of observed variables.

We can carry this principle further by generalizing the concept of v-structure. Whereas in Markovian models a v-structure is defined as two converging arrows whose tails are not connected by a link, we now define v-structure as any two converging arrowheads whose tails are "separable." By *separable* we mean that there exists a conditioning set S capable of d-separating the two tails. Clearly, the two tails will not be separable if they are connected by an arrow or by a bidirected arc. But a pair of nodes in a semi-Markovian model can be inseparable even when not connected by an edge (Verma and Pearl 1990). With this generalization in mind, we can state necessary conditions for edge replacement as follows.

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[9] Verma and Pearl (1990) presented an example using a nonparametric model, and Richardson devised an example using linear models with correlated errors (Spirtes and Richardson 1996).

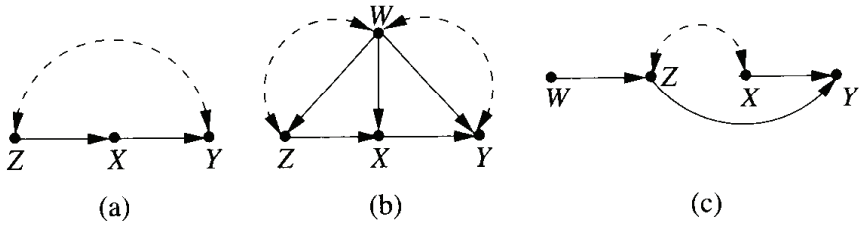

Figure 5.4 Models permitting ((a) and (b)) and forbidding (c) the reversal of $X \rightarrow Y$.

Rule 1: An arrow $X \rightarrow Y$ is interchangeable with $X \dashleftarrow\!\dashrightarrow Y$ only if every neighbor or parent of X is inseparable from Y. (By *neighbor* we mean a node connected (to X) through a bidirected arc.)

Rule 2: An arrow $X \rightarrow Y$ can be reversed into $X \leftarrow Y$ only if, before reversal, (i) every neighbor or parent of Y (excluding X) is inseparable from X and (ii) every neighbor or parent of X is inseparable from Y.

For example, consider the model $Z \dashleftarrow\!\dashrightarrow X \rightarrow Y$. The arrow $X \rightarrow Y$ cannot be replaced with a bidirected arc $X \dashleftarrow\!\dashrightarrow Y$ because Z (a neighbor of X) is separable from Y by the set $S = \{X\}$. Indeed, the new v-structure created at X would render X and Y marginally independent, contrary to the original model.

As another example, consider the graph in Figure 5.4(a). Here, it is legitimate to replace $X \rightarrow Y$ with $X \dashleftarrow\!\dashrightarrow Y$ or with a reversed arrow $X \leftarrow Y$ because X has no neighbors and Z, the only parent of X, is inseparable from Y. The same considerations apply to Figure 5.4(b); variables Z and Y, though nonadjacent, are inseparable, because the paths going from Z to Y through W cannot be blocked.

A more complicated example, one that demonstrates that rules 1 and 2 are not sufficient to ensure the legitimacy of a transformation, is shown in Figure 5.4(c). Here, it appears that replacing $X \rightarrow Y$ with $X \dashleftarrow\!\dashrightarrow Y$ would be legitimate because the (latent) v-structure at X is shunted by the arrow $Z \rightarrow Y$. However, the original model shows the path from W to Y to be d-connected given Z, whereas the postreplacement model shows the same path d-separated given Z. Consequently, the partial correlation $\rho_{WY \cdot Z}$ vanishes in the postreplacement model but not in the prereplacement model. A similar disparity also occurs relative to the partial correlation $\rho_{WY \cdot ZX}$. The original model shows that the path from W to Y is blocked, given $\{Z, X\}$, but the postreplacement model shows that path to be d-connected, given $\{Z, X\}$. Consequently, the partial correlation $\rho_{WY \cdot ZX}$ vanishes in the prereplacement model but is unconstrained in the postreplacement model.[10] Evidently, it is not enough to impose rules on the parents and neighbors of X; remote ancestors (e.g. W) should be considered, too.

These rules are just a few of the implications of the d-separation criterion when applied to semi-Markovian models. A necessary and sufficient criterion for testing the d-separation equivalence of two semi-Markovian models was devised by Spirtes and Verma (1992). Spirtes and Richardson (1996) extended that criterion to include models with feedback cycles. However, we should keep in mind that, because two semi-Markovian

[10] This example was brought to my attention by Jin Tian, and a similar one by two anonymous reviewers.

models can be zero-partial-correlation equivalent and yet not covariance equivalent, criteria based on d-separation can provide merely the necessary conditions for model equivalence.

The Significance of Equivalent Models

Theorem 5.2.6 is methodologically significant because it clarifies what it means to claim that structural models are "testable" (Bollen 1989, p. 78).[11] It asserts that we never test *a* model but rather a whole *class* of observationally equivalent models from which the hypothesized model cannot be distinguished by any statistical means. It asserts as well that this equivalence class can be constructed (by inspection) from the graph, which thus provides the investigator with a vivid representation of competing alternatives for consideration. Graphs representing all models in a given equivalence class have been devised by Verma and Pearl (1990) (see Section 2.6), Spirtes et al. (1993), and Andersson et al. (1999). Richardson (1996) discusses the representation of equivalence classes of models with cycles.

Although it is true that (overidentified) structural equation models have testable implications, those implications are but a small part of what the model represents: a set of claims, assumptions, and implications. Failure to distinguish among causal assumptions, statistical implications, and policy claims has been one of the main reasons for the suspicion and confusion surrounding quantitative methods in the social sciences (Freedman 1987, p. 112; Goldberger 1992; Wermuth 1992). However, because they make the distinctions among these components vivid and crisp, graphical methods promise to make SEM more acceptable to researchers from a wide variety of disciplines.

By and large, the SEM literature has ignored the explicit analysis of equivalent models. Breckler (1990), for example, found that only one of 72 articles in the areas of social and personality psychology even acknowledged the existence of an equivalent model. The general attitude has been that the combination of data fitness and model over-identification is sufficient to confirm the hypothesized model. Recently, however, the existence of multiple equivalent models seems to have jangled the nerves of some SEM researchers. MacCallum et al. (1993, p. 198) concluded that "the phenomenon of equivalent models represents a serious problem for empirical researchers using CSM" and "a threat to the validity of interpretation of CSM results" (CSM denotes "covariance structure modeling"; this does not differ from SEM, but the term is used by some social scientists to disguise euphemistically the causal content of their models). Breckler (1990, p. 262) reckoned that "if one model is supported, so too are all of its equivalent models" and hence ventured that "the term *causal modeling* is a misnomer."

Such extremes are not justifiable. The existence of equivalent models is logically inevitable if we accept the fact that causal relations cannot be inferred from statistical data alone; as Wright (1921) stated, "prior knowledge of the causal relations is assumed as prerequisite" in SEM. But this does not make SEM useless as a tool for causal modeling.

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[11] In response to an allegation that "path analysis does not derive the causal theory from the data, or test any major part of it against the data" (Freedman 1987, p. 112), Bollen (1989, p. 78) stated, "we can test and reject structural models.... Thus the assertion that these models cannot be falsified has little basis."

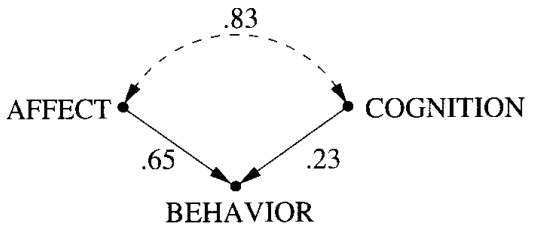

Figure 5.5 Untestable model displaying quantitative causal information derived.

The move from the qualitative causal premises represented by the structure of a path diagram (see note 3) to the quantitative causal conclusions advertised by the coefficients in the diagram is neither useless nor trivial. Consider, for example, the model depicted in Figure 5.5, which Bagozzi and Burnkrant (1979) used to illustrate problems associated with equivalent models. Although this model is saturated (i.e., just identified) and although it has (at least) 27 semi-Markovian equivalent models, finding that the influence of AFFECT on BEHAVIOR is almost three times stronger (on a standardized scale) than the influence of COGNITION on BEHAVIOR is still very illuminating – it tells us about the relative effectiveness of different behavior modification policies if some are known to influence AFFECT and others COGNITION. The significance of this quantitative analysis on policy analysis may be more dramatic when a path coefficient turns negative while the corresponding correlation coefficient measures positive. Such quantitative results may have profound impact on policy decisions, and learning that these results are logically implied by the data and the qualitative premises embedded in the diagram should make the basis for policy decisions more transparent to defend or criticize.

In summary, social scientists need not abandon SEM altogether; they need only abandon the notion that SEM is a method of *testing* causal models. Structural equation modeling is a method of testing a tiny fraction of the premises that make up a causal model and, in cases where that fraction is found to be compatible with the data, the method elucidates the necessary quantitative consequences of both the premises and the data. It follows, then, that users of SEM should concentrate on examining the implicit theoretical premises that enter into a model. As we will see in Section 5.4, graphical methods make these premises vivid and precise.

5.3 GRAPHS AND IDENTIFIABILITY

5.3.1 Parameter Identification in Linear Models

Consider a directed edge $X \rightarrow Y$ embedded in a path diagram G, and let α stand for the path coefficient associated with that edge. It is well known that the regression coefficient $r_{YX} = \rho_{XY}\sigma_Y/\sigma_X$ can be decomposed into the sum

$$r_{YX} = \alpha + I_{YX},$$

where I_{YX} is not a function of α, since it is computed (e.g., using Wright's rules) from other paths connecting X and Y excluding the edge $X \rightarrow Y$. (Such paths traverse both unidirected and bidirected arcs.) Thus, if we remove the edge $X \rightarrow Y$ from the path diagram and find that the resulting subgraph entails zero correlation between X and Y, then

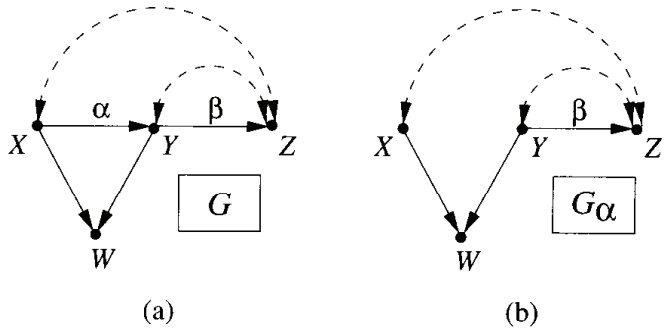

Figure 5.6 Test of whether structural parameter α can be equated with regression coefficient r_{YX}.

we know that $I_{YX} = 0$ and $\alpha = r_{YX}$; hence, α is identified. Such entailment can be established graphically by testing whether X is d-separated from Y (by the empty set $Z = \{\emptyset\}$) in the subgraph. Figure 5.6 illustrates this simple test for identification: all paths between X and Y in the subgraph G_α are blocked by converging arrows, and α can immediately be equated with r_{YX}.

We can extend this basic idea to cases where I_{YX} is not zero but can be made zero by adjusting for a set of variables $Z = \{Z_1, Z_2, \ldots, Z_k\}$ that lie on various d-connected paths between X and Y. Consider the partial regression coefficient $r_{YX \cdot Z} = \rho_{YX \cdot Z}\sigma_{Y \cdot Z}/\sigma_{X \cdot Z}$, which represents the residual correlation between Y and X after Z is "partialled out." If Z contains no descendant of Y, then again we can write[12]

$$r_{YX \cdot Z} = \alpha + I_{YX \cdot Z},$$

where $I_{YX \cdot Z}$ represents the partial correlation between X and Y resulting from setting α to zero, that is, the partial correlation in a model whose graph G_α lacks the edge $X \rightarrow Y$ but is otherwise identical to G. If Z d-separates X from Y in G_α, then $I_{YX \cdot Z}$ would indeed be zero in such a model and so we can conclude that, in our original model, α is identified and is equal to $r_{YX \cdot Z}$. Moreover, since $r_{YX \cdot Z}$ is given by the coefficient of x in the regression of Y on X and Z, α can be estimated using the regression

$$y = \alpha x + \beta_1 z_1 + \cdots + \beta_k z_k + \varepsilon.$$

This result provides a simple graphical answer to the questions, alluded to in Section 5.1.3, of (i) what constitutes an adequate set of regressors and (ii) when a regression coefficient provides a consistent estimate of a path coefficient. The answers are summarized in the following theorem.[13]

Theorem 5.3.1 (Single-Door Criterion for Direct Effects)
Let G be any path diagram in which α is the path coefficient associated with link $X \rightarrow Y$, and let G_α denote the diagram that results when $X \rightarrow Y$ is deleted from G. The coefficient α is identifiable if there exists a set of variables Z such that (i) *Z contains no*

[12] This can be seen when the relation between Y and its parents, $Y = \alpha x + \sum_i \beta_i w_i + \varepsilon$, is substituted into the expression for $r_{YX \cdot Z}$, which yields α plus an expression $I_{YX \cdot Z}$ involving partial correlations among the variables $\{X, W_1, \ldots, W_k, Z, \varepsilon\}$. Because Y is assumed not to be an ancestor of any of these variables, their joint density is unaffected by the equation for Y; hence, $I_{YX \cdot Z}$ is independent of α.

[13] This result is presented in Pearl (1998a) and Spirtes et al. (1998).

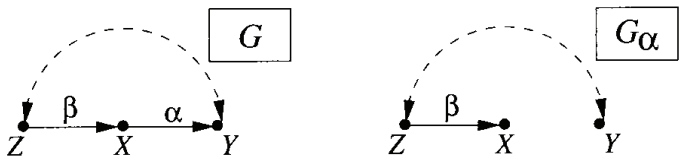

Figure 5.7 The identification of α with $r_{YX \cdot Z}$ (Theorem 5.3.1) is confirmed by G_α.

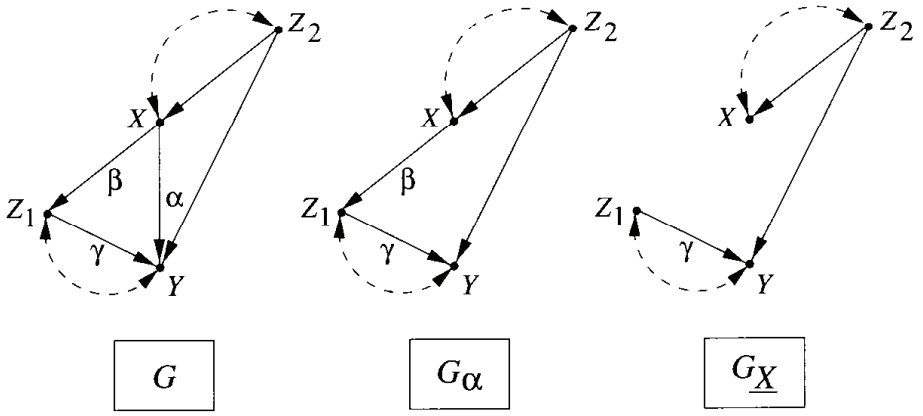

Figure 5.8 Graphical identification of the total effect of X on Y, yielding $\alpha + \beta\gamma = r_{YX \cdot Z_2}$.

descendant of Y and (ii) *Z d-separates X from Y in* G_α. *If Z satisfies these two conditions, then* α *is equal to the regression coefficient* $r_{YX \cdot Z}$. *Conversely, if Z does not satisfy these conditions, then* $r_{YX \cdot Z}$ *is not a consistent estimand of* α (*except in rare instances of measure zero*).

The use of Theorem 5.3.1 can be illustrated as follows. Consider the graphs G and G_α in Figure 5.7. The only path connecting X and Y in G_α is the one traversing Z, and since that path is d-separated (blocked) by Z, α is identifiable and is given by $\alpha = r_{YX \cdot Z}$. The coefficient β is identifiable, of course, since Z is d-separated from X in G_β (by the empty set $\emptyset$) and thus $\beta = r_{XZ}$. Note that this "single-door" test differs slightly from the back-door criterion for total effects (Definition 3.3.1); the set Z here must block *all* indirect paths from X to Y, not only back-door paths. Condition (i) is identical to both cases, because if X is a parent of Y then every descendant of Y must also be a descendant of X.

We now extend the identification of structural parameters through the identification of total effects (rather than direct effects). Consider the graph G in Figure 5.8. If we form the graph G_α by removing the link $X \rightarrow Y$, we observe that there is no set Z of nodes that d-separates all paths from X to Y. If Z contains Z_1, then the path $X \rightarrow Z_1 \dashleftarrow\dashrightarrow Y$ will be unblocked through the converging arrows at Z_1. If Z does not contain Z_1, the path $X \rightarrow Z_1 \rightarrow Y$ is unblocked. Thus we conclude that α cannot be identified using our previous method. However, suppose we are interested in the total effect of X on Y, which is given by $\alpha + \beta\gamma$. For this sum to be identified by r_{YX}, there should be no contribution to r_{YX} from paths other than those leading from X to Y. However, we see that two such paths, called *confounding* or *back-door* paths, exist in the graph – namely, $X \leftarrow Z_2 \rightarrow Y$ and $X \dashleftarrow\dashrightarrow Z_2 \rightarrow Y$. Fortunately, these paths are blocked by Z_2 and so we may conclude that adjusting for Z_2 would render $\alpha + \beta\gamma$ identifiable; thus we have

$$\alpha + \beta\gamma = r_{YX \cdot Z_2}.$$

This line of reasoning is captured by the back-door criterion of Definition 3.3.1, which we restate here for completeness.

Theorem 5.3.2 (Back-Door Criterion)
For any two variables X and Y in a causal diagram G, the total effect of X on Y is identifiable if there exists a set of measurements Z such that

1. *no member of Z is a descendant of X; and*
2. *Z d-separates X from Y in the subgraph $G_{\underline{X}}$ formed by deleting from G all arrows emanating from X.*

Moreover, if the two conditions are satisfied, then the total effect of X on Y is given by $r_{YX \cdot Z}$.

The two conditions of Theorem 5.3.2, as we have seen in Section 3.3.1, are also valid in nonlinear non-Gaussian models as well as in models with discrete variables. The test ensures that, after adjustment for Z, the variables X and Y are not associated through confounding paths, which means that the regression coefficient $r_{YX \cdot Z}$ is equal to the total effect. In fact, we can view Theorems 5.3.1 and 5.3.2 as special cases of a more general scheme: In order to identify any *partial effect,* as defined by a select bundle of causal paths from X to Y, we ought to find a set Z of measured variables that block all nonselected paths between X and Y. The partial effect will then equal the regression coefficient $r_{YX \cdot Z}$.

Figure 5.8 demonstrates that some total effects can be determined directly from the graphs without having to identify their individual components. Standard SEM methods (Bollen 1989; Chou and Bentler 1995) that focus on the identification and estimation of individual parameters may miss the identification and estimation of effects such as the one in Figure 5.8, which can be estimated reliably even though some of the constituents remain unidentified.

Some total effects cannot be determined directly as a unit but instead require the determination of each component separately. In Figure 5.7, for example, the effect of Z on Y $(= \alpha\beta)$ does not meet the back-door criterion, yet this effect can be determined from its constituents α and β, which meet the back-door criterion individually and evaluate to

$$\beta = r_{XZ}, \qquad \alpha = r_{YX \cdot Z}.$$

There is yet a third kind of causal parameter: one that cannot be determined either directly or through its constituents but rather requires the evaluation of a broader causal effect of which it is a part. The structure shown in Figure 5.9 represents an example of this case. The parameter α cannot be identified either directly or from its constituents (it has none), yet it can be determined from $\alpha\beta$ and β, which represent the effect of Z on Y and of Z on X, respectively. These two effects can be identified directly, since there are no back-door paths from Z to either Y or X; therefore, $\alpha\beta = r_{YZ}$ and $\beta = r_{XZ}$. It follows that

$$\alpha = r_{YZ}/r_{XZ},$$

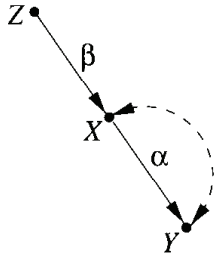

Figure 5.9 Graphical identification of α using instrumental variable Z.

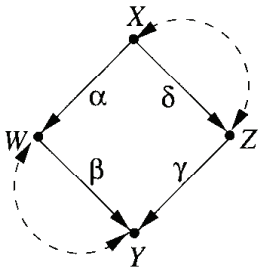

Figure 5.10 Graphical identification of α, β, and γ.

which is familiar to us as the *instrumental variable* formula (Bowden and Turkington 1984; see also Section 3.5, equation (3.46)).

The example shown in Figure 5.10 combines all three methods considered thus far. The total effect of X on Y is given by $\alpha\beta + \gamma\delta$, which is not identifiable because it does not meet the back-door criterion and is not part of another identifiable structure. However, suppose we wish to estimate β. By conditioning on Z, we block all paths going through Z and obtain $\alpha\beta = r_{YX \cdot Z}$, which is the effect of X on Y mediated by W. Because there are no back-door paths from X to W, α itself evaluates directly to $\alpha = r_{WX}$. We therefore obtain

$$\beta = r_{YX \cdot Z} / r_{WX}.$$

On the other hand, γ can be evaluated directly by conditioning on X (thus blocking all back-door paths from Z to Y through X), which gives

$$\gamma = r_{YZ \cdot X}.$$

The methods that we have been using suggest the following systematic procedure for recognizing identifiable coefficients in a graph.

1. Start by searching for identifiable causal effects among pairs of variables in the graph, using the back-door criterion and Theorem 5.3.1. These can be either direct effects, total effects, or partial effects (i.e., effects mediated by specific sets of variables).
2. For any such identified effect, collect the path coefficients involved and put them in a bucket.
3. Begin labeling the coefficients in the buckets according to the following procedure:
   (a) if a bucket is a singleton, label its coefficient I (denoting *identifiable*);
   (b) if a bucket is not a singleton but contains only a single unlabeled element, label that element I.

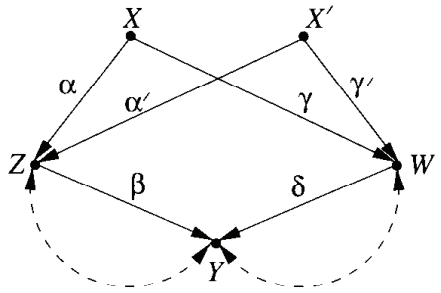

Figure 5.11 Identifying β and δ using two instrumental variables.

4. Repeat this process until no new labeling is possible.
5. List all labeled coefficients; these are identifiable.

The process just described is not complete, because our insistence on labeling coefficients one at a time may cause us to miss certain opportunities. This is shown in Figure 5.11. Starting with the pairs (X, Z), (X, W), (X', Z), and (X', W), we discover that α, γ, α', and γ' are identifiable. Going to (X, Y), we find that $\alpha\beta + \delta\gamma$ is identifiable; likewise, from (X', Y) we see that $\alpha'\beta + \gamma'\delta$ is identifiable. This does not yet enable us to label β or δ, but we can solve two equations for the unknowns β and δ as long as the determinant $\begin{vmatrix} \alpha & \gamma \\ \alpha' & \gamma' \end{vmatrix}$ is nonzero. Since we are not interested in identifiability at a point but rather in identifiability "almost everywhere" (Koopmans et al. 1950; Simon 1953), we need not compute this determinant. We merely inspect the symbolic form of the determinant's rows to make sure that the equations are nonredundant; each imposes a new constraint on the unlabeled coefficients for at least one value of the labeled coefficients.

With a facility to detect redundancies, we can increase the power of our procedure by adding the following rule:

3*. If there are k nonredundant buckets that contain at most k unlabeled coefficients, label these coefficients and continue.

Another way to increase the power of our procedure is to list not only identifiable effects but also expressions involving correlations due to bidirected arcs, in accordance with Wright's rules. Finally, one can endeavor to list effects of several variables jointly as is done in Section 4.4. However, such enrichments tend to make the procedure more complex and might compromise our main objective of providing investigators with a way to immediately recognize the identified coefficients in a given model and immediately understand those features in the model that influence the identifiability of the target quantity. We now relate these results to the identification in nonparametric models, such as those treated in Section 3.3.

5.3.2 Comparison to Nonparametric Identification

The identification results of the previous section are significantly more powerful than those obtained in Chapters 3 and 4 for nonparametric models. Nonparametric models should nevertheless be studied by parametric modelers for both practical and conceptual reasons. On the practical side, investigators often find it hard to defend the assumptions of

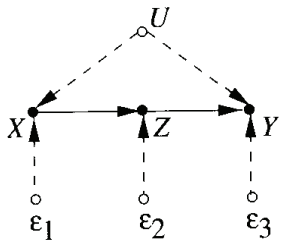

Figure 5.12 Path diagram corresponding to equations (5.4)–(5.6), where $\{X, Z, Y\}$ are observed and $\{U, \varepsilon_1, \varepsilon_2, \varepsilon_3\}$ are unobserved.

linearity and normality (or other functional–distributional assumptions), especially when categorical variables are involved. Because nonparametric results are valid for nonlinear functions and for any distribution of errors, having such results allows us to gauge how sensitive standard techniques are to assumptions of linearity and normality. On the conceptual side, nonparametric models illuminate the distinctions between structural and algebraic equations. The search for nonparametric quantities analogous to path coefficients forces explication of what path coefficients really mean, why one should labor at their identification, and why structural models are not merely a convenient way of encoding covariance information.

In this section we cast the problem of nonparametric causal effect identification (Chapter 3) in the context of parameter identification in linear models.

Parametric versus Nonparametric Models: An Example

Consider the set of structural equations

$$x = f_1(u, \varepsilon_1), \tag{5.4}$$

$$z = f_2(x, \varepsilon_2), \tag{5.5}$$

$$y = f_3(z, u, \varepsilon_3), \tag{5.6}$$

where X, Z, Y are observed variables, f_1, f_2, f_3 are unknown arbitrary functions, and $U, \varepsilon_1, \varepsilon_2, \varepsilon_3$ are unobservables that we can regard either as latent variables or as disturbances. For the sake of this discussion, we will assume that $U, \varepsilon_1, \varepsilon_2, \varepsilon_3$ are mutually independent and arbitrarily distributed. Graphically, these influences can be represented by the path diagram of Figure 5.12.

The problem is as follows. We have drawn a long stream of independent samples of the process defined by (5.4)–(5.6) and have recorded the values of the observed variables X, Z, and Y; we now wish to estimate the unspecified quantities of the model to the greatest extent possible.

To clarify the scope of the problem, we consider its linear version, which is given by

$$x = u + \varepsilon_1, \tag{5.7}$$

$$z = \alpha x + \varepsilon_2, \tag{5.8}$$

$$y = \beta z + \gamma u + \varepsilon_3, \tag{5.9}$$

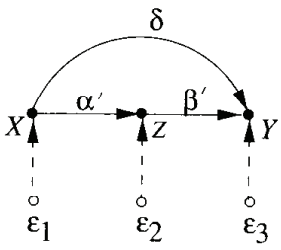

Figure 5.13 Diagram representing model M' of (5.12)–(5.14).

where $U, \varepsilon_1, \varepsilon_2, \varepsilon_3$ are uncorrelated, zero-mean disturbances.[14] It is not hard to show that parameters α, β, and γ can be determined uniquely from the correlations among the observed quantities X, Z, and Y. This identification was demonstrated already in the example of Figure 5.7, where the back-door criterion yielded

$$\beta = r_{YZ \cdot X}, \qquad \alpha = r_{ZX}, \tag{5.10}$$

and hence

$$\gamma = r_{YX} - \alpha\beta. \tag{5.11}$$

Thus, returning to the nonparametric version of the model, it is tempting to generalize that, for the model to be identifiable, the functions $\{f_1, f_2, f_3\}$ must be determined uniquely from the data. However, the prospect of this happening is unlikely, because the mapping between functions and distributions is known to be many-to-one. In other words, given any nonparametric model M, if there exists one set of functions $\{f_1, f_2, f_3\}$ compatible with a given distribution $P(x, y, z)$, then there are infinitely many such functions (see Figure 1.6). Thus, it seems that nothing useful can be inferred from loosely specified models such as the one given by (5.4)–(5.6).

Identification is not an end in itself, however, even in linear models. Rather, it serves to answer practical questions of prediction and control. At issue is not whether the data permit us to identify the form of the equations but, instead, whether the data permit us to provide unambiguous answers to questions of the kind traditionally answered by parametric models.

When the model given by (5.4)–(5.6) is used strictly for prediction (i.e., to determine the probabilities of some variables given a set of observations on other variables), the question of identification loses much (if not all) of its importance; all predictions can be estimated directly from either the covariance matrices or the sample estimates of those covariances. If dimensionality reduction is needed (e.g., to improve estimation accuracy) then the covariance matrix can be encoded in a variety of simultaneous equation models, all of the same dimensionality. For example, the correlations among X, Y, and Z in the linear model M of (5.7)–(5.9) might well be represented by the model M' (Figure 5.13):

$$x = \varepsilon_1, \tag{5.12}$$

$$z = \alpha' x + \varepsilon_2, \tag{5.13}$$

$$y = \beta' z + \delta x + \varepsilon_3. \tag{5.14}$$

[14] An equivalent version of this model is obtained by eliminating U from the equations and allowing ε_1 and ε_3 to be correlated, as in Figure 5.7.

This model is as compact as (5.7)–(5.9) and is covariance equivalent to M with respect to the observed variables X, Y, Z. Upon setting $\alpha' = \alpha$, $\beta' = \beta$, and $\delta = \gamma$, model M' will yield the same probabilistic predictions as those of the model of (5.7)–(5.9). Still, when viewed as data-generating mechanisms, the two models are not equivalent. Each tells a different story about the processes generating X, Y, and Z, so naturally their predictions differ concerning the changes that would result from subjecting these processes to external interventions.

5.3.3 Causal Effects: The Interventional Interpretation of Structural Equation Models

The differences between models M and M' illustrate precisely where the structural reading of simultaneous equation models comes into play, and why even causally shy researchers consider structural parameters more "meaningful" than covariances and other statistical parameters. Model M', defined by (5.12)–(5.14), regards X as a direct participant in the process that determines the value of Y, whereas model M, defined by (5.7)–(5.9), views X as an indirect factor whose effect on Y is mediated by Z. This difference is not manifested in the data itself but rather in the way the data would change in response to outside interventions. For example, suppose we wish to predict the expectation of Y after we intervene and fix the value of X to some constant x; this is denoted $E(Y \mid do(X = x))$. After $X = x$ is substituted into (5.13) and (5.14), model M' yields

$$E[Y \mid do(X = x)] = E[\beta'\alpha' x + \beta'\varepsilon_2 + \delta x + \varepsilon_3] \tag{5.15}$$

$$= (\beta'\alpha' + \delta)x; \tag{5.16}$$

model M yields

$$E[Y \mid do(X = x)] = E[\beta\alpha x + \beta\varepsilon_2 + \gamma u + \varepsilon_3] \tag{5.17}$$

$$= \beta\alpha x. \tag{5.18}$$

Upon setting $\alpha' = \alpha$, $\beta' = \beta$, and $\delta = \gamma$ (as required for covariance equivalence; see (5.10) and (5.11)), we see clearly that the two models assign different magnitudes to the (total) causal effect of X on Y: model M predicts that a unit change in x will change $E(Y)$ by the amount $\beta\alpha$, whereas model M' puts this amount at $\beta\alpha + \gamma$.

At this point, it is tempting to ask whether we should substitute $x - \varepsilon_1$ for u in (5.9) prior to taking expectations in (5.17). If we permit the substitution of (5.8) into (5.9), as we did in deriving (5.17), why not permit the substitution of (5.7) into (5.9) as well? After all (the argument runs), there is no harm in upholding a mathematical equality, $u = x - \varepsilon_1$, that the modeler deems valid. This argument is fallacious, however.[15] Structural equations are not meant to be treated as immutable mathematical equalities. Rather, they are meant to define a state of equilibrium – one that is *violated* when the equilibrium is perturbed by outside interventions. In fact, the power of structural equation models is

[15] Such arguments have led to Newcomb's paradox in the so-called evidential decision theory (see Section 4.1.1).

that they encode not only the initial equilibrium state but also the information necessary for determining which equations must be violated in order to account for a new state of equilibrium. For example, if the intervention consists merely of holding X constant at x, then the equation $x = u + \varepsilon_1$, which represents the preintervention process determining X, should be overruled and replaced with the equation $X = x$. The solution to the new set of equations then represents the new equilibrium. Thus, the essential characteristic of structural equations that sets them apart from ordinary mathematical equations is that the former stand not for one but for many sets of equations, each corresponding to a subset of equations taken from the original model. Every such subset represents some hypothetical physical reality that would prevail under a given intervention.

If we take the stand that the value of structural equations lies not in summarizing distribution functions but in encoding causal information for predicting the effects of policies (Haavelmo 1943; Marschak 1950; Simon 1953), it is natural to view such predictions as the proper generalization of structural coefficients. For example, the proper generalization of the coefficient β in the linear model M would be the answer to the control query, "What would be the change in the expected value of Y if we were to intervene and change the value of Z from z to $z + 1$," which is different, of course, from the observational query, "What would be the difference in the expected value of Y if we were to *find* Z at level $z + 1$ instead of level z." Observational queries, as we discussed in Chapter 1, can be answered directly from the joint distribution $P(x, y, z)$, while control queries require causal information as well. Structural equations encode this causal information in their syntax by treating the variable on the left-hand side of the equality sign as the effect and treating those on the right as causes. In Chapter 3 we distinguished between the two types of queries through the symbol $do(\cdot)$. For example, we wrote

$$E(Y \mid do(x)) \triangleq E[Y \mid do(X = x)] \tag{5.19}$$

for the controlled expectation and

$$E(Y \mid x) \triangleq E(Y \mid X = x) \tag{5.20}$$

for the standard conditional or observational expectation. That $E(Y \mid do(x))$ does not equal $E(Y \mid x)$ can easily be seen in the model of (5.7)–(5.9), where $E(Y \mid do(x)) = \alpha\beta x$ but $E(Y \mid x) = r_{YX}x = (\alpha\beta + \gamma)x$. Indeed, the passive observation $X = x$ should not violate any of the equations, and this is the justification for substituting both (5.7) and (5.8) into (5.9) before taking the expectation.

In linear models, the answers to questions of direct control are encoded in the path (or structural) coefficients, which can be used to derive the total effect of any variable on another. For example, the value of $E(Y \mid do(x))$ in the model defined by (5.7)–(5.9) is $\alpha\beta x$, that is, x times the product of the path coefficients along the path $X \rightarrow Z \rightarrow Y$. Computation of $E(Y \mid do(x))$ would be more complicated in the nonparametric case, even if we knew the functions f_1, f_2, and f_3. Nevertheless, this computation is well-defined; it requires the solution (for the expectation of Y) of a modified set of equations in which f_1 is "wiped out" and X is replaced by the constant x:

$$z = f_2(x, \varepsilon_2), \tag{5.21}$$

$$y = f_3(z, u, \varepsilon_3). \tag{5.22}$$

Thus, computation of $E(Y \mid do(x))$ requires evaluation of

$$E(Y \mid do(x)) = E\{f_3[f_2(x, \varepsilon_2), u, \varepsilon_3]\},$$

where the expectation is taken over U, ε_2, and ε_3. Graphical methods for performing this computation were discussed in Section 3.3.2.

What, then, is an appropriate definition of identifiability for nonparametric models? One reasonable definition is that answers to interventional queries are *unique,* and this is precisely how Definition 3.2.3 interprets the identification of the causal effect $P(y \mid do(x))$. As we have seen in Chapters 3 and 4, many aspects of nonparametric identification can be determined graphically, almost by inspection, from the diagrams that accompany the equations. These include tests for deciding whether a given interventional query is identifiable as well as formulas for estimating such queries.

5.4 SOME CONCEPTUAL UNDERPINNINGS

5.4.1 What Do Structural Parameters Really Mean?

Every student of SEM has stumbled on the following paradox at some point in his or her career. If we interpret the coefficient β in the equation

$$y = \beta x + \varepsilon$$

as the change in $E(Y)$ per unit change of X, then, after rewriting the equation as

$$x = (y - \varepsilon)/\beta,$$

we ought to interpret $1/\beta$ as the change in $E(X)$ per unit change of Y. But this conflicts both with intuition and with the prediction of the model: the change in $E(X)$ per unit change of Y ought to be *zero* if Y does not appear as an independent variable in the original, structural equation for X.

Teachers of SEM generally evade this dilemma via one of two escape routes. One route involves denying that β has any causal reading and settling for a purely statistical interpretation, in which β measures the reduction in the variance of Y explained by X (see e.g. Muthen 1987). The other route permits causal reading of only those coefficients that meet the "isolation" restriction (Bollen 1989; James et al. 1982): the explanatory variable must be uncorrelated with the error in the equation. Because ε cannot be uncorrelated with both X and Y (or so the argument goes), β and $1/\beta$ cannot both have causal meaning, and the paradox dissolves.

The first route is self-consistent, but it compromises the founders' intent that SEM function as an aid to policy making and clashes with the intuition of most SEM users. The second is vulnerable to attack logically. It is well known that every pair of bivariate normal variables, X and Y, can be expressed in two equivalent ways,

$$y = \beta x + \varepsilon_1 \quad \text{and} \quad x = \alpha y + \varepsilon_2,$$

where $\text{cov}(X, \varepsilon_1) = \text{cov}(Y, \varepsilon_2) = 0$ and $\alpha = r_{XY} = \beta\sigma_X^2/\sigma_Y^2$. Thus, if the condition $\text{cov}(X, \varepsilon_1) = 0$ endows β with causal meaning, then $\text{cov}(Y, \varepsilon_2) = 0$ ought to endow α

with causal meaning as well. But this, too, conflicts with both intuition and the intentions behind SEM; the change in $E(X)$ per unit change of Y ought to be zero, not r_{XY}, if there is no causal path from Y to X.

What then *is* the meaning of a structural coefficient? Or a structural equation? Or an error term? The interventional interpretation of causal effects, when coupled with the $do(x)$ notation, provides simple answers to these questions. The answers explicate the operational meaning of structural equations and thus should end, I hope, an era of controversy and confusion regarding these entities.

Structural Equations: Operational Definition

Definition 5.4.1 (Structural Equations)
An equation $y = \beta x + \varepsilon$ is said to be structural *if it is to be interpreted as follows: In an ideal experiment where we control X to x and any other set Z of variables (not containing X or Y) to z, the value y of Y is given by $\beta x + \varepsilon$, where ε is not a function of the settings x and z.*

This definition is operational because all quantities are observable, albeit under conditions of controlled manipulation. That manipulations cannot be performed in most observational studies does not negate the operationality of the definition, much as our inability to observe bacteria with the naked eye does not negate their observability under a microscope. The challenge of SEM is to extract the maximum information concerning what we wish to observe from the little we actually can observe.

Note that the operational reading just given makes no claim about how X (or any other variable) will behave when we control Y. This asymmetry makes the equality signs in structural equations different from algebraic equality signs; the former act symmetrically in relating observations on X and Y (e.g., observing $Y = 0$ implies $\beta x = -\varepsilon$), but they act asymmetrically when it comes to interventions (e.g., setting Y to zero tells us nothing about the relation between x and ε). The arrows in path diagrams make this dual role explicit, and this may account for the insight and inferential power gained through the use of diagrams.

The strongest empirical claim of the equation $y = \beta x + \varepsilon$ is made by excluding other variables from the r.h.s. of the equation, thus proclaiming X the *only* immediate cause of Y. This translates into a testable claim of *invariance:* the statistics of Y under condition $do(x)$ should remain invariant to the manipulation of any other variable in the model (see Section 1.3.2).[16] This claim can be written symbolically as

$$P(y \mid do(x), do(z)) = P(y \mid do(x)) \tag{5.23}$$

for all Z disjoint of $\{X \cup Y\}$.[17]

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[16] The basic notion that structural equations remain invariant to certain changes in the system goes back to Marschak (1950) and Simon (1953), and it has received mathematical formulation at various levels of abstraction in Hurwicz (1962), Mesarovic (1969), Sims (1977), Cartwright (1989), Hoover (1990), and Woodward (1995). The simplicity, precision, and clarity of (5.23) is unsurpassed, however.

[17] This claim is, in fact, only part of the message conveyed by the equation; the other part consists of a dynamic or counterfactual claim: If we were to control X to x' instead of x, then Y would attain

Note that this invariance holds relative to manipulations, not observations, of Z. The statistics of Y under condition $do(x)$ given the measurement $Z = z$, written $P(y \mid do(x), z)$, would certainly depend on z if the measurement were taken on a consequence (i.e. descendant) of Y. Note also that the ordinary conditional probability $P(y \mid x)$ does not enjoy such a strong property of invariance, since $P(y \mid x)$ is generally sensitive to manipulations of variables other than X in the model (unless X and ε are independent). Equation (5.23), in contrast, remains valid regardless of the statistical relationship between ε and X.

Generalized to a set of several structural equations, (5.23) explicates the assumptions underlying a given causal diagram. If G is the graph associated with a set of structural equations, then the assumptions are embodied in G as follows: (1) every missing arrow – say, between X and Y – represents the assumption that X has no causal effect on Y once we intervene and hold the parents of Y fixed; and (2) every missing bidirected link between X and Y represents the assumption that the omitted factors that (directly) influence X are uncorrelated with those that (directly) influence Y. We shall define the operational meaning of the latter assumption in (5.25)–(5.27).

The Structural Parameters: Operational Definition

The interpretation of a structural equation as a statement about the behavior of Y under a hypothetical intervention yields a simple definition for the structural parameters. The meaning of β in the equation $y = \beta x + \varepsilon$ is simply

$$\beta = \frac{\partial}{\partial x} E[Y \mid do(x)], \tag{5.24}$$

that is, the rate of change (relative to x) of the expectation of Y in an experiment where X is held at x by external control. This interpretation holds regardless of whether ε and X are correlated in nonexperimental studies (e.g., via another equation $x = \alpha y + \delta$).

We hardly need to add at this point that β has nothing to do with the regression coefficient r_{YX} or, equivalently, with the conditional expectation $E(Y \mid x)$, as suggested in many textbooks. The conditions under which β coincides with the regression coefficient are spelled out in Theorem 5.3.1.

It is important nevertheless to compare the definition of (5.24) with theories that acknowledge the invariant character of β but have difficulties explicating which changes β is invariant to. Cartwright (1989, p. 194), for example, characterizes β as an invariant of nature that she calls "capacity." She states correctly that β remains constant under change but explains that, as the statistics of X changes, "it is the ratio [$\beta = E(YX)/E(X^2)$] which remains fixed no matter how the variances shift." This characterization is imprecise on two accounts. First, β may in general not be equal to the stated ratio nor to any other combination of statistical parameters. Second – and this is the main point of Definition 5.4.1 – structural parameters are invariant to local interventions (i.e., changes in

the value $\beta x' + \varepsilon$. In other words, plotting the value of Y under various hypothetical controls of X, and under the same external conditions (ε), should result in a straight line with slope β. Such deterministic dynamic claims concerning system behavior under successive control conditions can only be tested under the assumption that ε, representing external conditions or properties of experimental units, remains unaltered as we switch from x to x'. Such counterfactual claims constitute the empirical content of every scientific law (see Section 7.2.2).

specific equations in the system) and not to general changes in the statistics of the variables. If we start with $\text{cov}(X, \varepsilon) = 0$ and the variance of X changes because we (or Nature) locally modify the *process* that generates X, then Cartwright is correct; the ratio $\beta = E(YX)/E(X^2)$ will remain constant. However, if the variance of X changes for any other reason – say, because we observed some evidence $Z = z$ that depends on both X and Y or because the process generating X becomes dependent on a wider set of variables – then that ratio will not remain constant.

The Mystical Error Term: Operational Definition

The interpretations given in Definition 5.4.1 and (5.24) provide an operational definition for that mystical error term

$$\varepsilon = y - E[Y \mid do(x)], \tag{5.25}$$

which, despite being unobserved in nonmanipulative studies, is far from being metaphysical or definitional as suggested by some researchers (e.g. Richard 1980; Holland 1988, p. 460; Hendry 1995, p. 62). Unlike errors in regression equations, ε measures the deviation of Y from its controlled expectation $E[Y \mid do(x)]$ and not from its conditional expectation $E[Y \mid x]$. The statistics of ε can therefore be measured from observations on Y once X is controlled. Alternatively, because β remains the same regardless of whether X is manipulated or observed, the statistics of $\varepsilon = y - \beta x$ can be measured in observational studies if we know β.

Likewise, correlations among errors can be estimated empirically. For any two nonadjacent variables X and Y, (5.25) yields

$$E[\varepsilon_Y \varepsilon_X] = E[YX \mid do(pa_Y, pa_X)] - E[Y \mid do(pa_Y)]E[X \mid do(pa_X)]. \tag{5.26}$$

Once we have determined the structural coefficients, the controlled expectations $E[Y \mid do(pa_Y)]$, $E[X \mid do(pa_X)]$, and $E[YX \mid do(pa_Y, pa_X)]$ become known linear functions of the observed variables pa_Y and pa_X; hence, the expectations on the r.h.s. of (5.26) can be estimated in observational studies. Alternatively, if the coefficients are not determined, then the expression can be assessed directly in interventional studies by holding pa_X and pa_Y fixed (assuming X and Y are not in parent–child relationship) and estimating the covariance of X and Y from data obtained under such conditions.

Finally, we are often interested not in assessing the numerical value of $E[\varepsilon_Y \varepsilon_X]$ but rather in determining whether ε_Y and ε_X can be assumed to be uncorrelated. For this determination, it suffices to test whether the equality

$$E[Y \mid x, do(s_{XY})] = E[Y \mid do(x), do(s_{XY})] \tag{5.27}$$

holds true, where s_{XY} stands for (any setting of) all variables in the model excluding X and Y. This test can be applied to any two variables in the model *except* when Y is a parent of X, in which case the symmetrical equation (with X and Y interchanged) is applicable.

The Mystical Error Term: Conceptual Interpretation

The authors of SEM textbooks usually interpret error terms as representing the influence of omitted factors. Many SEM researchers are reluctant to accept this interpretation,

however, partly because unspecified omitted factors open the door to metaphysical speculations and partly because arguments based on such factors were improperly used as a generic, substance-free license to omit bidirected arcs from path diagrams (McDonald 1997). Such concerns are answered by the operational interpretation of error terms, (5.25), since it prescribes how errors are measured, not how they originate.

It is important to note, though, that this operational definition is no substitute for the omitted-factors conception when it comes to deciding whether pairs of error terms can be assumed to be uncorrelated. Because such decisions are needed at a stage when the model's parameters are still "free," they cannot be made on the basis of numerical assessments of correlations but must rest instead on qualitative structural knowledge about how mechanisms are tied together and how variables affect each other. Such judgmental decisions are hardly aided by the operational criterion of (5.26), which instructs the investigator to assess whether two deviations – taken on two different variables under complex experimental conditions – would be correlated or uncorrelated. Such assessments are cognitively unfeasible.

In contrast, the omitted-factors conception instructs the investigator to judge whether there could be factors that simultaneously influence several observed variables. Such judgments are cognitively manageable because they are qualitative and rest on purely structural knowledge – the only knowledge available during this phase of modeling.

Another source of error correlation that should be considered by investigators is *selection bias.* If two uncorrelated unobserved factors have a common effect that is omitted from the analysis but influences the selection of samples for the study, then the corresponding error terms will be correlated in the sampled population; hence, the expectation in (5.26) will not vanish when taken over the sampled population (see discussion of Berkson's paradox in Section 1.2.3).

We should emphasize, however, that the arcs *missing* from the diagram, not those *in* the diagram, demand the most attention and careful substantive justification. Adding an extra bidirected arc can at worst compromise the identifiability of parameters, but deleting an existing bidirected arc may produce erroneous conclusions as well as a false sense of model testability. Thus, bidirected arcs should be assumed to exist, by default, between any two nodes in the diagram. They should be deleted only by well-motivated justifications, such as the unlikely existence of a common cause for the two variables and the unlikely existence of selection bias. Although we can never be cognizant of all the factors that may affect our variables, substantive knowledge sometimes permits us to state that the influence of a possible common factor is not likely to be significant.

Thus, as often happens in the sciences, the way we measure physical entities does not offer the best way of thinking about them. The omitted-factor conception of errors, because it rests on structural knowledge, is a more useful guide than the operational definition when building, evaluating, and thinking about causal models.

5.4.2 Interpretation of Effect Decomposition

Structural equation modeling prides itself, and rightly so, for providing principled methodology for distinguishing direct from indirect effects. We have seen in Section 4.5 that such distinction is important in many applications, ranging from process control to legal disputes, and that SEM indeed provides a coherent methodology of defining, identifying, and

estimating direct and indirect effects. However, the reluctance of most SEM researchers to admit the causal reading of structural parameters – coupled with their preoccupation with algebraic manipulations – has resulted in inadequate definitions of direct and indirect effects, as pointed out by Freedman (1987) and Sobel (1990). In this section we hope to correct this confusion by adhering to the operational meaning of the structural coefficients.

We start with the general notion of a causal effect $P(y \mid do(x))$ as in Definition 3.2.1. We then specialize it to define direct effect, as in Section 4.5, and finally express the definitions in terms of structural coefficients.

Definition 5.4.2 (Total Effect)
The total effect *of X on Y is given by* $P(y \mid do(x))$, *namely, the distribution of Y while X is held constant at x and all other variables are permitted to run their natural course.*

Definition 5.4.3 (Direct Effect)
The direct effect *of X on Y is given by* $P(y \mid do(x), do(s_{XY}))$, *where* S_{XY} *is the set of all observed variables in the system except X and Y.*

In linear analysis, Definitions 5.4.2 and 5.4.3 yield, after differentiation with respect to x, the familiar path coefficients in terms of which direct and indirect effects are usually defined. Yet they differ from conventional definitions in several important aspects. First, direct effects are defined in terms of hypothetical experiments in which intermediate variables are held constant by *physical intervention,* not by statistical adjustment (which is often disguised under the misleading phrase "control for"). Figure 5.10 depicts a simple example where adjusting for the intermediate variables (Z and W) would not give the correct value of zero for the direct effect of X on Y, whereas $\frac{\partial}{\partial_x} E(Y \mid do(x, y, w))$ does yield the correct value: $\frac{\partial}{\partial_x}(\beta w + \gamma z) = 0$. Section 4.5.3 (Table 4.1) provides another such example, one that involves dichotomous variables.

Second, there is no need to limit control to only intermediate variables; *all* variables in the system may be held constant (except for X and Y). Hypothetically, the scientist controls for all possible conditions S_{XY}, and measurements may commence without knowing the structure of the diagram. Finally, our definitions differ from convention by interpreting total and direct effects independently of each other, as outcomes of two different experiments. Textbook definitions (e.g. Bollen 1989, p. 376; Mueller 1996, p. 141; Kline 1998, p. 175) usually equate the total effect with a power series of path coefficient matrices. This algebraic definition coincides with the operational definition (Definition 5.4.2) in recursive (semi-Markovian) systems, but it yields erroneous expressions in models with feedback. For instance, given the pair of equations $\{y = \beta x + \varepsilon,\ x = \alpha y + \delta\}$, the total effect of X on Y is simply β, not $\beta(1 - \alpha\beta)^{-1}$ as stated in Bollen (1989, p. 379). The latter has no operational significance worthy of the phrase "effect of X."[18]

We end this section of effect decomposition with a few remarks that should be of interest to researchers dealing with dichotomous variables. The relations among such

[18] This error was noted by Sobel (1990) but, perhaps because constancy of path coefficients was presented as a new and extraneous assumption, Sobel's correction has not brought about a shift in practice or philosophy.

variables are usually nonlinear, so the results of Section 4.5 should be applicable. In particular, the direct effect of X on Y will depend on the levels at which we hold the other parents of Y. If we wish to average over these values, we obtain the expression given in Section 4.5.4.

In standard linear analysis, an indirect effect may be defined as the difference between the total effect and the direct effects (Bollen 1989). In nonlinear analysis, differences lose their significance, and one must isolate the contribution of mediating paths in some other way. Expressions of the form $P(y \mid do(x), do(z))$ cannot be used to isolate such contributions because there is no physical means of selectively disabling a direct causal link from X to Y by holding some variables constant. This suggests that the notion of indirect effect has no intrinsic operational meaning apart from providing a comparison between the direct and the total effects. In other words, a policy maker who asks for that part of the total effect transmitted by a particular intermediate variable or by a group Z of such variables is really asking for a comparison of the effects of two policies, one where Z is held constant versus the other where it is not. The expressions corresponding to these policies are $P(y \mid do(x), do(z))$ and $P(y \mid do(x))$, and this pair of distributions should be taken as the most general representation of indirect effects. Similar conclusions have been expressed by Robins (1986) and Robins and Greenland (1992).

5.4.3 Exogeneity, Superexogeneity, and Other Frills

Economics textbooks invariably warn readers that the distinction between exogenous and endogenous variables is, on the one hand, "most important for model building" (Darnell 1994, p. 127) and, on the other hand, "a subtle and sometimes controversial complication" (Greene 1997, p. 712). Economics students would naturally expect the concepts and tools developed in this chapter to shed some light on the subject, and rightly so. We next offer a simple definition of exogeneity that captures the important nuances appearing in the literature and that is both palatable and precise.

It is fashionable today to distinguish three types of exogeneity: weak, strong, and super (Engle et al. 1983); the former two are statistical and the latter causal. However, the importance of exogeneity – and the reason for its controversial status – lies in its implications for policy interventions. Some economists believe, therefore, that only the causal aspect (i.e. superexogeneity) deserves the exogenous title and that the statistical versions are unwarranted intruders that tend to confuse issues of identification and interpretability with those of estimation efficiency (Ed Leamer, personal communication).[19] I will serve both camps by starting with a simple definition of causal exogeneity and then offering a more general definition, from which both the causal and the statistical aspects would follow as special cases. Thus, what we call "exogeneity" corresponds to what Engle et al. called "superexogeneity," a notion that captures economists' interest in the structural invariance of certain relationships under policy intervention.

Suppose that we consider intervening on a set of variables X and that we wish to characterize the statistical behavior of a set Y of outcome variables under the intervention

[19] Similiar opinions have also been communicated by John Aldrich and James Heckman. See also Aldrich (1993).

$do(X = x)$. Denote the postintervention distribution of Y by the usual expression $P(y \mid do(x))$. If we are interested in a set λ of parameters of that distribution, then our task is to estimate $\lambda[P(y \mid do(x)]$ from the available data. However, the data available is typically generated under a different set of conditions: X was not held constant but instead was allowed to vary with whatever economical pressures and expectations prompted decision makers to set X in the past. Denoting the process that generated data in the past by M and the probability distribution associated with M by $P_M(v)$, we ask whether $\lambda[P_M(y \mid do(x)]$ can be estimated consistently from samples drawn from $P_M(v)$, given our background knowledge T (connoting "theory") about M. This is essentially the problem of identification that we have analyzed in this and previous chapters, with one important difference; we now ask whether $\lambda[P(y \mid do(x)]$ can be identified from the conditional distribution $P(y \mid x)$ alone, instead of from the entire joint distribution $P(v)$. When identification holds under this restricted condition, X is said to be *exogenous* relative to (Y, λ, T).

We may state this formally as follows.

Definition 5.4.4 (Exogeneity)
Let X and Y be two sets of variables, and let λ be any set of parameters of the postintervention probability $P(y \mid do(x))$. We say that X is exogenous *relative to (Y, λ, T) if λ is identifiable from the conditional distribution $P(y \mid x)$, that is, if*

$$P_{M_1}(y \mid x) = P_{M_2}(y \mid x) \implies \lambda[P_{M_1}(y \mid do(x))] = \lambda[P_{M_2}(y \mid do(x))] \tag{5.28}$$

for any two models, M_1 and M_2, satisfying theory T.

In the special case where λ constitutes a complete specification of the postintervention probabilities, (5.28) reduces to the implication

$$P_{M_1}(y \mid x) = P_{M_2}(y \mid x) \implies P_{M_1}(y \mid do(x)) = P_{M_2}(y \mid do(x)). \tag{5.29}$$

If we further assume that, for every $P(y \mid x)$, our theory T does not a priori exclude some model M_2 satisfying $P_{M_2}(y \mid do(x)) = P_{M_2}(y \mid x)$,[20] then (5.29) reduces to the equality

$$P(y \mid do(x)) = P(y \mid x), \tag{5.30}$$

a condition we recognize as "no confounding" (see Sections 3.3 and 6.2). Equation (5.30) follows (from (5.29)) because (5.29) must hold for all M_1 in T. Note that, since the theory T is not mentioned explicitly, (5.30) can be applied to any individual model M and can be taken as yet another definition of exogeneity – albeit a stronger one than (5.28).

The motivation for insisting that λ be identifiable from the conditional distribution $P(y \mid x)$ alone, even though the marginal distribution $P(x)$ is available, lies in its ramification for the process of estimation. As stated in (5.30), discovering that X is exogenous

[20] For example, if T stands for all models possessing the same graph structure, then such M_2 is not a priori excluded.

permits us to predict the effect of interventions (in X) directly from passive observations, without even adjusting for confounding factors. Our analyses in Sections 3.3 and 5.3 further provide a graphical test of exogeneity: X is exogenous for Y if there is no unblocked back-door path from X to Y (Theorem 5.3.2). This test supplements the declarative definition of (5.30) with a procedural definition and thus completes the formalization of exogeneity. That the invariance properties usually attributable to superexogeneity are discernible from the topology of the causal diagram should come as no surprise, considering that each causal diagram represents a structural model and that each structural model already embodies the invariance assumptions necessary for policy predictions (see Definition 5.4.1).

Leamer (1985) defined X to be exogenous if $P(y \mid x)$ remains invariant to changes in the "process that generates" X. This definition coincides[21] with (5.30) because $P(y \mid do(x))$ is governed by a structural model in which the equations determining X are wiped out; thus, $P(y \mid x)$ must be insensitive to the nature of those equations. In contrast, Engle et al. (1983) defined exogeneity (i.e., their superexogeneity) in terms of changes in the "marginal density" of X; as usual, the transition from process language to statistical terminology leads to ambiguities. According to Engle et al. (1983, p. 284), exogeneity requires that all the parameters of the conditional distribution $P(y \mid x)$ be "invariant for any change in the distribution of the conditioning variables"[22] (i.e. $P(x)$). This requirement of constancy under *any* change in $P(x)$ is too strong – changing conditions or new observations can easily alter both $P(x)$ and $P(y \mid x)$ even when X is perfectly exogenous. (To illustrate, consider a change that turns a randomized experiment, where X is indisputably exogenous, into a nonrandomized experiment; we should not insist on $P(y \mid x)$ remaining invariant under such change.) The class of changes considered must be restricted to local modification of the mechanisms (or equations) that determine X, as stated by Leamer, and this restriction must be incorporated into any definition of exogeneity. In order to make this restriction precise, however, the vocabulary of SEMs must be invoked as in the definition of $P(y \mid do(x))$; the vocabulary of marginal and conditional densities is far too coarse to properly define the changes against which $P(y \mid x)$ ought to remain invariant.

We are now ready to define a more general notion of exogeneity, one that includes "weak" and "super" exogeneities under the same umbrella.[23] Toward that end, we remove from Definition 5.4.4 the restriction that λ must represent features of the postintervention distribution. Instead, we allow λ to represent *any* feature of the underlying model M, including structural features such as path coefficients, causal effects, and counterfactuals, and including statistical features (which could, of course, be ascertained from the joint distribution alone). With this generalization, we also obtain a simpler definition of exogeneity.

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[21] Provided that changes are confined to modification of functions without changing the set of arguments (i.e. parents) in each function.

[22] This requirement is repeated verbatim in Darnell (1994, p. 131) and Maddala (1992, p. 192).

[23] We leave out discussion of "strong" exogeneity, which is a slightly more involved version of weak exogeneity applicable to time-series analysis.

Definition 5.4.5 (General Exogeneity)
Let X and Y be two sets of variables, and let λ be any set of parameters defined on a structural model M in a theory T. We say that X is exogenous *relative to (Y, λ, T) if λ is identifiable from the conditional distribution $P(y \mid x)$, that is, if*

$$P_{M_1}(y \mid x) = P_{M_2}(y \mid x) \implies \lambda(M_1) = \lambda(M_2) \tag{5.31}$$

for any two models, M_1 and M_2, satisfying theory T.

When λ consists of structural parameters, such as path coefficients or causal effects, (5.31) expresses invariance to a variety of interventions, not merely $do(X = x)$. Although the interventions themselves are not mentioned explicitly in (5.31), the equality $\lambda(M_1) = \lambda(M_2)$ reflects such interventions through the structural character of λ. In particular, if λ stands for the values of the causal effect function $P(y \mid do(x))$ at selected points of x and y, then (5.31) reduces to the implication

$$P_{M_1}(y \mid x) = P_{M_2}(y \mid x) \implies P_{M_1}(y \mid do(x)) = P_{M_2}(y \mid do(x)), \tag{5.32}$$

which is identical to (5.29). Hence the causal properties of exogeneity follow.

When λ consists of strictly statistical parameters – such as means, modes, regression coefficients, or other distributional features – the structural features of M do not enter into consideration; we have $\lambda(M) = \lambda(P_M)$ and so (5.31) reduces to

$$P_1(y \mid x) = P_2(y \mid x) \implies \lambda(P_1) = \lambda(P_2) \tag{5.33}$$

for any two probability distributions $P_1(x, y)$ and $P_2(x, y)$ that are consistent with T. We have thus obtained a statistical notion of exogeneity that permits us to ignore the marginal $P(x)$ in the estimation of λ and that we may call "weak exogeneity."[24]

Finally, if λ consists of causal effects among variables in Y (excluding X), we obtain a generalized definition of *instrumental variables.* For example, if our interest lies in the causal effect $\lambda = P(w \mid do(z))$, where W and Z are two sets of variables in Y, then the exogeneity of X relative to this parameter ensures the identification of $P(w \mid do(z))$ from the conditional probability $P(z, w \mid x)$. This is indeed the role of an instrumental variable – to assist in the identification of causal effects not involving the instrument. (See Figure 5.9, with Z, X, Y representing X, Z, W, respectively.)

A word of caution regarding the language used in most textbooks: exogeneity is frequently defined by asking whether parameters "enter" into the expressions of the conditional or the marginal density. For example, Maddala (1992, p. 392) defined weak exogeneity as the requirement that the marginal distribution $P(x)$ "does not involve" λ. Such definitions are not unambiguous, because the question of whether a parameter "enters" a density or whether a density "involves" a parameter are syntax-dependent; different algebraic representations may make certain parameters explicit or obscure. For example,

[24] Engle et al. (1983) further imposed a requirement called "variation-free," which is satisfied by default when dealing with genuinely structural models M in which mechanisms do not constrain one another.

if X and Y are dichotomous, then the marginal probability $P(x)$ certainly "involves" parameters such as

$$\lambda_1 = P(x_0, y_0) + P(x_0, y_1) \quad \text{and} \quad \lambda_2 = P(x_0, y_0),$$

as well as their ratio:

$$\lambda = \lambda_2/\lambda_1.$$

Therefore, writing $P(x_0) = \lambda_2/\lambda$ shows that both λ and λ_2 are involved in the marginal probability $P(x_0)$, and one may be tempted to conclude that X is not exogenous relative to λ. Yet X *is* in fact exogenous relative to λ, because the ratio $\lambda = \lambda_2/\lambda_1$ is none other than $P(y_0 \mid x_0)$; hence it is determined uniquely by $P(y_0 \mid x_0)$ as required by (5.33).[25]

The advantage of the definition given in (5.31) is that it depends not on the syntactic representation of the density function but rather on its semantical content alone. Parameters are treated as quantities *computed from* a model, and not as mathematical symbols that *describe* a model. Consequently, the definition applies to both statistical and structural parameters and, in fact, to any quantity λ that can be computed from a structural model M, regardless of whether it serves (or may serve) in the description of the marginal or conditional densities.

The Mystical Error Term Revisited

Historically, the definition of exogeneity that has evoked most controversy is the one expressed in terms of correlation between variables and errors. It reads as follows.

Definition 5.4.6 (Error-Based Exogeneity)
A variable X is exogenous (relative to $\lambda = P(y \mid do(x))$) if X is independent of all errors that influence Y, except those mediated by X.

This definition, which Hendry and Morgan (1995) trace to Orcutt (1952), became standard in the econometric literature between 1950 and 1970 (e.g. Christ 1966, p. 156; Dhrymes 1970, p. 169) and still serves to guide the thoughts of most econometricians (as in the selection of instrumental variables; Bowden and Turkington 1984). However, it came under criticism in the early 1980s when the distinction between structural errors (equation (5.25)) and regression errors became obscured (Richard 1980). (Regression errors, by definition, are orthogonal to the regressors.) The Cowles Commission logic of structural equations (see Section 5.1) has not reached full mathematical maturity and – by denying notational distinction between structural and regressional parameters – has left all notions based on error terms suspect of ambiguity. The prospect of establishing an entirely new foundation of exogeneity – seemingly free of theoretical terms such as "errors" and "structure" (Engle et al. 1983) – has further dissuaded economists from tidying up the Cowles Commission logic, and criticism of the error-based definition of exogeneity has become increasingly fashionable. For example, Hendry and Morgan (1995) wrote that

[25] Engle et al. (1983, p. 281) and Hendry (1995, pp. 162–3) attempted to overcome this ambiguity by using "reparameterization" – an unnecessary complication.

"the concept of exogeneity rapidly evolved into a loose notion as a property of an observable variable being uncorrelated with an unobserved error," and Imbens (1997) readily agreed that this notion "is inadequate."[26]

These critics are hardly justified if we consider the precision and clarity with which structural errors can be defined when using the proper notation (e.g. (5.25)). When applied to structural errors, the standard error-based criterion of exogeneity coincides formally with that of (5.30), as can be verified using the back-door test of Theorem 5.3.2 (with $Z = \emptyset$). Consequently, the standard definition conveys the same information as that embodied in more complicated and less communicable definitions of exogeneity. I am therefore convinced that the standard definition will eventually regain the acceptance and respectability that it has always deserved.

Relationships between graphical and counterfactual definitions of exogeneity and instrumental variables will be discussed in Chapter 7 (Section 7.4.5).

5.5 CONCLUSION

Today the enterprise known as structural equation modeling is increasingly under fire. The founding fathers have retired, their teachings are forgotten, and practitioners, teachers, and researchers currently find the methodology they inherited difficult to either defend or supplant. Modern SEM textbooks are preoccupied with parameter estimation and rarely explicate the role that those parameters play in causal explanations or in policy analysis; examples dealing with the effects of interventions are conspicuously absent, for instance. Research in SEM now focuses almost exclusively on model fitting, while issues pertaining to the meaning and usage of SEM's models are subjects of confusion and controversy.

I am thoroughly convinced that the contemporary crisis in SEM originates in the lack of a mathematical language for handling the causal information embedded in structural equations. Graphical models have provided such a language. They have thus helped us answer many of the unsettled questions that drive the current crisis:

1. Under what conditions can we give causal interpretation to structural coefficients?
2. What are the causal assumptions underlying a given structural equation model?
3. What are the statistical implications of any given structural equation model?
4. What is the operational meaning of a given structural coefficient?
5. What are the policy-making claims of any given structural equation model?
6. When is an equation not structural?

This chapter has described the conceptual developments that now resolve such foundational questions. In addition, we have presented several tools to be used in answering questions of practical importance:

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[26] Imbens prefers definitions in terms of experimental metaphors such as "random assignment assumption," fearing, perhaps, that "[t]ypically the researcher does not have a firm idea what these disturbances really represent" (Angrist et al. 1996, p. 446).

1. When are two structural equation models observationally indistinguishable?
2. When do regression coefficients represent path coefficients?
3. When would the addition of a regressor introduce bias?
4. How can we tell, prior to collecting any data, which path coefficients can be identified?
5. When can we dispose of the linearity–normality assumption and still extract causal information from the data?

I remain hopeful that researchers will recognize the benefits of these concepts and tools and use them to revitalize causal analysis in the social and behavioral sciences.

Acknowledgments

This chapter owes its inspiration to the generations of statisticians who have asked, with humor and disbelief, how SEM's methodology could make sense to any rational being – and to the social scientists who (perhaps unwittingly) have saved the SEM tradition from drowning in statistical interpretations. The comments of Herman Ader, Peter Bentler, Jacques Hagenaars, Rod McDonald, and Stan Mulaik have helped me gain a greater understanding of SEM practice and vocabulary. John Aldrich, Arthur Goldberger, James Heckman, Kevin Hoover, Ed Leamer, and Herbert Simon helped me penetrate the mazes of structural equations and exogeneity in econometrics. Jin Tian was instrumental in revising Sections 5.2.3 and 5.3.1.

CHAPTER SIX

Simpson's Paradox, Confounding, and Collapsibility

He who confronts the paradoxical
exposes himself to reality.
Friedrick Durrenmatt (1962)

Preface

Confounding represents one of the most fundamental impediments to the elucidation of causal inferences from empirical data. As a result, the consideration of confounding underlies much of what has been written or said in areas that critically rely on causal inferences; this includes epidemiology, econometrics, biostatistics, and the social sciences. Yet, apart from the standard analysis of randomized experiments, the topic is given little or no discussion in most statistics texts. The reason for this is simple: confounding is a causal concept and hence cannot be expressed in standard statistical models. When formal statistical analysis is attempted, it often leads to confusions or complexities that make the topic extremely hard for the nonexpert to comprehend, let alone master.

One of my main objectives in writing this book is to see these confusions resolved – to see problems involving the control of confounding reduced to simple mathematical routines. The mathematical techniques introduced in Chapter 3 have indeed culminated in simple graphical routines of detecting the presence of confounding and of identifying variables that need be controlled in order to obtain unconfounded effect estimates. In this chapter, we address the difficulties encountered when we attempt to define and control confounding by using statistical criteria.

We start by analyzing the interesting history of Simpson's paradox (Section 6.1) and use it as a magnifying glass to examine the difficulties that generations of statisticians have had in their attempts to capture causal concepts in the language of statistics. In Sections 6.2 and 6.3, we examine the feasibility of replacing the causal definition of confounding with statistical criteria that are based solely on frequency data and measurable statistical associations. We will show that, although such replacement is generally not feasible (Section 6.3), a certain kind of nonconfounding conditions, called *stable*, can be given statistical or semistatistical characterization (Section 6.4). This characterization leads to operational tests, similar to collapsibility tests, that can alert investigators to the existence of either instability or bias in a given effect estimate (Section 6.4.3). Finally, Section 6.5 clarifies distinctions between collapsibility and no-confounding, confounders and confounding, and between the structural and exchangeability approaches to representing problems of confounding.

6.1 SIMPSON'S PARADOX: AN ANATOMY

The reversal effect known as Simpson's paradox has been briefly discussed twice in this book: first in connection with the covariate selection problem (Section 3.3) and then in connection with the definition of direct effects (Section 4.5.3). In this section we analyze the reasons why the reversal effect has been (and still is) considered paradoxical and why its resolution has been so late in coming.

6.1.1 A Tale of a Non-Paradox

Simpson's paradox (Simpson 1951; Blyth 1972), first encountered by Pearson in 1899 (Aldrich 1995), refers to the phenomenon whereby an event C increases the probability of E in a given population p and, at the same time, decreases the probability of E in every subpopulation of p. In other words, if F and $\neg F$ are two complementary properties describing two subpopulations, we might well encounter the inequalities

$$P(E \mid C) > P(E \mid \neg C), \tag{6.1}$$

$$P(E \mid C, F) < P(E \mid \neg C, F), \tag{6.2}$$

$$P(E \mid C, \neg F) < P(E \mid \neg C, \neg F). \tag{6.3}$$

Although such order reversal might not surprise students of probability, it is paradoxical when given causal interpretation. For example, if we associate C (connoting *cause*) with taking a certain drug, E (connoting *effect*) with recovery, and F with being a female, then – under the causal interpretation of (6.2)–(6.3) – the drug seems to be harmful to both males and females yet beneficial to the population as a whole (equation (6.1)). Intuition deems such a result impossible, and correctly so.

The tables in Figure 6.1 represent Simpson's reversal numerically. We see that, overall, the recovery rate for patients receiving the drug (C) at 50% exceeds that of the control ($\neg C$) at 40% and so the drug treatment is apparently to be preferred. However, when we inspect the separate tables for males and females, the recovery rate for the untreated patients is 10% higher than that for the treated ones, for males and females both.

The explanation for Simpson's paradox should be clear to readers of this book, since we have taken great care in distinguishing *seeing* from *doing*. The conditioning operator in probability calculus stands for the evidential conditional "given that we see," whereas the $do(\cdot)$ operator was devised to represent the causal conditional "given that we do." Accordingly, the inequality

$$P(E \mid C) > P(E \mid \neg C)$$

is not a statement about C being a positive causal factor for E, properly written

$$P(E \mid do(C)) > P(E \mid do(\neg C)),$$

but rather about C being positive *evidence* for E, which may be due to spurious confounding factors that cause both C and E. In our example, the drug appears beneficial

	Combined	E	$\neg E$		Recovery Rate
(a)	Drug (C)	20	20	40	50%
	No drug ($\neg C$)	16	24	40	40%
		36	44	80	
	Males	E	$\neg E$		Recovery Rate
(b)	Drug (C)	18	12	30	60%
	No drug ($\neg C$)	7	3	10	70%
		25	15	40	
	Females	E	$\neg E$		Recovery Rate
(c)	Drug (C)	2	8	10	20%
	No drug ($\neg C$)	9	21	30	30%
		11	29	40	

Figure 6.1 Recovery rates under treatment (C) and control ($\neg C$) for males, females, and combined.

overall because the males, who recover (regardless of the drug) more often than the females, are also more likely than the females to use the drug. Indeed, finding a drug-using patient (C) of unknown gender, we would do well inferring that the patient is more likely to be a male and hence more likely to recover, in perfect harmony with (6.1)–(6.3).

The standard method for dealing with potential confounders of this kind is to "hold them fixed,"[1] namely, to condition the probabilities on any factor that might cause both C and E. In our example, if being a male ($\neg F$) is perceived to be a cause for both recovery (E) and drug usage (C), then the effect of the drug needs to be evaluated separately for men and women (as in (6.2)–(6.3)) and then averaged accordingly. Thus, assuming F is the only confounding factor, (6.2)–(6.3) properly represent the efficacy of the drug in the respective populations while (6.1) represents merely its evidential weight in the absence of gender information, and the paradox dissolves.

6.1.2 A Tale of Statistical Agony

Thus far, we have described the paradox as it is understood, or should be understood, by modern students of causality (see e.g. Cartwright 1983;[2] Holland and Rubin 1983; Greenland and Robins 1986; Pearl 1993; Spirtes et al. 1993; Meek and Glymour 1994). Most

[1] The phrases "hold F fixed" and "control for F," used both by philosophers (e.g. Eells 1991) and statisticians (e.g. Pratt and Schlaifer 1988), connote external interventions and may therefore be misleading. In statistical analysis, all one can do is *simulate* "holding F fixed" by considering cases with equal values of F – that is, "conditioning" on F and $\neg F$ – an operation that I will call "adjusting for F."

[2] Cartwright states, though, that the third factor F should be "held fixed" if and only if F is causally relevant to E (p. 37); the correct (back-door) criterion is somewhat more involved (see Definition 3.3.1).

statisticians, however, are reluctant to entertain the idea that Simpson's paradox emerges from causal considerations. The general attitude is as follows: The reversal is real and disturbing, because it actually shows up in the numbers and may actually mislead statisticians into incorrect conclusions. If something is real then it cannot be causal, because causality is a mental construct that is not well-defined. Thus, the paradox must be a statistical phenomenon that can be detected, understood, and avoided using the tools of statistical analysis. *The Encyclopedia of Statistical Sciences,* for example, warns us sternly of the dangers lurking from Simpson's paradox with no mention of the words "cause" or "causality" (Agresti 1983). *The Encyclopedia of Biostatistics* (Dong 1998) and *The Cambridge Dictionary of Statistics in Medical Sciences* (Everitt 1995) uphold the same conception.

I know of only two articles in the statistical literature that explicitly attribute the peculiarity of Simpson's reversal to causal interpretations. The first is Pearson et al. (1899), where the discovery of the phenomenon[3] is enunciated in these terms:

> To those who persist on looking upon all correlation as cause and effect, the fact that correlation can be produced between two quite uncorrelated characters A and B by taking an artificial mixture of the two closely allied races, must come as rather a shock.

Influenced by Pearson's life-long campaign, statisticians have refrained from causal talk whenever possible and, for over half a century, the reversal phenomenon has been treated as a curious mathematical property of 2×2 tables, stripped of its causal origin. Finally, Lindley and Novick (1981) analyzed the problem from a new angle, and made the second published connection to causality:

> In the last paragraph the concept of a "cause" has been introduced. One possibility would be to use the language of causation, rather than that of exchangeability or identification of populations. We have not chosen to do this; nor to discuss causation, because the concept, although widely used, does not seem to be well-defined. (p. 51)

What is amazing about the history of Simpson's reversal is that, from Pearson et al. to Lindley and Novick, none of the many authors who wrote on the subject dared ask why the phenomenon should warrant our attention and why it evokes surprise. After all, seeing probabilities change magnitude upon conditionalization is commonplace, and seeing such changes turn into sign reversal (by taking differences and mixtures of those probabilities) is not uncommon either. Thus, if it were not for some misguided yet persistent illusion, what is so shocking about inequalities reversing direction?

Pearson understood that the shock originates with distorted causal interpretations, which he set out to correct through the prisms of statistical correlations and contingency tables (see the Epilogue following Chapter 10). His disciples took him rather seriously, and some even asserted that causation is none but a species of correlation (Niles 1922). In so denying any attention to causal intuition, researchers often had no choice but to attribute Simpson's reversal to some evil feature of the data, one that ought to be avoided

[3] Pearson et al. (1899) and Yule (1903) reported a weaker version of the paradox in which (6.2)–(6.3) are satisfied with equality. The reversal was discovered later by Cohen and Nagel (1934, p. 449).

by scrupulous researchers. Dozens of papers have been written since the 1950s on the statistical aspects of Simpson's reversal; some dealt with the magnitude of the effect (Blyth 1972; Zidek 1984), some established conditions for its disappearance (Bishop et al. 1975; Whittemore 1978; Good and Mittal 1987; Wermuth 1987), and some even proposed remedies as drastic as replacing $P(E \mid C)$ with $P(C \mid E)$ as a measure of treatment efficacy (Barigelli and Scozzafava 1984) – the reversal had to be avoided at all cost.

A typical treatment of the topic can be found in the influential book of Bishop, Fienberg, and Holland (1975). Bishop et al. (1975, pp. 41–2) presented an example whereby an apparent association between amount of prenatal care and infant survival disappears when the data are considered separately for each clinic participating in the study. They concluded: "If we were to look only at this [the combined] table we would erroneously conclude that survival *was related* [my italics] to the amount of care received." Ironically, survival *was* in fact *related* to the amount of care received in the study considered. What Bishop et al. meant to say is that, looking uncritically at the combined table, we would erroneously conclude that survival was *causally* related to the amount of care received. However, since causal vocabulary had to be avoided in the 1970s, researchers like Bishop et al. were forced to use statistical surrogates such as "related" or "associated" and so naturally fell victim to the limitations of the language; statistical surrogates could not express the causal relationships that researchers meant to convey.

Simpson's paradox helps us to appreciate both the agony and the achievement of this tormented generation of statisticians. Driven by healthy causal intuition, yet culturally forbidden from admitting it and mathematically disabled from expressing it, they managed nevertheless to extract meaning from dry tables and to make statistical methods the standard in the empirical sciences. But the spice of Simpson's paradox turned out to be nonstatistical after all.

6.1.3 Causality versus Exchangeability

Lindley and Novick (1981) were the first to demonstrate the nonstatistical character of Simpson's paradox – that there is no statistical criterion that would warn the investigator against drawing the wrong conclusions or would indicate which table represents the correct answer.

In the tradition of Bayesian decision theory, they first shifted attention to the practical side of the phenomenon and boldly asked: A new patient comes in; do we use the drug or do we not? Equivalently: Which table do we consult, the combined or the gender-specific? "The apparent answer is," confesses Novick (1983, p. 45), "that when we know that the gender of the patient is male or when we know that it is female we do not use the treatment, but if the gender is unknown we should use the treatment! Obviously that conclusion is ridiculous." Lindley and Novick then go through lengthy informal discussion, concluding (as we did in Section 6.1.1) that we should consult the gender-specific tables and not use the drug.

The next step was to ask whether some additional statistical information could in general point us to the right table. This question Lindley and Novick answered in the negative by showing that, with the very same data, we sometimes should decide the opposite and

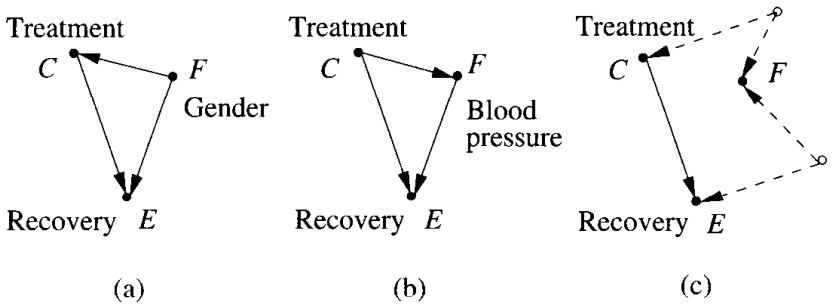

Figure 6.2 Three causal models capable of generating the data in Figure 6.1. Model (a) dictates use of the gender-specific tables, whereas (b) and (c) dictate use of the combined table.

consult the combined table. They asked: Suppose we keep the same numbers and merely change the story behind the data, imagining that F stands for some property that is affected by C – say, low blood pressure, as shown in Figure 6.2(b).[4] By inspecting the diagram in Figure 6.2(b), the reader should immediately conclude that the combined table represents the answer we want; we should not condition on F because it resides on the very causal pathway that we wish to evaluate. (Equivalently, by comparing patients with the same posttreatment blood pressure, we mask the effect of one of the two pathways through which the drug operates to bring about recovery.)

When two causal models generate the same statistical data (Figures 6.2(a) and (b) are observationally equivalent) and in one we decide to use the drug yet in the other not to use it, it is obvious that our decision is driven by causal and not by statistical considerations. Some readers might suspect that temporal information is involved in the decision, noting that gender is established before the treatment and blood pressure afterwards. But this is not the case; Figure 6.2(c) shows that F may occur before *or* after C and still the correct decision should remain to consult the combined table (i.e., not to condition on F, as can be seen from the back-door criterion).

We have just demonstrated by example what we already knew in Section 6.1.1 – namely, that every question related to the effect of actions must be decided by causal considerations; statistical information alone is insufficient. Moreover, the question of choosing the correct table on which to base our decision is a special case of the covariate selection problem that was given a general solution in Section 3.3 using causal calculus. Lindley and Novick, on the other hand, stopped short of this realization and attributed the difference between the two examples to a meta-statistical[5] concept called *exchangeability,* first proposed by De Finetti (1974).

Exchangeability concerns the question of choosing an appropriate reference class, or subpopulation, for making predictions about an individual unit. Insurance companies, for example, would like to estimate the life expectancy of a new customer using mortality records of a class of persons most closely resembling the characteristics of the

[4] The example used in Lindley and Novick (1981) was taken from agriculture, and the causal relationship between C and F was not mentioned, but the structure was the same as in Figure 6.2(b).

[5] By "meta-statistical" I mean a criterion – not itself discernible from statistical data – for judging the adequacy of a certain statistical method.

new customer. De Finetti gave this question a formal twist by translating judgment about resemblance into judgment of probabilities. According to this criterion, an $(n+1)$th unit is *exchangeable* in property X, relative to a group of n other units, if the joint probability distribution $P(X_1, \ldots, X_n, X_{n+1})$ is invariant under permutation. To De Finetti, the question of how such invariance can be established was a psychological question of secondary importance; the main point was to cast the target of this psychological exercise in the form of mathematical expression so that it could be communicated and discussed in scientific terms. It is this concept that Lindley and Novick tried to introduce into Simpson's reversal phenomenon and with which they hoped to show that the appropriate subpopulations in the F = female example are the male and female whereas, in the F = blood pressure example, the whole population of patients should be considered.

Readers of Lindley and Novick's article would quickly realize that, although these authors decorate their discussion with talks of *exchangeability* and *subpopulations,* what they actually do is present informal cause–effect arguments for their intuitive conclusions. Meek and Glymour (1994) keenly observed that the only comprehensible part of Lindley and Novick's discussion of exchangeability is the one based on causal considerations, which suggests that "an explicit account of the interaction of causal beliefs and probabilities is necessary to understand when exchangeability should and should not be assumed" (Meek and Glymour 1994, p. 1013).

This is indeed the case; exchangeability in experimental studies depends on causal understanding of the mechanisms that generate the data. The determination of whether the response of a new unit should be judged by previous response of a group of units is predicated upon the question of whether the experimental conditions to which we contemplate subjecting the new unit are equal to those prevailing while the group was observed. The reason we cannot use the combined table (Figure 6.1(a)) for determining the response of a new patient (with unknown gender) is that the experimental conditions have changed; whereas the group was studied with patients selecting treatment by choice, the new patient will be given treatment by decree, perhaps against his or her natural inclination. A mechanism will therefore be altered in the new experiment, and no judgment of exchangeability is feasible without first making causal assumptions regarding whether the probabilities involved would or would not remain invariant to such alteration. The reason we could use the combined table in the blood pressure example of Figure 6.2(b) is that the altered treatment selection mechanism in that setup is assumed to have no effect on the conditional probability $P(E \mid C)$; that is, C is assumed to be exogenous. (This can clearly be seen in the absence of any back-door path in the graph.)

Note that the same consideration holds if the next patient is a member of the group under study (assuming hypothetically that treatment and effect can be replicated and that the next patient is of unknown gender and identity); a randomly selected sample from a population is not "exchangeable" with that population if we subject the sample to new experimental conditions. Alteration of causal mechanisms must be considered in order to determine whether exchangability holds under the new circumstances. And once causal mechanisms are considered, separate judgment of exchangeability is not needed.

But why did Lindley and Novick choose to speak so elliptically (via exchangeability) when they could have articulated their ideas directly by talking openly about causal

relations? They partially answered this question as follows: "[causality], although widely used, does not seem to be well-defined." One may naturally wonder how exchangeability can be more "well-defined" than the very considerations by which it is judged! The answer can only be understood when we consider the mathematical tools available to statisticians in 1981. When Lindley and Novick wrote that causality is not well-defined, what they really meant is that causality cannot be written down in any mathematical form to which they were accustomed. The potentials of path diagrams, structural equations, and Neyman–Rubin notation as mathematical languages were generally unrecognized in 1981, for reasons described in Sections 5.1 and 7.4.3. Indeed, had Lindley and Novick wished to convey their ideas in causal terms, they would have been unable to express mathematically even the simple yet crucial fact that gender is not affected by the drug and a fortiori to derive less obvious truths from that fact.[6] The only formal language with which they were familiar was probability calculus, but as we have seen on several occasions already, this calculus cannot adequately handle causal relationships without the proper extensions.

Fortunately, the mathematical tools that have been developed in the past ten years permit a more systematic and friendly resolution of Simpson's paradox.

6.1.4 A Paradox Resolved (Or: What Kind of Machine Is Man?)

Paradoxes, like optical illusions, are often used by psychologists to reveal the inner workings of the mind, for paradoxes stem from (and amplify) dormant clashes among implicit sets of assumptions. In the case of Simpson's paradox, we have a clash between (i) the assumption that causal relationships are governed by the laws of probability calculus and (ii) the set of implicit assumptions that drive our causal intuitions. The first assumption tells us that the three inequalities in (6.1)–(6.3) are consistent, and it even presents us with a probability model to substantiate the claim (Figure 6.1). The second tells us that no miracle drug can ever exist that is harmful to both males and females and is simultaneously beneficial to the population at large.

To resolve the paradox we must either (a) show that our causal intuition is misleading or incoherent or (b) deny the premise that causal relationships are governed by the laws of standard probability calculus. As the reader surely suspects by now, we will choose the second option; our stance here, as well as in the rest of the book, is that causality is governed by its own logic and that this logic requires a major extension of probability calculus. This still behooves us to explicate the logic that governs our causal intuition and to show, formally, that this logic precludes the existence of such a miracle drug.

The logic of the $do(\cdot)$ operator is perfectly suitable for this purpose. Let us first translate the statement that our miracle drug C has harmful effect on both males and females into formal statements in causal calculus:

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[6] Lindley and Novick (1981, p. 50) did try to express this fact in probabilistic notation. But not having the $do(\cdot)$ operator at their disposal, they improperly wrote $P(F \mid C)$ instead of $P(F \mid do(C))$ and argued unconvincingly that we should equate $P(F \mid C)$ and $P(F)$: "Instead [y]ou might judge that the decision to use the treatment or the control is not affected by the unknown sex, so that F and C are independent." Oddly, this decision is also not affected by the unknown blood pressure and yet, if we write $P(F \mid C) = P(F)$ in the example of Figure 6.2(b), we obtain the wrong result.

$$P(E \mid do(C), F) < P(E \mid do(\neg C), F), \tag{6.4}$$

$$P(E \mid do(C), \neg F) < P(E \mid do(\neg C), \neg F). \tag{6.5}$$

We need to demonstrate that C must be harmful to the population at large; that is, the inequality

$$P(E \mid do(C)) > P(E \mid do(\neg C)) \tag{6.6}$$

must be shown to be inconsistent with what we know about drugs and gender.

Theorem 6.1.1 (Sure-Thing Principle[7])
An action C that increases the probability of an event E in each subpopulation must also increase the probability of E in the population as a whole, provided that the action does not change the distribution of the subpopulations.

Proof
We will prove Theorem 6.1.1 in the context of our example, where the population is partitioned into males and females; generalization to multiple partitions is straightforward. In this context, we need to prove that the reversal in the inequalities of (6.4)–(6.6) is inconsistent with the assumption that drugs have no effect on gender:

$$P(F \mid do(C)) = P(F \mid do(\neg C)) = P(F). \tag{6.7}$$

Expanding $P(E \mid do(C))$ and using (6.7) yields

$$\begin{aligned} P(E \mid do(C)) &= P(E \mid do(C), F)P(F \mid do(C)) \\ &\quad + P(E \mid do(C), \neg F)P(\neg F \mid do(C)) \\ &= P(E \mid do(C), F)P(F) + P(E \mid do(C), \neg F)P(\neg F). \end{aligned} \tag{6.8}$$

Similarly, for $do(\neg C)$ we obtain

$$\begin{aligned} P(E \mid do(\neg C)) &= P(E \mid do(\neg C), F)P(F) \\ &\quad + P(E \mid do(\neg C), \neg F)P(\neg F). \end{aligned} \tag{6.9}$$

Since every term on the right-hand side of (6.8) is smaller than the corresponding term in (6.9), we conclude that

[7] Savage (1954, p. 21) proposed the sure-thing principle as a basic postulate of preferences (on actions), tacitly assuming the no-change provision in the theorem. Blyth (1972) used this omission to devise an apparent counterexample. Theorem 6.1.1 shows that the sure-thing principle need not be stated as a separate postulate – it follows logically from the semantics of actions as modifiers of structural equations (or mechanisms). See Gibbard and Harper (1976) for a counterfactual analysis. Note that the no-change provision is probabilistic; it permits the action to change the classification of individual units as long as the relative sizes of the subpopulations remain unaltered.

$$P(E \mid do(C)) < P(E \mid do(\neg C)),$$

proving Theorem 6.1.1. □

We thus see where our causal intuition comes from: an obvious but crucial assumption in our intuitive logic has been that drugs do not influence gender. This explains why our intuition changes so drastically when F is interpreted as an intermediate event affected by the drug, as in Figure 6.2(b). In this case, our intuitive logic tells us that it is perfectly consistent to find a drug satisfying the three inequalities of (6.4)–(6.6) and, moreover, that it would be inappropriate to adjust for F. If F is affected by the C, then (6.8) cannot be derived and the difference $P(E \mid do(C)) - P(E \mid do(\neg C))$ may be positive or negative, depending on the relative magnitudes of $P(F \mid do(C))$ and $P(F \mid do(\neg C))$. Provided C and E have no common cause, we should then assess the efficacy of C directly from the combined table (equation (6.1)) and not from the F-specific tables (equations (6.2)–(6.3)).

Note that nowhere in our analysis have we assumed either that the data originate from a randomized study (i.e., $P(E \mid do(C)) = P(E \mid C)$) or from a balanced study (i.e., $P(C \mid F) = P(C \mid \neg F)$). On the contrary, given the tables of Figure 6.1, our causal logic accepts gracefully that we are dealing with unbalanced study but nevertheless refuses to accept the consistency of (6.4)–(6.6). People, likewise, can see clearly from the tables that the males were more likely to take the drug than the females; still, when presented with the reversal phenomenon, people are "shocked" to discover that differences of recovery rates can be reversed by combining tables.

The conclusions we may draw from these observations are that humans are generally oblivious to rates and proportions (which are transitory) and that they constantly search for causal relations (which are invariant). Once people interpret proportions as causal relations, they continue to process those relations by causal calculus and not by the calculus of proportions. Were our minds governed by the calculus of proportions, Figure 6.1 would have evoked no surprise at all and Simpson's paradox would never have generated the attention that it did.

6.2 WHY THERE IS NO STATISTICAL TEST FOR CONFOUNDING, WHY MANY THINK THERE IS, AND WHY THEY ARE ALMOST RIGHT

6.2.1 Introduction

Confounding is a simple concept. If we undertake to estimate the effect[8] of one variable (X) on another (Y) by examining the statistical association between the two, we ought to ensure that the association is not produced by factors other than the effect under study. The presence of spurious association, due for example to the influence of extraneous variables, is called *confounding* because it tends to confound our reading and to

[8] We will confine the use of the terms "effect," "influence," and "affect" to their causal interpretations; the term "association" will be set aside for statistical dependencies.

bias our estimate of the effect studied. Conceptually, therefore, we can say that X and Y are confounded when there is a third variable Z that influences both X and Y; such a variable is then called a *confounder* of X and Y.

As simple as this concept is, it has resisted formal treatment for decades, and for good reason: The very notions of "effect" and "influence" – relative to which "spurious association" must be defined – have resisted mathematical formulation. The empirical definition of effect as an association that *would* prevail in a controlled randomized experiment cannot easily be expressed in the standard language of probability theory, because that theory deals with static conditions and does not permit us to predict, even from a full specification of a population density function, what relationships would prevail if conditions were to change – say, from observational to controlled studies. Such predictions require extra information in the form of causal or counterfactual assumptions, which are not discernible from density functions (see Sections 1.3 and 1.4). The $do(\cdot)$ operator used in this book was devised specifically for distinguishing and managing this extra information.

These difficulties notwithstanding, epidemiologists, biostatisticians, social scientists, and economists[9] have made numerous attempts to define confounding in statistical terms, partly because statistical definitions – free of theoretical terms of "effect" or "influence" – can be expressed in conventional mathematical form and partly because such definitions may lead to practical tests of confounding and thereby alert investigators to possible bias and need for adjustment. These attempts have converged in the following basic criterion.

Associational Criterion

Two variables X and Y are not confounded if and only if every variable Z that is not affected by X is either

(U_1) *unassociated with X or*

(U_2) *unassociated with Y, conditional on X.*

This criterion, with some variations and derivatives (often avoiding the "only if" part), can be found in almost every epidemiology textbook (Schlesselman 1982; Rothman 1986; Rothman and Greenland 1998) and in almost every article dealing with confounding. In fact, the criterion has become so deeply entrenched in the literature that authors (e.g. Gail 1986; Hauck et al. 1991; Becher 1992; Steyer et al. 1996) often take it to be the *definition* of no-confounding, forgetting that ultimately confounding is useful only so far as it tells us about effect bias.[10]

The purpose of this and the next section is to highlight several basic limitations of the associational criterion and its derivatives. We will show that the associational criterion

[9] In econometrics, the difficulties have focused on the notion of "exogeneity" (Engle et al. 1983; Leamer 1985; Aldrich 1993), which stands essentially for "no confounding" (see Section 5.4.3).

[10] Hauck et al. (1991) dismiss the effect-based definition of confounding as "philosophic" and consider a difference between two measures of association to be a "bias." Grayson (1987) even goes so far as to state that the change-in-parameter method, a derivative of the associational criterion, is the only fundamental definition of confounding (see Greenland et al. 1989 for critiques of Grayson's position).

neither ensures unbiased effect estimates nor follows from the requirement of unbiasedness. After demonstrating, by examples, the absence of logical connections between the statistical and the causal notions of confounding, we will define a stronger notion of unbiasedness, called "stable" unbiasedness, relative to which a modified statistical criterion will be shown necessary and sufficient. The necessary part will then yield a practical test for stable unbiasedness that, remarkably, does not require knowledge of all potential confounders in a problem. Finally, we will argue that the prevailing practice of substituting statistical criteria for the effect-based definition of confounding is not entirely misguided, because stable unbiasedness is in fact (i) what investigators have been (and perhaps should be) aiming to achieve and (ii) what statistical criteria can test.

6.2.2 Causal and Associational Definitions

In order to facilitate the discussion, we shall first cast the causal and statistical definitions of no-confounding in mathematical forms.[11]

Definition 6.2.1 (No-Confounding; Causal Definition)
Let M be a causal model of the data-generating process – that is, a formal description of how the value of each observed variable is determined. Denote by $P(y \mid do(x))$ the probability of the response event $Y = y$ under the hypothetical intervention $X = x$, calculated according to M. We say that X and Y are not confounded *in M if and only if*

$$P(y \mid do(x)) = P(y \mid x) \tag{6.10}$$

for all x and y in their respective domains, where $P(y \mid x)$ is the conditional probability generated by M.

For the purpose of our discussion here, we take this causal definition as the meaning of the expression "no confounding." The probability $P(y \mid do(x))$ was defined in Chapter 3 (Definition 3.2.1, also abbreviated $P(y \mid \hat{x})$); it may be interpreted as the conditional probability $P^*(Y = y \mid X = x)$ corresponding to a controlled experiment in which X is randomized. We recall that this probability can be calculated from a causal model M either directly, by simulating the intervention $do(X = x)$, or (if $P(x, s) > 0$) via the adjustment formula (equation (3.19))

$$P(y \mid do(x)) = \sum_s P(y \mid x, s)P(s),$$

where S stands for any set of variables, observed as well as unobserved, that satisfy the back-door criterion (Definition 3.3.1). Equivalently, $P(y \mid do(x))$ can be written $P(Y(x) = y)$, where $Y(x)$ is the potential-outcome variable as defined in (3.51) or in

[11] For simplicity, we will limit our discussion to unadjusted confounding; extensions involving measurement of auxiliary variables are straightforward and can be obtained from Section 3.3. We also use the abbreviated expression "X and Y are not confounded," though "the effect of X on Y is not confounded" is more exact.

Rubin (1974). We bear in mind that the operator $do(\cdot)$, and hence also effect estimates and confounding, must be defined relative to a specific causal or data-generating model M because these notions are not statistical in character and cannot be defined in terms of joint distributions.

Definition 6.2.2 (No-Confounding; Associational Criterion)
Let T be the set of variables in a problem that are not affected by X. We say that X and Y are not confounded *in the presence of T if each member Z of T satisfies at least one of the following conditions:*

(U_1) *Z is not associated with X (i.e., $P(x \mid z) = P(x)$);*

(U_2) *Z is not associated with Y, conditional on X (i.e., $P(y \mid z, x) = P(y \mid x)$).*

Conversely, X and Y are said to be confounded *if any member Z of T violates both (U_1) and (U_2).*

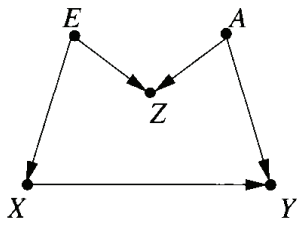
Note that the associational criterion in Definition 6.2.2 is not purely statistical in that it invokes the predicate "affected by," which is not discernible from probabilities but rests instead on causal information. This exclusion of variables that are affected by treatments (or exposures) is unavoidable and has long been recognized as a necessary judgmental input to every analysis of treatment effect in observational and experimental studies alike (Cox 1958, p. 48; Greenland and Neutra 1980). We shall assume throughout that investigators possess the knowledge required for distinguishing variables that are affected by the treatment X from those that are not. We shall then explore what additional causal knowledge is needed, if any, for establishing a test of confounding.

6.3 HOW THE ASSOCIATIONAL CRITERION FAILS

We will say that a criterion for no-confounding is *sufficient* if it never errs when it classifies a case as no-confounding and *necessary* if it never errs when it classifies a case as confounding. There are several ways that the associational criterion of Definition 6.2.2 fails to match the causal criterion of Definition 6.2.1. Failures with respect to sufficiency and necessity will be addressed in turn.

6.3.1 Failing Sufficiency via Marginality

The criterion in Definition 6.2.2 is based on testing each element of T individually. A situation may well be present where two factors, Z_1 and Z_2, jointly confound X and Y (in the sense of Definition 6.2.2) and yet each factor separately satisfies (U_1) or (U_2). This may occur because statistical independence between X and individual members of T does not guarantee the independence of X and groups of variables taken from T. For example, let Z_1 and Z_2 be the outcomes of two independent fair coins, each affecting both X and Y. Assume that X occurs when Z_1 and Z_2 are equal and that Y occurs whenever Z_1 and Z_2 are unequal. Clearly, X and Y are highly confounded by the pair $T = (Z_1, Z_2)$; they are, in fact, perfectly correlated (negatively) without causally affecting



X- exposure
Y- disease
Z= type of car owned by patient
E= education
A= age

Figure 6.3 X and Y are not confounded, though Z is associated with both.

each other. Yet, neither Z_1 nor Z_2 is associated with either X or Y; discovering the outcome of any one coin does not change the probability of X (or of Y) from its initial value of $\frac{1}{2}$.

An attempt to remedy Definition 6.2.2 by replacing Z with arbitrary subsets of T in (U_1) and (U_2) would be much too restrictive, because the set of *all* causes of X and Y, when treated as a group, would almost surely fail the tests of (U_1) and (U_2). In Section 6.5.2 we identify the subsets that should replace Z in (U_1) and (U_2) if sufficiency is to be restored.

6.3.2 Failing Sufficiency via Closed-World Assumptions

By "closed-world" assumption I mean the assumption that our model accounts for all relevant variables and, specifically to Definition 6.2.2, that the set T of variables consists of *all* potential confounders in a problem. In order to correctly classify every case of no-confounding, the associational criterion requires that condition (U_1) or (U_2) be satisfied for every potential confounder Z in a problem. In practice, since investigators can never be sure whether a given set T of potential confounders is complete, the associational criterion will falsely classify certain confounded cases as unconfounded.

This limitation actually implies that any statistical test whatsoever is destined to be insufficient. Since practical tests always involve proper subsets of T, the most we can hope to achieve by statistical means is *necessity* – that is, a test that would correctly label cases as confounding when criteria such as (U_1) and (U_2) are violated by an arbitrary subset of T. This prospect, too, is not fulfilled by Definition 6.2.2, as we now demonstrate.

6.3.3 Failing Necessity via Barren Proxies

Example 6.3.1 Imagine a situation where exposure (X) is influenced by a person's education (E), disease (Y) is influenced by both exposure and age (A), and car type (Z) is influenced by both age (A) and education (E). These relationships are shown schematically in Figure 6.3.

The car-type variable (Z) violates the two conditions in Definition 6.2.2 because: (1) car type is indicative of education and hence is associated with the exposure variable; and (2) car type is indicative of age and hence is associated with the disease among the exposed and the nonexposed. However, in this example the effect of X on Y is not confounded; the type of car owned by a person has no effect on either exposure or disease and is merely one among many irrelevant properties that are associated with both via intermediaries. The analysis of Chapter 3 establishes that,

indeed, (6.10) is satisfied in this model[12] and that, moreover, adjustment for Z would generally yield a biased result:

$$\sum_z P(Y = y \mid X = x,\ Z = z)P(Z = z) \neq P(Y = y \mid do(x)).$$

Thus we see that the traditional criterion based on statistical association fails to identify an unconfounded effect and would tempt one to adjust for the wrong variable. This failure occurs whenever we apply (U_1) and (U_2) to a variable Z that is a *barren proxy* – that is, a variable that has no influence on X or Y but is a proxy for factors that do have such influence.

Readers may not consider this failure to be too serious, because experienced epidemiologists would rarely regard a variable as confounder unless it is suspect of having some influence on either X or Y. Nevertheless, adjustment for proxies is a prevailing practice in epidemiology and should be done with great caution (Greenland and Neutra 1980; Weinberg 1993). To regiment this caution, the associational criterion must be modified to exclude barren proxies from the test set T. This yields the following modified criterion in which T consists only of variables that (causally) influence Y (possibly through X).

Definition 6.3.2 (No-Confounding; Modified Associational Criterion)
Let T be the set of variables in a problem that are not affected by X but may potentially affect Y. We say that X and Y are unconfounded *by the presence of T if and only if every member Z of T satisfies either (U_1) or (U_2) of Definition 6.2.2.*

Stone (1993) and Robins (1997) proposed alternative modifications of Definition 6.2.2 that avoid the problems created by barren proxies without requiring one to judge whether a variable has an effect on Y. Instead of restricting the set T to potential causes of Y, we let T remain the set of *all* variables unaffected by X,[13] requiring instead that T be composed of two disjoint subsets, T_1 and T_2, such that

(U_1^*) T_1 is unassociated with X *and*

(U_2^*) T_2 is unassociated with Y given X and T_1.

In the model of Figure 6.3, for instance, conditions (U_1^*) and (U_2^*) are satisfied by the choice $T_1 = A$ and $T_2 = \{Z, E\}$, because (using the d-separation test) A is independent of X and E is independent of Y, given $\{X, A\}$.

This modification of the associational criterion further rectifies the problem associated with marginality (see Section 6.3.1) because (U_1^*) and (U_2^*) treat T_1 and T_2 as compound

[12] Because the (back-door) path $X \leftarrow E \rightarrow Z \leftarrow A \rightarrow Y$ is blocked by the colliding arrows at Z (see Definition 3.3.1).

[13] Alternatively, T can be confined to any set S of variables sufficient for control of confounding:

$$P(y \mid do(x)) = \sum_s P(y \mid x, s)P(s).$$

Again, however, we can never be sure if the measured variables in the model contain such a set, or which of T's subsets possess this property.

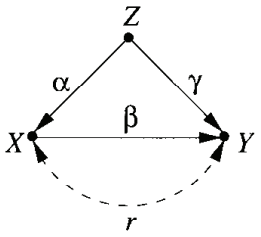


Figure 6.4 Z is associated with both X and Y, yet the effect of X on Y is not confounded (when $r = -\alpha\gamma$).

variables. However, the modification falls short of restoring necessity. Because the set $T = (T_1, T_2)$ must include *all* variables unaffected by X (see note 13) and because practical tests are limited to proper subsets of T, we cannot conclude that confounding is present solely upon the failure of (U_1^*) and (U_2^*), as specified in Section 6.3.2. This criterion, too, is thus inadequate as a basis for practical detection of confounding.

We now discuss another fundamental limitation on our ability to detect confounding by statistical means.

6.3.4 Failing Necessity via Incidental Cancellations

Here we present a case that is devoid of barren proxies and in which the effect of X on Y (i) is not confounded in the sense of (6.10) but (ii) is confounded according to the modified associational criterion of Definition 6.3.2.

Example 6.3.3 Consider a causal model defined by the linear equations

$$x = \alpha z + \varepsilon_1, \tag{6.11}$$

$$y = \beta x + \gamma z + \varepsilon_2, \tag{6.12}$$

where ε_1 and ε_2 are correlated unmeasured variables with $\text{cov}(\varepsilon_1, \varepsilon_2) = r$ and where Z is an exogenous variable that is uncorrelated with ε_1 or ε_2. The diagram associated with this model is depicted in Figure 6.4. The effect of X on Y is quantified by the path coefficient β, which gives the rate of change of $E(Y \mid do(x))$ per unit change in x.[14]

It is not hard to show (assuming standardized variables) that the regression of Y on X gives

$$y = (\beta + r + \alpha\gamma)x + \varepsilon,$$

where $\text{cov}(x, \varepsilon) = 0$. Thus, whenever the equality $r = -\alpha\gamma$ holds, the regression coefficient of $r_{YX} = \beta + r + \alpha\gamma$ is an unbiased estimate of β, meaning that the effect of X on Y is unconfounded (no adjustment is necessary). Yet the associational conditions (U_1) and (U_2) are both violated by the variable Z; Z is associated with X (if $\alpha \neq 0$) and conditionally associated with Y, given X (except for special values of γ for which $\rho_{yz \cdot x} = 0$).

[14] See Sections 3.5–3.6 or (5.24) in Section 5.4.1.

This example demonstrates that the condition of unbiasedness (Definition 6.2.1) does not imply the modified criterion of Definition 6.3.2. The associational criterion might falsely classify some unconfounded situations as confounded and, worse yet, adjusting for the false confounder (Z in our example) will introduce bias into the effect estimate.[15]

6.4 STABLE VERSUS INCIDENTAL UNBIASEDNESS

6.4.1 Motivation

The failure of the associational criterion in the previous example calls for a reexamination of the notion of confounding and unbiasedness as defined in (6.10). The reason that X and Y were classified as unconfounded in Example 6.3.3 was that, by setting $r = -\alpha\gamma$, we were able to make the spurious association represented by r *cancel* the one mediated by Z. In practice, such perfect cancellation would be an incidental event specific to a peculiar combination of study conditions, and it would not persist when the parameters of the problem (i.e., α, γ, and r) undergo slight changes – say, when the study is repeated in a different location or at a different time. In contrast, the condition of no-confounding found in Example 6.3.1 does not exhibit such volatility. In this example, the unbiasedness expressed in (6.10) would continue to hold regardless of the strength of connection between education and exposure and regardless on how education and age influence the type of car that a patient owns. We call this type of unbiasedness *stable,* since it is robust to change in parameters and remains intact as long as the configuration of causal connections in the model remains the same.

In light of this distinction between stable and incidental unbiasedness, we need to reexamine whether we should regard a criterion as inadequate if it misclassifies (as confounded) cases that are rendered unconfounded by mere incidental cancellation and, more fundamentally, whether we should insist on including such peculiar cases in the definition of unbiasedness (given the precarious conditions under which (6.10) would be satisfied in these cases). Although answers to these questions are partly a matter of choice, there is ample evidence that our intuition regarding confounding is driven by considerations of stable unbiasedness, not merely incidental ones. How else can we explain why generations of epidemiologists and biostatisticians would advocate confounding criteria that fail in cases involving incidental cancellation? On the pragmatic side, failing to detect situations of incidental unbiasedness should not introduce appreciable error in observational studies because those situations are short-lived and are likely to be refuted by subsequent studies, under slightly different conditions.[16]

Assuming that we are prepared to classify as unbiased only cases in which unbiasedness remains robust to changes in parameters, two questions remain: (1) How can we give this new notion of "stable unbiasedness" a formal, nonparametric formulation? (2) Are practical statistical criteria available for testing stable unbiasedness? Both questions can be answered using structural models.

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[15] Note that the Stone–Robins modifications of Definition 6.3.2 would also fail in this example, unless we can measure the factors responsible for the correlation between ε_1 and ε_2.

[16] As we have seen in Example 6.3.3, any statistical test capable of recognizing such cases would require measurement of *all* variables in T.

Chapter 3 describes a graphical criterion, called the "back-door criterion," for identifying conditions of unbiasedness in a causal diagram.[17] In the simple case of no adjustment (for measured covariates), the criterion states that X and Y are unconfounded if every path between X and Y that contains an arrow pointing into X must also contain a pair of arrows pointing head-to-head (as in Figure 6.3); this criterion is valid whenever the missing links in the diagram represent absence of causal connections among the corresponding variables. Because the causal assumptions embedded in the missing links are so explicit, the back-door criterion has two remarkable features. First, no statistical information is needed; the topology of the diagram suffices for reliably determining whether an effect is unconfounded (in the sense of Definition 6.2.1) and whether an adjustment for a set of variables is sufficient for removing confounding when one exists. Second, any model that meets the back-door criterion would in fact satisfy (6.10) for an infinite class of models (or situations), each generated by assigning different parameters to the causal connections in the diagram.

To illustrate, consider the diagram depicted in Figure 6.3. The back-door criterion will identify the pair (X, Y) as unconfounded, because the only path ending with an arrow into X is the one traversing (X, E, Z, A, Y), and this path contains two arrows pointing head-to-head at Z. Moreover, since the criterion is based only on graphical relationships, it is clear that (X, Y) will continue to be classified as unconfounded regardless of the strength or type of causal relationships that are represented by the arrows in the diagram. In contrast, consider Figure 6.4 in Example 6.3.3, where two paths end with arrows into X. Since none of these paths contains head-to-head arrows, the back-door criterion will fail to classify the effect of X on Y as unconfounded, acknowledging that an equality $r = -\alpha\gamma$ (if it prevails) would not represent a stable case of unbiasedness.

The vulnerability of the back-door criterion to causal assumptions can be demonstrated in the context of Figure 6.3. Assume the investigator suspects that variable Z (car type) has some influence on the outcome variable Y. This would amount to adding an arrow from Z to Y in the diagram, classifying the situation as confounded, and suggesting an adjustment for E (or $\{A, Z\}$). Yet no adjustment is necessary if, owing to the specific experimental conditions in the study, Z has in fact no influence on Y. It is true that the adjustment suggested by the back-door criterion would introduce no bias, but such adjustment could be costly if it calls for superfluous measurements in a no-confounding situation.[18] The added cost is justified in light of (i) the causal information at hand (i.e., that Z may potentially influence Y) and (ii) our insistence on ensuring stable unbiasedness – that is, avoiding bias in all situations compatible with the information at hand.

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[17] A gentle introduction to applications of the back-door criterion in epidemiology can be found in Greenland et al. (1999a).

[18] On the surface, it appears as though the Stone–Robins criterion would correctly recognize the absence of confounding in this situation, since it is based on associations that prevail in the probability distribution that actually generates the data (according to which $\{E, Z\}$ should be independent of Y, given $\{A, X\}$). However, these associations are of no help in deciding whether certain measurements can be *avoided;* such decisions must be made prior to gathering the data and must rely therefore on subjective assumptions about the disappearance of conditional associations. Such assumptions are normally supported by causal, not associational, knowledge (see Section 1.3).

6.4.2 Formal Definitions

To formally distinguish between *stable* and *incidental* unbiasedness, we use the following general definition.

Definition 6.4.1 (Stable Unbiasedness)
Let A be a set of assumptions (or restrictions) on the data-generating process, and let C_A be a class of causal models satisfying A. The effect estimate of X on Y is said to be stably unbiased *given A if $P(y \mid do(x)) = P(y \mid x)$ holds in every model M in C_A. Correspondingly, we say that the pair (X, Y) is* stably unconfounded *given A.*

The assumptions commonly used to specify causal models can be either parametric or topological. For example, the structural equation models used in the social sciences and economics are usually restricted by the assumptions of linearity and normality. In this case, C_A would consist of all models created by assigning different values to the unspecified parameters in the equations and in the covariance matrix of the error terms. Weaker, nonparametric assumptions emerge when we specify merely the topological structure of the causal diagram but let the error distributions and the functional form of the equations remain undetermined. We now explore the statistical ramifications of these nonparametric assumptions.

Definition 6.4.2 (Structurally Stable No-Confounding)
Let A_D be the set of assumptions embedded in a causal diagram D. We say that X and Y are stably unconfounded *given A_D if $P(y \mid do(x)) = P(y \mid x)$ holds in every parameterization of D. By "parameterization" we mean an assignment of functions to the links of the diagram and prior probabilities to the background variables in the diagram.*

Explicit interpretation of the assumptions embedded in a causal diagram are given in Chapters 3 and 5. Put succinctly, if D is the diagram associated with the causal model, then:

1. every missing arrow (between, say, X and Y) represents the assumption that X has no effect on Y once we intervene and hold the parents of Y fixed;
2. every missing bidirected link between X and Y represents the assumption that there are no common causes for X and Y, except those shown in D.

Whenever the diagram D is acyclic, the back-door criterion provides a necessary and sufficient test for stable no-confounding, given A_D. In the simple case of no adjustment for covariates, the criterion reduces to the nonexistence of a common ancestor, observed or latent, of X and Y.[19] Thus, we have our next theorem.

[19] The colloquial term "common ancestors" should exclude nodes that have no other connection to Y except through X (e.g., node E in Figure 6.3) and include latent nodes for correlated errors. In the diagram of Figure 6.4, for example, X and Y are understood to have two common ancestors; the first is Z and the second is the (implicit) latent variable responsible for the double-arrowed arc between X and Y (i.e., the correlation between ε_1 and ε_2).

Theorem 6.4.3 (Common-Cause Principle)
Let A_D be the set of assumptions embedded in an acyclic causal diagram D. Variables X and Y are stably unconfounded *given A_D if and only if X and Y have no common ancestor in D.*

Proof
The "if" part follows from the validity of the back-door criterion (Theorem 3.3.2). The "only if" part requires the construction of a specific model in which (6.10) is violated whenever X and Y have a common ancestor in D. This is easily done using linear models and Wright's rules for path coefficients. □

Theorem 6.4.3 provides a necessary and sufficient condition for stable no-confounding without invoking statistical data, since it relies entirely on the information embedded in the diagram. Of course, the diagram itself has statistical implications that can be tested (Sections 1.2.3 and 5.2.1), but those tests do not specify the diagram uniquely (see Chapter 2 and Section 5.2.3).

Suppose, however, that we do not possess all the information required for constructing a causal diagram and instead know merely for each variable Z whether it is safe to assume that Z has no effect on Y and whether X has no effect on Z. The question now is whether this more modest information, together with statistical data, is sufficient to qualify or disqualify a pair (X, Y) as stably unconfounded. The answer is positive.

6.4.3 Operational Test for Stable No-Confounding

Theorem 6.4.4 (Criterion for Stable No-Confounding)
Let A_Z denote the assumptions that (i) *the data are generated by some (unspecified) acyclic model M and* (ii) *Z is a variable in M that is unaffected by X but may possibly affect Y.*[20] *If both of the associational criteria (U_1) and (U_2) of Definition 6.2.2 are violated, then (X, Y) are not stably unconfounded given A_Z.*

Proof
Whenever X and Y are stably unconfounded, Theorem 6.4.3 rules out the existence of a common ancestor of X and Y in the diagram associated with the underlying model. The absence of a common ancestor, in turn, implies the satisfaction of either (U_1) or (U_2) whenever Z satisfies A_Z. This is a consequence of the d-separation rule (Section 1.2.3) for reading the conditional independence relationships entailed by a diagram.[21] □

Theorem 6.4.4 implies that the traditional associational criteria (U_1) and (U_2) could be used in a simple operational test for stable no-confounding, a test that does not require us to know the causal structure of the variables in the domain or even to enumerate the set of relevant variables. Finding just *any* variable Z that satisfies A_Z and violates (U_1)

[20] By "possibly affecting Y" we mean: A_Z does not contain the assumption that Z does not affect Y. In other words, the diagram associated with M must contain a directed path from Z to Y.
[21] It also follows from Theorem 7(a) in Robins (1997).

and (U_2) permits us to disqualify (X, Y) as stably unconfounded (though (X, Y) may be incidentally unconfounded in the particular experimental conditions prevailing in the study).

Theorem 6.4.4 communicates a formal connection between statistical associations and confounding that is not based on the closed-world assumption.[22] It is remarkable that the connection can be formed under such weak set of added assumptions: the qualitative assumption that a variable may have influence on Y and is not affected by X suffices to produce a necessary statistical test for stable no-confounding.

6.5 CONFOUNDING, COLLAPSIBILITY, AND EXCHANGEABILITY

6.5.1 Confounding and Collapsibility

Theorem 6.4.4 also establishes a formal connection between confounding and "collapsibility" – a criterion under which a measure of association remains invariant to the omission of certain variables.

Definition 6.5.1 (Collapsibility)
Let $g[P(x, y)]$ be any functional[23] *that measures the association between Y and X in the joint distribution $P(x, y)$. We say that g is* collapsible *on a variable Z if*

$$E_z g[P(x, y \mid z)] = g[P(x, y)].$$

It is not hard to show that if g stands for any linear functional of $P(y \mid x)$ – for example, the risk difference $P(y \mid x_1) - P(y \mid x_2)$ – then collapsibility holds whenever Z is either unassociated with X or unassociated with Y given X. Thus, any violation of collapsibility implies violation of the two statistical criteria of Definition 6.2.2, and that is probably why many believed noncollapsibility to be intimately connected with confounding. However, the examples in this chapter demonstrate that violation of these two conditions is neither sufficient nor necessary for confounding. Thus, noncollapsibility and confounding are in general two distinct notions; neither implies the other.

Some authors tend to believe that this distinction is a peculiar property of nonlinear effect measures g, such as the odds or likelihood ratios, and that "when the effect measure is an expectation over population units, confounding and noncollapsibility are algebraically equivalent" (Greenland 1998, p. 906). This chapter shows that confounding and noncollapsibility need not correspond even in linear functionals. For example, the effect measure $P(y \mid x_1) - P(y \mid x_2)$ (the risk difference) is not collapsible over Z in Figure 6.3 (for almost every parameterization of the graph) and yet the effect measure is unconfounded (for every parameterization).

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[22] I am not aware of another such connection in the literature.

[23] A *functional* is an assignment of a real number to any function from a given set of functions. For example, the mean $E(X) = \sum_x xP(x)$ is a functional, since it assigns a real number $E(X)$ to each probability function $P(x)$.

The logical connection between confounding and collapsibility is formed through the notion of *stable no-confounding,* as formulated in Definition 6.4.2 and Theorem 6.4.4. Because any violation of collapsibility means violation of (U_1) and (U_2) in Definition 6.2.2, it also implies (by Theorem 6.4.4) violation of stable unbiasedness (or stable no-confounding). Thus we can state the following corollary.

Corollary 6.5.2 (Stable No-Confounding Implies Collapsibility)
Let Z be any variable that is not affected by X and that may possibly affect Y. Let $g[P(x, y)]$ be any linear functional that measures the association between X and Y. If g is not collapsible on Z, then X and Y are not stably unconfounded.

This corollary provides a rationale for the widespread practice of testing confoundedness by the change-in-parameter method, that is, labeling a variable Z a confounder whenever the "crude" measure of association, $g[P(x, y)]$, is not equal to the Z-specific measures of association averaged over the levels of Z (Breslow and Day 1980; Kleinbaum et al. 1982; Yanagawa 1984; Grayson 1987). Theorem 6.4.4 suggests that the intuitions responsible for this practice were shaped by a quest for a stable condition of no-confounding, not merely an incidental one. Moreover, condition A_Z in Theorem 6.4.4 justifies a requirement made by some authors that a confounder must be a causal determinant of, and not merely associated with, the outcome variable Y.

6.5.2 Confounding versus Confounders

The focus of our discussion in this chapter has been the phenomenon of confounding, which we equated with that of effect bias (Definition 6.2.1). Much of the literature on this topic has been concerned with the presence or absence of *confounders,* presuming that some variables possess the capacity to confound and some do not. This notion may be misleading if interpreted literally, and caution should be exercised before we label a variable as a confounder.

Rothman and Greenland (1998, p. 120), for example, offer this definition: "The extraneous factors responsible for difference in disease frequency between the exposed and unexposed are called *confounders*"; they go on to state that: "In general, a confounder must be associated with both the exposure under study and the disease under study to be confounding" (p. 121). Rothman and Greenland qualify their statement with "In general," and for good reason: We have seen (in the two-coin example of Section 6.3.1) that each individual variable in a problem can be *unassociated* with both the exposure (X) and the disease (Y) under study and still the effect of X on Y remains confounded. A similar situation can also be seen in the linear model depicted in Figure 6.5. Although Z is clearly a confounder for the effect of X on Y and must therefore be controlled, the association between Z and Y may actually vanish (at each level of X) and the association between Z and X may vanish as well. This can occur if the indirect association mediated by the path $Z \leftarrow A \rightarrow Y$ happens to cancel the direct association carried by the arrow $Z \rightarrow Y$. This cancellation does not imply the absence of confounding, because the path $X \leftarrow E \rightarrow Z \rightarrow Y$ is unblocked while $X \leftarrow E \rightarrow Z \leftarrow A \rightarrow Y$ is blocked. Thus, Z is a confounder that is associated neither with the exposure (X) nor with the disease (Y).

The intuition behind Rothman and Greenland's statement just quoted can be explicated formally through the notion of stability: a variable that is *stably* unassociated with

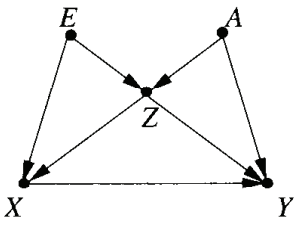

Figure 6.5 Z may be unassociated with both X and Y and still be a confounder (i.e., a member of every sufficient set).

either X or Y can safely be excluded from adjustment. Alternatively, Rothman and Greenland's statement can be supported (without invoking stability) by using the notion of *minimal sufficient set* (Section 3.3) – a minimal set of variables for which adjustment will remove confounding bias. It can be shown (see the end of this section) that each such sufficient set S, taken as a unit, must indeed be associated with X and be conditionally associated with Y, given X. Thus, Rothman and Greenland's condition is valid for minimal sufficient sets but not for the individual variables in a problem.

The practical ramifications of this condition are as follows. If we are given a set S of variables that is claimed to be minimally sufficient (for removing bias by adjustment), then that claim can be given a necessary statistical test: S as a compound variable must be associated both with X and with Y (given X). In Figure 6.5, for example, the minimal sufficient sets are $S_1 = \{A, Z\}$ and $S_2 = \{E, Z\}$; both must satisfy the condition stated.

Note that, although this test can be used for screening sets claimed to be minimally sufficient, it does not constitute a test for detecting confounding. Even if we find a set S in a problem that is associated with both X and Y, we are still unable to conclude that X and Y are confounded. Our finding merely qualifies S as a candidate for minimally sufficient status in case confounding exists, but we cannot rule out the possibility that the problem is unconfounded to start with. (The sets $S = \{E, A\}$ or $S = \{Z\}$ in Figure 6.1 illustrate this point.) Observing a discrepancy between adjusted and unadjusted associations (between X and Y) does not help us either, because (recalling our discussion of collapsibility) we do not know which – the preadjustment or postadjustment association – is unbiased (see Figure 6.4).

Proof of Necessity

To prove that (U_1) and (U_2) must be violated whenever Z stands for a minimally sufficient set S, consider the case where X has no effect on Y. In this case, confounding amounts to a nonvanishing association between X and Y. A well-known property of conditional independence, called *contraction* (Section 1.1.5), states that violation of (U_1), $X \not\perp\!\!\!\perp S$, together with sufficiency, $X \perp\!\!\!\perp Y \mid S$, implies violation of minimality, $X \not\perp\!\!\!\perp Y$:

$$X \not\perp\!\!\!\perp S \;\&\; X \perp\!\!\!\perp Y \mid S \implies X \not\perp\!\!\!\perp Y.$$

Likewise, another property of conditional independence, called *intersection,* states that violation of (U_2), $S \not\perp\!\!\!\perp Y \mid X$, together with sufficiency, $X \not\perp\!\!\!\perp Y \mid S$, also implies violation of minimality, $X \not\perp\!\!\!\perp Y$:

$$S \not\perp\!\!\!\perp Y \mid X \;\&\; X \not\perp\!\!\!\perp Y \mid S \implies X \not\perp\!\!\!\perp Y.$$

Thus, both (U_1) and (U_2) must be violated by any minimally sufficient set Z in Definition 6.2.2.

Note, however, that intersection holds only for strictly positive probability distributions, which means that the Rothman–Greenland condition may be violated if deterministic relationships hold among some variables in a problem. This can be seen from a simple example in which both X and Y stand in a one-to-one functional relationship to a third variable, Z. Clearly, Z is a minimally sufficient set yet is not associated with Y given X; once we know the value of X, the probability of Y is determined and would no longer change with learning the value of Z.

6.5.3 Exchangeability versus Structural Analysis of Confounding

Students of epidemiology complain bitterly about the confusing way in which the fundamental concept of confounding has been treated in the literature. A few authors have acknowledged the confusion (e.g. Greenland and Robins 1986; Wickramaratne and Holford 1987; Weinberg 1993) and have suggested new ways of looking at the problem that might lead to more systematic analysis. Greenland and Robins (GR), in particular, have recognized the same basic principles and results that we have expounded here in Sections 6.2 and 6.3. Their analysis represents one of the few bright spots in the vast literature on confounding in that it treats confounding as an unknown causal quantity that is not directly measurable from observed data. They further acknowledge (as do Miettinen and Cook 1981) that the presence or absence of confounding should not be equated with absence or presence of collapsibility and that confounding should not be regarded as a parameter-dependent phenomenon.

However, the structural analysis presented in this chapter differs in a fundamental way from that of GR, who have pursued an approach based on judgment of "exchangeability." In Section 6.1 we encountered a related notion of exchangeability, one with which Lindley and Novick (1981) attempted to view Simpson's paradox; GR's idea of exchangeability is more concrete and more clearly applicable. Conceptually, the connection between confounding and exchangeability is as follows. If we undertake to assess the effect of some treatment, we ought to make sure that any response differences between the treated and the untreated group is due to the treatment itself and not to some intrinsic differences between the groups that are unrelated to the treatment. In other words, the two groups must resemble each other in all characteristics that have bearing on the response variable. In principle, we could have ended the definition of confounding at this point, declaring simply that the effect of treatment is unconfounded if the treated and untreated groups resemble each other in all relevant features. This definition, however, is too verbal in the sense that it is highly sensitive to interpretation of the terms "resemblance" and "relevance." To make it less informal, GR used De Finetti's twist of hypothetical permutation; instead of judging whether two groups are similar, the investigator is instructed to imagine a hypothetical *exchange* of the two groups (the treated group becomes untreated, and vice versa) and then to judge whether the observed data under the swap would be distinguishable from the actual data.

One can justifiably ask what has been gained by this mental exercise, relative to judging directly if the two groups are effectively identical. The gain is twofold. First, people are quite good in envisioning dynamic processes and can simulate the outcome of this swapping scenario from basic understanding of the processes that govern the response

to treatment and the factors that affect the choice of treatment. Second, moving from judgment about resemblance to judgment about probabilities permits us to cast those judgments in probabilistic notation and hence to invite the power and respectability of probability calculus.

Greenland and Robins made an important first step toward this formalization by bringing notation closer to where judgment originates – the human understanding of causal processes. The structural approach pursued in this book takes the next, natural step: formalizing the causal processes themselves.

Let A and B stand (respectively) for the treated and untreated groups, and let $P_{A1}(y)$ and $P_{A0}(y)$ stand (respectively) for the response distribution of group A under two hypothetical conditions, treatment and no treatment.[24] If our interest lies in some parameter μ of the response distribution, we designate by μ_{A1} and μ_{A0} the values of that parameter in the corresponding distribution $P_{A1}(y)$ and $P_{A0}(y)$, with μ_{B1} and μ_{B0} defined similarly for group B. In actuality, we measure the pair (μ_{A1}, μ_{B0}); after the hypothetical swap, we would measure (μ_{B1}, μ_{A0}). We define the groups to be *exchangeable* relative to parameter μ if the two pairs are indistinguishable, that is, if

$$(\mu_{A1}, \mu_{B0}) = (\mu_{B1}, \mu_{A0}).$$

In particular, if we define the causal effect by the difference $\text{CE} = \mu_{A1} - \mu_{A0}$, then exchangeability permits us to replace μ_{A0} with μ_{B0} and so obtain $\text{CE} = \mu_{A1} - \mu_{B0}$, which is measurable because both quantities are observed. Greenland and Robins thus declare the causal effect CE to be *unconfounded* if $\mu_{A0} = \mu_{B0}$.

If we compare this definition to that of (6.10), $P(y \mid do(x)) = P(y \mid x)$, we find that the two coincide if we rewrite the latter as $\mu[P(y \mid do(x))] = \mu[P(y \mid x)]$, where μ is the parameter of interest in the response distribution. However, the major difference between the structural and the GR approaches lies in the level of analysis. Structural modeling extends the formalization of confounding in two important directions. First, (6.10) is not submitted to direct human judgment but is derived mathematically from more elementary judgments concerning causal processes.[25] Second, the input judgments needed for the structural model are both qualitative and stable.

A simple example will illustrate the benefits of these features. Consider the following statement (Greenland 1998):

(Q^*) "if the effect measure is the difference or ratio of response proportions, then the above phenomenon – noncollapsibility without confounding – cannot occur, nor can confounding occur without noncollapsibility."

We have seen in this chapter that statement (Q^*) should be qualified in several ways and that, in general, noncollapsibility and confounding are two distinct notions – neither implying the other, regardless of the effect measure (Section 6.5.1). However, the

[24] In $do(\cdot)$ notation, we would write $P_{A1}(y) = P_A(y \mid do(X = 1))$.

[25] Recall that the $do(\cdot)$ operator is defined mathematically in terms of equation deletion in structural equation models; consequently, the verification of the nonconfounding condition $P(y \mid do(x)) = P(y \mid x)$ in a given model is not a matter of judgment but a subject of mathematical analysis.

question we wish to discuss here is methodological: What formalism would be appropriate for validating, refuting, or qualifying statements of this sort? Clearly, since (Q^*) makes a general claim about all instances, one counterexample would suffice to refute its general validity. But how do we construct such a counterexample? More generally, how do we construct examples that embody properties of confounding, effect bias, causal effects, experimental versus nonexperimental data, counterfactuals, and other causality-based concepts?

In probability theory, if we wish to refute a general statement about parameters and their relationship we need only present one density function f for which that relationship fails to hold. In propositional logic, in order to show that a sentence is false, we need only present one truth table T that satisfies the premises and violates the conclusions. What, then, is the mathematical object that should replace f or T when we wish to refute causal claims like statement (Q^*)? The corresponding object used in the exchangeability framework of Greenland and Robins is a counterfactual contingency table (see e.g. Greenland et al. 1999b, p. 905, or Figure 1.7 in Section 1.4.4). For instance, to illustrate confounding, we need two such tables: one describing the hypothetical response of the treated group A to both treatment and nontreatment, and one describing the hypothetical response of the untreated group B to both treatment and nontreatment. If the tables show that the parameter μ_{A0}, computed from the hypothetical response of the treated group to no treatment, differs from μ_{B0}, computed from the actual response of the untreated group, then we have confounding on our hands.

Tables of this type can easily be constructed for simple problems involving one treatment and one response variable, but they become a nightmare when several covariates are involved or when we wish to impose certain constraints on those covariates. For example, we may wish to incorporate the standard assumption that a covariate Z does not lie on the causal pathway between treatment and response, or that Z has causal influence on Y, but such assumptions cannot conveniently be expressed in counterfactual contingency tables. As a result, the author of the claim to be refuted could always argue that the tables used in the counterexample may be inconsistent with the agreed assumptions.[26]

Such difficulties do not plague the structural representation of confounding. In this formalism, the appropriate object for exemplifying or refuting causal statements is a causal model, as defined in Chapter 3 and used throughout this book. Here, hypothetical responses (μ_{A0} and μ_{B0}) and contingency tables are not the primitive quantities but rather are derivable from a set of equations that already embody the assumptions we wish to respect. Every parameterization of a structural model implies (using the $do(\cdot)$ operator) a specific set of counterfactual contingency tables that satisfies the input assumptions and exhibits the statistical properties displayed in the graph. For example, any parameterization of the graph in Figure 6.3 generates a set of counterfactual contingency tables that already embodies the assumptions that Z is not on the causal pathway between X and Y and that Z has no causal effect on Y, and almost every such parameterization will generate a counterexample to claim (Q^*). Moreover, we can also disprove (Q^*) by a casual inspection of the diagram and without generating numerical counterexamples. In

[26] Readers who attempt to construct a counterexample to statement (Q^*) using counterfactual contingency tables will certainly appreciate this difficulty.

Figure 6.3, for example, shows vividly that the risk difference $P(y \mid x_1) - P(y \mid x_2)$ is not collapsible on Z and, simultaneously, that X and Y are (stably) unconfounded.

The difference between the two formulations is even more pronounced when we come to substantiate, not refute, generic claims about confounding. Here it is not enough to present a single contingency table; instead, we must demonstrate the validity of the claim for all tables that can possibly be constructed in compliance with the input assumptions. This task, as the reader surely realizes, is a hopeless exercise within the framework of contingency tables; it calls for a formalism in which assumptions can be stated succinctly and in which conclusions can be deduced by mathematical derivations. The structural semantics offers such formalism, as demonstrated by the many generic claims proven in this book (examples include Theorem 6.4.4 and Corollary 6.5.2).

As much as I admire the rigor introduced by Greenland and Robins's analysis through the framework of exchangeability, I am thoroughly convinced that the opacity and inflexibility of counterfactual contingency tables are largely responsible for the slow acceptance of the GR framework among epidemiologists and, as a byproduct, for the lingering confusion that surrounds confounding in the statistical literature at large. I am likewise convinced that formulating claims and assumptions in the language of structural models will make the mathematical analysis of causation accessible to rank-and-file researchers and thus lead eventually to a total and natural disconfounding of confounding.

6.6 CONCLUSIONS

Past efforts to establish a theoretical connection between statistical associations (or collapsibility) and confounding have been unsuccessful for three reasons. First, the lack of mathematical language for expressing claims about causal relationships and effect bias has made it difficult to assess the disparity between the requirement of effect unbiasedness (Definition 6.2.1) and statistical criteria purporting to capture unbiasedness.[27] Second, the need to exclude barren proxies (Figure 6.3) from consideration has somehow escaped the attention of researchers. Finally, the distinction between stable and incidental unbiasedness has not received the attention it deserves and, as we observed in Example 6.3.3, no connection can be formed between associational criteria (or collapsibility) and confounding without a commitment to the notion of stability. Such commitment rests critically on the conception of a causal model as an assembly of autonomous mechanisms that may vary independently of one another (Aldrich 1989). It is only in anticipation of such independent variations that we are not content with incidental unbiasedness but rather seek conditions of stable unbiasedness. The mathematical formalization of this conception has led to related notions of *DAG-isomorph* (Pearl 1988b, p. 128), *stability*

[27] The majority of papers on collapsibility (e.g. Bishop 1971; Whittemore 1978; Wermuth 1987; Becher 1992; Geng 1992) motivate the topic by citing Simpson's paradox and the dangers of obtaining confounded effect estimates. Of these, only a handful pursue the study of confounding or effect estimates; most prefer to analyze the more manageable phenomenon of collapsibility as a stand-alone target. Some go as far as naming collapsibility "nonconfoundedness" (Grayson 1987; Steyer et al. 1997).

(Pearl and Verma 1991), and *faithfulness* (Spirtes et al. 1993), which assist in the elucidation of causal diagrams from sparse statistical associations (see Chapter 2). The same conception has evidently been shared by authors who aspired to connect associational criteria with confounding.

The advent of structural model analysis, assisted by graphical methods, offers a mathematical framework in which considerations of confounding can be formulated and managed more effectively. Using this framework, this chapter explicates the criterion of stable unbiasedness and shows that this criterion (i) has implicitly been the target of many investigations in epidemiology and biostatistics, and (ii) can be given operational statistical tests similar to those invoked in testing collapsibility. We further show (Section 6.5.3) that the structural framework overcomes basic cognitive and methodological barriers that have made confounding one of the most confused topics in the literature. It is therefore natural to predict that this framework will become the primary mathematical basis for future studies of confounding.

Acknowledgment

Sections 6.2–6.3 began as a commentary on Sander Greenland's 1997 manuscript entitled "Causation, Confounding, and Collapsibility." Greenland's paper was motivated by considerations similar to those exposed in this chapter, and it was based on a counterfactual–exchangeability approach that he and James Robins introduced to epidemiology in the mid-1980s. I have since joined Sander and Jamie as co-author on "Confounding and Collapsibility in Causal Inference" (Greenland et al. 1999b). However, space limitations and other constraints did not permit the ideas presented in this chapter to be fully expressed in our joint paper.

Technical discussions with James Robin and Sander Greenland were extremely valuable. Sander, in particular, gave many constructive comments on two early drafts and helped to keep them comprehensible to epidemiologists. Jan Koster called my attention to the connection between Stone's and Robins's criteria of no-confounding and caught several oversights in an earlier draft. Other helpful discussants were Michelle Pearl, Bill Shipley, Rolf Steyer, Stephen Stigler, and David Trichler.

CHAPTER SEVEN

The Logic of Structure-Based Counterfactuals

And the Lord said,
"If I find in the city of Sodom fifty good men,
I will pardon the whole place for their sake."
Genesis 18:26

Preface

This chapter provides a formal analysis of structure-based *counterfactuals,* a concept introduced briefly in Chapter 1 that will occupy the rest of our discussion in this book. Through this analysis, we will obtain sharper mathematical definitions of other concepts that were introduced in earlier chapters, including causal models, action, causal effects, causal relevance, error terms, and exogeneity.

After casting the concepts of causal model and counterfactuals in abstract mathematical terms, we will demonstrate by examples how counterfactual questions can be answered from both deterministic and probabilistic causal models (Section 7.1). In Section 7.2.1, we will argue that policy analysis is an exercise in counterfactual reasoning and demonstrate this thesis in a simple example taken from econometrics. This will set the stage for our discussion in Section 7.2.2, where we explicate the empirical content of counterfactuals in terms of policy predictions. Section 7.2.3 discusses the role of counterfactuals in the interpretation and generation of causal explanations. Section 7.2 concludes with discussions of how causal relationships emerge from actions and mechanisms (Section 7.2.4) and how causal directionality can be induced from a set of symmetric equations (Section 7.2.5).

In Section 7.3 we develop an axiomatic characterization of counterfactual and causal relevance relationships as they emerge from the structural model semantics. Section 7.3.1 will identify a set of properties, or axioms, that allow us to derive new counterfactual relations from assumptions, and Section 7.3.2 demonstrates the use of these axioms in algebraic derivation of causal effects. Section 7.3.3 introduces axioms for the relationship of causal relevance and, using their similarity to the axioms of graphs, describes the use of graphs for verifying relevance relationships.

The axiomatic characterization developed in Section 7.3 enables us to compare structural models with other approaches to causality and counterfactuals, most notably those based on Lewis's closest-world semantics (Sections 7.4.1–7.4.4). The formal equivalence of the structural approach and the Neyman–Rubin potential-outcome framework is discussed in Section 7.4.4. Finally, we revisit the topic of exogeneity and extend our discussion of Section 5.4.3 with counterfactual definitions of exogenous and instrumental variables in Section 7.4.5.

The final part of this chapter (Section 7.5) compares the structural account of causality with that based on probabilistic relationships. We elaborate our preference toward the structural account and highlight the difficulties that the probabilistic account is currently facing.

7.1 STRUCTURAL MODEL SEMANTICS

How do scientists predict the outcome of one experiment from the results of other experiments run under totally different conditions? Such predictions require us to envision what the world would be like under various hypothetical changes and so invoke *counterfactual* inference. Though basic to scientific thought, counterfactual inference cannot easily be formalized in the standard languages of logic, algebraic equations, or probability. The formalization of counterfactual inference requires a language within which the invariant relationships in the world are distinguished from transitory relationships that represent one's beliefs about the world, and such distinction is not supported by standard algebras, including the algebra of equations, Boolean algebra, and probability calculus. Structural models offer such distinction, and this section presents a structural model semantics of counterfactuals as defined in Balke and Pearl (1995), Galles and Pearl (1997, 1998), and Halpern (1998).[1] Related approaches have been proposed in Simon and Rescher (1966), Robins (1986), and Ortiz (1999).

We start with a deterministic definition of a causal model, which consists (as we have discussed in earlier chapters) of functional relationships among variables of interest, each relationship representing an autonomous mechanism. Causal and counterfactual relationships are defined in this model in terms of response to local modifications of those mechanisms. Probabilistic relationships emerge naturally by assigning probabilities to background conditions. After demonstrating, by examples, how this model facilitates the computation of counterfactuals in both deterministic and probabilistic contexts (Section 7.1.2), we then present a general method of computing probabilities of counterfactual expressions using causal diagrams (Section 7.1.3).

7.1.1 Definitions: Causal Models, Actions, and Counterfactuals

A "model," in the common use of the word, is an idealized representation of reality that highlights some aspects and ignores others. In logical systems, however, a model is a mathematical object that assigns truth values to sentences in a given language, where each sentence represents some aspect of reality. Truth tables, for example, are models in propositional logic; they assign a truth value to any Boolean expression, which may represent an event or a set of conditions in the domain of interest. A joint probability function, as another example, is a model in probability logic; it assigns a truth value to any sentence of the form $P(A \mid B) < p$, where A and B are Boolean expressions representing events. A *causal model,* naturally, should encode the truth values of sentences

[1] Similar models, called "neuron diagrams" (Lewis 1986, p. 200; Hall 1998) are used informally by philosophers to illustrate chains of causal processes.

that deal with causal relationships; these include action sentences (e.g., "A will be true if we do B"), counterfactuals (e.g., "A would have been different were it not for B"), and plain causal utterances (e.g., "A may cause B" or "B occurred because of A"). Such sentences cannot be interpreted in standard propositional logic or probability calculus because they deal with changes that occur in the external world rather than with changes in our beliefs about a static world. Causal models encode and distinguish information about external changes through an explicit representation of the mechanisms that are altered in such changes.

Definition 7.1.1 (Causal Model)
A causal model *is a triple*

$$M = \langle U, V, F \rangle,$$

where:

(i) *U is a set of* background *variables, (also called* exogenous[2]*), that are determined by factors outside the model;*

(ii) *V is a set $\{V_1, V_2, \ldots, V_n\}$ of variables, called* endogenous, *that are determined by variables in the model – that is, variables in $U \cup V$; and*

(iii) *F is a set of functions $\{f_1, f_2, \ldots, f_n\}$ such that each f_i is a mapping from (the respective domains of) $U \cup (V \setminus V_i)$ to V_i and such that the entire set F forms a mapping from U to V. In other words, each f_i tells us the value of V_i given the values of all other variables in $U \cup V$, and the entire set F has a unique solution $V(u)$.[3] Symbolically, the set of equations F can be represented by writing*

$$v_i = f_i(pa_i, u_i), \quad i = 1, \ldots, n,$$

where pa_i is any realization of the unique minimal set of variables PA_i in $V \setminus V_i$ (connoting parents*) sufficient for representing f_i. Likewise, $U_i \subseteq U$ stands for the unique minimal set of variables in U sufficient for representing f_i.*[4]

Every causal model M can be associated with a directed graph, $G(M)$, in which each node corresponds to a variable and the directed edges point from members of PA_i and U_i toward V_i. We call such a graph the *causal diagram* associated with M. This graph merely identifies the endogenous and background variables that have direct influence on each V_i; it does not specify the functional form of f_i. The convention of confining the parent set PA_i to variables in V stems from the fact that the background variables are often unobservable. In general, however, we can extend the parent sets to include observed variables in U.

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[2] We will try to refrain from using the term "exogenous" in referring to background conditions, because this term has acquired more refined technical connotations (see Sections 5.4.3 and 7.4). The term "predetermined" is used in the econometric literature.

[3] Uniqueness is ensured in recursive (i.e. acyclic) systems. Halpern (1998) allows multiple solutions in nonrecursive systems.

[4] A set of variables X is *sufficient* for representing a function $y = f(x, z)$ if f is trivial in Z – that is, if for every x, z, z' we have $f(x, z) = f(x, z')$.

Definition 7.1.2 (Submodel)
Let M be a causal model, X a set of variables in V, and x a particular realization of X. A submodel M_x *of M is the causal model*

$$M_x = \langle U, V, F_x \rangle,$$

where

$$F_x = \{f_i : V_i \notin X\} \cup \{X = x\}. \tag{7.1}$$

In words, F_x is formed by deleting from F all functions f_i corresponding to members of set X and replacing them with the set of constant functions $X = x$.

Submodels are useful for representing the effect of local actions and hypothetical changes, including those implied by counterfactual antecedents. If we interpret each function f_i in F as an independent physical mechanism and define the action $do(X = x)$ as the minimal change in M required to make $X = x$ hold true under any u, then M_x represents the model that results from such a minimal change, since it differs from M by only those mechanisms that directly determine the variables in X. The transformation from M to M_x modifies the algebraic content of F, which is the reason for the name "modifiable structural equations" used in Galles and Pearl (1998).[5]

Definition 7.1.3 (Effect of Action)
Let M be a causal model, X a set of variables in V, and x a particular realization of X. The effect of action $do(X = x)$ *on M is given by the submodel* M_x.

Definition 7.1.4 (Potential Response)
Let X and Y be two subsets of variables in V. The potential response *of Y to action* $do(X = x)$, *denoted* $Y_x(u)$, *is the solution for Y of the set of equations* F_x.[6]

We will confine our attention to actions in the form of $do(X = x)$. Conditional actions of the form "$do(X = x)$ if $Z = z$" can be formalized using the replacement of equations by functions of Z, rather than by constants (Section 4.2). We will not consider disjunctive actions of the form "$do(X = x \text{ or } Z = z)$," since these complicate the probabilistic treatment of counterfactuals.

Definition 7.1.5 (Counterfactual)
Let X and Y be two subsets of variables in V. The counterfactual sentence "The value that Y would have obtained, had X been x" is interpreted as denoting the potential response $Y_x(u)$.

---

[5] Structural modifications date back to Marschak (1950) and Simon (1953). An explicit translation of interventions into "wiping out" equations from the model was first proposed by Strotz and Wold (1960) and later used in Fisher (1970), Sobel (1990), Spirtes et al. (1993), and Pearl (1995a). A similar notion of submodel was introduced by Fine (1985), though not specifically for representing actions and counterfactuals.

[6] If Y is a set of variables $Y = (Y_1, Y_2, \ldots)$, then $Y_x(u)$ stands for a vector of functions $(Y_{1_x}(u), Y_{2_x}(u), \ldots)$.

Definition 7.1.5 thus interprets the counterfactual phrase "had X been x" in terms of a hypothetical modification of the equations in the model; it simulates an external action (or spontaneous change) that modifies the actual course of history and enforces the condition "$X = x$" with minimal change of mechanisms. This is a crucial step in the semantics of counterfactuals (Balke and Pearl 1994b), as it permits x to differ from the current value of $X(u)$ without creating logical contradiction; it also suppresses abductive inferences (or backtracking) from the counterfactual antecedent $X = x$.[7] In Chapter 3 (Section 3.6.3) we used the notation $Y(x, u)$ to denote the subjunctive conditional "the value that Y would obtain in unit u, had X been x" (as used in the Neyman–Rubin potential-outcome model). Throughout the rest of this book we will use the notation $Y_x(u)$ to denote counterfactuals tied specifically to the structural model interpretation of Definition 7.1.5 (paralleling (3.51)); $Y(x, u)$ will be reserved for generic subjunctive conditionals, uncommitted to any specific semantics.

Definition 7.1.5 endows the atomic mechanisms $\{f_i\}$ themselves with interventional–counterfactual interpretation, because $v_i = f_i(pa_i, u_i)$ is the value of V_i in the submodel $M_{v\setminus v_i}$. In other words, $f_i(pa_i, u_i)$ stands for the potential response of V_i when we hold constant *all* other variables in V.

This formulation generalizes naturally to probabilistic systems as follows.

Definition 7.1.6 (Probabilistic Causal Model)
A probabilistic causal model *is a pair*

$$\langle M, P(u)\rangle,$$

where M is a causal model and $P(u)$ is a probability function defined over the domain of U.

The function $P(u)$, together with the fact that each endogenous variable is a function of U, defines a probability distribution over the endogenous variables. That is, for every set of variables $Y \subseteq V$, we have

$$P(y) \triangleq P(Y = y) = \sum_{\{u \mid Y(u)=y\}} P(u). \tag{7.2}$$

The probability of counterfactual statements is defined in the same manner, through the function $Y_x(u)$ induced by the submodel M_x:

$$P(Y_x = y) = \sum_{\{u \mid Y_x(u)=y\}} P(u). \tag{7.3}$$

Likewise, a causal model defines a joint distribution on counterfactual statements. That is, $P(Y_x = y, Z_w = z)$ is defined for any (not necessarily disjoint) sets of variables Y, X, Z, and W. In particular, $P(Y_x = y, X = x')$ and $P(Y_x = y, Y_{x'} = y')$ are well-defined for $x \neq x'$ and are given by

[7] Simon and Rescher (1966, p. 339) did not include this step in their account of counterfactuals and noted that backward inferences triggered by the antecedents can lead to ambiguous interpretations.

$$P(Y_x = y,\ X = x') = \sum_{\{u \mid Y_x(u)=y\ \&\ X(u)=x'\}} P(u) \tag{7.4}$$

and

$$P(Y_x = y,\ Y_{x'} = y') = \sum_{\{u \mid Y_x(u)=y\ \&\ Y_{x'}(u)=y'\}} P(u). \tag{7.5}$$

If x and x' are incompatible then Y_x and $Y_{x'}$ cannot be measured simultaneously, and it may seem meaningless to attribute probability to the joint statement "Y would be y if $X = x$ *and* Y would be y' if $X = x'$." Such concerns have been a source of recent objections to treating counterfactuals as jointly distributed random variables (Dawid 1997). The definition of Y_x and $Y_{x'}$ in terms of two distinct submodels, driven by a standard probability space over U, explains away these objections (see Section 7.2.2) and further illustrates that joint probabilities of counterfactuals can be encoded rather parsimoniously using $P(u)$ and F.

Of particular interest to us would be probabilities of counterfactuals that are conditional on actual observations. For example, the probability that event $X = x$ "was the cause" of event $Y = y$ may be interpreted as the probability that Y would not be equal to y had X not been x, given that $X = x$ and $Y = y$ have in fact occurred (see Chapter 9 for an in-depth discussion of the probabilities of causation). Such probabilities are well-defined in the model just described; they require the evaluation of expressions of the form $P(Y_{x'} = y' \mid X = x,\ Y = y)$ with x' and y' incompatible with x and y, respectively. Equation (7.4) allows the evaluation of this quantity as follows:

$$\begin{aligned} P(Y_{x'} = y' \mid X = x,\ Y = y) &= \frac{P(Y_{x'} = y',\ X = x,\ Y = y)}{P(X = x,\ Y = y)} \\ &= \sum_u P(Y_{x'}(u) = y') P(u \mid x, y). \end{aligned} \tag{7.6}$$

In other words, we first update $P(u)$ to obtain $P(u \mid x, y)$ and then use the updated distribution $P(u \mid x, y)$ to compute the expectation of the index function $Y_{x'}(u) = y'$.

This substantiates the three-step procedure introduced in Section 1.4, which we now summarize in a theorem.

Theorem 7.1.7

Given model $\langle M, P(u)\rangle$, the conditional probability $P(B_A \mid e)$ of a counterfactual sentence "If it were A then B," given evidence e, can be evaluated using the following three steps.

1. ***Abduction*** *– Update $P(u)$ by the evidence e to obtain $P(u \mid e)$.*
2. ***Action*** *– Modify M by the action $do(A)$, where A is the antecedent of the counterfactual, to obtain the submodel M_A.*
3. ***Prediction*** *– Use the modified model $\langle M_A, P(u \mid e)\rangle$ to compute the probability of B, the consequence of the counterfactual.*

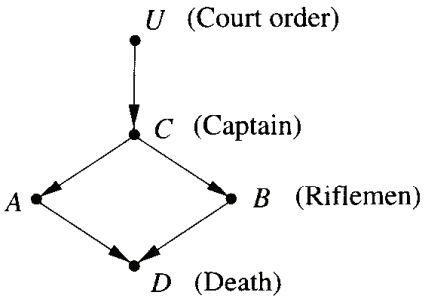

Figure 7.1 Causal relationships in the example of the two-man firing squad.

To complete this section, we introduce two additional objects that will prove useful in subsequent discussions: *worlds*[8] and *theories.*

Definition 7.1.8 (Worlds and Theories)
A causal world *w is a pair* $\langle M, u \rangle$, *where M is a causal model and u is a particular realization of the background variables U. A causal* theory *is a set of causal worlds.*

A world w can be viewed as a degenerate probabilistic model for which $P(u) = 1$. Causal theories will be used to characterize partial specifications of causal models, for example, models sharing the same causal diagram or models in which the functions f_i are linear with undetermined coefficients.

7.1.2 Evaluating Counterfactuals: Deterministic Analysis

In Section 1.4.1 we presented several examples demonstrating the interpretation of actions and counterfactuals in structural models. We now apply the definitions of Section 7.1.1 to demonstrate how counterfactual queries, both deterministic and probabilistic, can be answered formally using structural model semantics.

Example 1: The Firing Squad

Consider a two-man firing squad as depicted in Figure 7.1, where A, B, C, D, and U stand for the following propositions:

U = court orders the execution;
C = captain gives a signal;
A = rifleman A shoots;
B = rifleman B shoots;
D = prisoner dies.

Assume that the court's decision is unknown, that both riflemen are accurate, alert, and law-abiding, and that the prisoner is not likely to die from fright or other extraneous causes. We wish to construct a formal representation of the story, so that the following sentences can be evaluated mechanically.

[8] Adnan Darwiche called my attention to the importance of this object.

S1 *Prediction* – If rifleman A did not shoot then the prisoner is alive:

$$\neg A \implies \neg D.$$

S2 *Abduction* – If the prisoner is alive, then the captain did not signal:

$$\neg D \implies \neg C.$$

S3 *Transduction* – If rifleman A shot, then B shot as well:

$$A \implies B.$$

S4 *Action* – If the captain gave no signal and rifleman A decides to shoot, then the prisoner will die and B will not shoot.

$$\neg C \implies D_A \ \& \ \neg B_A.$$

S5 *Counterfactual* – If the prisoner is dead, then the prisoner would be dead even if rifleman A had not shot:

$$D \implies D_{\neg A}.$$

Evaluating Standard Sentences

To prove the first three sentences we need not invoke causal models; these sentences involve standard logical connectives and thus can be handled using standard logical deduction. The story can be captured in any convenient logical theory (a set of propositional sentences), for example,

$$T_1\colon\ U \iff C,\ C \iff A,\ C \iff B,\ A \vee B \iff D$$

or

$$T_2\colon\ U \iff C \iff A \iff B \iff D,$$

where each theory admits the two logical models

$$m_1\colon\ \{U, C, A, B, D\} \quad \text{and} \quad m_2\colon\ \{\neg U, \neg C, \neg A, \neg B, \neg D\}.$$

In words, any theory T that represents our story should imply that either all five propositions are true or all are false; models m_1 and m_2 present these two possibilities explicitly. The validity of S1–S3 can easily be verified, either by derivation from T or by noting that the antecedent and consequent in each sentence are both part of the same model.

Two remarks are worth making before we go on to analyze sentences S4 and S5. First, the two-way implications in T_1 and T_2 are necessary for supporting abduction; if we were to use one-way implications (e.g. $C \implies A$) then we would not be able to conclude C from A. In standard logic, this symmetry removes all distinctions between the tasks of prediction (reasoning forward in time), abduction (reasoning from evidence to explanation), and transduction (reasoning from evidence to explanation and then from explanation to predictions). Using two-way implication, these three modes of reasoning differ only in the interpretations they attach to antecedents and consequents of conditional sentences – not in their methods of inference. In nonstandard logics (e.g., logic programming), where the implication sign dictates the direction of inference and even contraposition is not licensed, metalogical inference machinery must be invoked to perform abduction (Eshghi and Kowalski 1989).

Second, the feature that renders S1-S3 manageable in standard logic is that they all deal with *epistemic* inference – that is, inference from beliefs to beliefs about a static world. Sentence S2, for example, can be explicated to state: If we find that the prisoner is alive then we have the license to believe that the captain did not give the signal. The material implication sign ($\Longrightarrow$) in logic does not extend beyond this narrow meaning, to be contrasted next with the counterfactual implication.

Evaluating Action Sentences

Sentence S4 invokes a deliberate action, "rifleman A decides to shoot." From our discussion of actions (see e.g. Chapter 4 or Definition 7.1.3), any such action must violate some premises, or mechanisms, in the initial theory of the story. To formally identify what remains invariant under the action, we must incorporate causal relationships into the theory; logical relationships alone are not sufficient. The causal model corresponding to our story is as follows.

Model M

$$\begin{array}{ll} & (U) \\ C = U & (C) \\ A = C & (A) \\ B = C & (B) \\ D = A \vee B & (D) \end{array}$$

Here we use equality rather than implication in order to (i) permit two-way inference and (ii) stress that, unlike logical sentences, each equation represents an autonomous mechanism (an "integrity constraint" in the language of databases) – it remains invariant unless specifically violated. We further use parenthetical symbols next to each equation in order to identify explicitly the dependent variable (on the l.h.s.) in the equation, thus representing the causal asymmetry associated with the arrows in Figure 7.1.

To evaluate S4, we follow Definition 7.1.3 and form the submodel M_A, in which the equation $A = C$ is replaced by A (simulating the decision of of rifleman A to shoot regardless of signals).

Model M_A

$$\begin{array}{ll} & (U) \\ C = U & (C) \\ A & (A) \\ B = C & (B) \\ D = A \vee B & (D) \end{array}$$

Facts: $\neg C$

Conclusions: $A, D, \neg B, \neg U, \neg C$

We see that, given $\neg C$, we can easily deduce D and $\neg B$ and thus confirm the validity of S4.

It is important to note that "problematic" sentences like S4, whose antecedent violates one of the basic premises in the story (i.e., that both riflemen are law-abiding) are handled naturally in the same deterministic setting in which the story is told. Traditional

logicians and probabilists tend to reject sentences like S4 as contradictory and insist on reformulating the problem probabilistically so as to tolerate exceptions to the law $A = C$.[9] Such reformulations are unnecessary; the structural approach permits us to process commonplace causal statements in their natural deterministic habitat without first immersing them in nondeterministic decor. In this framework, all laws are understood to represent "defeasible" default expressions – subject to breakdown by deliberate intervention. The basic laws of physics remain immutable, of course, but their applicability to any given scenario is subject to modification by agents' actions or external intervention.

Evaluating Counterfactuals

We are now ready to evaluate the counterfactual sentence S5. Following Definition 7.1.5, the counterfactual $D_{\neg A}$ stands for the value of D in submodel $M_{\neg A}$. This value is ambiguous because it depends on the value of U, which is not specified in $M_{\neg A}$. The observation D removes this ambiguity; upon finding the prisoner dead we can infer that the court has given the order (U) and, consequently, if rifleman A had refrained from shooting then rifleman B would have shot and killed the prisoner, thus confirming $D_{\neg A}$.

Formally, we can derive $D_{\neg A}$ by using the steps of Theorem 7.1.7 (though no probabilities are involved). We first add the fact D to the original model M and evaluate U; then we form the submodel $M_{\neg A}$ and reevaluate the truth of D in $M_{\neg A}$, using the value of U found in the first step. These steps are explicated as follows.

Step 1

Model M

	(U)
$C = U$	(C)
$A = C$	(A)
$B = C$	(B)
$D = A \vee B$	(D)

Facts: D

Conclusions: U, A, B, C, D

Step 2

Model $M_{\neg A}$

	(U)
$C = U$	(C)
$\neg A$	(A)
$B = C$	(B)
$D = A \vee B$	(D)

Facts: U

Conclusions: $U, \neg A, C, B, D$

[9] This problem, I speculate, was one of the primary forces for the emergence of probabilistic causality in the 1960s (see Section 7.5 for review).

Note that it is only the value of U, the background variable, that is carried over from step 1 to step 2; all other propositions must be reevaluated subject to the new modification of the model. This reflects the understanding that background factors U are not affected by either the variables or the mechanisms in the model $\{f_i\}$; hence, the counterfactual consequent (in our case, D) must be evaluated under the same background conditions as those prevailing in the actual world. In fact, the background variables are the main carriers of information from the actual world to the hypothetical world; they serve as the "guardians of invariance" (or persistence) in the dynamic process that transforms the former into the latter (an observation by David Heckerman, personal communication).

Note also that this two-step procedure of evaluating counterfactuals can be combined into one. If we use an asterisk to distinguish postmodification from premodification variables, then we can combine M and M_x into one logical theory and prove the validity of S5 by purely logical deduction in the combined theory. To illustrate, we write S5 as $D \Longrightarrow D^*_{\neg A^*}$ (read: If D is true in the actual world, then D would also be true in the hypothetical world created by the modification $\neg A^*$) and prove the validity of D^* in the combined theory as follows.

Combined Theory

		(U)
$C^* = U$	$C = U$	(C)
$\neg A^*$	$A = C$	(A)
$B^* = C^*$	$B = C$	(B)
$D^* = A^* \vee B^*$	$D = A \vee B$	(D)

Facts: D

Conclusions: $U, A, B, C, D, \neg A^*, C^*, B^*, D^*$

Note that U need not be "starred," reflecting the assumption that background conditions remain unaltered.

It is worth reflecting at this point on the difference between S4 and S5. The two appear to be syntactically identical, as both involve a fact implying a counterfactual, and yet we labeled S4 an "action" sentence and S5 a "counterfactual" sentence. The difference lies in the relationship between the given fact and the antecedent of the counterfactual (i.e., the "action" part). In S4, the fact given ($\neg C$) is not affected by the antecedent (A); in S5, the fact given (D) is potentially affected by the antecedent ($\neg A$). The difference between these two situations is fundamental, as can be seen from their methods of evaluation. In evaluating S4, we knew in advance that C would not be affected by the model modification $do(A)$; therefore, we were able to add C directly to the modified model M_A. In evaluating S5, on the other hand, we were contemplating a possible reversal, from D to $\neg D$, attributable to the modification $do(\neg A)$. As a result, we first had to add fact D to the preaction model M, summarize its impact via U, and reevaluate D once the modification $do(\neg A)$ takes place. Thus, although the causal effect of actions can be expressed syntactically as a counterfactual sentence, this need to route the impact of known facts through U makes counterfactuals a different species than actions (see Section 1.4).

We should also emphasize that most counterfactual utterances in natural language presume, often implicitly, knowledge of facts that are affected by the antecedent. For

example, when we say that "B would be different were it not for A," we imply knowledge of what the actual value of B is and that B is susceptible to A. It is this sort of relationship that gives counterfactuals their unique character – distinct from action sentences – and, as we saw in Section 1.4, it is this sort of sentence that would require a more detailed specification for its evaluation: some knowledge of the functional mechanisms $f_i(pa_i, u_i)$ would be necessary.

7.1.3 Evaluating Counterfactuals: Probabilistic Analysis

To demonstrate the probabilistic evaluation of counterfactuals (equations (7.3)–(7.5)), let us modify the firing-squad story slightly, assuming that:

1. there is a probability $P(U) = p$ that the court has ordered the execution;
2. rifleman A has a probability q of pulling the trigger out of nervousness; and
3. rifleman A's nervousness is independent of U.

With these assumptions, we wish to compute the quantity $P(\neg D_{\neg A} \mid D)$ – namely, the probability that the prisoner would be alive if A had not shot, given that the prisoner is in fact dead.

Intuitively, we can figure out the answer by noting that $\neg D_{\neg A}$ is true if and only if the court has not issued an order. Thus, our task amounts to that of computing $P(\neg U \mid D)$, which evaluates to $q(1-p)/[1-(1-q)(1-p)]$. However, our aim is to demonstrate a general and formal method of deriving such probabilities, based on (7.4), that makes little use of intuition.

The probabilistic causal model (Definition 7.1.6) associated with the new story contains two background variables, U and W, where W stands for rifleman A's nervousness. This model is given as follows.

Model $\langle M, P(u, w)\rangle$

$$
\begin{array}{ll}
 & (U, W) \sim P(u, w) \\
C = U & (C) \\
A = C \vee W & (A) \\
B = C & (B) \\
D = A \vee B & (D)
\end{array}
$$

In this model, the background variables are distributed as

$$
P(u, w) = \begin{cases} pq & \text{if } u = 1,\ w = 1, \\ p(1-q) & \text{if } u = 1,\ w = 0, \\ (1-p)q & \text{if } u = 0,\ w = 1, \\ (1-p)(1-q) & \text{if } u = 0,\ w = 0. \end{cases} \tag{7.7}
$$

Following Theorem 7.1.7, our first step (abduction) is to compute the posterior probability $P(u, w \mid D)$, accounting for the fact that the prisoner is found dead. This is easily evaluated to:

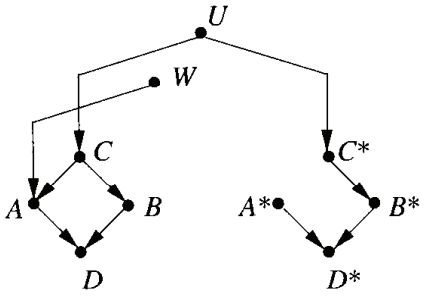

Figure 7.2 Twin network representation of the firing squad.

$$P(u, w \mid D) = \begin{cases} \frac{P(u,w)}{1-(1-p)(1-q)} & \text{if } u = 1 \text{ or } w = 1, \\ 0 & \text{if } u = 0 \text{ and } w = 0. \end{cases} \tag{7.8}$$

The second step (action) is to form the submodel $M_{\neg A}$ while retaining the posterior probability of (7.8).

Model $\langle M_{\neg A}, P(u, w \mid D)\rangle$

$$\begin{aligned} & (U, W) \sim P(u, w \mid D) \\ C = U & \quad (C) \\ \neg A & \quad (A) \\ B = C & \quad (B) \\ D = A \vee B & \quad (D) \end{aligned}$$

The last step (prediction) is to compute $P(\neg D)$ in this probabilistic model. Noting that $\neg D = \neg U$, the result (as expected) is

$$P(\neg D_{\neg A} \mid D) = P(\neg U \mid D) = \frac{q(1-p)}{1-(1-q)(1-p)}.$$

7.1.4 The Twin Network Method

A major practical difficulty in the procedure just described is the need to compute, store, and use the posterior distribution $P(u \mid e)$, where u stand for the set of all background variables in the model. As illustrated in the preceding example, even when we start with Markovian model in which the background variables are mutually independent, conditioning on e normally destroys this independence and so makes it necessary to carry over a full description of the joint distribution of U, conditional on e. Such description may be prohibitively large if encoded in the form of a table, as we have done in (7.8).

A graphical method of overcoming this difficulty is described in Balke and Pearl (1994b); it uses two networks, one to represent the actual world and one to represent the hypothetical world. Figure 7.2 illustrates this construction for the firing-squad story analyzed.

The two networks are identical in structure, save for the arrows entering A^*, which have been deleted to mirror the equation deleted from $M_{\neg A}$. Like Siamese twins, the two networks share the background variables (in our case, U and W), since those remain invariant under modification. The endogenous variables are replicated and labeled distinctly, because they may obtain different values in the hypothetical versus the actual

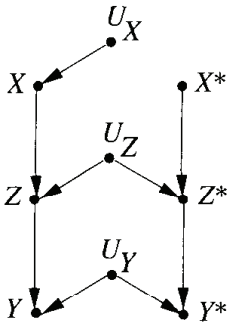

Figure 7.3 Twin network representation of the counterfactual Y_x in the model $X \rightarrow Z \rightarrow Y$.

world. The task of computing $P(\neg D)$ in the model $\langle M_{\neg A}, P(u, v \mid D)\rangle$ thus reduces to that of computing $P(\neg D^* \mid D)$ in the twin network shown, setting A^* to false.

In general, if we wish to compute the counterfactual probability $P(Y_x = y \mid z)$, where X, Y, and Z are arbitrary sets of variables (not necessarily disjoint), Theorem 7.1.7 instructs us to compute $P(y)$ in the submodel $\langle M_x, P(u \mid z)\rangle$, which reduces to computing an ordinary conditional probability $P(y^* \mid z)$ in an augmented Bayesian network. Such computation can be performed by standard evidence propagation techniques. The advantages of delegating this computation to inference in a Bayesian network are that the distribution $P(u \mid z)$ need not be explicated, conditional independencies can be exploited, and local computation methods can be employed (such as those summarized in Section 1.2.4).

The twin network representation also offers a useful way of testing independencies among counterfactual quantities. To illustrate, suppose that we have a chainlike causal diagram, $X \rightarrow Z \rightarrow Y$, and that we wish to test whether Y_x is independent of X given Z (i.e., $Y_x \perp\!\!\!\perp X \mid Z$). The twin network associated with this chain is shown in Figure 7.3. To test whether $Y_x \perp\!\!\!\perp X \mid Z$ holds in the original model, we test whether Z d-separates X from Y^* in the twin network. As can be easily seen (via Definition 1.2.3), conditioning on Z renders the path between X and Y^* d-connected through the collider at Z and hence $Y_x \perp\!\!\!\perp X \mid Z$ does not hold in the model. This conclusion is not easily discernible from the chain model itself or from the equations in that model. In the same fashion, we can see that whenever we condition on either Y or on $\{Y, Z\}$, we form a connection between Y^* and X; hence, Y_x and X are not independent conditional on those variables. The connection is disrupted, however, if we do not condition on either Y or Z, in which case $Y_x \perp\!\!\!\perp X$.

The twin network reveals an interesting interpretation of counterfactuals of the form Z_{pa_Z}, where Z is any variable and PA_Z stands for the set of Z's parents. Consider the question of whether Z_x is independent of some given set of variables in the model of Figure 7.3. The answer to this question depends on whether Z^* is d-separated from that set of variables. However, any variable that is d-separated from Z^* would also be d-separated from U_Z, so the node representing U_Z can serve as a proxy for representing the counterfactual variable Z_x. This is not a coincidence, considering that Z is governed by the equation $z = f_Z(x, u_Z)$. By definition, the distribution of Z_x is equal to the distribution of Z under the condition where X is held fixed at x. Under such condition, Z may vary only if U_Z varies. Therefore, if U_Z obeys a certain independence relationship then Z_x (more generally, Z_{pa_Z}) must obey that relationship as well. We thus obtain a simple graphical

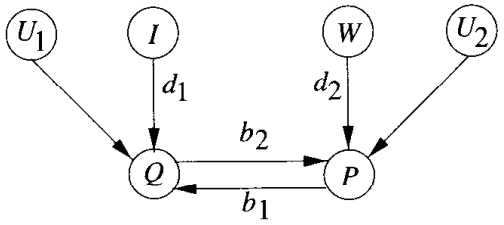

Figure 7.4 Causal diagram illustrating the relationship between price (P) and demand (Q).

representation for any counterfactual variable of the form Z_{pa_Z}. Using this representation, we can easily verify from Figure 7.3 that $(Y^* \perp\!\!\!\perp X \mid \{Z, U_Z, Y\})_G$ and $(Y^* \perp\!\!\!\perp X \mid \{U_Y, U_Z, Y\})_G$ both hold in the twin network and therefore

$$Y_x \perp\!\!\!\perp X \mid \{Z, Z_x, Y\} \quad \text{and} \quad Y_x \perp\!\!\!\perp X \mid \{Y_z, Z_x, Y\}$$

must hold in the model. The verification of such independencies is important for deciding the identification of plans, because these independencies permit us to reduce counterfactual probabilities to ordinary probabilistic expression on observed variables (see Section 7.3.2).

7.2 APPLICATIONS AND INTERPRETATION OF STRUCTURAL MODELS

7.2.1 Policy Analysis in Linear Econometric Models: An Example

In Section 1.4 we illustrated the nature of structural equations modeling using the canonical economic problem of demand and price equilibrium (see Figure 7.4). In this chapter, we use this problem to answer policy-related questions.

To recall, this example consists of the two equations

$$q = b_1 p + d_1 i + u_1, \tag{7.9}$$

$$p = b_2 q + d_2 w + u_2, \tag{7.10}$$

where q is the quantity of household demand for a product A, p is the unit price of product A, i is household income, w is the wage rate for producing product A, and u_1 and u_2 represent error terms – unmodeled factors that affect quantity and price, respectively (Goldberger 1992).

This system of equations constitutes a causal model (Definition 7.1.1) if we define $V = \{Q, P\}$ and $U = \{U_1, U_2, I, W\}$ and assume that each equation represents an autonomous process in the sense of Definition 7.1.3. It is normally assumed that I and W are observed, while U_1 and U_2 are unobservable and independent in I and W. Since the error terms U_1 and U_2 are unobserved, a complete specification of the model must include the distribution of these errors, which is usually taken to be Gaussian with the covariance matrix $\Sigma_{ij} = \text{cov}(u_i, u_j)$. It is well known in economics (dating back to Wright 1928) that the assumptions of linearity, normality, and the independence of $\{I, W\}$ and $\{U_1, U_2\}$ permit consistent estimation of all model parameters, including the covariance matrix Σ_{ij}. However, the focus of this book is not the estimation of parameters but rather their

utilization in policy predictions. Accordingly, we will demonstrate how to evaluate the following three queries.

1. What is the expected value of the demand Q if the price is *controlled at* $P = p_0$?
2. What is the expected value of the demand Q if the price is *reported to be* $P = p_0$?
3. Given that the current price is $P = p_0$, what would be the expected value of the demand Q if we *were to control* the price at $P = p_1$?

The reader should recognize these queries as representing (respectively) actions, predictions, and counterfactuals – our three-level hierarchy. The second query, representing prediction, is standard in the literature and can be answered directly from the covariance matrix without reference to causality, structure, or invariance. The first and third queries rest on the structural properties of the equations and, as expected, are not treated in the standard literature of structural equations.[10]

In order to answer the first query, we replace (7.10) with $p = p_0$, leaving

$$q = b_1 p + d_1 i + u_1, \tag{7.11}$$

$$p = p_0, \tag{7.12}$$

with the statistics of U_1 and I unaltered. The controlled demand is then $q = b_1 p_0 + d_1 i + u_1$, and its expected value (conditional on $I = i$) is given by

$$E[Q \mid do(P = p_0), i] = b_1 p_0 + d_1 i + E(U_1 \mid i). \tag{7.13}$$

Since U_1 is independent of I, the last term evaluates to

$$E(U_1 \mid i) = E(U_1) = E(Q) - b_1 E(P) - d_1 E(I)$$

and, substituted into (7.13), yields

$$E[Q \mid do(P = p_0), i] = E(Q) + b_1(p_0 - E(P)) + d_1(i - E(I)).$$

The answer to the second query is obtained by conditioning (7.9) on the current observation $\{P = p_0, I = i, W = w\}$ and taking the expectation,

$$E(Q \mid p_0, i, w) = b_1 p_0 + d_1 i + E(U_1 \mid p_0, i, w). \tag{7.14}$$

The computation of $E[U_1 \mid p_0, i, w]$ is a standard procedure once Σ_{ij} is given (Whittaker 1990, p. 163). Note that, although U_1 was assumed to be independent of I and W, this independence no longer holds once $P = p_0$ is observed. Note also that (7.9) and (7.10)

[10] I have presented this example to well over a hundred econometrics students and faculty across the United States. Respondents had no problem answering question 2, one person was able to solve question 1, and none managed to answer question 3. Chapter 5 (Section 5.1) suggests an explanation.

both participate in the solution and that the observed value p_0 will affect the expected demand Q (through $E(U_1 \mid p_0, i, w)$) even when $b_1 = 0$, which is not the case in query 1.

The third query requires the expectation of the counterfactual quantity $Q_{P=p_1}$, conditional on the current observations $\{P = p_0, I = i, W = w\}$. According to Definition 7.1.5, $Q_{P=p_1}$ is governed by the submodel

$$q = b_1 p + d_1 i + u_1, \tag{7.15}$$

$$p = p_1; \tag{7.16}$$

the density of u_1 should be conditioned on the observations $\{P = p_0, I = i, W = w\}$. We therefore obtain

$$E(Q_{p=p_1} \mid p_0, i, w) = b_1 p_1 + d_1 i + E(U_1 \mid p_0, i, w). \tag{7.17}$$

The expected value $E(U_1 \mid p_0, i, w)$ is the same as in the solution to the second query; the latter differs only in the term $b_1 p_1$. A general matrix method for evaluating counterfactual queries in linear Gaussian models is described in Balke and Pearl (1995).

At this point, it is worth emphasizing that the problem of computing counterfactual expectations is not an academic exercise; it represents in fact the typical case in almost every decision-making situation. Whenever we undertake to predict the effect of policy, two considerations apply. First, the policy variables (e.g., price and interest rates in economics, pressure and temperature in process control) are rarely exogenous. Policy variables are endogenous when we observe a system under operation; they become exogenous in the planning phase, when we contemplate actions and changes. Second, policies are rarely evaluated in the abstract; rather, they are brought into focus by certain eventualities that demand remedial correction. In troubleshooting, for example, we observe undesirable effects e that are influenced by other conditions $X = x$ and wish to predict whether an action that brings about a change in X would remedy the situation. The information provided by e is extremely valuable, and it must be processed (using abduction) before we can predict the effect of any action. This step of abduction endows practical queries about actions with a counterfactual character, as we have seen in the evaluation of the third query (7.17).

The current price p_0 reflects economic conditions (e.g. Q) that prevail at the time of decision, and these conditions are presumed to be changeable by the policies considered. Thus, the price P represents an endogenous decision variable (as shown in Figure 7.4) that becomes exogenous in deliberation, as dictated by the submodel $M_{P=p_1}$. The hypothetical mood of query 3 translates into a practical problem of policy analysis: "Given that the current price is $P = p_0$, find the expected value of the demand (Q) if we change the price *today* to $P = p_1$." The reasons for using hypothetical phrases in practical decision-making situations are discussed in the next section.

7.2.2 The Empirical Content of Counterfactuals

The word "counterfactual" is a misnomer, since it connotes a statement that stands contrary to facts or, at the very least, a statement that escapes empirical verification. Counterfactuals are in neither category; they are fundamental to scientific thought and carry as clear an empirical message as any scientific law.

Consider Ohm's law, $V = IR$. The empirical content of this law can be encoded in two alternative forms.

1. *Predictive form:* If at time t_0 we measure current I_0 and voltage V_0 then, ceteris paribus, at any future times $t > t_0$, if the current flow is $I(t)$ then the voltage will be
$$V(t) = \frac{V_0}{I_0} I(t).$$
2. *Counterfactual form:* If at time t_0 we measure current I_0 and voltage V_0 then, had the current flow at time t_0 been I' instead of I_0, the voltage would have been
$$V' = \frac{V_0 I'}{I_0}.$$

On the surface, it seems that the predictive form makes meaningful and testable empirical claims whereas the counterfactual form merely speculates about events that have not (and could not have) occurred, since it is impossible to apply two different currents into the same resistor at the same time. However, if we interpret the counterfactual form to be neither more nor less than a conversational shorthand of the predictive form, the empirical content of the former shines through clearly. Both enable us to make an infinite number of predictions from just one measurement (I_0, V_0), and both derive their validity from a scientific law that ascribes a time-invariant property (the ratio V/I) to any object that conducts electricity.

But if counterfactual statements are merely a roundabout way of stating sets of predictions, why do we resort to such convoluted modes of expression instead of using the predictive mode directly? One obvious answer is that we often use counterfactuals to convey not the predictions themselves but rather the logical ramifications of those predictions. For example, the intent of saying: "if A were not to have shot, then the prisoner would still be alive" may be merely to convey the factual information that B did not shoot. The counterfactual mood, in this case, serves to supplement the fact conveyed with logical justification based on a general law. The less obvious answer rests with the ceteris paribus (all else held equal) qualification that accompanies the predictive claim, which is not entirely free of ambiguities. What should be held constant when we change the current in a resistor – the temperature? the laboratory equipment? the time of day? Certainly not the reading on the voltmeter!

Such matters must be carefully specified when we pronounce predictive claims and take them seriously. Many of these specifications are implicit (and hence superfluous) when we use counterfactual expressions, especially when we agree on the underlying causal model. For example, we do not need to specify under what temperature and pressure the predictions should hold true; these are implied by the statement "had the current flow at time t_0 been I', instead of I_0." In other words, we are referring to precisely those conditions that prevailed in our laboratory at time t_0. The statement also implies that we do not really mean for anyone to hold the reading on the voltmeter constant; variables should run their natural course, and the only change we should envision is in the mechanism that (according to our causal model) is currently determining the current.

To summarize, a counterfactual statement might well be interpreted as conveying a set of predictions under a well-defined set of conditions – those prevailing in the factual part of the statement. For these predictions to be valid, two components must remain invariant: the laws (or mechanisms) and the boundary conditions. Cast in the language of structural models, the laws correspond to the equations $\{f_i\}$ and the boundary conditions correspond to the state of the background variables U. Thus, a precondition for the validity of the predictive interpretation of a counterfactual statement is the assumption that U will not change when our predictive claim is to be applied or tested.

This is best illustrated by using a betting example. We must bet heads or tails on the outcome of a fair coin toss; we win a dollar if we guess correctly and lose one if we don't. Suppose we bet heads and win a dollar, without glancing at the outcome of the coin. Consider the counterfactual "Had I bet differently I would have lost a dollar." The predictive interpretation of this sentence translates into the implausible claim: "If my next bet is tails, I will lose a dollar." For this claim to be valid, two invariants must be assumed: the payoff policy and the outcome of the coin. Whereas the former is a plausible assumption in a betting context, the latter would be realized in only rare circumstances. It is for this reason that the predictive utility of the statement "Had I bet differently I would have lost a dollar" is rather low, and some would even regard it as hindsighted nonsense. It is the persistence across time of U and $f(x, u)$ that endows counterfactual expressions with predictive power; absent this persistence, the counterfactual loses its obvious predictive utility.

However, there is an element of utility in counterfactuals that does not translate immediately to predictive payoff and thus may serve to explain the ubiquity of counterfactuals in human discourse. I am thinking of explanatory value. Suppose, in the betting story, coins were tossed afresh for every bet. Is there no value whatsoever to the statement "Had I bet differently I would have lost a dollar?" I believe there is; it tells us that we are not dealing here with a whimsical bookie but instead with one who at least glances at the bet, compares it to some standard, and decides a win or a loss using a consistent policy. This information may not be very useful to us as players, but it may be useful to, say, state inspectors who come every so often to calibrate the gambling machines and so ensure the state's take of the profit. More significantly, it may be useful to us players, too, if we venture to cheat slightly – say, by manipulating the trajectory of the coin, or by installing a tiny transmitter to tell us which way the coin landed. For such cheating to work, we should know the payoff policy $y = f(x, u)$, and the statement "Had I bet differently I would have lost a dollar" reveals important aspects of that policy.

Is it far-fetched to argue for the merit of counterfactuals by hypothesizing unlikely situations where players cheat and rules are broken? I suggest that such unlikely operations are precisely the norm for gauging the explanatory value of sentences. It is the nature of any causal explanation that its utility be proven not over standard situations but rather over novel settings that require innovative manipulations of the standards. The utility of understanding how television works comes not from turning the knobs correctly but from the ability to repair a TV set when it breaks down. Recall that every causal model advertises not one but rather a host of submodels, each created by violating some laws. The autonomy of the mechanisms in a causal model thus stands for an open invitation to

remove or replace those mechanisms, and it is only natural that the explanatory value of sentences be judged by how well they predict the ramifications of such replacements.

Counterfactuals with Intrinsic Nondeterminism

Recapping our discussion, we see that counterfactuals may earn predictive value under two conditions: (1) when the unobserved uncertainty-producing variables (U) remain constant (until our next prediction or action); or (2) when the uncertainty-producing variables offer the potential of being observed sometime in the future (before our next prediction or action). In both cases, we also need to ensure that the outcome-producing mechanism $f(x, u)$ persists unaltered.

These conclusions raise interesting questions regarding the use of counterfactuals in microscopic phenomena, as none of these conditions holds for the type of uncertainty that we encounter in quantum theory. Heisenberg's die is rolled afresh billions of times each second, and our measurement of U will never be fine enough to remove all uncertainty from the response equation $y = f(x, u)$. Thus, when we include quantum-level processes in our analysis we face a dilemma: either dismiss all talk of counterfactuals (a strategy recommended by some researchers, including Dawid 1997) or continue to use counterfactuals but limit their usage to situations where they assume empirical meaning. This amounts to keeping in our analysis only those U that satisfy conditions (1) and (2) of the previous paragraph. Instead of hypothesizing U that completely remove all uncertainties, we admit only those U that are either (1) persistent or (2) potentially observable.

Naturally, coarsening the granularity of the background variables has its price: the mechanism equations $v_i = f_i(pa_i, u_i)$ lose their deterministic character and hence should be made stochastic. Instead of constructing causal models from a set of deterministic equations $\{f_i\}$, we should consider models made up of stochastic functions $\{f_i^*\}$, where each f_i^* is a mapping from $V \cup U$ to some intrinsic probability distribution $P^*(v_i)$ over the states of V_i. This option leads to a causal Bayesian network (Section 1.3) in which the conditional probabilities $P^*(v_i \mid pa_i, u_i)$ represent intrinsic nondeterminism (sometimes called "objective chance"; Skyrms 1980) and in which the root nodes represent background variables U that are either persistent or potentially observable. In this representation, counterfactual probabilities $P(Y_x = y \mid e)$ can still be evaluated using the three steps (abduction, action, and prediction) of Theorem 7.1.7. In the abduction phase, we condition the prior probability $P(u)$ of the root nodes on the evidence available, e, and so obtain $P(u \mid e)$. In the action phase, we delete the arrows entering variables in set X and instantiate their values to $X = x$. Finally, in the prediction phase, we compute the probability of $Y = y$ resulting from the updated manipulated network.

This evaluation can, of course, be implemented in ordinary causal Bayesian networks (i.e., not only in ones that represent intrinsic nondeterminism), but in that case the results computed would not represent the probability of the counterfactual $Y_x = y$. Such evaluation amounts to assuming that units are homogeneous, with each possessing the stochastic properties of the population – namely, $P(v_i \mid pa_i, u) = P(v_i \mid pa_i)$. Such an assumption may be adequate in quantum-level phenomena, where units stands for specific experimental conditions, but it will not be adequate in macroscopic phenomena, where units may differ appreciably from each other. In the example of Chapter 1 (Section 1.4.4, Figure 1.6), the stochastic attribution amounts to assuming that no individual

is affected by the drug (as dictated by model 1) while ignoring the possibility that some individuals may, in fact, be more sensitive to the drug than others (as in model 2).

7.2.3 Causal Explanations, Utterances, and Their Interpretation

It is a commonplace wisdom that explanation improves understanding and that he who understands more can reason and learn more effectively. It is also generally accepted that the notion of explanation cannot be divorced from that of causation; for example, a symptom may explain our *belief* in a disease, but it does not explain the disease itself. However, the precise relationship between causes and explanations is still a topic of much discussion (Cartwright 1989; Woodward 1997). Having a formal theory of causality and counterfactuals in both deterministic and probabilistic settings casts new light on the question of what constitutes an adequate explanation, and it opens new possibilities for automatic generation of explanations by machine.

A natural starting point for generating explanations would be to use a causal Bayesian network (Section 1.3) in which the events to be explained (explanadum) consist of some combination e of instantiated nodes in the network, and where the task is to find an instantiation c of a subset of e's ancestors (i.e. causes) that maximizes some measure of "explanatory power," namely, the degree to which c explains e. However, the proper choice of this measure is unsettled. Many philosophers and statisticians argue for the likelihood ratio $L = \frac{P(e|c)}{P(e|c')}$ as the proper measure of the degree to which c is a better explanation of e than c'. In Pearl (1988b, chap. 5) and Peng and Reggia (1986), the best explanation is found by maximizing the posterior probability $P(c \mid e)$. Both measures have their faults and have been criticized by several researchers, including Pearl (1988b), Shimony (1991, 1993), Suermondt and Cooper (1993), and Chajewska and Halpern (1997). To remedy these faults, more intricate combinations of the probabilistic parameters $[P(e \mid c), P(e \mid c'), P(c), P(c')]$ have been suggested, none of which seems to capture well the meaning people attach to the word "explanation."

The problem with probabilistic measures is that they cannot capture the strength of a *causal* connection between c and e; any proposition h whatsoever can, with a small stretch of imagination, be thought of as having some influence on e, however feeble. This would then qualify h as an ancestor of e in the causal network and would permit h to compete and win against genuine explanations by virtue of h having strong spurious association with e.

To rid ourselves of this difficulty, we must go beyond probabilistic measures and concentrate instead on causal parameters, such as causal effects $P(y \mid do(x))$ and counterfactual probabilities $P(Y_{x'} = y' \mid x, y)$, as the basis for defining explanatory power. Here x and x' range over the set of alternative explanations, and Y is the set of response variables observed to take on the value y. The expression $P(Y_{x'} = y' \mid x, y)$ is read as: the probability that Y would take on a different value, y', had X been x' (instead of the actual values x). (Note that $P(y \mid do(x)) \triangleq P(Y_x = y)$.) The developments of computational models for evaluating causal effects and counterfactual probabilities now make it possible to combine these parameters with standard probabilistic parameters and so synthesize a more faithful measure of explanatory power that may guide the selection and generation of adequate explanations.

These possibilities trigger an important basic question: Is "explanation" a concept based on *general* causes (e.g., "Drinking hemlock causes death") or *singular* causes (e.g., "Socrates' drinking hemlock caused his death")? Causal effect expressions $P(y \mid do(x))$ belong to the first category whereas counterfactual expressions $P(Y_{x'} = y' \mid x, y)$ belong to the second, since conditioning on x and y narrows down world scenarios to those compatible with the most specific information at hand: $X = x$ and $Y = y$.

The classification of causal statements into general and singular categories has been the subject of intensive research in philosophy (see e.g. Good 1961; Kvart 1986; Cartwright 1989; Eells 1991; see also discussions in Sections 7.5.4 and 10.1.1). This research has attracted little attention in cognitive science and artificial intelligence, partly because it has not entailed practical inferential procedures and partly because it is based on problematic probabilistic semantics (see Section 7.5 for discussion of probabilistic causality). In the context of machine-generated explanations, this classification assumes both cognitive and computational significance. We discussed in Chapter 1 (Section 1.4) the sharp demarcation line between two types of causal queries, those that are answerable from the pair $\langle P(M), G(M) \rangle$ (the probability and diagram, respectively, associated with model M) and those that require additional information in the form of functional specification. Generic causal statements (e.g., $P(y \mid do(x))$) often fall into the first category (as in Chapter 3) whereas counterfactual expressions (e.g., $P(Y_{x'} = y \mid x, y)$) fall into the second, thus demanding more detailed specifications and higher computational resources.

The proper classification of explanation into a general or singular category depends on whether the cause c attains its explanatory power relative to its effect e by virtue of c's general *tendency* to produce e (as compared with the weaker tendencies of c's alternatives) or by virtue of c being *necessary* for triggering a specific chain of events leading to e in the specific situation at hand (as characterized by e and perhaps other facts and observations). Formally, the difference hinges on whether, in evaluating explanatory powers of various hypotheses, we should condition our beliefs on the events c and e that actually occurred.

Formal analysis of these alternatives is given in Chapters 9 and 10, where we discuss the necessary and sufficient aspects of causation as well as the notion of single-event causation. In the balance of this section we will be concerned with the interpretation and generation of explanatory utterances, taking the necessary aspect as a norm.

The following list, taken largely from Galles and Pearl (1997), provides examples of utterances used in explanatory discourse and their associated semantics within the modifiable structural model approach described in Section 7.1.1.

- "X is a cause of Y" if there exist two values x and x' of X and a value u of U such that $Y_x(u) \neq Y_{x'}(u)$.
- "X is a cause of Y in the context $Z = z$" if there exist two values x and x' of X and a value u of U such that $Y_{xz}(u) \neq Y_{x'z}(u)$.
- "X is a direct cause of Y" if there exist two values x and x' of X and a value u of U such that $Y_{xr}(u) \neq Y_{x'r}(u)$, where r is some realization of $V \setminus \{X, Y\}$.
- "X is an indirect cause of Y" if X is a cause of Y and X is not a direct cause of Y.

- "Event $X = x$ always causes $Y = y$" if:
  (i) $Y_x(u) = y$ for all u; and
  (ii) there exists a value u' of U such that $Y_{x'}(u') \neq y$ for some $x' \neq x$.
- "Event $X = x$ may have caused $Y = y$" if:
  (i) $X = x$ and $Y = y$ are true; and
  (ii) there exists a value u of U such that $X(u) = x$, $Y(u) = y$, and $Y_{x'}(u) \neq y$ for some $x' \neq x$.
- "The unobserved event $X = x$ is a likely cause of $Y = y$" if:
  (i) $Y = y$ is true; and
  (ii) $P(Y_x = y, Y_{x'} \neq y \mid Y = y)$ is high for all $x' \neq x$.
- "Event $Y = y$ occurred despite $X = x$" if:
  (i) $X = x$ and $Y = y$ are true; and
  (ii) $P(Y_x = y)$ is low.

The preceding list demonstrates the flexibility of modifiable structural models in formalizing nuances of causal expressions. Additional nuances (invoking such notions as enabling, preventing, sustaining, producing, etc.) will be analyzed in Chapters 9 and 10. Related expressions include: "Event A explains the occurrence of event B"; "A would explain B if C were the case"; "B occurred despite A because C was true." The ability to interpret and generate such explanatory sentences, or to select the expression most appropriate for the context, is one of the most intriguing challenges of research in man–machine conversation.

7.2.4 From Mechanisms to Actions to Causation

The structural model semantics described in Section 7.1.1 suggests solutions to two problems in cognitive science and artificial intelligence: the representation of actions and the role of causal ordering. We will discuss these problems in turn, since the second builds on the first.

Action, Mechanisms, and Surgeries

Whether we take the probabilistic paradigm that actions are transformations from probability distributions to probability distributions or the deterministic paradigm that actions are transformations from states to states, such transformations could in principle be infinitely complex. Yet in practice, people teach each other rather quickly the normal results of actions in the world, and people predict the consequences of most actions without much trouble. How?

Structural models answer this question by assuming that the actions we normally invoke in common reasoning can be represented as *local surgeries*. The world consists of a huge number of autonomous and invariant linkages or mechanisms, each corresponding to a physical process that constrains the behavior of a relatively small group of variables. If we understand how the linkages interact with each other (usually, they simply share variables), then we should also be able to understand what the effect of any given action would be: simply respecify those few mechanisms that are perturbed by the action; then let the mechanisms in the modified assembly interact with one another and see what state

will evolve at equilibrium. If the specification is complete (i.e., if M and U are given), then a single state will evolve. If the specification is probabilistic (i.e., if $P(u)$ is given), then a new probability distribution will emerge; if the specification is partial (i.e., if some f_i are not given), then a new, partial theory will be created. In all three cases we should be able to answer queries about postaction states of affair, albeit with decreasing level of precision.

The ingredient that makes this scheme operational is the *locality* of actions. Standing alone, locality is a vague concept because what is local in one space may not be local in another. A speck of dust, for example, appears extremely diffused in the frequency (or Fourier) representation; conversely, a pure musical tone requires a long stretch of time to be appreciated. Structural semantics emphasizes that actions are local in the space of mechanisms and not in the space of variables or sentences or time slots. For example, tipping the leftmost object in an array of domino tiles does not appear to be "local" in physical space, yet it is quite local in the mechanism domain: only one mechanism is perturbed, the gravitational restoring force that normally keeps that tile in a stable erect position; all other mechanisms remain unaltered, as specified, obedient to the usual equations of physics. Locality makes it easy to specify this action without enumerating all its ramifications. The listener, assuming she shares our understanding of domino physics, can figure out for herself the ramifications of this action, or any action of the type: "tip the ith domino tile to the right." By representing the domain in the form of an assembly of stable mechanisms, we have in fact created an oracle capable of answering queries about the effects of a huge set of actions and action combinations – without us having to explicate those effects.

Laws versus Facts

This surgical procedure sounds trivial when expressed in the context of structural equation models. However, it has encountered great difficulties when attempts were made to implement such schemes in classical logic. In order to implement surgical procedures in mechanism space, we need a language in which some sentences are given different status than others. Sentences describing mechanisms should be treated differently than those describing other facts of life (e.g., observations, assumptions, and conclusions), because the former are presumed to be stable whereas the latter are transitory. Indeed, the equations describing how the domino tiles interact with one another remain unaltered even though the states of the tiles themselves are free to vary with circumstances.

Admitting the need for this distinction has been a difficult transition in the logical approach to actions and causality, perhaps because much of the power of classical logic stems from its representational uniformity and syntactic invariance, where no sentence commands special status. Probabilists were much less reluctant to embrace the distinction between laws and facts, because this distinction has already been programmed into probability language by Reverend Bayes in 1763: Facts are expressed as ordinary propositions and hence can obtain probability values and can be conditioned on; laws, on the other hand, are expressed as conditional probability sentences (e.g., P(accident | careless driving) = high) and hence should not be assigned probabilities and cannot be conditioned on. It is because of this tradition that probabilists have always attributed nonpropositional character to conditional sentences (e.g., birds fly), refused to allow nested

conditionals (Levi 1988), and insisted on interpreting one's confidence in a conditional sentence as a conditional probability judgment (Adams 1975; see also Lewis 1976). Remarkably, these constraints, which some philosophers view as limitations, are precisely the safeguards that have kept probabilists from confusing laws and facts, protecting them from some of the traps that have ensnared logical approaches.[11]

Mechanisms and Causal Relationships

From our discussion thus far, it may seem that one can construct an effective representation for computing the ramification of actions without appealing to any notion of causation. This is indeed feasible in many areas of physics and engineering. For instance, if we have a large electric circuit consisting of resistors and voltage sources, and if we are interested in computing the effect of changing one resistor in the circuit, then the notion of causality hardly enters the computation. We simply insert the modified value of the resistor into Ohm's and Kirchhoff's equations and proceed to solve the set of (symmetric) equations for the variables needed. This computation can be performed effectively without committing to any directional causal relationship between the currents and voltages.

To understand the role of causality, we should note that (unlike our electrical circuit example) most mechanisms do not have names in common everyday language. We say: "raise taxes," or "make him laugh," or "press the button" – in general, $do(q)$, where q is a proposition, not a mechanism. It would be meaningless to say "increase this current" or "if this current were higher ..." in the electrical circuit example, because there are many ways of (minimally) increasing that current, each with different ramifications. Evidently, common-sense knowledge is not as entangled as a resistor network. In the STRIPS language (Fikes and Nilsson 1971), to give another example, an action is not characterized by the name of the mechanisms it modifies but rather by the action's immediate effects (the ADD and DELETE lists), and these effects are expressed as ordinary propositions. Indeed, if our knowledge is organized causally then this specification is sufficient, because each variable is governed by one and only one mechanism (see Definition 7.1.1). Thus, we should be able to figure out for ourselves which mechanism it is that must be perturbed in realizing the effect specified, and this should enable us to predict the rest of the scenario.

This linguistic abbreviation defines a new relation among events, a relation we normally call "causation": Event A causes B if the perturbation needed for realizing A entails the realization of B.[12] Causal abbreviations of this sort are used very effectively for specifying domain knowledge. Complex descriptions of what relationships are stable and how mechanisms interact with one another are rarely communicated explicitly in terms of mechanisms. Instead, they are communicated in terms of cause–effect relationships

[11] The distinction between laws and facts was proposed by Poole (1985) and Geffner (1992) as a fundamental principle for nonmonotonic reasoning. In database theory, laws are expressed by special sentences called *integrity constraints* (Reiter 1987). The distinction seems to be gaining broader support as a necessary requirement for formulating actions in artificial intelligence (Sandewall 1994; Lin 1995).

[12] The word "needed" connotes minimality and can be translated as: "... if every minimal perturbation realizing A entails B." The necessity and sufficiency aspects of this entailment relationship are formalized in Chapter 9 (Section 9.2).

between events or variables. We say, for example: "If tile i is tipped to the right, it causes tile $i + 1$ to tip to the right as well"; we do not communicate such knowledge in terms of the tendencies of each domino tile to maintain its physical shape, to respond to gravitational pull, and to obey Newtonian mechanics.

7.2.5 Simon's Causal Ordering

Our ability to talk directly in terms of one event causing another, (rather than an action altering a mechanism and the alteration, in turn, producing the effect) is computationally very useful, but at the same time it requires that the assembly of mechanisms in our domain satisfy certain conditions that accommodate causal directionality. Indeed, the formal definition of causal models given in Section 7.1.1 assumes that each equation is designated a distinct privileged variable, situated on its left-hand side, that is considered "dependent" or "output." In general, however, a mechanism may be specified as a functional constraint

$$G_k(x_1, \ldots, x_l; u_1, \ldots, u_m) = 0$$

without identifying any "dependent" variable.

Simon (1953) devised a procedure for deciding whether a collection of such symmetric G functions dictates a unique way of selecting an endogenous dependent variable for each mechanisms (excluding the background variables, since they are determined outside the system). Simon asked: When can we order the variables $(V_1, V_2, \ldots, V_n)$ in such a way that we can solve for each V_i without solving for any of V_i's successors? Such an ordering, if it exists, dictates the direction we attribute to causation. This criterion might at first sound artificial, since the order of solving equations is a matter of computational convenience whereas causal directionality is an objective attribute of physical reality. (For discussion of this issue see De Kleer and Brown 1986; Iwasaki and Simon 1986; Druzdzel and Simon 1993.) To justify the criterion, let us rephrase Simon's question in terms of actions and mechanisms. Assume that each mechanism (i.e. equation) can be modified independently of the others, and let A_k be the set of actions capable of modifying equation G_k (while leaving other equations unaltered). Imagine that we have chosen an action a_k from A_k and that we have modified G_k in such a way that the set of solutions $(V_1(u), V_2(u), \ldots, V_n(u))$ to the entire system of equations differs from what it was prior to the action. If X is the set of endogenous variables constrained by G_k, then we can ask which members of X would change by the modification. If only one member of X changes, say X_k, and if the identity of that distinct member remains the same for all choices of a_k and u, then we designate X_k as the dependent variable in G_k.

Formally, this property means that changes in a_k induce a *functional mapping* from the domain of X_k to the domain of $\{V \setminus X_k\}$; all changes in the system (generated by a_k) can be attributed to changes in X_k. It would make sense, in such a case, to designate X_k as a "representative" of the mechanism G_k, and we would be justified in replacing the sentence "action a_k caused event $Y = y$" with "event $X_k = x_k$ caused $Y = y$" (where Y is any variable in the system). The invariance of X_k to the choice of a_k is the basis for treating an action as a modality $do(X_k = x_k)$ (Definition 7.1.3). It provides a license for characterizing an action by its immediate consequence(s), independent of the instrument

that actually brought about those consequences, and it defines in fact the notion of "local action" or "local surgery."

It can be shown (Nayak 1994) that the uniqueness of X_k can be determined by a simple criterion that involves purely topological properties of the equation set (i.e., how variables are grouped into equations). The criterion is that one should be able to form a one-to-one correspondence between equations and variables and that the correspondence be unique. This can be decided by solving the "matching problem" (Serrano and Gossard 1987) between equations and variables. If the matching is unique, then the choice of dependent variable in each equation is unique and the directionality induced by that choice defines a directed acyclic graph (DAG). In Figure 7.1, for example, the directionality of the arrows need not be specified externally; they can be determined mechanically from the set of symmetrical constraints (i.e., logical propositions)

$$S = \{G_1(C, U), G_2(A, C), G_3(B, C), G_4(A, B, D)\} \tag{7.18}$$

that characterizes the problem. The reader can easily verify that the selection of a privileged variable from each equation is unique and hence that the causal directionality of the arrows shown in Figure 7.1 is inevitable.

Thus, we see that causal directionality, according to Simon, emerges from two assumptions: (1) the partition of variables into background (U) and endogenous (V) sets; and (2) the overall configuration of mechanisms in the model. Accordingly, a variable designated as "dependent" in a given mechanism may well be labeled "independent" when that same mechanism is embedded in a different model. Indeed, the engine causes the wheels to turn when the train goes uphill but changes role in going downhill.

Of course, if we have no way of determining the background variables, then several causal orderings may ensue. In (7.18), for example, if we were not given the information that U is a background variable, then either one of $\{U, A, B, C\}$ could be chosen as background, and each such choice would induce a different ordering on the remaining variables. (Some would conflict with common-sense knowledge, e.g., that the captain's signal influences the court's decision.) However, the directionality of $A \rightarrow D \leftarrow B$ would be maintained in all those orderings. The question of whether there exists a partition $\{U, V\}$ of the variables that would yield a causal ordering in a system of symmetric constraints can also be solved (in polynomial time) by topological means (Dechter and Pearl 1991).

Simon's ordering criterion fails when we are unable to solve the equations one at a time and so must solve a block of k equations simultaneously. In such a case, all the k variables determined by the block would be mutually unordered, though their relationships with other blocks may still be ordered. This occurs, for example, in the economic model of Figure 7.4, where (7.9) and (7.10) need to be solved simultaneously for P and Q and hence the correspondence between equations and variables is not unique; either Q or P could be designated as "independent" in either of the two equations. Indeed, the information needed for classifying (7.9) as the "demand" equation (and, respectively, (7.10) as the "price" equation) comes not from the way variables are assigned to equations but rather from subject-matter considerations. Our understanding that household income directly affects household demand (and not prices) plays a major role in this classification.

In cases where we tend to assert categorically that the flow of causation in a feedback loop goes clockwise, this assertion is normally based on the relative magnitudes of forces. For example, turning the faucet would lower the water level in the water tank, but there is practically nothing we can do to the water tank that would turn the faucet. When such information is available, causal directionality is determined by appealing, again, to the notion of hypothetical intervention and asking whether an external control over one variable in the mechanism necessarily affects the others. This consideration then constitutes the operational semantics for identifying the dependent variables V_i in nonrecursive causal models (Definition 7.1.1).

The asymmetry that characterizes causal relationships in no way conflicts with the symmetry of physical equations. By saying that "X causes Y and Y does not cause X," we mean to say that changing a mechanism in which X is normally the dependent variable has a different effect on the world than changing a mechanism in which Y is normally the dependent variable. Because two separate mechanisms are involved, the statement stands in perfect harmony with the symmetry we find in the equations of physics.

Simon's theory of causal ordering has profound repercussions on Hume's problem of causal induction, that is, how causal knowledge is acquired from experience (see Chapter 2). The ability to deduce causal directionality from an assembly of symmetrical mechanisms (together with a selection of a set of endogenous variables) means that the acquisition of causal relationships is no different than the acquisition (e.g., by experiments) of ordinary physical laws, such as Hooke's law of suspended springs or Newton's law of acceleration. This does not imply that acquiring physical laws is a trivial task, free of methodological and philosophical subtleties. It does imply that the problem of causal induction – one of the toughest in the history of philosophy – can be reduced to the more familiar problem of scientific induction.

7.3 AXIOMATIC CHARACTERIZATION

Axioms play important roles in the characterization of formal systems. They provide a parsimonious description of the essential properties of the system, thus allowing comparisons among alternative formulations and easy tests of equivalence or subsumption among such alternatives. Additionally, axioms can often be used as rules of inference for deriving (or verifying) new relationships from a given set of premises. In the next subsection, we will establish a set of axioms that characterize the relationships among counterfactual sentences of the form $Y_x(u) = y$ in both recursive and nonrecursive systems. Using these axioms, we will then demonstrate (in Section 7.3.2) how the identification of causal effects can be verified by symbolic means, paralleling the derivations of Chapter 3 (Section 3.4). Finally, Section 7.3.3 establishes axioms for the notion of *causal relevance,* contrasting those that capture informational relevance.

7.3.1 The Axioms of Structural Counterfactuals

We present three properties of counterfactuals – composition, effectiveness, and reversibility – that hold in all causal models.

Property 1 (Composition)
For any three sets of endogenous variables X, Y, and W in a causal model, we have

$$W_x(u) = w \implies Y_{xw}(u) = Y_x(u). \tag{7.19}$$

Composition states that, if we force a variable (W) to a value w that it would have had without our intervention, then the intervention will have no effect on other variables in the system. That invariance holds in all fixed conditions $do(X = x)$.

Since composition allows for the removal of a subscript (i.e., reducing $Y_{xw}(u)$ to $Y_x(u)$), we need an interpretation for a variable with an empty set of subscripts, which (naturally) we identify with the variable under no interventions.

Definition 7.3.1 (Null Action)

$$Y_\emptyset(u) \triangleq Y(u).$$

Corollary 7.3.2 (Consistency)
For any set of variables Y and X in a causal model, we have

$$X(u) = x \implies Y(u) = Y_x(u). \tag{7.20}$$

Proof
Substituting X for W and $\emptyset$ for X in (7.19), we obtain $X_\emptyset(u) = x \implies Y_\emptyset(u) = Y_x(u)$. Null action (Definition 7.3.1) allows us to drop the $\emptyset$, leaving $X(u) = x \implies Y(u) = Y_x(u)$. □

The implication in (7.20) was called "consistency" by Robins (1987).[13]

Property 2 (Effectiveness)
For all sets of variables X and W, $X_{xw}(u) = x$.

Effectiveness specifies the effect of an intervention on the manipulated variable itself – namely, that if we force a variable X to have the value x, then X will indeed take on the value x.

Property 3 (Reversibility)
For any two variables Y and W and any set of variables X,

$$(Y_{xw}(u) = y) \ \& \ (W_{xy}(u) = w) \implies Y_x(u) = y. \tag{7.21}$$

Reversibility precludes multiple solutions due to feedback loops. If setting W to a value w results in a value y for Y, and if setting Y to the value y results in W achieving the

[13] Consistency and composition are used routinely in economics (Manski 1990; Heckman 1996) and statistics (Rosenbaum 1995) within the potential-outcome framework (Section 3.6.3). Consistency was stated formally by Gibbard and Harper (1976, p. 156) and Robins (1987) (see equation (3.52)). Composition is stated in Holland (1986, p. 968) and was brought to my attention by J. Robins.

value w, then W and Y will naturally obtain the values w and y (respectively), without any external setting. In recursive systems, reversibility follows directly from composition. This can easily be seen by noting that, in a recursive system, either $Y_{xw}(u) = Y_x(u)$ or $W_{xy}(u) = W_x(u)$. Thus, reversibility reduces to $(Y_{xw}(u) = y)$ & $(W_x(u) = w) \Longrightarrow Y_x(u) = y$ (another form of composition) or to $(Y_x(u) = y)$ & $(W_{xy}(u) = w) \Longrightarrow Y_x(u) = y$ (which is trivially true).

Reversibility reflects "memoryless" behavior: the state of the system, V, tracks the state of U regardless of U's history. A typical example of irreversibility is a system of two agents who adhere to a "tit-for-tat" strategy (e.g., the prisoners' dilemma). Such a system has two stable solutions – cooperation and defection – under the same external conditions U, and thus it does not satisfy the reversibility condition; forcing either one of the agents to cooperate results in the other agent's cooperation $(Y_w(u) = y, W_y(u) = w)$, yet this does not guarantee cooperation from the start $(Y(u) = y, W(u) = w)$. In such systems, irreversibility is a product of using a state description that is too coarse, one where not all of the factors that determine the ultimate state of the system are included in U. In a tit-for-tat system, a complete state description should include factors such as the previous actions of the players, and reversibility is restored once the missing factors are included.

In general, the properties of composition, effectiveness, and reversibility are independent – none is a consequence of the other two. This can be shown (Galles and Pearl 1997) by constructing specific models in which two of the properties hold and the third does not. In recursive systems, composition and effectiveness are independent while reversibility holds trivially, as just shown.

The next theorem asserts the *soundness*[14] of properties 1–3, that is, their validity.

Theorem 7.3.3 (Soundness)
Composition, effectiveness, and reversibility are sound *in structural model semantics; that is, they hold in all causal models.*

A proof of Theorem 7.3.3 is given in Galles and Pearl (1997).

Our next theorem establishes the *completeness* of the three properties when treated as axioms or rules of inference. Completeness amounts to sufficiency; *all* other properties of counterfactual statements follow from these three. Another interpretation of completeness is as follows: Given any set S of counterfactual statements that is consistent with properties 1–3, there exists a causal model M in which S holds true.

A formal proof of completeness requires the explication of two technical properties – existence and uniqueness – that are implicit in the definition of causal models (Definition 7.1.1).

Property 4 (Existence)
For any variable X and set of variables Y,

$$\exists x \in X \text{ s.t. } X_y(u) = x. \tag{7.22}$$

[14] The terms *soundness* and *completeness* are sometimes referred to as *necessity* and *sufficiency*, respectively.

Property 5 (Uniqueness)
For every variable X and set of variables Y,

$$X_y(u) = x \ \& \ X_y(u) = x' \implies x = x'. \tag{7.23}$$

Definition 7.3.4 (Recursiveness)
A model M is recursive *if, for any two variables Y and W and for any set of variables X, we have*

$$Y_{xw}(u) = Y_x(u) \quad \text{or} \quad W_{xy}(u) = W_x(u). \tag{7.24}$$

In words, recursiveness means that either Y does not affect W or W does not affect Y. Clearly, any model M for which the causal diagram $G(M)$ is acyclic must be recursive.

Theorem 7.3.5 (Recursive Completeness)
Composition, effectiveness, and recursiveness are complete (Galles and Pearl 1998; Halpern 1998).[15]

Theorem 7.3.6 (Completeness)
Composition, effectiveness, and reversibility are complete for all causal models (Halpern 1998).

The practical importance of soundness and completeness surfaces when we attempt to test whether a certain set of conditions is sufficient for the identifiability of some counterfactual quantity Q. Soundness, in this context, guarantees that if we symbolically manipulate Q using the three axioms and manage to reduce it to an expression that involves ordinary probabilities (free of counterfactual terms), then Q is identifiable (in the sense of Definition 3.2.3). Completeness guarantees the converse: if we do not succeed in reducing Q to a probabilistic expression, then Q is nonidentifiable – our three axioms are as powerful as can be.

The next section demonstrates a proof of identifiability that uses effectiveness and decomposition as axioms.

7.3.2 Causal Effects from Counterfactual Logic: An Example

We revisit the smoking–cancer example analyzed in Section 3.4.3. The model associated with this example is assumed to have the following structure (see Figure 7.5):

$$V = \{X \text{ (smoking)}, Y \text{ (lung cancer)}, Z \text{ (tar in lungs)}\},$$
$$U = \{U_1, U_2\},\ U_1 \perp\!\!\!\perp U_2,$$

[15] Galles and Pearl (1997) proved recursive completeness assuming that, for any two variables, one knows which of the two (if any) is an ancestor of the other. Halpern (1998) proved recursive completeness without this assumption, provided only that (7.24) is known to hold for any two variables in the model. Halpern further provided a set of axioms for cases where the solution of $Y_x(u)$ is not unique or does not exist.

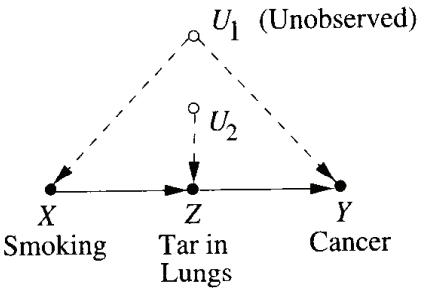

Figure 7.5 Causal diagram illustrating the effect of smoking on lung cancer.

$$x = f_1(u_1),$$
$$z = f_2(x, u_2),$$
$$y = f_3(z, u_1).$$

This model embodies several assumptions, all of which are represented in the diagram of Figure 7.5. The missing link between X and Y represents the assumption that the effect of smoking cigarettes (X) on the production of lung cancer (Y) is entirely mediated through tar deposits in the lungs. The missing connection between U_1 and U_2 represents the assumption that even if a genotype (U_1) is aggravating the production of lung cancer, it nevertheless has no effect on the amount of tar in the lungs except indirectly (through cigarette smoking). We wish to use the assumptions embodied in the model to derive an estimable expression for the causal effect $P(Y = y \mid do(x)) \triangleq P(Y_x = y)$ that is based on the joint distribution $P(x, y, z)$.

This problem was solved in Section 3.4.3 by a graphical method, using the axioms of *do* calculus (Theorem 3.4.1). Here we show how the counterfactual expression $P(Y_x = y)$ can be reduced to ordinary probabilistic expression (involving no counterfactuals) by purely symbolic operations, using only probability calculus and two rules of inference: effectiveness and composition. Toward this end, we first need to translate the assumptions embodied in the graphical model into the language of counterfactuals. In Section 3.6.3 it was shown that the translation can be accomplished systematically, using two simple rules (Pearl 1995a, p. 704).

Rule 1 (*exclusion restrictions*): For every variable Y having parents PA_Y and for every set of variables $Z \subset V$ disjoint of PA_Y, we have

$$Y_{pa_Y}(u) = Y_{pa_Y z}(u). \tag{7.25}$$

Rule 2 (*independence restrictions*): If $Z_1, \ldots, Z_k$ is any set of nodes in V not connected to Y via paths containing only U variables, we have

$$Y_{pa_Y} \perp\!\!\!\perp \{Z_{1pa_{Z_1}}, \ldots, Z_{kpa_{Z_k}}\}. \tag{7.26}$$

Equivalently, (7.26) holds if the corresponding U terms $(U_{Z_1}, \ldots, U_{Z_k})$ are jointly independent of U_Y.

Rule 1 reflects the insensitivity of Y to any manipulation in V, once its direct causes PA_Y are held constant; it follows from the identity $v_i = f_i(pa_i, u_i)$ in Definition 7.1.1. Rule 2 interprets independencies among U variables as independencies between the counterfactuals of the corresponding V variables, with their parents held fixed. Indeed, the statistics

of Y_{pa_Y} is governed by the equation $Y = f_Y(pa_Y, u_Y)$; therefore, once we hold PA_Y fixed, the residual variations of Y are governed solely by the variations in U_Y.

Applying these two rules to our example, we see that the causal diagram in Figure 7.5 encodes the following assumptions:

$$Z_x(u) = Z_{yx}(u), \tag{7.27}$$

$$X_y(u) = X_{zy}(u) = X_z(u) = X(u), \tag{7.28}$$

$$Y_z(u) = Y_{zx}(u), \tag{7.29}$$

$$Z_x \perp\!\!\!\perp \{Y_z, X\}. \tag{7.30}$$

Equations (7.27)–(7.29) follow from the exclusion restrictions of (7.25), using

$$PA_X = \emptyset, \quad PA_Y = \{Z\}, \quad \text{and} \quad PA_Z = \{X\}.$$

Equation (7.27), for instance, represents the absence of a causal link from Y to Z, while (7.28) represents the absence of a causal link from Z or Y to X. In contrast, (7.30) follows from the independence restriction of (7.26), since the lack of a connection between (i.e., the independence of) U_1 and U_2 rules out any path between Z and $\{X, Y\}$ that contains only U variables.

We now use these assumptions (which embody recursiveness), together with the properties of composition and effectiveness, to compute the tasks analyzed in Section 3.4.3.

Task 1

Compute $P(Z_x = z)$ (i.e., the causal effect of smoking on tar).

$$\begin{aligned} P(Z_x = z) &= P(Z_x = z \mid x) && \text{from (7.30)} \\ &= P(Z = z \mid x) && \text{by composition} \\ &= P(z \mid x). \end{aligned} \tag{7.31}$$

Task 2

Compute $P(Y_z = y)$ (i.e., the causal effect of tar on cancer).

$$P(Y_z = y) = \sum_x P(Y_z = y \mid x)P(x). \tag{7.32}$$

Since (7.30) implies $Y_z \perp\!\!\!\perp Z_x \mid X$, we can write

$$\begin{aligned} P(Y_z = y \mid x) &= P(Y_z = y \mid x, Z_x = z) && \text{from (7.30)} \\ &= P(Y_z = y \mid x, z) && \text{by composition} \\ &= P(y \mid x, z). && \text{by composition} \end{aligned} \tag{7.33}$$

Substituting (7.33) into (7.32) yields

$$P(Y_z = y) = \sum_x P(y \mid x, z)P(x). \tag{7.34}$$

Task 3

Compute $P(Y_x = y)$ (i.e., the causal effect of smoking on cancer).

For any variable Z, by composition we have

$$Y_x(u) = Y_{xz}(u) \quad \text{if } Z_x(u) = z.$$

Since $Y_{xz}(u) = Y_z(u)$ (from (7.29)),

$$Y_x(u) = Y_{xz_x}(u) = Y_z(u), \quad \text{where } z_x = Z_x(u). \tag{7.35}$$

Thus,

$$\begin{aligned} P(Y_x = y) &= P(Y_{z_x} = y) && \text{from (7.35)} \\ &= \sum_z P(Y_{z_x} = y \mid Z_x = z) P(Z_x = z) \\ &= \sum_z P(Y_z = y \mid Z_x = z) P(Z_x = z) && \text{by composition} \\ &= \sum_z P(Y_z = y) P(Z_x = z) && \text{from (7.30)} \end{aligned} \tag{7.36}$$

The probabilities $P(Y_z = y)$ and $P(Z_x = z)$ were computed in (7.34) and (7.31), respectively. Substituting gives us

$$P(Y_x = y) = \sum_z P(z \mid x) \sum_{x'} P(y \mid z, x') P(x'). \tag{7.37}$$

The right-hand side of (7.37) can be computed from $P(x, y, z)$ and coincides with the front-door formula derived in Section 3.4.3 (equation (3.42)).

Thus, $P(Y_x = y)$ can be reduced to expressions involving probabilities of observed variables and is therefore identifiable. More generally, our completeness result (Theorem 7.3.5) implies that *any* identifiable counterfactual quantity can be reduced to the correct expression by repeated application of composition and effectiveness (assuming recursiveness).

7.3.3 Axioms of Causal Relevance

In Section 1.2 we presented a set of axioms for a class of relations called *graphoids* (Pearl and Paz 1987; Geiger et al. 1990) that characterize informational relevance.[16] We now develop a parallel set of axioms for *causal relevance,* that is, the tendency of certain events to affect the occurrence of other events in the physical world, independent of the observer–reasoner. Informational relevance is concerned with questions of the form: "Given that we know Z, would gaining information about X gives us new information

[16] "Relevance" will be used primarily as a generic name for the relationship of being relevant or irrelevant. It will be clear from the context when "relevance" is intended to negate "irrelevance."

about Y?" Causal relevance is concerned with questions of the form: "Given that Z is fixed, would changing X alter Y?" We show that causal relevance complies with all the axioms of path interception in directed graphs except transitivity.

The notion of causal relevance has its roots in the philosophical works of Suppes (1970) and Salmon (1984), who attempted to give probabilistic interpretations to cause–effect relationships and recognized the need to distinguish causal from statistical relevance (see Section 7.5). Although these attempts did not produce a probabilistic definition of causal relevance, they led to methods for testing the consistency of relevance statements against a given probability distribution and a given temporal ordering among the variables (see Section 7.5.2). Here we aim at axiomatizing relevance statements in themselves – with no reference to underlying probabilities or temporal orderings.

The axiomization of causal relevance may be useful to experimental researchers in domains where exact causal models do not exist. If we know, through experimentation, that some variables have no causal influence on others in a system, then we may wish to determine whether other variables will exert causal influence (perhaps under different experimental conditions) or may ask what additional experiments could provide such information. For example, suppose we find that a rat's diet has no effect on tumor growth while the amount of exercise is kept constant and, conversely, that exercise has no effect on tumor growth while diet is kept constant. We would like to be able to infer that controlling only diet (while paying no attention to exercise) would still have no influence on tumor growth. A more subtle inference problem is deciding whether changing the ambient temperature in the cage would have an effect on the rat's physical activity, given that we have established that temperature has no effect on activity when diet is kept constant and that temperature has no effect on (the rat's choice of) diet when activity is kept constant.

Galles and Pearl (1997) analyzed both probabilistic and deterministic interpretations of causal irrelevance. The probabilistic interpretation, which equates causal irrelevance with inability to change the probability of the effect variable, has intuitive appeal but is inferentially very weak; it does not support a very expressive set of axioms unless further assumptions are made about the underlying causal model. If we add the stability assumption (i.e., that no irrelevance can be destroyed by changing the nature of the individual processes in the system), then we obtain the same set of axioms for probabilistic causal irrelevance as the set governing path interception in directed graphs.

In this section we analyze a deterministic interpretation that equates causal irrelevance with inability to change the effect variable in any state u of the world. This interpretation is governed by a rich set of axioms without our making any assumptions about the causal model: many of the path interception properties in directed graphs hold for deterministic causal irrelevance.

Definition 7.3.7 (Causal Irrelevance)
A variable X is causally irrelevant *to Y, given Z (written $X \not\rightarrow Y \mid Z$) if, for every set W disjoint of $X \cup Y \cup Z$, we have*

$$\forall(u, z, x, x', w), \quad Y_{xzw}(u) = Y_{x'zw}(u), \tag{7.38}$$

where x and x' are two distinct values of X.

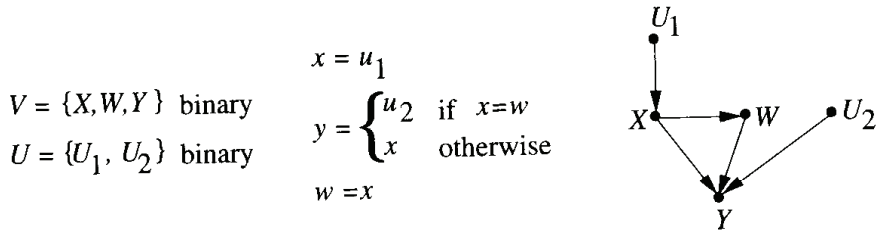

Figure 7.6 Example of a causal model that requires the examination of submodels to determine causal relevance.

This definition captures the intuition "If X is causally irrelevant to Y, then X cannot affect Y under any circumstance u or under any modification of the model that includes $do(Z = z)$."

To see why we require the equality $Y_{xzw}(u) = Y_{x'zw}(u)$ to hold in every context $W = w$, consider the causal model of Figure 7.6. In this example, $Z = \emptyset$, W follows X, and hence Y follows X; that is, $Y_{X=0}(u) = Y_{X=1}(u) = u_2$. However, since $y(x, w, u_2)$ is a nontrivial function of x, X is perceived to be causally relevant to Y. Only holding W constant would reveal the causal influence of X on Y. To capture this intuition, we must consider all contexts $W = w$ in Definition 7.3.7.

With this definition of causal irrelevance, we have the following theorem.

Theorem 7.3.8
For any causal model, the following sentences must hold.

Weak Right Decomposition:[17]

$$(X \not\to YW \mid Z) \;\&\; (X \to Y \mid ZW) \Longrightarrow (X \not\to Y \mid Z).$$

Left Decomposition:

$$(XW \not\to Y \mid Z) \Longrightarrow (X \not\to Y \mid Z) \;\&\; (W \not\to Y \mid Z).$$

Strong Union:

$$(X \not\to Y \mid Z) \Longrightarrow (X \not\to Y \mid ZW)\ \forall W.$$

Right Intersection:

$$(X \not\to Y \mid ZW) \;\&\; (X \not\to W \mid ZY) \Longrightarrow (X \not\to YW \mid Z).$$

Left Intersection:

$$(X \not\to Y \mid ZW) \;\&\; (W \not\to Y \mid ZX) \Longrightarrow (XW \not\to Y \mid Z).$$

This set of axioms bears a striking resemblance to the properties of path interception in a directed graph. Paz and Pearl (1994) showed that the axioms of Theorem 7.3.8, together with transitivity and right decomposition, constitute a complete characterization of th

[17] Galles and Pearl (1997) used a stronger version of right decomposition: $(X \not\to YW \mid Z) \Longrightarrow (X \not\to Y \mid Z)$. But Bonet (1999) showed that it must be weakened to render the axiom system sound.

relation $(X \not\to Y \mid Z)_G$ when interpreted to mean that every directed path from X to Y in a directed graph G contains at least one node in Z (see also Paz et al. 1996).

Galles and Pearl (1997) showed that, despite the absence of transitivity, Theorem 7.3.8 permits one to infer certain properties of causal irrelevance from properties of directed graphs. For example, suppose we wish to validate a generic statement such as: "If X has an effect on Y, but ceases to have an effect when we fix Z, then Z must have an effect on Y." That statement can be proven from the fact that, in any directed graph, if all paths from X to Y are intercepted by Z and there are no paths from Z to Y, then there is no path from X to Y.

Remark on the Transitivity of Causal Dependence

That causal dependence is not transitive is clear from Figure 7.6. In any state of (U_1, U_2), X is capable of changing the state of W and W is capable of changing Y, yet X is incapable of changing Y. Galles and Pearl (1997) gave examples where causal relevance in the weak sense of Definition 7.3.7 is also nontransitive, even for binary variables. The question naturally arises as to why transitivity is so often conceived of as an inherent property of causal dependence or, more formally, what assumptions we tacitly make when we classify causal dependence as transitive.

One plausible answer is that we normally interpret transitivity to mean the following: "If (1) X causes Y and (2) Y causes Z regardless of X, then (3) X causes Z." The suggestion is that questions about transitivity bring to mind chainlike processes, where X influences Y and Y influences Z but where X does not have a *direct* influence over Z. With this qualification, transitivity for binary variables can be proven immediately from composition (equation (7.19)) as follows.

Let the sentence "$X = x$ causes $Y = y$," denoted $x \to y$, be interpreted as the joint condition $\{X(u) = x, Y(u) = y, Y_{x'}(u) = y' \neq y\}$ (in words, x and y hold, but changing x to x' would change y to y'). We can now prove that if X has no direct effect on Z, that is, if

$$Z_{y'x'} = Z_{y'}, \tag{7.39}$$

then

$$x \to y \;\&\; y \to z \implies x \to z. \tag{7.40}$$

Proof

The l.h.s. of (7.40) reads

$$X(u) = x, \quad Y(u) = y, \quad Z(u) = z, \quad Y_{x'}(u) = y', \quad Z_{y'}(u) = z'.$$

From (7.39) we can rewrite the last term as $Z_{y'x'}(u) = z'$. Composition further permits us to write

$$Y_{x'}(u) = y' \;\&\; Z_{y'x'}(u) = z' \implies Z_{x'}(u) = z',$$

which, together with $X(u) = x$ and $Z(u) = z$, implies $x \to z$. □

Weaker forms of causal transitivity are discussed in Chapter 9 (Lemmas 9.2.7 and 9.2.8).

7.4 STRUCTURAL AND SIMILARITY-BASED COUNTERFACTUALS

7.4.1 Relations to Lewis's Counterfactuals

Causality from Counterfactuals

In one of his most quoted sentences, David Hume tied together two aspects of causation, regularity of succession and counterfactual dependency:

> we may define a cause to be an object followed by another, and where all the objects, similar to the first, are followed by object similar to the second, Or, in other words, where, if the first object had not been, the second never had existed. (Hume 1748/1959, sec. VII).

This two-faceted definition is puzzling on several accounts. First, regularity of succession, or "correlation" in modern terminology, is not sufficient for causation, as even nonstatisticians know by now. Second, the expression "in other words" is a too strong, considering that regularity rests on observations whereas counterfactuals rest on mental exercise. Third, Hume had introduced the regularity criterion nine years earlier,[18] and one wonders what jolted him into supplementing it with a counterfactual companion. Evidently, Hume was not completely happy with the regularity account, and must have felt that the counterfactual criterion is less problematic and more illuminating. But how can convoluted expressions of the type "if the first object had not been, the second never had existed" illuminate simple commonplace expressions like "A caused B"?

The idea of basing causality on counterfactuals is further echoed by John Stuart Mill (1843), and it reached fruition in the works of David Lewis (1973b, 1986). Lewis called for abandoning the regularity account altogether and for interpreting "A has caused B" as "B would not have occurred if it were not for A." Lewis (1986, p. 161) asked: "Why not take counterfactuals at face value: as statements about possible alternatives to the actual situation ...?"

Implicit in this proposal lies a claim that counterfactual expressions are less ambiguous to our mind than causal expressions. Why else would the expression "B would be false if it were not for A" be considered an *explication* of "A caused B," and not the other way around, unless we could discern the truth of the former with greater certitude than that of the latter? Taken literally, discerning the truth of counterfactuals requires generating and examining possible alternatives to the actual situation as well as testing whether certain propositions hold in those alternatives – a mental task of nonnegligible proportions. Nonetheless, Hume, Mill, and Lewis apparently believed that going through this mental exercise is simpler than intuiting directly on whether it was A that caused B. How can this be done? What mental representation allows humans to process counterfactuals so swiftly and reliably, and what logic governs that process so as to maintain uniform standards of coherence and plausibility?

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[18] In *Treatise of Human Nature,* Hume wrote: "We remember to have had frequent instances of the existence of one species of objects; and also remember, that the individuals of another species of objects have always attended them, and have existed in a regular order of contiguity and succession with regard to them" (Hume 1739, p. 156).

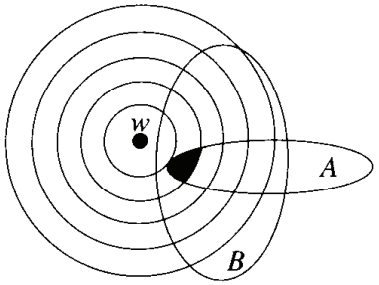

Figure 7.7 Graphical representation of Lewis's closest-world semantics. Each circular region corresponds to a set of worlds that are equally similar to w. The shaded region represents the set of closest A-worlds; since all these worlds satisfy B, the counterfactual sentence $A \mathrel{\square\!\!\rightarrow} B$ is declared true in w.

Structure versus Similarity

According to Lewis's account (1973b), the evaluation of counterfactuals involves the notion of *similarity*: one orders possible worlds by some measure of similarity, and the counterfactual $A \mathrel{\square\!\!\rightarrow} B$ (read: "B if it were A") is declared true in a world w just in case B is true in all the closest A-worlds to w (see Figure 7.7).[19]

This semantics still leaves questions of representation unsettled. What choice of similarity measure would make counterfactual reasoning compatible with ordinary conceptions of cause and effect? What mental representation of worlds ordering would render the computation of counterfactuals manageable and practical (in both man and machine)?

In his initial proposal, Lewis was careful to keep the formalism as general as possible; save for the requirement that every world be closest to itself, he did not impose any structure on the similarity measure. However, simple observations tell us that similarity measures cannot be arbitrary. The very fact that people communicate with counterfactuals already suggests that they share a similarity measure, that this measure is encoded parsimoniously in the mind, and hence that it must be highly structured. Kit Fine (1975) further demonstrated that similarity of appearance is inadequate. Fine considers the counterfactual "Had Nixon pressed the button, a nuclear war would have started," which is generally accepted as true. Clearly, a world in which the button happened to be disconnected is many times more similar to our world, as we know it, than the one yielding a nuclear blast. Thus we see not only that similarity measures could not be arbitrary but also that they must respect our conception of causal laws.[20] Lewis (1979) subsequently set up an intricate system of weights and priorities among various aspects of similarity – size of "miracles" (violations of laws), matching of facts, temporal precedence, and so forth – in attempting to bring similarity closer to causal intuition. But these priorities are rather post hoc and still yield counterintuitive inferences (J. Woodward, personal communication).

Such difficulties do not enter the structural account. In contrast with Lewis's theory, counterfactuals are not based on an abstract notion of similarity among hypothetical worlds; instead, they rest directly on the mechanisms (or "laws," to be fancy) that produce those worlds and on the invariant properties of those mechanisms. Lewis's elusive "miracles" are replaced by principled minisurgeries, $do(X = x)$, which represent the minimal change (to a model) necessary for establishing the antecedent $X = x$ (for all u).

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[19] Related possible-world semantics were introduced in artificial intelligence to represent actions and database updates (Ginsberg 1986; Ginsberg and Smith 1987; Winslett 1988; Katsuno and Mendelzon 1991).

[20] In this respect, Lewis's reduction of causes to counterfactuals is somewhat circular.

Thus, similarities and priorities – if they are ever needed – may be read into the $do(\cdot)$ operator as an afterthought (see discussion following (3.11) and Goldszmidt and Pearl 1992), but they are not basic to the analysis.

The structural account answers the mental representation question by offering a parsimonious encoding of knowledge from which causes, counterfactuals, and probabilities of counterfactuals can be derived by effective algorithms. However, this parsimony is acquired at the expense of generality; limiting the counterfactual antecedent to conjunction of elementary propositions prevents us from analyzing disjunctive hypotheticals such as "if Bizet and Verdi were compatriots."

7.4.2 Axiomatic Comparison

If our assessment of interworld distances comes from causal knowledge, the question arises of whether that knowledge does not impose its own structure on distances, a structure that is not captured in Lewis's logic. Phrased differently: By agreeing to measure closeness of worlds on the basis of causal relations, do we restrict the set of counterfactual statements we regard as valid? The question is not merely theoretical. For example, Gibbard and Harper (1976) characterized decision-making conditionals (i.e., sentences of the form "If we do A, then B") using Lewis's general framework, whereas our $do(\cdot)$ operator is based directly on causal mechanisms; whether the two formalisms are identical is uncertain.[21]

We now show that the two formalisms are identical for recursive systems; in other words, composition and effectiveness hold with respect to Lewis's closest-world framework whenever recursiveness does. We begin by providing a version of Lewis's logic for counterfactual sentences (from Lewis 1973c).

Rules

(1) If A and $A \Longrightarrow B$ are theorems, then so is B.

(2) If $(B_1 \ \& \ \cdots) \Longrightarrow C$ is a theorem, then so is $((A \boxrightarrow B_1)\cdots) \Longrightarrow (A \boxrightarrow C)$.

Axioms

(1) All truth-functional tautologies.

(2) $A \boxrightarrow A$.

(3) $(A \boxrightarrow B) \ \& \ (B \boxrightarrow A) \Longrightarrow (A \boxrightarrow C) \equiv (B \boxrightarrow C)$.

(4) $((A \vee B) \boxrightarrow A) \vee ((A \vee B) \boxrightarrow B) \vee$
$(((A \vee B) \boxrightarrow C) \equiv (A \boxrightarrow C) \ \& \ (B \boxrightarrow C))$.

(5) $A \boxrightarrow B \Longrightarrow A \Longrightarrow B$.

(6) $A \ \& \ B \Longrightarrow A \boxrightarrow B$.

[21] Ginsberg and Smith (1987) and Winslett (1988) have also advanced theories of actions based on closest-world semantics; they have not imposed any special structure for the distance measure to reflect causal considerations.

The statement $A \square\!\!\rightarrow B$ stands for "In all closest worlds where A holds, B holds as well." To relate Lewis's axioms to those of causal models, we must translate his syntax. We will equate Lewis's world with an instantiation of all the variables, including those in U, in a causal model. Values assigned to subsets of variables in a causal model will stand for Lewis's propositions (e.g., A and B in the stated rules and axioms). Thus, let A stand for the conjunction $X_1 = x_1, \ldots, X_n = x_n$, and let B stand for the conjunction $Y_1 = y_1, \ldots, Y_m = y_m$. Then

$$\begin{aligned} A \square\!\!\rightarrow B \equiv\ & Y_{1_{x_1,\ldots,x_n}}(u) = y_1 \\ & \&\ Y_{2_{x_1,\ldots,x_n}}(u) = y_2 \\ & \vdots \\ & \&\ Y_{m_{x_1,\ldots,x_n}}(u) = y_m. \end{aligned} \tag{7.41}$$

Conversely, we need to translate causal statements such as $Y_x(u) = y$ into Lewis's notation. Let A stand for the proposition $X = x$ and B for the proposition $Y = y$. Then

$$Y_x(u) = y \equiv A \square\!\!\rightarrow B. \tag{7.42}$$

Axioms (1)–(6) follow from the closest-world interpretation without imposing any restrictions on the distance measured, except for the requirement that each world w be no further from itself than any other world $w' \neq w$. Because structural semantics defines an obvious distance measure among worlds, $d(w, w')$, given by the minimal number of local interventions needed for transforming w into w', all of Lewis's axioms should hold in causal models and must follow logically from effectiveness, composition, and (for nonrecursive systems) reversibility. This will be shown explicitly first. However, to guarantee that structural semantics does not introduce new constraints we need to show the converse: that the three axioms of structural semantics follow from Lewis's axioms. This will be shown second.

To show that Axioms (1)–(6) hold in structural semantics, we examine each axiom in turn.

(1) This axiom is trivially true.

(2) This axiom is the same as effectiveness: If we force a set of variables X to have the value x, then the resulting value of X is x. That is, $X_x(u) = x$.

(3) This axiom is a weaker form of reversibility, which is relevant only for nonrecursive causal models.

(4) Because actions in structural models are restricted to conjunctions of literals, this axiom is irrelevant.

(5) This axiom follows from composition.

(6) This axiom follows from composition.

To show that composition and effectiveness follow from Lewis's axioms, we note that composition is a consequence of axiom (5) and rule (1) in Lewis's formalism, while effectiveness is the same as Lewis's axiom (2).

In sum, for recursive models, the causal model framework does not add any restrictions to counterfactual statements beyond those imposed by Lewis's framework; the very general concept of closest worlds is sufficient. Put another way, the assumption of recursiveness is so strong that it already embodies all other restrictions imposed by structural semantics. When we consider nonrecursive systems, however, we see that reversibility is not enforced by Lewis's framework. Lewis's axiom (3) is similar to but not as strong as reversibility; that is, even though $Y = y$ may hold in all closest w-worlds and $W = w$ in all closest y-worlds, $Y = y$ still may not hold in the actual world. Nonetheless, we can safely conclude that, in adopting the causal interpretation of counterfactuals (together with the representational and algorithmic machinery of modifiable structural equation models), we are not introducing any restrictions on the set of counterfactual statements that are valid relative to recursive systems.

7.4.3 Imaging versus Conditioning

If action is a transformation from one probability function to another, one may ask whether every such transformation corresponds to an action, or if there are some constraints that are peculiar to those transformations that originate from actions. Lewis's (1976) formulation of counterfactuals indeed identifies such constraints: the transformation must be an *imaging* operator.

Whereas Bayes conditioning $P(s \mid e)$ transfers the entire probability mass from states excluded by e to the remaining states (in proportion to their current $P(s)$), imaging works differently; each excluded state s transfers its mass individually to a select set of states $S^*(s)$ that are considered "closest" to s. Indeed, we saw in (3.11) that the transformation defined by the action $do(X_i = x_i')$ can be interpreted in terms of such a mass-transfer process; each excluded state (i.e., one in which $X_i \neq x_i'$) transferred its mass to a select set of nonexcluded states that shared the same value of pa_i. This simple characterization of the set $S^*(s)$ of closest states is valid for Markovian models, but imaging generally permits the selection of any such set.

The reason why imaging is a more adequate representation of transformations associated with actions can be seen through a representation theorem due to Gardenfors (1988, thm. 5.2, p. 113; strangely, the connection to actions never appears in Gardenfors's analysis). Gardenfors's theorem states that a probability update operator $P(s) \rightarrow P_A(s)$ is an imaging operator if and only if it preserves mixtures; that is,

$$[\alpha P(s) + (1 - \alpha)P'(s)]_A = \alpha P_A(s) + (1 - \alpha)P_A'(s) \tag{7.43}$$

for all constants $1 > \alpha > 0$, all propositions A, and all probability functions P and P'. In other words, the update of any mixture is the mixture of the updates.[22]

This property, called *homomorphy,* is what permits us to specify actions in terms of transition probabilities, as is usually done in stochastic control and Markov decision processes. Denoting by $P_A(s \mid s')$ the probability resulting from acting A on a known state s', the homomorphism (7.43) dictates that

[22] Property (7.43) is reflected in the (U8) postulate of Katsuno and Mendelzon (1991): $(K_1 \vee K_2)o\mu = (K_1 o\mu) \vee (K_2 o\mu)$, where o is an update operator, similar to our $do(\cdot)$ operator.

$$P_A(s) = \sum_{s'} P_A(s \mid s')P(s'); \tag{7.44}$$

this means that, whenever s' is not known with certainty, $P_A(s)$ is given by a weighted sum of $P_A(s \mid s')$ over s', with the weight being the current probability function $P(s')$.

This characterization, however, is too permissive; although it requires any action-based transformation to be describable in terms of transition probabilities, it also accepts any transition probability specification, howsoever whimsical, as a descriptor of some action. The valuable information that actions are defined as *local* surgeries is ignored in this characterization. For example, the transition probability associated with the atomic action $A_i = do(X_i = x_i)$ originates from the deletion of just one mechanism in the assembly. Hence, the transition probabilities associated with the set of atomic actions would normally constrain one another. Such constraints emerge from the axioms of effectiveness, composition, and reversibility when probabilities are assigned to the states of U (Galles and Pearl 1997).

7.4.4 Relations to the Neyman–Rubin Framework

A Language in Search of a Model

The notation $Y_x(u)$ that we used for denoting counterfactual quantities is borrowed from the potential-outcome framework of Neyman (1923) and Rubin (1974), briefly introduced in Section 3.6.3, which was devised for statistical analysis of treatment effects.[23] In that framework, $Y_x(u)$ (often written $Y(x, u)$) stands for the outcome of experimental unit u (e.g., an individual or an agricultural lot) under a hypothetical experimental condition $X = x$. In contrast to the structural modeling, however, the variable $Y_x(u)$ in the potential-outcome framework is not a derived quantity but is taken as a primitive – that is, as an undefined symbol that represents the English phrase "the value that Y would assume in u, had X been x." Researchers pursuing the potential-outcome framework (e.g. Robins 1987; Manski 1995; Angrist et al. 1996) have used this interpretation as a guide for expressing subject-matter information and for devising plausible relationships between counterfactual and observed variables, including Robins's consistency rule $X = x \implies Y_x = Y$ (equation (7.20)). However, the potential-outcome framework does not provide a mathematical model from which such relationships could be derived or on the basis of which the question of completeness could be decided – that is, whether the relationships at hand are sufficient for managing all valid inferences.

The structural equation model formulated in Section 7.1 provides a formal semantics for the potential-outcome framework, since each such model assigns coherent truth values to the counterfactual quantities used in potential-outcome studies. From the structural perspective, the quantity $Y_x(u)$ is not a primitive but rather is derived mathematically from a set of equations F that is modified by the operator $do(X = x)$ (see Definition 7.1.4). Subject-matter information is expressed directly through the variables participating in those equations, without committing to their precise functional form. The variable

[23] A related (if not identical) framework that has been used in economics is the *switching regression*. For a brief review of such models, see Heckman (1996; see also Heckman and Honoré 1990 and Manski 1995). Winship and Morgan (1999) provided an excellent overview of the two schools.

U represents any set of background factors relevant to the analysis, not necessarily the identity of a specific individual in the population.

Using this semantics, in Section 7.3 we established an axiomatic characterization of the potential-response function $Y_x(u)$ and its relationships with the observed variables $X(u)$ and $Y(u)$. These basic axioms include or imply restrictions such as Robins's consistency rule (equation (7.20)), which were taken as given by potential-outcome researchers.

The completeness result further assures us that derivations involving counterfactual relationships in recursive models may safely be managed with two axioms only, effectiveness and composition. All truths implied by structural equation semantics are also derivable using these two axioms. Likewise – in constructing hypothetical contingency tables for recursive models (see Section 6.5.3) – we are guaranteed that, once a table satisfies effectiveness and composition, there exists at least one causal model that would generate that table. In essence, this establishes the formal equivalence of structural equation modeling, which is popular in economics and the social sciences (Goldberger 1991), and the potential-outcome framework as used in statistics (Rubin 1974; Holland 1986; Robins 1986).[24] In nonrecursive models, however, this is not the case. Attempts to evaluate counterfactual statements using only composition and effectiveness may fail to certify some valid conclusions (i.e., true in all causal models) whose validity can only be recognized through the use of reversibility.

Graphical versus Counterfactual Analysis

This formal equivalence between the structural and potential-outcome frameworks covers issues of semantics and expressiveness but does not imply equivalence in conceptualization or practical usefulness. Structural equations and their associated graphs are particularly useful as means of expressing assumptions about cause–effect relationships. Such assumptions rest on prior experiential knowledge, which – as suggested by ample evidence – is encoded in the human mind in terms of interconnected assemblies of autonomous mechanisms. These mechanisms are thus the building blocks from which judgments about counterfactuals are derived. Structural equations $\{f_i\}$ and their graphical abstraction $G(M)$ provide direct mappings for these mechanisms and therefore constitute a natural language for articulating or verifying causal knowledge or assumptions. The major weakness of the potential-outcome framework lies in the requirement that assumptions be articulated as conditional independence relationships involving counterfactual variables. For example, an assumption such as the one expressed in (7.30) is not easily comprehended even by skilled investigators, yet its structural image $U_1 \perp\!\!\!\perp U_2$ evokes an immediate process-based interpretation.[25]

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[24] This equivalence was anticipated in Holland (1988), Pratt and Schlaifer (1988), Pearl (1995a), and Robins (1995). Note, though, that counterfactual claims and the equation deletion part of our model (Definition 7.1.3) are not made explicit in the standard literature on structural equation modeling.

[25] These views are diametrically opposite to those expressed by Angrist et al. (1996), who stated: "Typically the researcher does not have a firm idea what these disturbances really represent, and therefore it is difficult to draw realistic conclusions or communicate results based on their properties." I have found that researchers who are knowledgeable in their respective subjects have a very clear idea what these disturbances really represent, and those who don't would certainly not be able to make realistic judgments about counterfactual dependencies.

A happy symbiosis between graphs and counterfactual notation was demonstrated in Section 7.3.2. In that example, assumptions were expressed in graphical form, then translated into counterfactual notation (using the rules of (7.25) and (7.26)), and finally submitted to algebraic derivation. Such symbiosis offers a more effective method of analysis than methods that insist on expressing assumptions directly as counterfactuals. Additional examples will be demonstrated in Chapter 9, where we analyze probability of causation. Note that, in the derivation of Section 7.3.2, the graph continued to assist the procedure by displaying independence relationships that are not easily derived by algebraic means alone. For example, it is hardly straightforward to show that the assumptions of (7.27)–(7.30) imply the conditional independence $(Y_z \perp\!\!\!\perp Z_x \mid \{Z, X\})$ but do not imply the conditional independence $(Y_z \perp\!\!\!\perp Z_x \mid Z)$. Such implications can, however, easily be tested in the graph of Figure 7.5 or in the twin network construction of Section 7.1.3 (see Figure 7.3).

The most compelling reason for molding causal assumptions in the language of graphs is that such assumptions are needed before the data are gathered, at a stage when the model's parameters are still "free" (i.e., still to be determined from the data). The usual temptation is to mold those assumptions in the language of statistical independence, which carries an aura of testability and hence of scientific legitimacy. (Chapter 6 exemplifies the difficulties associated with such temptations.) However, conditions of statistical independence – regardless of whether they relate to V variables, U variables, or counterfactuals – are generally sensitive to the values of the model's parameters, which are not available at the model construction phase. The substantive knowledge available at the modeling phase cannot support such assumptions unless they are *stable,* that is, insensitive to the values of the parameters involved. The implications of graphical models, which rest solely on the interconnections among mechanisms, satisfy this stability requirement and can therefore be ascertained from generic substantive knowledge *before* data are collected. For example, the assertion $(X \perp\!\!\!\perp Y \mid Z, U_1)$, which is implied by the graph of Figure 7.5, remains valid for any substitution of functions in $\{f_i\}$ and for any assignment of prior probabilities to U_1 and U_2.

These considerations apply not only to the formulation of causal assumptions but also to the language in which causal concepts are defined and communicated. Many concepts in the social and medical sciences are defined in terms of relationships among unobserved U variables, also known as "errors" or "disturbance terms." We have seen in Chapter 5 (Section 5.4.3) that key econometric notions such as exogeneity and instrumental variables have traditionally been defined in terms of absence of correlation between certain observed variables and certain error terms. Naturally, such definitions attract criticism from strict empiricists, who regard unobservables as metaphysical or definitional (Richard 1980; Engle et al. 1983; Holland 1988), and also (more recently) from potential-outcome analysts, who regard the use of structural models as an unwarranted commitment to a particular functional form (Angrist et al. 1996). This new criticism will be considered in the following section.

7.4.5 Exogeneity Revisited: Counterfactual and Graphical Definitions

The analysis of this chapter provides a counterfactual interpretation of the error terms in structural equation models, supplementing the operational definition of (5.25). We have

seen that the meaning of the error term u_Y in the equation $Y = f_Y(pa_Y, u_Y)$ is captured by the counterfactual variable Y_{pa_Y}. In other words, the variable U_Y can be interpreted as a modifier of the functional mapping from PA_Y to Y. The statistics of such modifications is observable when pa_Y is held fixed. This translation into counterfactual notation may facilitate algebraic manipulations of U_Y without committing to the functional form of f_Y. However, from the viewpoint of model specification, the error terms should be still viewed as (summaries of) omitted factors.

Armed with this interpretation, we can obtain graphical and counterfactual definitions of causal concepts that were originally given error-based definitions. Examples of such concepts are causal influence, exogeneity, and instrumental variables (Section 5.4.3). In clarifying the relationships among error-based, counterfactual, and graphical definitions of these concepts, we should first note that these three modes of description can be organized in a simple hierarchy. Since graph separation implies independence but independence does not imply graph separation (Theorem 1.2.4), definitions based on graph separation should imply those based on error-term independence. Likewise, since for any two variables X and Y the independence relation $U_X \perp\!\!\!\perp U_Y$ implies the counterfactual independence $X_{pa_X} \perp\!\!\!\perp Y_{pa_Y}$ (but not the other way around), it follows that definitions based on error independence should imply those based on counterfactual independence. Overall, we have the following hierarchy:

$$\text{graphical criteria} \Longrightarrow \text{error-based criteria} \Longrightarrow \text{counterfactual criteria}.$$

The concept of exogeneity may serve to illustrate this hierarchy. The pragmatic definition of exogeneity is best formulated in counterfactual or interventional terms as follows.

Exogeneity (Counterfactual Criterion)
A variable X is exogenous relative to Y if and only if the effect of X on Y is identical to the conditional probability of Y given X – that is, if

$$P(Y_x = y) = P(y \mid x) \tag{7.45}$$

or, equivalently,

$$P(Y = y \mid do(x)) = P(y \mid x); \tag{7.46}$$

this in turn is equivalent to the independence condition $Y_x \perp\!\!\!\perp X$, named "weak ignorability" in Rosenbaum and Rubin (1983).[26]

This definition is pragmatic in that it highlights the reasons economists should be concerned with exogeneity by explicating the policy-analytic benefits of discovering that a variable is exogenous. However, this definition fails to guide an investigator toward

[26] We focus the discussion in this section on the causal component of exogeneity, which the econometric literature has unfortunately renamed "superexogeneity" (see Section 5.4.3). We also postpone discussion of "strong ignorability," defined as the joint independence $\{Y_x, Y_{x'}\} \perp\!\!\!\perp X$, to Chapter 9 (Definition 9.2.3).

verifying, from substantive knowledge of the domain, whether this independence condition holds in any given system, especially when many equations are involved. To facilitate such judgments, economists (e.g. Koopmans 1950; Orcutt 1952) have adopted the error-based criterion of Definition 5.4.6.

Exogeneity (Error-Based Criterion)
A variable X is exogenous in M relative to Y if X is independent of all error terms that have an influence on Y that is not mediated by X.[27]

This definition is more transparent to human judgment because the reference to error terms tends to focus attention on specific factors, potentially affecting Y, with which scientists are familiar. Still, to judge whether such factors are statistically independent is a difficult mental task unless the independencies considered are dictated by topological considerations that assure their stability. Indeed, the most popular conception of exogeneity is encapsulated in the notion of "common cause"; this may be stated formally as follows.

Exogeneity (Graphical Criterion)
A variable X is exogenous relative to Y if X and Y have no common ancestor in $G(M)$ or, equivalently, if all back-door paths between X and Y are blocked (by colliding arrows).[28]

It is easy to show that the graphical condition implies the error-based condition, which in turn implies the counterfactual (or pragmatic) condition of (7.46). The converse implications do not hold. For example, Figure 6.4 illustrates a case where the graphical criterion fails and both the error-based and counterfactual criteria classify X as exogenous. We argued in Section 6.4 that this type of exogeneity (there called "no confounding") is unstable or incidental, and we have raised the question of whether such cases were meant to be embraced by the definition. If we exclude unstable cases from consideration, then our three-level hierarchy collapses and all three definitions coincide.

Instrumental Variables: Three Definitions

A three-level hierarchy similarly characterizes the notion of instrumental variables (Bowden and Turkington 1984; Pearl 1995c; Angrist et al. 1996), illustrated in Figure 5.9. The traditional definition qualifies a variable Z as *instrumental* (relative to the pair (X, Y)) if (i) Z is independent of all error terms that have an influence on Y that is not mediated by X and (ii) Z is not independent of X.

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[27] Independence relative to *all* errors is sometimes required in the literature (e.g. Dhrymes 1970, p. 169), but this is obviously too strong.

[28] As in Chapter 6 (note 19), the expression "common ancestors" should exclude nodes that have no other connection to Y except through X and should include latent nodes for every pair of dependent errors. Generalization to conditional exogeneity relative to observed covariates is straightforward in all three definitions.

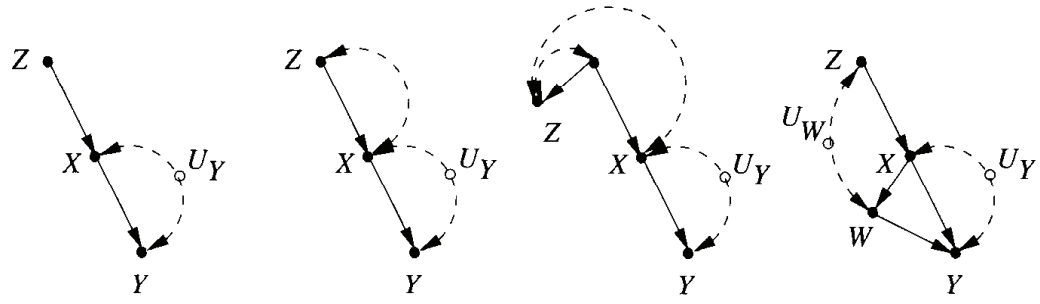

Figure 7.8 Z is a proper instrumental variable in the (linear) models of (a), (b), and (c), since it satisfies $Z \perp\!\!\!\perp U_Y$. It is not an instrument in (d) because it is correlated with U_W, which influences Y.

The counterfactual definition[29] replaces condition (i) with (i′): Z is independent of Y_x. The graphical definition replaces condition (i) with (i″): every unblocked path connecting Z and Y must contain an arrow pointing into X (alternatively, $(Z \perp\!\!\!\perp Y)_{G_{\bar{X}}}$). Figure 7.8 illustrates this definition through examples.

When a set S of covariates is measured, these definitions generalize as follows.

Definition 7.4.1 (Instrument)
A variable Z is an instrument *relative to the total effect of X on Y if there exists a set of measurements $S = s$, unaffected by X, such that either of the following criteria holds.*

1. ***Counterfactual** criterion*:
   (i) $Z \perp\!\!\!\perp Y_x \mid S = s$;
   (ii) $Z \not\perp\!\!\!\perp X \mid S = s$.
2. ***Graphical** criterion*:
   (i) $(Z \perp\!\!\!\perp Y \mid S)_{G_{\bar{X}}}$;
   (ii) $(Z \not\perp\!\!\!\perp X \mid S)_G$.

In concluding this section, I should reemphasize that it is because graphical definitions are insensitive to the values of the model's parameters that graphical vocabulary guides and expresses so well our intuition about causal effects, exogeneity, instruments, confounding, and even (I speculate) more technical notions such as randomness and statistical independence.

[29] There is, in fact, no agreed-upon generalization of instrumental variables to nonlinear systems. The definition here, taken from Galles and Pearl (1998), follows by translating the error-based definition into counterfactual vocabulary. Angrist et al. (1996), who expressly rejected all reference to graphs or error terms, assumed two unnecessary restrictions: that Z be ignorable (i.e. randomized; this is violated in Figures 7.8(b) and (c)) and that Z affect X (violated in Figure 7.8(c)). Similar assumptions were made by Heckman and Vytlacil (1999), who used both counterfactuals and structural equation models.

7.5 STRUCTURAL VERSUS PROBABILISTIC CAUSALITY

Probabilistic causality is a branch of philosophy that attempts to explicate causal relationships in terms of probabilistic relationships. This attempt is motivated by several ideas and expectations. First and foremost, probabilistic causality promises a solution to the centuries-old puzzle of causal discovery – that is, how humans discover genuine causal relationships from bare empirical observations, free of any causal preconceptions. Given the Humean dictum that all knowledge originates with human experience and the (less compelling but fashionable) assumption that human experience is encoded in the form of a probability function, it is natural to expect that causal knowledge be reducible to a set of relationships in some probability distribution that is defined over the variables of interest. Second, in contrast to deterministic accounts of causation, probabilistic causality offers substantial cognitive economy. Physical states and physical laws need not be specified in minute detail because instead they can be summarized in the form of probabilistic relationships among macro states so as to match the granularity of natural discourse. Third, probabilistic causality is equipped to deal with the modern (i.e. quantum-theoretical) conception of uncertainty, according to which determinism is merely an epistemic fiction and nondeterminism is the fundamental feature of physical reality.

The formal program of probabilistic causality owes its inception to Reichenbach (1956) and Good (1961), and it has subsequently been pursued by Suppes (1970), Skyrms (1980), Spohn (1980), Otte (1981), Salmon (1984), Cartwright (1989), and Eells (1991). The current state of this program is rather disappointing, considering its original aspirations. Salmon has abandoned the effort altogether, concluding that "causal relations are not appropriately analyzable in terms of statistical relevance relations" (1984, p. 185); instead, he has proposed an analysis in which "causal processes" are the basic building blocks. More recent accounts by Cartwright and Eells have resolved some of the difficulties encountered by Salmon, but at the price of either complicating the theory beyond recognition or compromising its original goals. The following is a brief account of the major achievements, difficulties, and compromises of probabilistic causality as portrayed in Cartwright (1989) and Eells (1991).

7.5.1 The Reliance on Temporal Ordering

Standard probabilistic accounts of causality assume that, in addition to a probability function P, we are also given the temporal order of the variables in the analysis. This is understandable, considering that causality is an asymmetric relation whereas statistical relevance is symmetric. Lacking temporal information, it would be impossible to decide which of two dependent variables is the cause and which the effect, since every joint distribution $P(x, y)$ induced by a model in which X is a cause of Y can also be induced by a model in which Y is the cause of X. Thus, any method of inferring that X is a cause of Y must also infer, by symmetry, that Y is a cause of X. In Chapter 2 we demonstrated that, indeed, at least three variables are needed for determining the directionality of arrows in a DAG and, more serious yet, no arrow can be oriented from probability information

alone – that is, without the added assumptions of stability or minimality. By imposing the constraint that an effect never precede its cause, the symmetry is broken and causal inference can commence.

The reliance on temporal information has its price, as it excludes a priori the analysis of cases in which the temporal order is not well-defined, either because processes overlap in time or because they (appear to) occur instantaneously. For example, one must give up the prospect of determining (by uncontrolled methods) whether sustained physical exercise contributes to low cholesterol levels or if, conversely, low cholesterol levels enhance the urge to engage in physical exercise. Likewise, the philosophical theory of probabilistic causality would not attempt to distinguish between the claims "tall flag poles cause long shadows" and "long shadows cause tall flag poles" – where, for all practical purposes, the putative cause and effect occur simultaneously.

We have seen in Chapter 2 that some determination of causal directionality can be made from atemporal statistical information, if fortified with the assumptions of minimality or stability. These assumptions, however, implicitly reflect generic properties of physical processes – invariance and autonomy (see Section 2.9.1) – that constitute the basis of the structural approach to causality.

7.5.2 The Perils of Circularity

Despite the reliance on temporal precedence, the criteria that philosophers have devised for identifying causal relations suffer from glaring circularity: In order to determine whether an event C is a cause of event E, one must know in advance how other factors are causally related to C and E. Such circularity emerges from the need to define the "background context" under which a causal relation is evaluated, since the intuitive idea that causes should increase the probability of their effects must be qualified by the condition that other things are assumed equal. For example, "studying arithmetic" increases the probability of passing a science test, but only if we keep student age constant; otherwise, studying arithmetic may actually lower the probability of passing the test because it is indicative of young age. Thus, it seems natural to offer the following.

Definition 7.5.1

An event C is causally relevant *to E if there is at least one condition F in some background context K such that* $P(E \mid C, F) > P(E \mid \neg C, F)$.[30]

But what kind of conditions should we include in the background context? On the one hand, insisting on a complete description of the physical environment would reduce probabilistic causality to deterministic physics (barring quantum-level considerations). On the other hand, ignoring background factors altogether – or describing them too coarsely – would introduce spurious correlations and other confounding effects. A natural compromise is to require that the background context itself be "causally relevant" to the variables

[30] The reader can interpret K to be a set of variables and F a particular truth-value assignment to those variables.

in question, but this very move is the source of circularity in the definition of probabilistic causality.

The problem of choosing an appropriate set of background factors is similar to the problem of finding an appropriate adjustment for confounding, as discussed in several previous chapters in connection with Simpson's paradox (e.g., Sections 3.3, 5.1.3, and 6.1). We have seen (e.g., in Section 6.1) that the criterion for choosing an appropriate set of covariates for adjustment cannot be based on probabilistic relationships alone but must rely on causal information. In particular, we must make sure that factors listed as background are not affected by C; otherwise, no C would ever qualify as a cause of E, because we can always find factors F that are intermediaries between C and E and that screen off E from C.[31] Here we see the emergence of circularity: In order to determine the causal role of C relative to E (e.g., the effect of the drug on recovery), we must first determine the causal role of every factor F (e.g., gender) relative to C and E.

Factors affecting both C and E can be rescued from circularity by conditioning on *all* factors preceding C but, unfortunately, other factors that cannot be identified through temporal ordering alone must also be weighed. Consider the betting example used in Section 7.1.2. I must bet heads or tails on the outcome of a fair coin toss; I win if I guess correctly and lose if I don't. Naturally, once the coin is tossed (and while the outcome is still unknown), the bet is deemed causally relevant to winning, even though the probability of winning is the same whether I bet heads or tails. In order to reveal the causal relevance of the bet (C), we must include the outcome of the coin (F) in the background context even though F does not meet the common-cause criterion – it does not affect my bet (C) nor is it causally relevant to winning (E) (unless we first declare the bet *is* relevant to winning). Worse yet, we cannot justify including F in the background context by virtue of its occurring earlier than C because whether the coin is tossed before or after my bet is totally irrelevant to the problem at hand. We conclude that temporal precedence alone is insufficient for identifying the background context, and we must refine the definition of the background context to include what Eells (1991) called "interacting causes" – namely, (simplified) factors F that (i) are not affected causally by C and (ii) jointly with C (or $\neg C$) increase the probability of E.

Because of the circularity inherent in all definitions of causal relevance, probabilistic causality cannot be regarded as a program for extracting causal relations from temporal–probabilistic information; rather, it should be viewed as a program for validating whether a proposed set of causal relationships is consistent with the available temporal–probabilistic information. More formally, suppose someone gives us a probability distribution P and a temporal order O on a (complete) set of variables V. Furthermore, any pair of variable sets (say, X and Y) in V is annotated by a symbol R or I, where R stands for "causally relevant" and I for "causally irrelevant." Probabilistic causality deals with testing whether the proposed R and I labels are consistent with the pair $\langle P, O \rangle$ and with the restriction that causes should both precede and increase the probability of their effect.

[31] We say that F "screens off" E from C if C and E are conditionally independent, given both F and $\neg F$.

Currently, the most advanced consistency test is the one based on Eells's (1991) criterion of relevance, which may be translated as follows.

Consistency Test
For each pair of variables labeled $R(X, Y)$, test whether

(i) X precedes Y in O, and

(ii) there exist x, x', y such that $P(y \mid x, z) > P(y \mid x', z)$ for some z in Z, where Z is a set of variables in the background context K such that $I(X, Z)$ and $R(Z, Y)$.

This now raises additional questions.

(a) Is there a consistent label for every pair $\langle P, O \rangle$?

(b) When is the label unique?

(c) Is there a procedure for finding a consistent label when it exists?

Although some insights into these questions are provided by graphical methods (Pearl 1996), the point is that, owing to circularity, the mission of probabilistic causality has been altered: from discovery to consistency testing.

It should also be remarked that the basic program of defining causality in terms of conditionalization, even if it turns out to be successful, is at odds with the natural conception of causation as an oracle for interventions. This program first confounds the causal relation $P(E \mid do(C))$ with epistemic conditionalization $P(E \mid C)$ and then removes spurious correlations through steps of remedial conditionalization, yielding $P(E \mid C, F)$. The structural account, in contrast, defines causation directly in terms of Nature's invariants (i.e., submodel M_x in Definition 7.1.2); see the discussion following Theorem 3.2.2.

7.5.3 The Closed-World Assumption

By far the most critical and least defensible paradigm underlying probabilistic causality rests on the assumption that one is in the possession of a probability function on all variables relevant to a given domain. This assumption absolves the analyst from worrying about unmeasured spurious causes that might (physically) affect several variables in the analysis and still remain obscure to the analyst. It is well known that the presence of such "confounders" may reverse or negate any causal conclusion that might be drawn from probabilities. For example, observers might conclude that "bad air" is the cause of malaria if they are not aware of the role of mosquitoes, or that falling barometers are the cause of rain, or that speeding to work is the cause of being late to work, and so on. Because they are unmeasured (or even unsuspected), the confounding factors in such examples cannot be neutralized by conditioning or by "holding them fixed." Thus, taking seriously Hume's program of extracting causal information from raw data entails coping with the problem that the validity of any such information is predicated on the untestable assumption that all relevant factors have been accounted for.

This raises the question of how people ever acquire causal information from the environment and, more specifically, how children extract causal information from experience.

The proponents of probabilistic causality who attempt to explain this phenomenon through statistical theories of learning cannot ignore the fact that the child never operates in a closed, isolated environment. Unnoticed external conditions govern the operation of every learning environment, and these conditions often have the potential to confound cause and effect in unexpected and clandestine ways.

Fortunately, that children do not grow in closed, sterile environments does have its advantages. Aside from passive observations, a child possesses two valuable sources of causal information that are not available to the ordinary statistician: manipulative experimentation and linguistic advice. Manipulation subjugates the putative causal event to the sole influence of a known mechanism, thus overruling the influence of uncontrolled factors that might also produce the putative effect. "The beauty of independent manipulation is, of course, that other factors can be kept constant without their being identified" (Cheng 1992). The independence is accomplished by subjecting the object of interest to the whims of one's volition in order to ensure that the manipulation is not influenced by any environmental factor likely to produce the putative effect. Thus, for example, a child can infer that shaking a toy can produce a rattling sound because it is the child's hand, governed solely by the child's volition, that brings about the shaking of the toy and the subsequent rattling sound. The whimsical nature of free manipulation replaces the statistical notion of randomized experimentation and serves to filter sounds produced by the child's actions from those produced by uncontrolled environmental factors.

But manipulative experimentation cannot explain all of the causal knowledge that humans acquire and possess, simply because most variables in our environment are not subject to direct manipulation. The second valuable source of causal knowledge is linguistic advice: explicit causal sentences about the workings of things which we obtain from parents, friends, teachers, and books and which encode the manipulative experience of past generations. As obvious and uninteresting as this source of causal information may appear, it probably accounts for the bulk of our causal knowledge, and understanding how this transference of knowledge works is far from trivial. In order to comprehend and absorb causal sentences such as "The glass broke because you pushed it," the child must already possess a causal schema within which such inputs make sense. To further infer that pushing the glass will make someone angry at you and not at your brother, even though he was responsible for all previous breakage, requires a truly sophisticated inferential machinery. In most children, this machinery is probably innate.

Note, however, that linguistic input is by and large qualitative; we rarely hear parents explaining to children that placing the glass at the edge of the table increases the probability of breakage by a factor of 2.85. The probabilistic approach to causality embeds such qualitative input in an artificial numerical frame, whereas the structural approach to causality (Section 7.1) builds directly on the qualitative knowledge that we obtain and transmit linguistically.

7.5.4 Singular versus General Causes

In Section 7.2.3 we saw that the distinction between general causes (e.g., "Drinking hemlock causes death") and singular causes (e.g., "Socrates' drinking hemlock caused his death") plays an important role in understanding the nature of explanations. We have

also remarked that the notion of singular causation (also known as "token" or "single-event" causation) has not reached an adequate state of conceptualization or formalization in the probabilistic account of causation. In this section we elaborate the nature of these difficulties and conclude that they stem from basic deficiencies in the probabilistic account.

In Chapter 1 (Figure 1.6) we demonstrated that the evaluation of singular causal claims requires knowledge in the form of counterfactual or functional relationships and that such knowledge cannot be extracted from bare statistical data even when obtained under controlled experimentation. This limitation was attributed in Section 7.2.2 to the temporal persistence (or invariance) of information that is needed to sustain counterfactual statements – persistence that is washed out (by averaging) in statistical statements even when enriched with temporal and causally relevant information. The manifestations of this basic limitation have taken an interesting slant in the literature of probabilistic causation and have led to intensive debates regarding the relationships between singular and generic statements (see e.g. Good 1961; Cartwright 1989; Eells 1991; Hausman 1998).

According to one of the basic tenets of probabilistic causality, a cause should raise the probability of the effect. It is often the case, however, that we judge an event x to be the cause of y when the conditional probability $P(y \mid x)$ is *lower* than $P(y \mid x')$. For example, a vaccine (x) usually decreases the probability of the disease (y) and yet we often say (and can medically verify) that the vaccine itself caused the disease in a given person u. Such reversals would not be problematic to students of structural models, who can interpret the singular statement as saying that "had person u not taken the vaccine (x') then u would still be healthy (y')." The probability of this counterfactual statement $P(Y_{x'} = y' \mid x, y)$ can be high while the conditional probability $P(y \mid x)$ is low, with both probabilities evaluated formally from the same structural model (Section 9.2 provides precise relationships between the two quantities). However, this reversal is traumatic to students of probabilistic causation, who mistrust counterfactuals for various reasons – partly because counterfactuals carry an aura of determinism (Kvart 1986, pp. 256–63) and partly because counterfactuals are perceived as resting on shaky formal foundation "for which we have only the beginnings of a semantics (via the device of measures over possible worlds)" (Cartwright 1983, p. 34).

In order to reconcile the notion of probability increase with that of singular causation, probabilists claim that, if we look hard enough at any given scenario in which x is judged to be a cause of y, then we will always be able to find a subpopulation $Z = z$ in which x raises the probability of y – namely,

$$P(y \mid x, z) > P(y \mid x', z). \tag{7.47}$$

In the vaccine example, we might identify the desired subpopulation as consisting of individuals who are adversely susceptible to the vaccine; by definition, the vaccine would no doubt raise the probability of the disease in that subpopulation. Oddly, only few philosophers have noticed that factors such as being "adversely susceptible" are defined counterfactually and that, in permitting conditionalization on such factors, one opens a clandestine back door for sneaking determinism and counterfactual information back into the analysis.

Perhaps a less obvious appearance of counterfactuals surfaces in Hesslow's example of the birth-control pill (Hesslow 1976), discussed in Section 4.5.1. Suppose we find that

Mrs. Jones is not pregnant and ask whether taking a birth-control pill was the cause of her suffering from thrombosis. The population of pregnant women turns out to be too coarse for answering this question unequivocally. If Mrs. Jones belongs to the class of women who would have become pregnant *but for* the pill, then the pill might actually have lowered the probability of thrombosis in her case by preventing her pregnancy. If, on the other hand, she belongs to the class of women who would *not* have become pregnant regardless of the pill, then her taking the pill has surely increased the chance of thrombosis. This example is illuminating because the two classes of test populations do not have established names in the English language (unlike "susceptibility" of the vaccine example) and must be defined explicitly in counterfactual vocabulary. Whether a woman belongs to the former or latter class depends on many social and circumstantial contingencies, which are usually unknown and are not likely to define an invariant attribute of a given person. Still, we recognize the need to consider the two classes separately in evaluating whether the pill was the cause of Mrs. Jones's thrombosis.

Thus we see that there is no escape from counterfactuals when we deal with token-level causation. Probabilists' insistence on counterfactual-free syntax in defining token causal claims has led to subpopulations delineated by none other but counterfactual expressions: "adversely susceptible" in the vaccine example and "would not have become pregnant" in the case of Mrs. Jones.[32]

Probabilists can argue, of course, that there is no need to refine the subclasses $Z = z$ down to deterministic extremes, since one can stop the refinement as soon as one finds a subclass that increases the probability of y, as required in (7.47). This argument borders on the tautological, unless it is accompanied with formal procedures for identifying the test subpopulation $Z = z$ and for computing the quantities in (7.47) from some reasonable model of human knowledge, however hypothetical. Unfortunately, the probabilistic causality literature is silent on questions of procedures and representation.[33]

In particular, probabilists face a tough dilemma in explaining how people search for that rescuing subpopulation z so swiftly and consistently and how the majority of people end up with the same answer when asked whether it was x that caused y. For example (due to Debra Rosen, quoted in Suppes 1970), a tree limb (x) that fortuitously deflects a golf ball is immediately and consistently perceived as "the cause" for the ball finally ending up in the hole, though such collisions generally lower one's chances of reaching the hole (y). Clearly, if there is a subpopulation z that satisfies (7.47) in such examples (and I doubt it ever enters anyone's mind), it must have at least two features.

(1) It must contain events that occur both before and after x. For example, both the angle at which the ball hit the limb and the texture of the grass on which the ball bounced after hitting the limb should be part of z.

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[32] Cartwright (1989, chap. 3) recognized the insufficiency of observable partitions (e.g. pregnancy) for sustaining the thesis of increased probability, but she did not emphasize the inevitable counterfactual nature of the finer partitions that sustain that thesis. Not incidentally, Cartwright was a strong advocate of excluding counterfactuals from causal analysis (Cartwright 1983, pp. 34–5).

[33] Even Eells (1991, chap. 6) and Shafer (1996a), who endeavored to uncover discriminating patterns of increasing probabilities in the actual trajectory of the world leading to y, did not specify what information is needed either to select the appropriate trajectory or to compute the probabilities associated with a given trajectory.

(2) It must depend on x and y. For, surely, a different conditioning set z' would be necessary in (7.47) if we were to test whether the limb caused an alternative consequence y' – say, that the ball stopped two yards short of the hole.

And this brings us to a major methodological inconsistency in the probabilistic approach to causation: If ignorance of x and y leads to the wrong z and if awareness of x and y leads to the correct selection of z, then there must be some process by which people incorporate the occurrence of x and y into their awareness. What could that process be? According to the norms of probabilistic epistemology, evidence is incorporated into one's corpus of knowledge by means of conditionalization. How, then, can we justify excluding from z the very evidence that led to its selection – namely, the occurrence of x and y?

Inspection of (7.47) shows that the exclusion of x and y from z is compelled on syntactic grounds, since it would render $P(y \mid x', z)$ undefined and make $P(y \mid x, z) = 1$. Indeed, in the syntax of probability calculus we cannot ask what the probability of event y *would* be, given that y has in fact occurred – the answer is (trivially) 1. The best we can do is detach ourselves momentarily from the actual world, pretend that we are ignorant of the occurrence of y, and ask for the probability of y under such a state of ignorance. This corresponds precisely to the three steps (abduction, action, and prediction) that govern the evaluation of $P(Y_{x'} = y' \mid x, y)$ (see Theorem 7.1.7), which attains a high value (in our example) and correctly qualifies the tree limb (x) as the cause of making the hole (y). As we see, the desired quantity *can* be expressed and evaluated by ordinary conditionalization on x and y, without explicitly invoking any subpopulation z.[34]

Ironically, by denying counterfactual conditionals, probabilists deprived themselves of using standard conditionals – the very conditionals they were trying to preserve – and were forced to accommodate simple evidential information in roundabout ways. This syntactic barrier that probabilists erected around causation has created an artificial tension between singular and generic causes, but the tension disappears in the structural account. In Section 10.1.1 we show that, by accommodating both standard and counterfactual conditionals (i.e. Y_x), singular and generic causes no longer stand in need of separate analyses. The two types of causes differ merely in the level of scenario-specific information that is brought to bear on a problem, that is, in the specificity of the evidence e that enters the quantity $P(Y_x = y \mid e)$.

7.5.5 Summary

Cartwright (1983, p. 34) listed several reasons for pursuing the probabilistic versus the counterfactual approach to causation:

> [the counterfactual approach] requires us to evaluate the probability of counterfactuals for which we have only the beginnings of a semantics (via the device of measures over possible worlds) and no methodology, much less an account of why the methodology is suited

[34] The desired subpopulation z is equal to the set of all u that are mapped into $X(u) = x$, $Y(u) = y$, and $Y_{x'}(u) = y'$.

to the semantics. How do we test claims about probabilities of counterfactuals? We have no answer, much less an answer that fits with our nascent semantics. It would be preferable to have a measure of effectiveness that requires only probabilities over events that can be tested in the actual world in the standard ways.

Examining the progress of the probabilistic approach in the past two decades, it seems clear that Cartwright's aspirations have materialized not in the framework she advocated but rather in the competing framework of counterfactuals, as embodied in structural models. Full characterization of "effectiveness" ("causal effects" in our vocabulary) in terms of "events that can be tested" emerged from Simon's (1953) and Strotz and Wold's (1960) conception of modifiable structural models and led to the back-door criterion (Theorem 3.3.2) and to the more general Theorem 4.3.1, of which the probabilistic criteria (as in (3.13)) are but crude special cases. The interpretation of singular causation in terms of the counterfactual probability $P(Y_{x'} \neq y \mid x, y)$ has enlisted the support of meaningful formal semantics (Section 7.1) and effective evaluation methodology (Theorem 7.1.7 and Sections 7.1.3–7.2.1), while the probabilistic criterion of (7.47) lingers in vagueness and procedureless debates. The original dream of rendering causal claims testable was given up in the probabilistic framework as soon as unmeasured entities (e.g., state of the world, background context, causal relevance, susceptibility) were allowed to infiltrate the analysis, and methodologies for answering questions of testability have moved over to the structural–counterfactual framework (see Chapter 9).

The ideal of remaining compatible with the teachings of nondeterministic physics seems to be the only viable aspect remaining in the program of probabilistic causation, and this section questions whether maintaining this ideal justifies the sacrifices. It further suggests that the basic agenda of the probabilistic causality program is due for a serious reassessment. If the program is an exercise in epistemology, then the word "probabilistic" is oxymoronic – human perception of causality has remained quasi-deterministic, and these fallible humans are still the main consumers of causal talk. If the program is an exercise in modern physics, then the word "causality" is nonessential – quantum-level causality follows its own rules and intuitions, and another name (perhaps "qua-sality") might be more befitting. However, regarding artificial intelligence and cognitive science, I would venture to predict that robots programmed to emulate the quasi-deterministic macroscopic approximations of Laplace and Einstein would far outperform those built on the correct but counterintuitive theories of Born, Heisenberg, and Bohr.

Acknowledgment

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CHAPTER EIGHT

Imperfect Experiments: Bounding Effects and Counterfactuals

Would that I could discover truth
as easily as I can uncover falsehood.
Cicero (44 B.C.)

Preface

In this chapter we describe how graphical and counterfactual models (Sections 3.2 and 7.1 can combine to elicit causal information from imperfect experiments: experiments that deviate from the ideal protocol of randomized control. A common deviation occurs, for example, when subjects in a randomized clinical trial do not fully comply with their assigned treatment, thus compromising the identification of causal effects. When conditions for identification are not met, the best one can do is derive *bounds* for the quantities of interest – namely, a range of possible values that represents our ignorance about the data-generating process and that cannot be improved with increasing sample size. The aim of this chapter is to demonstrate (i) that such bounds can be derived by simple algebraic methods and (ii) that, despite the imperfection of the experiments, the derived bounds can yield significant and sometimes accurate information on the impact of a policy on the entire population as well as on a particular individual who participated in the study.

8.1 INTRODUCTION

8.1.1 Imperfect and Indirect Experiments

Standard experimental studies in the biological, medical, and behavioral sciences invariably invoke the instrument of randomized control; that is, subjects are assigned at random to various groups (or treatments or programs), and the mean differences between participants in different groups are regarded as measures of the efficacies of the associated programs. Deviations from this ideal setup may take place either by failure to meet any of the experimental requirements or by deliberate attempts to relax these requirements. *Indirect experiments* are studies in which randomized control is either unfeasible or undesirable. In such experiments, subjects are still assigned at random to various groups, but members of each group are simply encouraged (rather than forced) to participate in the program associated with the group; it is up to the individuals to select among the programs.

Recently, use of strict randomization in social and medical experimentation has been questioned for three major reasons.

1. Perfect control is hard to achieve or ascertain. Studies in which treatment is assumed to be randomized may be marred by uncontrolled *imperfect compliance.* For example, subjects experiencing adverse reactions to an experimental drug may decide to reduce the assigned dosage. Alternatively, if the experiment is testing a drug for a terminal disease, a subject suspecting that he or she is in the control group may obtain the drug from other sources. Such imperfect compliance renders the experiment indirect and introduces bias into the conclusions that researchers draw from the data. This bias cannot be corrected unless detailed models of compliance are constructed (Efron and Feldman 1991).
2. Denying subjects assigned to certain control groups the benefits of the best available treatment has moral and legal ramifications. For example, in AIDS research it is difficult to justify placebo programs because those patients assigned to the placebo group would be denied access to potentially life-saving treatment (Palca 1989).
3. Randomization, by its very presence, may influence participation as well as behavior (Heckman 1992). For example, eligible candidates may be wary of applying to a school once they discover that it deliberately randomizes its admission criteria. Likewise, as Kramer and Shapiro (1984) noted, subjects in drug trials may be less likely to participate in randomized trials than in nonexperimental studies, even when the treatments are equally nonthreatening.

Altogether, researchers are beginning to acknowledge that mandated randomization may undermine the reliability of experimental evidence and that experimentation with human subjects often involves – and sometimes *should* involve – an element of self-selection.

This chapter concerns the drawing of inferences from studies in which subjects have final choice of program; the randomization is confined to an indirect *instrument* (or *assignment*) that merely encourages or discourages participation in the various programs. For example, in evaluating the efficacy of a given training program, notices of eligibility may be sent to a randomly selected group of students or, alternatively, eligible candidates may be selected at random to receive scholarships for participating in the program. Similarly, in drug trials, subjects may be given randomly chosen advice on recommended dosage level, yet the final choice of dosage will be determined by the subjects to fit their individual needs.

Imperfect compliance poses a problem because simply comparing the fractions in the treatment and control groups may provide a misleading estimate for how effective the treatment would be if applied uniformly to the population. For example, if those subjects who declined to take the drug are precisely those who would have responded adversely, the experiment might conclude that the drug is more effective than it actually is. In Chapter 3 (see Section 3.5, Figure 3.7(b)), we showed that treatment effectiveness in such studies is actually *nonidentifiable.* That is, in the absence of additional modeling assumptions, treatment effectiveness cannot be estimated from the data without bias,

even when the number of subjects in the experiment approaches infinity and even when a record is available of the action and response of each subject.

The question we attempt to answer in this chapter is whether indirect randomization can provide information that allows approximate assessment of the intrinsic merit of a program, as would be measured, for example, if the program were to be extended and mandated uniformly to the population. The analysis presented shows that, given a minimal set of assumptions, such inferences are indeed possible – albeit in the form of *bounds,* rather than precise point estimates, for the causal effect of the program or treatment. These bounds can be used by the analyst to guarantee that the causal effect of a given program must be higher than one measurable quantity and lower than another.

Our most crucial assumption is that, for any given person, the encouraging instrument influences the treatment chosen by that person but has no effect on how that person would respond to the treatment chosen (see the definition of instrumental variables in Section 7.4.5). The second assumption, one which is always made in experimental studies, is that subjects respond to treatment independently of one other. Other than these two assumptions, our model places no constraints on how tendencies to respond to treatments may interact with choices among treatments.

8.1.2 Noncompliance and Intent to Treat

In a popular compromising approach to the problem of imperfect compliance, researchers perform an "intent to treat" analysis in which the control and treatment group are compared without regard to whether the treatment was actually received.[1] The result of such an analysis is a measure of how well the treatment *assignment* affects the disease, as opposed to the desired measure of how well the treatment *itself* affects the disease. Estimates based on intent-to-treat analyses are valid only as long as the experimental conditions perfectly mimic the conditions prevailing in the eventual usage of the treatment. In particular, the experiment should mimic subjects' incentives for receiving each treatment. In situations where field incentives are more compelling than experimental incentives, as is usually the case when drugs receive the approval of a government agency, treatment effectiveness may vary significantly from assignment effectiveness. For example, imagine a study in which (a) the drug has an adverse effect on a large segment of the population and (b) only those members of the segment who drop from the treatment "arm" (subpopulation) recover. The intent-to-treat analysis will attribute these cases of recovery to the drug because they are part of the intent-to-treat arm, although in reality these cases recovered by *avoiding* the treatment.

Another approach to the problem is to use a correction factor based on an instrumental variables formula (Angrist et al. 1996), according to which the intent-to-treat measure should be divided by the fraction of subjects who comply with the treatment assigned to them. Angrist et al. (1996) showed that, under certain conditions, the corrected formula is valid for the subpopulation of "responsive" subjects – that is, subjects who would have changed treatment status if given a different assignment. Unfortunately, this subpopulation cannot be identified and, more seriously, it cannot serve as a basis for policies

[1] This approach is currently used by the FDA to approve new drugs.

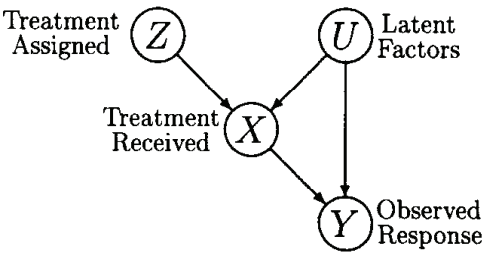


Figure 8.1 Graphical representation of causal dependencies in a randomized clinical trial with partial compliance.

involving the entire population because it is instrument-dependent: individuals who are responsive in the study may not remain responsive in the field, where the incentives for obtaining treatment differ from those used in the study. We therefore focus our analysis on the stable aspect of the treatment – the aspect that would remain invariant to changes in compliance behavior.

8.2 BOUNDING CAUSAL EFFECTS

8.2.1 Problem Formulation

The basic experimental setting associated with indirect experimentation is shown in Figure 8.1, which is isomorphic to Figures 3.7(b) and 5.9. To focus the discussion, we will consider a prototypical clinical trial with partial compliance, although in general the model applies to any study in which a randomized instrument encourages subjects to choose one program over another.

We assume that Z, X, Y are observed binary variables, where Z represents the (randomized) treatment assignment, X is the treatment actually received, and Y is the observed response. The U term represents all factors, both observed and unobserved, that influence the way a subject responds to treatments; hence, an arrow is drawn from U to Y. The arrow from U to X denotes that the U factors may also influence the subject's choice of treatment X; this dependence may represent a complex decision process standing between the assignment (Z) and the actual treatment (X).

To facilitate the notation, we let z, x, y represent (respectively) the values taken by the variables Z, X, Y, with the following interpretation:

$z \in \{z_0, z_1\}$, z_1 asserts that treatment has been assigned (z_0, its negation);

$x \in \{x_0, x_1\}$, x_1 asserts that treatment has been administered (x_0, its negation); and

$y \in \{y_0, y_1\}$, y_1 asserts a positive observed response (y_0, its negation).

The domain of U remains unspecified and may, in general, combine the spaces of several random variables, both discrete and continuous.

The graphical model reflects two assumptions.

1. The assigned treatment Z does not influence Y directly but rather through the actual treatment X. In practice, any direct effect Z might have on Y would be adjusted for through the use of a placebo.
2. The variables Z and U are marginally independent; this is ensured through the randomization of Z, which rules out a common cause for both Z and U.

These assumptions impose on the joint distribution the decomposition

$$P(y, x, z, u) = P(y \mid x, u)P(x \mid z, u)P(z)P(u), \tag{8.1}$$

which, of course, cannot be observed directly because U is unobserved. However, the marginal distribution $P(y, x, z)$ and, in particular, the conditional distributions

$$P(y, x \mid z) = \sum_u P(y \mid x, u)P(x \mid z, u)P(u), \quad z \in \{z_0, z_1\}, \tag{8.2}$$

are observed,[2] and the challenge is to assess from these distributions the average *change* in Y due to treatment.

Treatment effects are governed by the distribution $P(y \mid do(x))$, which – using the truncated factorization formula of (3.10) – is given by

$$P(y \mid do(x)) = \sum_u P(y \mid x, u)P(u); \tag{8.3}$$

here, the factors $P(y \mid x, u)$ and $P(u)$ are the same as those in (8.2). Therefore, if we are interested in the average change in Y due to treatment then we should compute the *average causal effect*, $\text{ACE}(X \rightarrow Y)$ (Holland 1988), which is given by

$$\begin{aligned} \text{ACE}(X \rightarrow Y) &= P(y_1 \mid do(x_1)) - P(y_1 \mid do(x_0)) \\ &= \sum_u [P(y_1 \mid x_1, u) - P(y_1 \mid x_0, u)]P(u). \end{aligned} \tag{8.4}$$

Our task is then to estimate or bound the expression in (8.4) given the observed probabilities $P(y, x \mid z_0)$ and $P(y, x \mid z_1)$, as expressed in (8.2). This task amounts to a constrained optimization exercise of finding the highest and lowest values of (8.4) subject to the equality constraint in (8.2), where the maximization ranges over all possible functions

$$P(u),\ P(y_1 \mid x_0, u),\ P(y_1 \mid x_1, u),\ P(x_1 \mid z_0, u),\ \text{and}\ P(x_1 \mid z_1, u)$$

that satisfy those constraints.

8.2.2 The Evolution of Potential-Response Variables

The bounding exercise described in Section 8.2.1 can be solved using conventional techniques of mathematical optimization. However, the continuous nature of the functions involved – as well as the unspecified domain of U – makes this representation inconvenient for computation. Instead, we can use the observation that U can always be replaced by a finite-state variable such that the resulting model is equivalent with respect to all observations and manipulations of Z, X, and Y (Pearl 1994a).

[2] In practice, of course, only a finite sample of $P(y, x \mid z)$ will be observed. But our task is one of identification, not estimation, so we make the large-sample assumption and consider $P(y, x \mid z)$ as given.

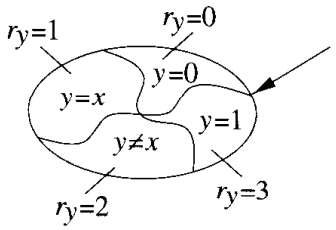


Figure 8.2 The partition of U into four equivalence classes, each inducing a distinct functional mapping from X to Y for any given function $y = f(x, u)$.

Consider the structural equation that connects two binary variables, Y and X, in a causal model:

$$y = f(x, u).$$

For any given u, the relationship between X and Y must be one of four functions:

$$\begin{aligned} &f_0 : y = 0, \quad f_1 : y = x, \\ &f_2 : y \neq x, \quad f_3 : y = 1. \end{aligned} \tag{8.5}$$

As u varies along its domain, regardless of how complex the variation, the only effect it can have on the model is to switch the relationship between X and Y among these four functions. This partitions the domain of U into four equivalence classes, as shown in Figure 8.2, where each class contains those points u that correspond to the same function. We can thus replace U by a four-state variable, $R(u)$, such that each state represents one of the four functions. The probability $P(u)$ would automatically translate into a probability function $P(r)$, $r = 0, 1, 2, 3$, that is given by the total weight assiged to the equivalence class corresponding to r. A state-minimal variable like R is called a "response" variable by Balke and Pearl (1994a,b) and a "mapping" variable by Heckerman and Shachter (1995).[3]

Because Z, X, and Y are all binary variables, the state space of U divides into 16 *equivalence classes:* each class dictates two functional mappings, one from Z to X and the other from X to Y. To describe these equivalence classes, it is convenient to regard each of them as a point in the joint space of two four-valued variables R_x and R_y. The variable R_x determines the compliance behavior of a subject through the mapping

[3] In the potential-outcome model (see Section 7.4.4), u stands for an experimental unit and $R(u)$ corresponds to the potential response of unit u to treatment x. The assumption that each experimental unit (e.g., an individual subject) possesses an intrinsic, seemingly "fatalistic" response function has met with some objections (Dawid 1997), owing to the complexity and inherent unobservability of the many factors that might govern an individual response to treatment. The equivalence-class formulation of $R(u)$ mitigates those objections by showing that $R(u)$ evolves naturally and mathematically from any complex system of stochastic latent variables, provided only that we acknowledge the existence of such variables through the equation $y = f(x, u)$. Those who invoke quantum-mechanical objections to the latter step as well (e.g. Salmon 1998) should regard the functional relationship $y = f(x, u)$ as an abstract mathematical construct that represents the extreme points (vertices) of the set of conditional probabilities $P(y \mid x, u)$ satisfying the constraints of (8.1) and (8.2).

$$x = f_X(z, r_x) = \begin{cases} x_0 & \text{if } r_x = 0; \\ x_0 & \text{if } r_x = 1 \text{ and } z = z_0, \\ x_1 & \text{if } r_x = 1 \text{ and } z = z_1; \\ x_1 & \text{if } r_x = 2 \text{ and } z = z_0, \\ x_0 & \text{if } r_x = 2 \text{ and } z = z_1; \\ x_1 & \text{if } r_x = 3. \end{cases} \tag{8.6}$$

Imbens and Rubin (1997) call a subject with compliance behavior $r_x = 0, 1, 2, 3$ (respectively) a *never-taker,* a *complier,* a *defier,* and an *always-taker.* Similarly, the variable R_y determines the response behavior of a subject through the mapping:

$$y = f_Y(x, r_y) = \begin{cases} y_0 & \text{if } r_y = 0; \\ y_0 & \text{if } r_y = 1 \text{ and } x = x_0, \\ y_1 & \text{if } r_y = 1 \text{ and } x = x_1; \\ y_1 & \text{if } r_y = 2 \text{ and } x = x_0, \\ y_0 & \text{if } r_y = 2 \text{ and } x = x_1; \\ y_1 & \text{if } r_y = 3. \end{cases} \tag{8.7}$$

Following Heckerman and Shachter (1995), we call the response behavior $r_y = 0, 1, 2, 3$ (respectively) *never-recover, helped, hurt,* and *always-recover.*

The correspondence between the states of variable R_y and the potential response variables, Y_{x_0} and Y_{x_1}, defined in Section 7.1 (Definition 7.1.4) is as follows:

$$Y_{x_1} = \begin{cases} y_1 & \text{if } r_y = 1 \text{ or } r_y = 3, \\ y_0 & \text{otherwise;} \end{cases}$$

$$Y_{x_0} = \begin{cases} y_1 & \text{if } r_y = 2 \text{ or } r_y = 3, \\ y_0 & \text{otherwise.} \end{cases}$$

In general, response and compliance may not be independent, hence the double arrow $R_x \dashleftarrow\dashrightarrow R_y$ in Figure 8.3. The joint distribution over $R_x \times R_y$ requires 15 independent parameters, and these parameters are sufficient for specifying the model of Figure 8.3, $P(y, x, z, r_x, r_y) = P(y \mid x, r_y)P(x \mid r_x, z)P(z)P(r_x, r_y)$, because Y and X stand in fixed functional relations to their parents in the graph. The causal effect of the treatment can now be obtained directly from (8.7), giving

$$P(y_1 \mid do(x_1)) = P(r_y = 1) + P(r_y = 3), \tag{8.8}$$

$$P(y_1 \mid do(x_0)) = P(r_y = 2) + P(r_y = 3), \tag{8.9}$$

and

$$\text{ACE}(X \rightarrow Y) = P(r_y = 1) - P(r_y = 2). \tag{8.10}$$

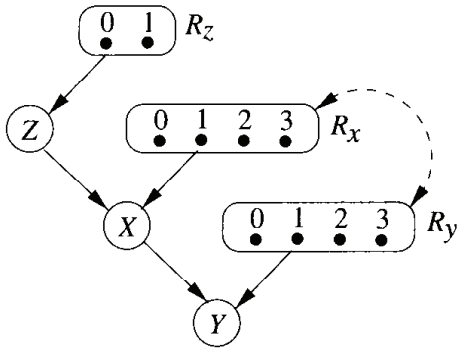


Figure 8.3 A structure equivalent to that of Figure 8.1 but employing finite-state response variables R_z, R_x, and R_y.

8.2.3 Linear Programming Formulation

By explicating the relationship between the parameters of $P(y, x \mid z)$ and those of $P(r_x, r_y)$, we obtain a set of linear constraints needed for minimizing or maximizing $\text{ACE}(X \rightarrow Y)$ given $P(y, x \mid z)$.

The conditional distribution $P(y, x \mid z)$ over the observable variables is fully specified by eight parameters, which will be written as follows:

$$p_{00.0} = P(y_0, x_0 \mid z_0), \quad p_{00.1} = P(y_0, x_0 \mid z_1),$$
$$p_{01.0} = P(y_0, x_1 \mid z_0), \quad p_{01.1} = P(y_0, x_1 \mid z_1),$$
$$p_{10.0} = P(y_1, x_0 \mid z_0), \quad p_{10.1} = P(y_1, x_0 \mid z_1),$$
$$p_{11.0} = P(y_1, x_1 \mid z_0), \quad p_{11.1} = P(y_1, x_1 \mid z_1).$$

The probabilistic constraints

$$\sum_{n=00}^{11} p_{n.0} = 1 \quad \text{and} \quad \sum_{n=00}^{11} p_{n.1} = 1$$

further imply that $\vec{p} = (p_{00.0}, \ldots, p_{11.1})$ can be specified by a point in 6-dimensional space. This space will be referred to as P.

The joint probability $P(r_x, r_y)$ has 16 parameters:

$$q_{jk} \triangleq P(r_x = j,\ r_y = k), \tag{8.11}$$

where $j, k \in \{0, 1, 2, 3\}$. The probabilistic constraint

$$\sum_{j=0}^{3} \sum_{k=0}^{3} q_{jk} = 1$$

implies that $\vec{q}$ specifies a point in 15-dimensional space. This space will be referred to as Q.

Equation (8.10) can now be rewritten as a linear combination of the Q parameters:

$$\text{ACE}(X \rightarrow Y) = q_{01} + q_{11} + q_{21} + q_{31} - q_{02} - q_{12} - q_{22} - q_{32}. \tag{8.12}$$

Applying (8.6) and (8.7), we can write the linear transformation from a point $\vec{q}$ in Q to a point $\vec{p}$ in P:

$$\begin{aligned}
p_{00.0} &= q_{00} + q_{01} + q_{10} + q_{11}, & p_{00.1} &= q_{00} + q_{01} + q_{20} + q_{21},\\
p_{01.0} &= q_{20} + q_{22} + q_{30} + q_{32}, & p_{01.1} &= q_{10} + q_{12} + q_{30} + q_{32},\\
p_{10.0} &= q_{02} + q_{03} + q_{12} + q_{13}, & p_{10.1} &= q_{02} + q_{03} + q_{22} + q_{23},\\
p_{11.0} &= q_{21} + q_{23} + q_{31} + q_{33}, & p_{11.1} &= q_{11} + q_{13} + q_{31} + q_{33},
\end{aligned}$$

this can be written in matrix form as $\vec{p} = \boldsymbol{R}\vec{q}$.

Given a point $\vec{p}$ in P-space, the strict lower bound on ACE($X \rightarrow Y$) can be determined by solving the following linear programming problem.

$$\text{Minimize } q_{01} + q_{11} + q_{21} + q_{31} - q_{02} - q_{12} - q_{22} - q_{32}$$

subject to:

$$\begin{aligned}
&\sum_{j=0}^{3}\sum_{k=0}^{3} q_{jk} = 1,\\
&\boldsymbol{R}\vec{q} = \vec{p},\\
&q_{jk} \geq 0 \quad \text{for } j,k \in \{0,1,2,3\}.
\end{aligned} \tag{8.13}$$

Moreover, for problems of this size, procedures are available for deriving symbolic expressions as well (Balke 1995), leading to the following lower bound on the treatment effect:

$$\text{ACE}(X \rightarrow Y) \geq \max\left\{\begin{array}{c}
p_{11.1} + p_{00.0} - 1\\
p_{11.0} + p_{00.1} - 1\\
p_{11.0} - p_{11.1} - p_{10.1} - p_{01.0} - p_{10.0}\\
p_{11.1} - p_{11.0} - p_{10.0} - p_{01.1} - p_{10.1}\\
-p_{01.1} - p_{10.1}\\
-p_{01.0} - p_{10.0}\\
p_{00.1} - p_{01.1} - p_{10.1} - p_{01.0} - p_{00.0}\\
p_{00.0} - p_{01.0} - p_{10.0} - p_{01.1} - p_{00.1}
\end{array}\right\}. \tag{8.14a}$$

Similarly, the upper bound is given by

$$\text{ACE}(X \rightarrow Y) \leq \min\left\{\begin{array}{c}
1 - p_{01.1} - p_{10.0}\\
1 - p_{01.0} - p_{10.1}\\
-p_{01.0} + p_{01.1} + p_{00.1} + p_{11.0} + p_{00.0}\\
-p_{01.1} + p_{11.1} + p_{00.1} + p_{01.0} + p_{00.0}\\
p_{11.1} + p_{00.1}\\
p_{11.0} + p_{00.0}\\
-p_{10.1} + p_{11.1} + p_{00.1} + p_{11.0} + p_{10.0}\\
-p_{10.0} + p_{11.0} + p_{00.0} + p_{11.1} + p_{10.1}
\end{array}\right\}. \tag{8.14b}$$

We may also derive bounds for (8.8) and (8.9) individually (under the same linear constraints), giving:

$$
\begin{aligned}
P(y_1 \mid do(x_0)) &\geq \max \left\{ \begin{array}{c} p_{10.0} + p_{11.0} - p_{00.1} - p_{11.1} \\ p_{10.1} \\ p_{10.0} \\ p_{01.0} + p_{10.0} - p_{00.1} - p_{01.1} \end{array} \right\}, \\
P(y_1 \mid do(x_0)) &\leq \min \left\{ \begin{array}{c} p_{01.0} + p_{10.0} + p_{10.1} + p_{11.1} \\ 1 - p_{00.1} \\ 1 - p_{00.0} \\ p_{10.0} + p_{11.0} + p_{01.1} + p_{10.1} \end{array} \right\}; \\
P(y_1 \mid do(x_1)) &\geq \max \left\{ \begin{array}{c} p_{11.0} \\ p_{11.1} \\ -p_{00.0} - p_{01.0} + p_{00.1} + p_{11.1} \\ -p_{01.0} - p_{10.0} + p_{10.1} + p_{11.1} \end{array} \right\}, \\
P(y_1 \mid do(x_1)) &\leq \min \left\{ \begin{array}{c} 1 - p_{01.1} \\ 1 - p_{01.0} \\ p_{00.0} + p_{11.0} + p_{10.1} + p_{11.1} \\ p_{10.0} + p_{11.0} + p_{00.1} + p_{11.1} \end{array} \right\}.
\end{aligned}
\tag{8.15, 8.16}
$$

These expressions give the tightest possible assumption-free[4] bounds on the quantities involved.

8.2.4 The Natural Bounds

The expression for ACE($X \rightarrow Y$) (equation (8.4)) can be bounded by two simple formulas, each made up of the first two terms in (8.14a) and (8.14b) (Robins 1989; Manski 1990; Pearl 1994a):

$$
\begin{aligned}
\text{ACE}(X \rightarrow Y) &\geq P(y_1 \mid z_1) - P(y_1 \mid z_0) - P(y_1, x_0 \mid z_1) - P(y_0, x_1 \mid z_0), \\
\text{ACE}(X \rightarrow Y) &\leq P(y_1 \mid z_1) - P(y_1 \mid z_0) + P(y_0, x_0 \mid z_1) + P(y_1, x_1 \mid z_0).
\end{aligned}
\tag{8.17}
$$

Because of their simplicity and wide range of applicability, the bounds given by (8.17) were named the *natural* bounds (Balke and Pearl 1997). The natural bounds guarantee that the causal effect of the actual treatment cannot be smaller than that of the encouragement ($P(y_1 \mid z_1) - P(y_1 \mid z_0)$) by more than the sum of two measurable quantities, $P(y_1, x_0 \mid z_1) + P(y_0, x_1 \mid z_0)$; they also guarantee that the causal effect of the treatment cannot exceed that of the encouragement by more than the sum of two other measurable

[4] "Assumption-transparent" might be a better term; we make no assumptions about factors that determine subjects' compliance, but we rely on the assumptions of (i) randomized assignment and (ii) no side effects, as displayed in the graph (e.g., Figure 8.1).

quantities, $P(y_0, x_0 \mid z_1) + P(y_1, x_1 \mid z_0)$. The width of the natural bounds, not surprisingly, is given by the rate of noncompliance: $P(x_1 \mid z_0) + P(x_0 \mid z_1)$.

The width of the sharp bounds in (8.14ab) can be substantially narrower, though. In Balke (1995) and Pearl (1995b), it is shown that – even under conditions of 50% noncompliance – these bounds may collapse to a point and thus permit consistent estimation of ACE($X \rightarrow Y$). This occurs whenever (a) the percentage of subjects complying with assignment z_0 is the same as those complying with z_1 and (b) Y and Z are perfectly correlated in at least one treatment arm x (see Table 8.1 in Section 8.5).

Although more complicated than the natural bounds of (8.17), the sharp bounds of (8.14ab) are nevertheless easy to assess once we have the frequency data in the eight cells of $P(y, x \mid z)$. It can also be shown (Balke 1995) that the natural bounds are optimal when we can safely assume that no subject is *contrarian* – in other words, that no subject would consistently choose a treatment arm contrary to the one assigned.

Note that, if the response Y is continuous, then one can associate y_1 and y_0 with the binary events $Y > t$ and $Y \leq t$ (respectively) and let t vary continuously over the range of Y. Equations (8.15) and (8.16) would then provide bounds on the entire distribution of the treatment effect $P(Y < t \mid do(x))$.

8.2.5 Effect of Treatment on the Treated

Much of the literature assumes that ACE($X \rightarrow Y$) is the parameter of interest, because ACE($X \rightarrow Y$) predicts the impact of applying the treatment uniformly (or randomly) over the population. However, if a policy maker is not interested in introducing new treatment policies but rather in deciding whether to maintain or terminate an existing program under its current incentive system, then the parameter of interest should measure the impact of the treatment *on the treated*, namely, the mean response of the treated subjects compared to the mean response of these same subjects had they not been treated (Heckman 1992). The appropriate formula for this parameter is

$$\begin{aligned} \text{ACE}^*(X \rightarrow Y) &= P(Y_{x_1} = y_1 \mid x_1) - P(Y_{x_0} = y_1 \mid x_1) \\ &= \sum_u [P(y_1 \mid x_1, u) - P(y_1 \mid x_0, u)] P(u \mid x_1), \end{aligned} \tag{8.18}$$

which is similar to (8.4) except for replacing the expectation over u with the conditional expectation given $X = x_1$.

The analysis of ACE$^*(X \rightarrow Y)$ reveals that, under conditions of *no intrusion* (i.e., $P(x_1 \mid z_0) = 0$, as in most clinical trials), ACE$^*(X \rightarrow Y)$ can be identified precisely (Bloom 1984; Angrist and Imbens 1991). The natural bounds governing ACE$^*(X \rightarrow Y)$ in the general case can be obtained by similar means, which yield

$$\begin{aligned} \text{ACE}^*(X \rightarrow Y) &\geq \frac{P(y_1 \mid z_1) - P(y_1 \mid z_0)}{P(x_1)/P(z_1)} - \frac{P(y_0, x_1 \mid z_0)}{P(x_1)}, \\ \text{ACE}^*(X \rightarrow Y) &\leq \frac{P(y_1 \mid z_1) - P(y_1 \mid z_0)}{P(x_1)/P(z_1)} + \frac{P(y_1, x_1 \mid z_0)}{P(x_1)}. \end{aligned} \tag{8.19}$$

The sharp bounds are presented in Balke (1995, p. 113). Clearly, in situations where treatment may be obtained only by those encouraged (by assignment), we have $P(x_1 \mid z_0) = 0$ and

$$\text{ACE}^*(X \rightarrow Y) = \frac{P(y_1 \mid z_1) - P(y_1 \mid z_0)}{P(x_1 \mid z_1)}. \tag{8.20}$$

Unlike $\text{ACE}(X \rightarrow Y)$, $\text{ACE}^*(X \rightarrow Y)$ is not an intrinsic property of the treatment, since it varies with the encouraging instrument. Hence, its significance lies in studies where it is desired to evaluate the efficacy of an existing program on its current participants.

8.2.6 Example: The Effect of Cholestyramine

To demonstrate by example how the bounds for $\text{ACE}(X \rightarrow Y)$ can be used to provide meaningful information about causal effects, consider the Lipid Research Clinics Coronary Primary Prevention Trial data (Program 1984). A portion (covering 337 subjects) of this data was analyzed in Efron and Feldman (1991) and is the focus of this example. Subjects were randomized into two treatment groups of roughly equal size; in one group, all subjects were prescribed cholestyramine (z_1), while subjects in the other group were prescribed a placebo (z_0). Over several years of treatment, each subject's cholesterol level was measured many times, and the average of these measurements was used as the posttreatment cholesterol level (continuous variable C_F). The compliance of each subject was determined by tracking the quantity of prescribed dosage consumed (a continuous quantity).

In order to apply the bounds of (8.17) to data from this study, the continuous data is first transformed, using thresholds, to binary variables representing treatment assignment (Z), received treatment (X), and treatment response (Y). The threshold for dosage consumption was selected as roughly the midpoint between minimum and maximum consumption; the threshold for cholesterol level reduction was set at 28 units. After this "thresholding" procedure, the data samples give rise to the following eight probabilities:[5]

$$P(y_0, x_0 \mid z_0) = 0.919, \quad P(y_0, x_0 \mid z_1) = 0.315,$$
$$P(y_0, x_1 \mid z_0) = 0.000, \quad P(y_0, x_1 \mid z_1) = 0.139,$$
$$P(y_1, x_0 \mid z_0) = 0.081, \quad P(y_1, x_0 \mid z_1) = 0.073,$$
$$P(y_1, x_1 \mid z_0) = 0.000, \quad P(y_1, x_1 \mid z_1) = 0.473.$$

These data represent a compliance rate of

$$P(x_1 \mid z_1) = 0.139 + 0.473 = 0.61,$$

[5] We make the large-sample assumption and take the sample frequencies as representing $P(y, x \mid z)$. To account for sample variability, all bounds should be supplemented with confidence intervals and significance levels, as in traditional analyses of controlled experiments. Section 8.5.1 assesses sample variability using Gibbs sampling.

a mean difference (using $P(z_1) = 0.50$) of

$$P(y_1 \mid x_1) - p(y_1 \mid x_0) = \frac{0.473}{0.473 + 0.139} - \frac{0.073 + 0.081}{1 + 0.315 + 0.073} = 0.662,$$

and an encouragement effect (intent to treat) of

$$P(y_1 \mid z_1) - P(y_1 \mid z_0) = 0.073 + 0.473 - 0.081 = 0.465.$$

According to (8.17), $\text{ACE}(X \rightarrow Y)$ can be bounded by

$$\text{ACE}(X \rightarrow Y) \geq 0.465 - 0.073 - 0.000 = 0.392,$$

$$\text{ACE}(X \rightarrow Y) \leq 0.465 + 0.315 + 0.000 = 0.780.$$

These are remarkably informative bounds: although 38.8% of the subjects deviated from their treatment protocol, the experimenter can categorically state that, when applied uniformly to the population, the treatment is guaranteed to increase by at least 39.2% the probability of reducing the level of cholesterol by 28 points or more.

The impact of treatment "on the treated" is equally revealing. Using equation (8.20), $\text{ACE}^*(X \rightarrow Y)$ can be evaluated precisely (since $P(x_1 \mid z_0) = 0$):

$$\text{ACE}^*(X \rightarrow Y) = \frac{0.465}{0.610} = 0.762.$$

In other words, those subjects who stayed in the program are much better off than they would have been if not treated: the treatment can be credited with reducing cholesterol levels by at least 28 units in 76.2% of these subjects.

8.3 COUNTERFACTUALS AND LEGAL RESPONSIBILITY

Evaluation of counterfactual probabilities could be enlightening in some legal cases in which a plaintiff claims that a defendant's actions were responsible for the plaintiff's misfortune. Improper rulings can easily be issued without an adequate treatment of counterfactuals. Consider the following hypothetical and fictitious case study, specially crafted in Balke and Pearl (1994a) to accentuate the disparity between causal effects and causal attribution.

The marketer of PeptAid (antacid medication) randomly mailed out product samples to 10% of the households in the city of Stress, California. In a follow-up study, researchers determined for each individual whether they received the PeptAid sample, whether they consumed PeptAid, and whether they developed peptic ulcers in the following month.

The causal structure for this scenario is identical to the partial compliance model given by Figure 8.1, where z_1 asserts that PeptAid was received from the marketer, x_1 asserts that PeptAid was consumed, and y_1 asserts that peptic ulceration occurred. The data showed the following distribution:

$$P(y_0, x_0 \mid z_0) = 0.32, \quad P(y_0, x_0 \mid z_1) = 0.02,$$
$$P(y_0, x_1 \mid z_0) = 0.32, \quad P(y_0, x_1 \mid z_1) = 0.17,$$
$$P(y_1, x_0 \mid z_0) = 0.04, \quad P(y_1, x_0 \mid z_1) = 0.67,$$
$$P(y_1, x_1 \mid z_0) = 0.32, \quad P(y_1, x_1 \mid z_1) = 0.14.$$

These data indicate a high correlation between those who consumed PeptAid and those who developed peptic ulcers:

$$P(y_1 \mid x_1) = 0.50, \quad P(y_1 \mid x_0) = 0.26.$$

In addition, the intent-to-treat analysis showed that those individuals who received the PeptAid samples had a 45% greater chance of developing peptic ulcers:

$$P(y_1 \mid z_1) = 0.81, \quad P(y_1 \mid z_0) = 0.36.$$

The plaintiff (Mr. Smith), having heard of the study, litigated against both the marketing firm and the PeptAid producer. The plaintiff's attorney argued against the producer, claiming that the consumption of PeptAid triggered his client's ulcer and resulting medical expenses. Likewise, the plaintiff's attorney argued against the marketer, claiming that his client would not have developed an ulcer if the marketer had not distributed the product samples.

The defense attorney, representing both the manufacturer and marketer of PeptAid, rebutted this argument, stating that the high correlation between PeptAid consumption and ulcers was attributable to a common factor, namely, pre-ulcer discomfort. Individuals with gastrointestinal discomfort would be much more likely both to use PeptAid and to develop stomach ulcers. To bolster his clients' claims, the defense attorney introduced expert analysis of the data showing that, on average, consumption of PeptAid actually decreases an individual's chances of developing ulcers by at least 15%.

Indeed, the application of (8.14ab) results in the following bounds on the average causal effect of PeptAid consumption on peptic ulceration:

$$-0.23 \leq \text{ACE}(X \rightarrow Y) \leq -0.15;$$

this proves that PeptAid is beneficial to the population as a whole.

The plaintiff's attorney, though, stressed the distinction between the average treatment effects for the entire population and for the subpopulation consisting of those individuals who, like his client, received the PeptAid sample, consumed it, and then developed ulcers. Analysis of the population data indicated that, had PeptAid not been distributed, Mr. Smith would have had at most a 7% chance of developing ulcers – regardless of any confounding factors such as pre-ulcer pain. Likewise, if Mr. Smith had not consumed PeptAid, he would have had at most a 7% chance of developing ulcers.

The damaging statistics against the marketer are obtained by evaluating the bounds on the counterfactual probability that the plaintiff would have developed a peptic ulcer if he had not received the PeptAid sample, given that he in fact received the sample PeptAid, consumed the PeptAid, and developed peptic ulcers. This probability may be written in terms of the parameters q_{13}, q_{31}, and q_{33} as

$$P(Y_{z_0} = y_1 \mid y_1, x_1, z_1) = \frac{P(r_z = 1)(q_{13} + q_{31} + q_{33})}{P(y_1, x_1, z_1)},$$

since only the combinations $\{r_x = 1, r_y = 3\}$, $\{r_x = 3, r_y = 1\}$, and $\{r_x = 3, r_y = 3\}$ satisfy the joint event $\{X = x_1, Y = y_1, Y_{z_0} = y_1\}$ (see (8.6), (8.7), and (8.11)). Therefore,

$$P(Y_{z_0} = y_1 \mid y_1, x_1, z_1) = \frac{q_{13} + q_{31} + q_{33}}{P(y_1, x_1 \mid z_1)}.$$

This expression is linear in the q parameters and may be bounded using linear programming to give

$$P(Y_{z_0} = y_1 \mid z_1, x_1, y_1) \geq \frac{1}{p_{11.1}} \max \left\{ \begin{array}{c} 0 \\ p_{11.1} - p_{00.0} \\ p_{11.0} - p_{00.1} - p_{10.1} \\ p_{10.0} - p_{01.1} - p_{10.1} \end{array} \right\},$$

$$P(Y_{z_0} = y_1 \mid z_1, x_1, y_1) \leq \frac{1}{p_{11.1}} \min \left\{ \begin{array}{c} p_{11.1} \\ p_{10.0} + p_{11.0} \\ 1 - p_{00.0} - p_{10.1} \end{array} \right\}.$$

Similarly, the damaging evidence against PeptAid's producer is obtained by evaluating the bounds on the counterfactual probability

$$P(Y_{x_0} = y_1 \mid y_1, x_1, z_1) = \frac{q_{13} + q_{33}}{p_{11.1}}.$$

If we minimize and maximize the numerator (subject to (8.13)), we obtain

$$P(Y_{x_0} = y_1 \mid y_1, x_1, z_1) \geq \frac{1}{p_{11.1}} \max \left\{ \begin{array}{c} 0 \\ p_{11.1} - p_{00.0} - p_{11.0} \\ p_{10.0} - p_{01.1} - p_{10.1} \end{array} \right\},$$

$$P(Y_{x_0} = y_1 \mid y_1, x_1, z_1) \leq \frac{1}{p_{11.1}} \min \left\{ \begin{array}{c} p_{11.1} \\ p_{10.0} + p_{11.0} \\ 1 - p_{00.0} - p_{10.1} \end{array} \right\}.$$

Substituting the observed distribution $P(y, x \mid z)$ into these formulas, the following bounds were obtained:

$$0.93 \leq P(Y_{z_0} = y_0 \mid z_1, x_1, y_1) \leq 1.00,$$

$$0.93 \leq P(Y_{x_0} = y_0 \mid z_1, x_1, y_1) \leq 1.00.$$

Thus, at least 93% of the people in the plaintiff's category would not have developed ulcers had they not been encouraged to take PeptAid (z_0) or, similarly, had they not taken PeptAid (x_0). This lends very strong support for the plaintiff's claim that he was adversely affected by the marketer and producer's actions and product.

In Chapter 9 we will continue the analysis of causal attribution in specific events, and we will establish conditions under which the probability of correct attribution can be identified from both experimental and nonexperimental data.

8.4 A TEST FOR INSTRUMENTS

As defined in Section 8.2, our model of imperfect experiment rests on two assumptions: Z is randomized, and Z has no side effect on Y. These two assumptions imply that Z is independent of U, a condition that economists call "exogeneity" and which qualifies Z as an instrumental variable (see Sections 5.4.3 and 7.4.5) relative to the relation between X and Y. For a long time, experimental verification of whether a variable Z is exogenous or instrumental has been thought to be impossible (Imbens and Angrist 1994), since the definition involves unobservable factors (or disturbances, as they are usually called) such as those represented by U.[6] The notion of exogeneity, like that of causation itself, has been viewed as a product of subjective modeling judgment, exempt from the scrutiny of nonexperimental data.

The bounds presented in (8.14ab) tell a different story. Despite its elusive nature, exogeneity can be given an empirical test. The test is not guaranteed to detect all violations of exogeneity, but it can (in certain circumstances) screen out very bad would-be instruments.

By insisting that each upper bound in (8.14b) be higher than the corresponding lower bound in (8.14a), we obtain the following testable constraints on the observed distribution:

$$\begin{aligned} &P(y_0, x_0 \mid z_0) + P(y_1, x_0 \mid z_1) \leq 1, \\ &P(y_0, x_1 \mid z_0) + P(y_1, x_1 \mid z_1) \leq 1, \\ &P(y_1, x_0 \mid z_0) + P(y_0, x_0 \mid z_1) \leq 1, \\ &P(y_1, x_1 \mid z_0) + P(y_0, x_1 \mid z_1) \leq 1. \end{aligned} \tag{8.21}$$

If any of these inequalities is violated, the investigator can deduce that at least one of the assumptions underlying our model is violated as well. If the assignment is carefully randomized, then any violation of these inequalities must be attributed to some direct influence that the assignment process has on subjects' responses (e.g., a traumatic experience). Alternatively, if direct effects of Z on Y can be eliminated – say, through an effective use of a placebo – then any observed violation of the inequalities can safely be attributed to spurious correlation between Z and U: namely, to assignment bias and hence loss of exogeneity.

The Instrumental Inequality

The inequalities in (8.21), when generalized to multivalued variables, assume the form

$$\max_x \sum_y \left[\max_z P(y, x \mid z)\right] \leq 1, \tag{8.22}$$

[6] The tests developed by economists (Wu 1973) merely compare estimates based on two or more instruments and, in case of discrepency, do not tell us objectively which estimate is incorrect.

which is called the *instrumental inequality*. A proof is given in Pearl (1995b,c). Extending the instrumental inequality to the case where Z or Y is continuous presents no special difficulty. If $f(y \mid x, z)$ is the conditional density function of Y given X and Z, then the inequality becomes

$$\int_y \max_z [f(y \mid x, z) P(x \mid z)]\, dy \leq 1 \quad \forall x. \tag{8.23}$$

However, the transition to a continuous X signals a drastic change in behavior, and it seems that the structure of Figure 8.1 induces no constraint whatsoever on the observed density (Pearl 1995c).

From (8.21) we see that the instrumental inequality is violated when the controlling instrument Z manages to produce significant changes in the response variable Y while the treatment X remains constant. Although such changes could in principle be explained by strong correlations between U, X, and Y (since X does not screen off Z from Y), the instrumental inequality sets a limit on the magnitude of the changes.

The similarity of the instrumental inequality to Bell's inequality in quantum physics (Suppes 1988; Cushing and McMullin 1989) is not accidental; both inequalities delineate a class of observed correlations that cannot be explained by hypothesizing latent common causes. The instrumental inequality can, in a sense, be viewed as a generalization of Bell's inequality for cases where direct causal connection is permitted to operate between the correlated observables, X and Y.

The instrumental inequality can be tightened appreciably if we are willing to make additional assumptions about subjects' behavior – for example, that no individual can be discouraged by the encouragement instrument or (mathematically) that, for all u, we have

$$P(x_1 \mid z_1, u) \geq P(x_1 \mid z_0, u).$$

Such an assumption amounts to having no contrarians in the population, that is, no subjects who will consistently choose treatment contrary to their assignment. Under this assumption, the inequalities in (8.21) can be tightened (Balke and Pearl 1997) to yield

$$\begin{aligned} P(y, x_1 \mid z_1) &\geq P(y, x_1 \mid z_0), \\ P(y, x_0 \mid z_0) &\geq P(y, x_0 \mid z_1) \end{aligned} \tag{8.24}$$

for all $y \in \{y_0, y_1\}$. Violation of these inequalities now means either selection bias or direct effect of Z on Y or the presence of defiant subjects.

8.5 CAUSAL INFERENCE FROM FINITE SAMPLES

8.5.1 Gibbs Sampling

This section describes a method of estimating causal effects and counterfactual probabilities from a finite sample, as presented in Chickering and Pearl (1997).[7] The method is

[7] A similar method, though lacking the graphical perspective, is presented in Imbens and Rubin (1997).

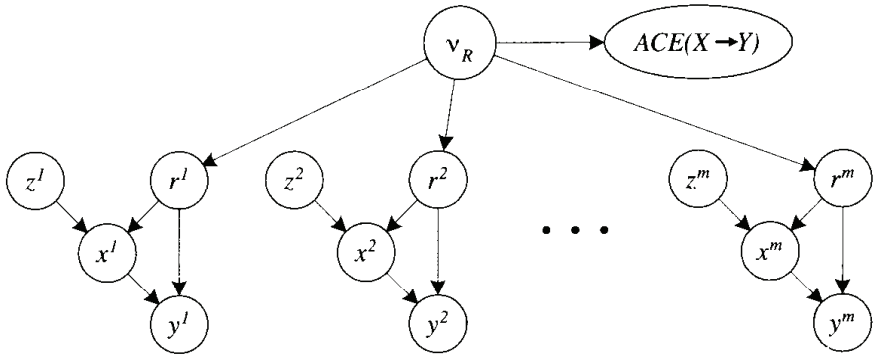


Figure 8.4 Model used to represent the independencies in $P(\{\mathcal{X}\} \cup \{\nu_R\} \cup \{\text{ACE}(X \rightarrow Y)\})$.

applicable within the Bayesian framework, according to which (i) any unknown statistical parameter can be assigned prior probability and (ii) the estimation of that parameter amounts to computing its posterior distribution, conditioned on the sampled data. In our case the parameter in question is the probability $P(r_x, r_y)$ (or $P(r)$ for short), from which we can deduce $\text{ACE}(X \rightarrow Y)$.

If we think of $P(r)$ not as probability but rather as the fraction ν_r of individuals in the population who possess response characteristics given by $R = r$, then the idea of assigning probability to such a quantity would fit the standard philosophy of Bayesian analysis; ν_r is a potentially measurable (albeit unknown) physical quantity and can therefore admit a prior probability, one that encodes our uncertainty in that quantity.

Assume there are m subjects in the experiment. We use z^i, x^i, y^i to denote the observed value of Z, X, Y, respectively, for subject i. Similarly, we use r^i to denote the (unobserved) compliance (r_x) and response (r_y) combination for subject i. We use $\mathcal{X}^i$ to denote the triple $\{z^i, x^i, y^i\}$.

Given the observed data $\mathcal{X}$ from the experiment and a prior distribution over the unknown fractions ν_r, our problem is to derive the posterior distribution for $\text{ACE}(X \rightarrow Y)$. The posterior distributions of both ν_R and $\text{ACE}(X \rightarrow Y)$ can be derived using the graphical model shown in Figure 8.4, which explicitly represents the independencies that hold in the joint (Bayesian) distribution defined over the variables $\{\mathcal{X}, \nu_R, \text{ACE}(X \rightarrow Y)\}$. The model can be understood as m realizations of the response-variable model (Figure 8.3), one for each triple in $\mathcal{X}$, connected together using the node representing the unknown fractions $\nu_R = (\nu_{r_1}, \nu_{r_2}, \ldots, \nu_{r_{16}})$. The model explicitly represents the assumption that, given the fractions ν_R, the probability of a subject belonging to any of the 16 compliance–response subpopulations does not depend on the compliance and response behavior of the other subjects in the experiment. From (8.10), $\text{ACE}(X \rightarrow Y)$ is a deterministic function of ν_R and consequently $\text{ACE}(X \rightarrow Y)$ is independent of all other variables in the domain once these fractions are known.

In principle, then, estimating $\text{ACE}(X \rightarrow Y)$ reduces to the standard inference task of computing the posterior probability for a variable in a fully specified Bayesian network. (The graphical techniques for this inferential computation are briefly summarized in Section 1.2.4.) In many cases, the independencies embodied in the graph can be exploited to render the inference task efficient. Unfortunately, because the r^i are never observed, deriving the posterior distribution for $\text{ACE}(X \rightarrow Y)$ is not tractable in our model, even with the given independencies. To obtain an estimate of the posterior distribution of $\text{ACE}(X \rightarrow Y)$, an approximation technique known as Gibbs sampling can be

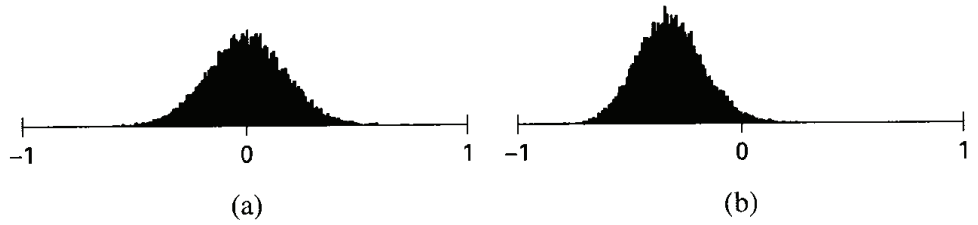


Figure 8.5 (a) The prior distribution of ACE($X \rightarrow Y$) induced by flat priors over the parameters v_{CR}. (b) The distribution for ACE($X \rightarrow Y$) induced by skewed priors over the parameters.

used (Robert and Casella 1999). A graphical version of this technique, called "stochastic simulation," is described in Pearl (1988b, p. 210); the details (as applied to the graph of Figure 8.4) are discussed in Chickering and Pearl (1997). Here we present typical results, in the form of histograms, that demonstrate the general applicability of this technique to problems of causal inference.

8.5.2 The Effects of Sample Size and Prior Distribution

The method takes as input (1) the observed data $\mathcal{X}$, expressed as the number of cases observed for each of the 8 possible realizations of $\{z, x, y\}$, and (2) a Dirichlet prior over the unknown fractions v_R, expressed in terms of 16 parameters. The system outputs the posterior distribution of ACE($X \rightarrow Y$), expressed in a histogram.

To show the effect of the prior distribution on the output, we present all the results using two different priors. The first is a flat (uniform) distribution over the 16-vector v_R that is commonly used to express ignorance about the domain. The second prior is skewed to represent a strong dependency between the compliance and response characteristics of the subjects. Figure 8.5 shows the distribution of ACE($X \rightarrow Y$) induced by these two prior distributions (in the absence of any data). We see that the skewed prior of Figure 8.5(b) assigns almost all the weight to negative values of ACE($X \rightarrow Y$).

To illustrate how increasing sample size washes away the effect of the prior distribution, we apply the method to simulated data drawn from a distribution $P(x, y \mid z)$ for which ACE is known to be identified. Such a distribution is shown Table 8.1. For this distribution, the resulting upper and lower bounds of (8.14ab) collapse to a single point: ACE($X \rightarrow Y$) = 0.55.

Figure 8.6 shows the output of the Gibbs sampler when applied to data sets of various sizes drawn from the distribution shown in Table 8.1, using both the flat and the skewed prior. As expected, as the number of cases increases, the posterior distributions become increasingly concentrated near the value 0.55. In general, because the skewed prior for ACE($X \rightarrow Y$) is concentrated further from 0.55 than the uniform prior, more cases are needed before the posterior distribution converges to the value 0.55.

8.5.3 Causal Effects from Clinical Data with Imperfect Compliance

In this section we analyze two clinical data sets obtained under conditions of imperfect compliance. Consider first the Lipid Research Clinics Coronary Primary Prevention data described in Section 8.2.6. The resulting data set (after thresholding) is shown in Table 8.2. Using the large-sample assumption, (8.14ab) gives the bounds $0.39 \leq \text{ACE}(X \rightarrow Y) \leq 0.78$.

Table 8.1. ***Distribution Resulting in an Identifiable*** **ACE($X \rightarrow Y$)**

z	x	y	$P(x, y, z)$
0	0	0	0.275
0	0	1	0.0
0	1	0	0.225
0	1	1	0.0
1	0	0	0.225
1	0	1	0.0
1	1	0	0.0
1	1	1	0.275

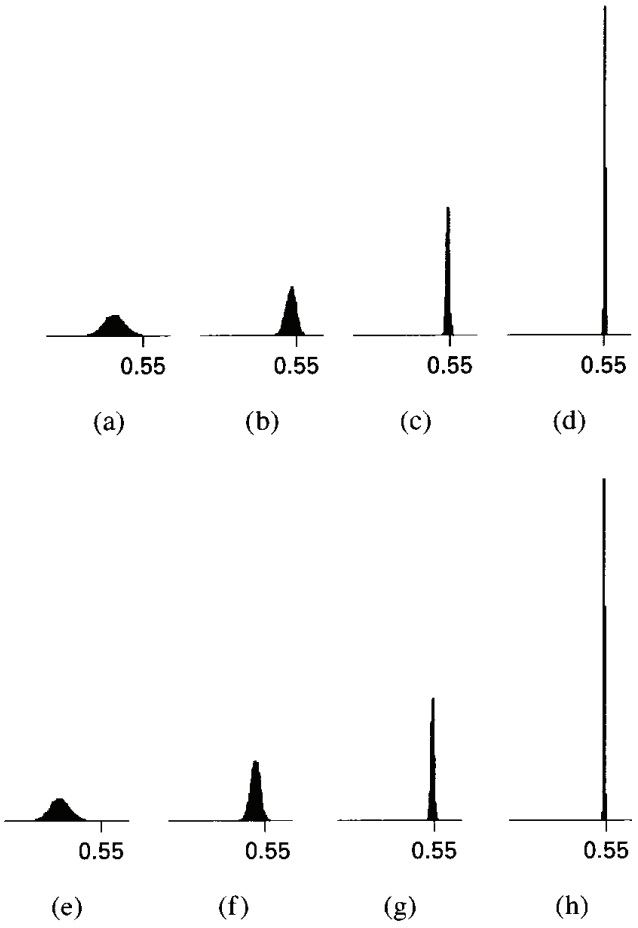


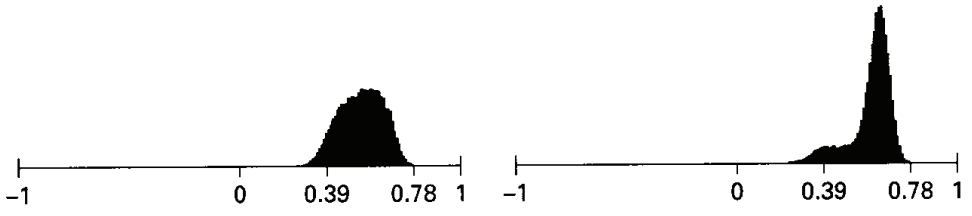
Figure 8.6 Output histograms for identified treatment effect using two priors. (a), (b), (c), and (d) show the posteriors for ACE($X \rightarrow Y$) using the flat prior and data sets that consisted of 10, 100, 1,000 and 10,000 subjects, respectively; (e), (f), (g), and (h) show the posteriors for ACE($X \rightarrow Y$) using the skewed prior with the same respective data sets. (Horizontal lines span the interval $(-1, +1)$.)

Figure 8.7 shows posterior densities for ACE($X \rightarrow Y$), based on these data. Rather remarkably, even with only 337 cases in the data set, both posterior distributions are highly concentrated within the large-sample bounds of 0.39 and 0.78.

As a second example, we consider an experiment described by Sommer et al. (1986) that was designed to determine the impact of vitamin A supplementation on childhood mortality. In the study, 450 villages in northern Sumatra were randomly assigned to participate in a vitamin A supplementation scheme or serve as a control group for one year. Children in the treatment group received two large doses of vitamin A (x_1), while those

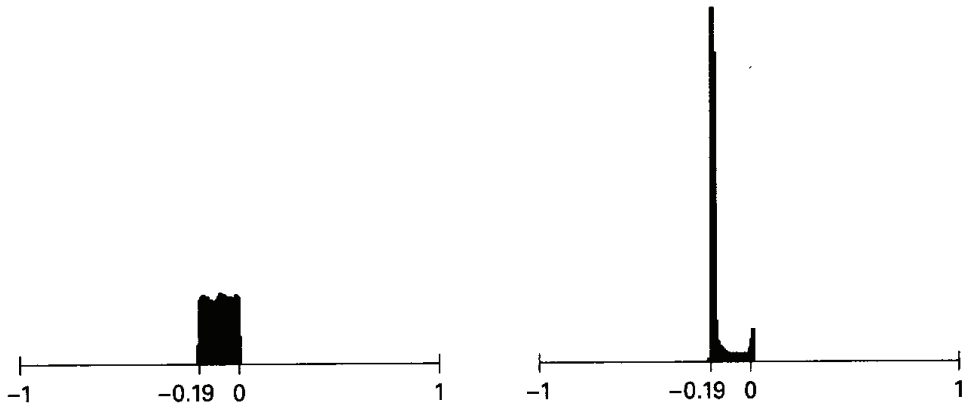
Table 8.2. ***Observed Data for the Lipid Study and the Vitamin A Study***

z	x	y	Lipid Study Observations	Vitamin A Study Observations
0	0	0	158	74
0	0	1	14	11,514
0	1	0	0	0
0	1	1	0	0
1	0	0	52	34
1	0	1	12	2,385
1	1	0	23	12
1	1	1	78	9,663



(a) (b)

Figure 8.7 Output histograms for the Lipid data: (a) using flat priors; (b) using skewed priors.



(a) (b)

Figure 8.8 Output histograms for the vitamin A data: (a) using flat priors; (b) using skewed priors.

in the control group received no treatment (x_0). After the year had expired, the number of deaths y_0 were counted for both groups. The results of this study are also shown in Table 8.2.

Under the large-sample assumption, the inequalities of (8.14ab) yield the bounds $-0.19 \leq \text{ACE}(X \rightarrow Y) \leq 0.01$. Figure 8.8 shows posterior densities for $\text{ACE}(X \rightarrow Y)$, given the data, for two priors. It is interesting to note that, for this study, the choice of the prior distribution has a significant effect on the posterior. This suggests that if the clinician is not very confident in the prior then a sensitivity analysis should be performed.

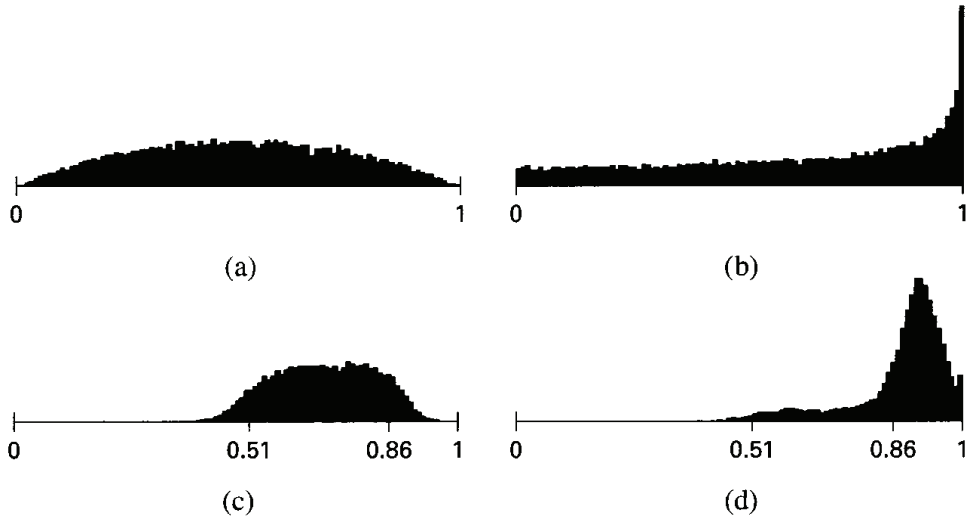


Figure 8.9 Prior ((a) and (b)) and posterior ((c) and (d)) distributions for a subpopulation $f(v_R)$ specified by the counterfactual query: "Would Joe have improved had he taken the drug, given that he did not improve without it?" Part (a) corresponds to the flat prior, (b) to the skewed prior.

In such cases, the asymptotic bounds are more informative than the Bayesian estimates, and the major role of the Gibbs sampler would be to give an indication of the sharpness of the boundaries around those bounds.

8.5.4 Bayesian Estimate of Single-Event Causation

In addition to assessing causal effects, the Bayesian method just described is also capable (with only minor modification) of answering a variety of counterfactual queries concerning individuals with specific characteristics. Queries of this type were analyzed and bounded in Section 8.3 under the large sample assumption. In this section, we demonstrate a Bayesian analysis of the following query. What is the probability that Joe would have had an improved cholesterol reading had he taken cholestyramine, given that: (1) Joe was in the control group of the Lipid study; (2) Joe took the placebo as prescribed; and (3) Joe's cholesterol level did not improve.

This query can be answered by running the Gibbs sampler on a model identical to that shown in Figure 8.4, except that the function ACE($X \rightarrow Y$) (equation (8.10)) is replaced by another function of v_R, one that represents our query. If Joe was in the control group and took the placebo, that means he is either a complier or a never-taker. Furthermore, because Joe's cholesterol level did not improve, Joe's response behavior is either never-recover or helped. Consequently, he must be a member of one of the following four compliance-response populations: $\{(r_x = 0, r_y = 1), (r_x = 0, r_y = 2), (r_x = 1, r_y = 1), (r_x = 1, r_y = 2)\}$. Joe would have improved had he taken cholestyramine if his response behavior is either helped ($r_y = 1$) or always-recover ($r_y = 3$). It follows that the query of interest is captured by the function

$$f(v_R) = \frac{v_{01} + v_{11}}{v_{01} + v_{02} + v_{11} + v_{12}}.$$

Figures 8.9(a) and (b) show the prior distribution of $f(v_R)$ that follows from the flat prior and the skewed prior, respectively. Figures 8.9(c) and (d) show the posterior

distribution $P(f(\nu_R \mid \mathcal{X}))$ obtained from the Lipid data when using the flat prior and the skewed prior, respectively. For reference, the bounds computed under the large-sample assumption are $0.51 \leq f(\nu_R \mid \mathcal{X}) \leq 0.86$.

Thus, despite 39% noncompliance in the treatment group and despite having just 337 subjects, the study strongly supports the conclusion that – given his specific history – Joe would have been better off taking the drug. Moreover, the conclusion holds for both priors.

8.6 CONCLUSION

This chapter has developed causal-analytic techniques for managing one of the major problems in clinical experiments: the assessment of treatment efficacy in the face of imperfect compliance. Estimates based solely on intent-to-treat analysis – as well as those based on instrumental variable formulas – can be misleading in that they may lie entirely outside the theoretical bounds. The formulas established in this chapter provide instrument-independent guarantees for policy analysis and, in addition, should enable analysts to determine the extent to which efforts to enforce compliance may increase overall treatment effectiveness.

The importance of indirect experimentation is not confined to studies involving human subjects. Experimental conditions equivalent to those of imperfect compliance occur whenever the variable whose causal effect we seek to assess cannot be manipulated directly yet could be partially influenced by indirect means. Typical applications involve the diagnosis of ongoing processes for which the source of malfunctioning behavior must be identified using indirect means because direct manipulation of suspected sources is either physically impossible or prohibitively expensive. An example of the latter would be interrupting the normal operation of a production line so as to achieve direct control over a physical parameter that is suspected of malfunctioning. Partial control over that parameter, in the form of indirect influence, would be much more convenient and would allow the production to continue.

Methodologically, the message of this chapter has been to demonstrate that, even in cases where causal quantities are not identifiable, reasonable assumptions about the salient relationships in the domain can be harnessed to yield useful quantitative information about the causal forces that operate in the domain. Once such assumptions are articulated in graphical form, they can easily be submitted to algebraic methods that yield the desired bounds or, alternatively, invite Gibbs sampling technique to facilitate Bayesian estimation of the causal quantities of interest.

Acknowledgment

The main results in this chapter are based on collaborative works with Alex Balke and David Chickering. The encouragements of James Robins and Miles Hollander helped us endure a long and agonizing publication process. These investigations have benefited from discussions with Phil Dawid, James Heckman, Guido Imbens, Steve Jacobsen, Steffen Lauritzen, and Charles Manski.

CHAPTER NINE

Probability of Causation: Interpretation and Identification

Come and let us cast lots to find out
who is to blame for this ordeal.
Jonah 1:7

Preface

Assessing the likelihood that one event *was the cause* of another guides much of what we understand about (and how we act in) the world. For example, according to common judicial standard, judgment in favor of the plaintiff should be made if and only if it is "more probable than not" that the defendant's action was the *cause* for the plaintiff's damage (or death). But causation has two faces, *necessary* and *sufficient;* which of the two have lawmakers meant us to consider? And how are we to evaluate their probabilities?

This chapter provides formal semantics for the probability that event x was a *necessary* or *sufficient* cause (or both) of another event y. We then explicate conditions under which the probability of necessary (or sufficient) causation can be learned from statistical data, and we show how data from both experimental and nonexperimental studies can be combined to yield information that neither study alone can provide.

9.1 INTRODUCTION

The standard counterfactual definition of causation (i.e., that E would not have occurred were it not for C) captures the notion of "necessary cause." Competing notions such as "sufficient cause" and "necessary and sufficient cause" are of interest in a number of applications, and these, too, can be given concise mathematical definitions in structural model semantics (Section 7.1). Although the distinction between necessary and sufficient causes goes back to J. S. Mill (1843), it has received semiformal explications only in the 1960s – via conditional probabilities (Good 1961) and logical implications (Mackie 1965). These explications suffer from basic semantical difficulties,[1] and they do not yield effective procedures for computing probabilities of causes as those provided by the structural account (Sections 7.1.3 and 8.3).

[1] The limitations of the probabilistic account are discussed in Section 7.5; those of the logical account will be discussed in Section 10.1.4.

In this chapter we explore the counterfactual interpretation of necessary and sufficient causes, illustrate the application of structural model semantics to the problem of identifying probabilities of causes, and present, by way of examples, new ways of estimating probabilities of causes from statistical data. Additionally, we argue that necessity and sufficiency are two distinct facets of causation and that both facets should take part in the construction of causal explanations.

Our results have applications in epidemiology, legal reasoning, artificial intelligence (AI), and psychology. Epidemiologists have long been concerned with estimating the probability that a certain case of disease is "attributable" to a particular exposure, which is normally interpreted counterfactually as "the probability that disease would not have occurred in the absence of exposure, given that disease and exposure did in fact occur." This counterfactual notion, which Robins and Greenland (1989) called the "probability of causation," measures how *necessary* the cause is for the production of the effect.[2] It is used frequently in lawsuits, where legal responsibility is at the center of contention (see e.g. Section 8.3). We shall denote this notion by the symbol PN, an acronym for probability of necessity.

A parallel notion of causation, capturing how *sufficient* a cause is for the production of the effect, finds applications in policy analysis, AI, and psychology. A policy maker may well be interested in the dangers that a certain exposure may present to the healthy population (Khoury et al. 1989). Counterfactually, this notion can be expressed as the "probability that a healthy unexposed individual would have contracted the disease had he or she been exposed," and it will be denoted by PS (probability of sufficiency). A natural extension would be to inquire for the probability of necessary and sufficient causation (PNS) – that is, how likely a given individual is to be affected both ways.

As the examples illustrate, PS assesses the presence of an active causal process capable of producing the effect, while PN emphasizes the absence of alternative processes – not involving the cause in question – that is still capable of expaining the effect. In legal settings, where the occurrence of the cause (x) and the effect (y) are fairly well established, PN is the measure that draws most attention, and the plaintiff must prove that y would not have occurred *but for* x (Robertson 1997). Still, lack of sufficiency may weaken arguments based on PN (Good 1993; Michie in press).

It is known that PN is in general nonidentifiable, that is, it cannot be estimated from frequency data involving exposures and disease cases (Greenland and Robins 1988; Robins and Greenland 1989). The identification is hindered by two factors.

1. *Confounding* – Exposed and unexposed subjects may differ in several relevant factors or, more generally, the cause and the effect may both be influenced by a third factor. In this case we say that the cause is not *exogenous* relative to the effect (see Section 7.4.5).

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[2] Greenland and Robins (1988) further distinguish between two ways of measuring probabilities of causation: the first (called "excess fraction") concerns only *whether* the effect (e.g. disease) occurs by a particular time; the second (called "etiological fraction") requires consideration of *when* the effect occurs. We will confine our discussion here to events occurring within a specified time period, or to "all or none" outcomes (such as birth defects) for which the probability of occurrence but not the time to occurrence is important.

2. *Sensitivity to the generative process* – Even in the absence of confounding, probabilities of certain counterfactual relationships cannot be identified from frequency information unless we specify the functional relationships that connect causes and effects. Functional specification is needed whenever the facts at hand (e.g. disease) might be affected by the counterfactual antecedent (e.g. exposure) (see the examples in Sections 1.4, 7.5, and 8.3).

Although PN is not identifiable in the general case, several formulas have nevertheless been proposed to estimate attributions of various kinds in terms of frequencies obtained in epidemiological studies (Breslow and Day 1980; Hennekens and Buring 1987; Cole 1997). Naturally, any such formula must be predicated upon certain implicit assumptions about the data-generating process. Section 9.2 explicates some of those assumptions and explores conditions under which they can be relaxed.[3] It offers new formulas for PN and PS in cases where causes are confounded (with outcomes) but their effects can nevertheless be estimated (e.g., from clinical trials or from auxiliary measurements). Section 9.3 exemplifies the use of these formulas in legal and epidemiological settings, while Section 9.4 provides a general condition for the identifiability of PN and PS when functional relationships are only partially known.

The distinction between necessary and sufficient causes has important implications in AI, especially in systems that generate verbal explanations automatically (see Section 7.2.3). As can be seen from the epidemiological examples, necessary causation is a concept tailored to a specific event under consideration (singular causation), whereas sufficient causation is based on the general tendency of certain event *types* to produce other event types. Adequate explanations should respect both aspects. If we base explanations solely on generic tendencies (i.e., sufficient causation) then we lose important specific information. For instance, aiming a gun at and shooting a person from 1,000 meters away will not qualify as an explanation for that person's death, owing to the very low tendency of shots fired from such long distances to hit their marks. This stands contrary to common sense, for when the shot does hit its mark on that singular day, regardless of the reason, the shooter is an obvious culprit for the consequence. If, on the other hand, we base explanations solely on singular-event considerations (i.e., necessary causation), then various background factors that are normally present in the world would awkwardly qualify as explanations. For example, the presence of oxygen in the room would qualify as an explanation for the fire that broke out, simply because the fire would not have occurred were it not for the oxygen. That we judge the match struck, not the oxygen, to be the actual cause of the fire indicates that we go beyond the singular event at hand (where each factor alone is both necessary and sufficient) and consider situations of the same general type – where oxygen alone is obviously insufficient to start a fire. Clearly, some balance must be struck between the necessary and the sufficient components of causal explanation, and the present chapter illuminates this balance by formally explicating the basic relationships between these two components.

[3] A set of sufficient conditions for the identification of etiological fractions are given in Robins and Greenland (1989). These conditions, however, are too restrictive for the identification of PN, which is oblivious to the temporal aspects associated with etiological fractions.

9.2 NECESSARY AND SUFFICIENT CAUSES: CONDITIONS OF IDENTIFICATION

9.2.1 Definitions, Notation, and Basic Relationships

Using the counterfactual notation and the structural model semantics introduced in Section 7.1, we give the following definitions for the three aspects of causation discussed in the introduction.

Definition 9.2.1 (Probability of Necessity, PN)
Let X and Y be two binary variables in a causal model M. Let x and y stand (respectively) for the propositions X = true and Y = true, and let x′ and y′ denote their complements. The probability of necessity *is defined as the expression*

$$\begin{aligned} \text{PN} &\triangleq P(Y_{x'} = \textit{false} \mid X = \textit{true},\ Y = \textit{true}) \\ &\triangleq P(y'_{x'} \mid x, y). \end{aligned} \tag{9.1}$$

In other words, PN stands for the probability of $y'_{x'}$ (that event y would not have occurred in the absence of event x), given that x and y did in fact occur.

Observe the slight change in notation relative to that used in Section 7.1. Lowercase letters (e.g., x and y) denoted values of variables in Section 7.1 but now stand for propositions (or events). Note also the abbreviations y_x for Y_x = true and y'_x for Y_x = false.[4] Readers accustomed to writing "$A > B$" for the counterfactual "B if it were A" can translate (9.1) to read PN $\triangleq P(x' > y' \mid x, y)$.[5]

Definition 9.2.2 (Probability of Sufficiency, PS)

$$\text{PS} \triangleq P(y_x \mid y', x'). \tag{9.2}$$

PS measures the capacity of x to *produce* y and, since "production" implies a transition from the absence to the presence of x and y, we condition the probability $P(y_x)$ on situations where x and y are both absent. Thus, mirroring the necessity of x (as measured by PN), PS gives the probability that setting x would produce y in a situation where x and y are in fact absent.

Definition 9.2.3 (Probability of Necessity and Sufficiency, PNS)

$$\text{PNS} \triangleq P(y_x, y'_{x'}). \tag{9.3}$$

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[4] These were proposed by Peyman Meshkat (in class homework) and substantially simplify the derivations.

[5] Definition 9.2.1 generalizes naturally to cases where X and Y are multivalued, say $x \in \{x_1, x_2, \ldots, x_k\}$ and $y \in \{y_1, y_2, \ldots, y_l\}$. We say that event $C = \bigvee_{i \in I}(X = x_i)$ is "counterfactually necessary" for $E = \bigvee_{j \in J}(Y = y_j)$, written $\bar{C} > \bar{E}$, if Y_x falls outside E whenever $X = x$ is outside C. Accordingly, the probability that C was a necessary cause of E is defined as PN $\triangleq P(\bar{C} > \bar{E} \mid C, E)$. For simplicity, however, we will pursue the analysis in the binary case.

PNS stands for the probability that y would respond to x both ways, and therefore measures both the sufficiency and necessity of x to produce y.

Associated with these three basic notions are other counterfactual quantities that have attracted either practical or conceptual interest. We will mention two such quantities but will not dwell on their analyses, since these can be easily inferred from our treatment of PN, PS, and PNS.

Definition 9.2.4 (Probability of Disablement, PD)

$$\text{PD} \triangleq P(y'_{x'} \mid y). \tag{9.4}$$

PD measures the probability that y would have been prevented if it were not for x; it is therefore of interest to policy makers who wish to assess the social effectiveness of various prevention programs (Fleiss 1981, pp. 75–6).

Definition 9.2.5 (Probability of Enablement, PE)

$$\text{PE} \triangleq P(y_x \mid y').$$

PE is similar to PS, save for the fact that we do not condition on x'. It is applicable, for example, when we wish to assess the danger of an exposure on the entire population of healthy individuals, including those who were already exposed.

Although none of these quantities is sufficient for determining the others, they are not entirely independent, as shown in the following lemma.

Lemma 9.2.6

The probabilities of causation (PNS, PN, *and* PS) *satisfy the following relationship:*

$$\text{PNS} = P(x, y)\text{PN} + P(x', y')\text{PS}. \tag{9.5}$$

Proof

The consistency conditions of (7.19), $X = x \implies Y_x = Y$, translate in our notation into

$$x \implies (y_x = y), \qquad x' \implies (y_{x'} = y).$$

Hence we can write

$$\begin{aligned} y_x \wedge y'_{x'} &= (y_x \wedge y'_{x'}) \wedge (x \vee x') \\ &= (y \wedge x \wedge y'_{x'}) \vee (y_x \wedge y' \wedge x'). \end{aligned}$$

Taking probabilities on both sides and using the disjointness of x and x', we obtain

$$\begin{aligned} P(y_x, y'_{x'}) &= P(y'_{x'}, x, y) + P(y_x, x', y') \\ &= P(y'_{x'} \mid x, y)P(x, y) + P(y_x \mid x', y')P(x', y'), \end{aligned}$$

which proves Lemma 9.2.6. □

To put into focus the aspects of causation captured by PN and PS, it is helpful to characterize those changes in the causal model that would leave each of the two measures invariant. The next two lemmas show that PN is insensitive to the introduction of potential inhibitors of y, while PS is insensitive to the introduction of alternative causes of y.

Lemma 9.2.7

Let $\mathrm{PN}(x, y)$ *stand for the probability that* x *is a necessary cause of* y. *Let* $z = y \wedge q$ *be a consequence of* y *that is potentially inhibited by* q'. *If* $q \perp\!\!\!\perp \{X, Y, Y_{x'}\}$, *then*

$$\mathrm{PN}(x, z) \triangleq P(z'_{x'} \mid x, z) = P(y'_{x'} \mid x, y) \triangleq \mathrm{PN}(x, y).$$

Cascading the process $Y_x(u)$ with the link $z = y \wedge q$ amounts to inhibiting the output of the process with probability $P(q')$. Lemma 9.2.7 asserts that, if q is randomized, we can add such a link without affecting PN. The reason is clear; conditioning on x and z implies that, in the scenario considered, the added link was not inhibited by q'.

Proof of Lemma 9.2.7

We have

$$\mathrm{PN}(x, z) = P(z'_{x'} \mid x, z) = \frac{P(z'_{x'}, x, z)}{P(x, z)}$$

$$= \frac{P(z'_{x'}, x, z \mid q)P(q) + P(z'_{x'}, x, z \mid q')P(q')}{P(z, x, q) + P(z, x, q')}. \tag{9.6}$$

Using $z = y \wedge q$, it follows that

$$q \implies (z = y), \quad q \implies (z'_{x'} = y'_{x'}), \quad \text{and} \quad q' \implies z';$$

therefore,

$$\mathrm{PN}(x, z) = \frac{P(y'_{x'}, x, y \mid q)P(q) + 0}{P(y, x, q) + 0}$$

$$= \frac{P(y'_{x'}, x, y)}{P(y, x)} = P(y'_{x'} \mid x, y) = \mathrm{PN}(x, y). \qquad \square$$

Lemma 9.2.8

Let $\mathrm{PS}(x, y)$ *stand for the probability that* x *is a sufficient cause of* y, *and let* $z = y \vee r$ *be a consequence of* y *that may also be triggered by* r. *If* $r \perp\!\!\!\perp \{X, Y, Y_x\}$, *then*

$$\mathrm{PS}(x, z) \triangleq P(z_x \mid x', z') = P(y_x \mid x', y') \triangleq \mathrm{PS}(x, y).$$

Lemma 9.2.8 asserts that we can add alternative (independent) causes (r) without affecting PS. The reason again is clear; conditioning on the event x' and y' implies that the added causes (r) were not active. The proof of Lemma 9.2.8 is similar to that of Lemma 9.2.7.

Since all the causal measures defined so far invoke conditionalization on y, and since y is presumed to be affected by x, we know that none of these quantities is identifiable from knowledge of the causal diagram $G(M)$ and the data $P(v)$ alone, even under conditions of no-confounding. Moreover, none of these quantities determines the others in the general case. However, simple interrelationships and useful bounds can be derived for these quantities under the assumption of no-confounding, an assumption that we call *exogeneity*.

9.2.2 Bounds and Basic Relationships under Exogeneity

Definition 9.2.9 (Exogeneity)
A variable X is said to be exogenous *relative to Y in model M if and only if*

$$\{Y_x, Y_{x'}\} \perp\!\!\!\perp X. \tag{9.7}$$

In other words, the way Y would potentially respond to conditions x or x' is independent of the actual value of X.

Equation (9.7) is a strong version of those used in Chapter 5 (equation (5.30)) and in Chapter 6 (equation (6.10)) in that it involves the joint variable $\{Y_x, Y_{x'}\}$. This definition was named "strong ignorability" in Rosenbaum and Rubin (1983), and it coincides with the classical error-based criterion for exogeneity (Christ 1966, p. 156; see Section 7.4.5) and with the back-door criterion of Definition 3.3.1. The weaker definition of (5.30) is sufficient for all the results in this chapter except equations (9.11), (9.12), and (9.19), for which strong exogeneity (9.7) is needed.

The importance of exogeneity lies in permitting the identification of $\{P(y_x), P(y_{x'})\}$, the *causal effect* of X on Y, since (using $x \implies (y_x = y)$)

$$P(y_x) = P(y_x \mid x) = P(y \mid x), \tag{9.8}$$

with similar reduction for $P(y_{x'})$.

Theorem 9.2.10
Under condition of exogeneity, PNS *is bounded as follows:*

$$\max[0, P(y \mid x) - P(y \mid x')] \leq \text{PNS} \leq \min[P(y \mid x), P(y' \mid x')]. \tag{9.9}$$

Both bounds are sharp in the sense that, for every joint distribution $P(x, y)$, there exists a model $y = f(x, u)$, with u independent of x, that realizes any value of PNS *permitted by the bounds.*

Proof
For any two events A and B, we have the sharp bounds

$$\max[0, P(A) + P(B) - 1] \leq P(A, B) \leq \min[P(A), P(B)]. \tag{9.10}$$

Equation (9.9) follows from (9.3) and (9.10) using $A = y_x$, $B = y'_{x'}$, $P(y_x) = P(y \mid x)$, and $P(y'_{x'}) = P(y' \mid x')$. □

Clearly, if exogeneity cannot be ascertained, then PNS is bound by inequalities similar to those of (9.9), with $P(y_x)$ and $P(y'_{x'})$ replacing $P(y \mid x)$ and $P(y' \mid x')$, respectively.

Theorem 9.2.11

Under condition of exogeneity, the probabilities PN, PS, *and* PNS *are related to each other as follows:*

$$\text{PN} = \frac{\text{PNS}}{P(y \mid x)}, \tag{9.11}$$

$$\text{PS} = \frac{\text{PNS}}{P(y' \mid x')}. \tag{9.12}$$

Thus, the bounds for PNS *in* (9.9) *provide corresponding bounds for* PN *and* PS.

The resulting bounds for PN,

$$\frac{\max[0,\, P(y \mid x) - P(y \mid x')]}{P(y \mid x)} \leq \text{PN} \leq \frac{\min[P(y \mid x),\, P(y' \mid x')]}{P(y \mid x)}, \tag{9.13}$$

place limits on our ability to identify PN in experimental studies, where exogeneity holds.

Corollary 9.2.12

If x and y occur in an experimental study and if $P(y_x)$ and $P(y_{x'})$ are the causal effects measured in that study, then for any point p in the range

$$\frac{\max[0,\, P(y_x) - P(y_{x'})]}{P(y_x)} \leq p \leq \frac{\min[P(y_x),\, P(y'_{x'})]}{P(y_x)} \tag{9.14}$$

there exists a causal model M that agrees with $P(y_x)$ and $P(y_{x'})$ and for which PN $= p$.

Other bounds can be established for nonexperimental events if we have data from both experimental and observational studies (as in Section 9.3.4). The nonzero widths of these bounds imply that probabilities of causation cannot be defined uniquely in stochastic (non-Laplacian) models where, for each u, $Y_x(u)$ is specified in probability $P(Y_x(u) = y)$ instead of a single number.[6]

Proof of Theorem 9.2.11

Using $x \implies (y_x = y)$, we can write $x \wedge y_x = x \wedge y$ and so obtain

[6] Robins and Greenland (1989), who used a stochastic model of $Y_x(u)$, defined the probability of causation as

$$\text{PN}(u) = [P(y \mid x, u) - P(y \mid x', u)]/P(y \mid x, u)$$

instead of the counterfactual definition in (9.1).

$$\text{PN} = P(y'_{x'} \mid x, y) = \frac{P(y'_{x'}, x, y)}{P(x, y)} \tag{9.15}$$

$$= \frac{P(y'_{x'}, x, y_x)}{P(x, y)} \tag{9.16}$$

$$= \frac{P(y'_{x'}, y_x)P(x)}{P(x, y)} \tag{9.17}$$

$$= \frac{\text{PNS}}{P(y \mid x)}, \tag{9.18}$$

which establishes (9.11). Equation (9.12) follows by identical steps. □

For completeness, we write the relationship between PNS and the probabilities of enablement and disablement:

$$\text{PD} = \frac{P(x)\text{PNS}}{P(y)}, \qquad \text{PE} = \frac{P(x')\text{PNS}}{P(y')}. \tag{9.19}$$

9.2.3 Identifiability under Monotonicity and Exogeneity

Before attacking the general problem of identifying the counterfactual quantities in (9.1)–(9.3), it is instructive to treat a special condition, called *monotonicity,* which is often assumed in practice and which renders these quantities identifiable. The resulting probabilistic expressions will be recognized as familiar measures of causation that often appear in the literature.

Definition 9.2.13 (Monotonicity)
A variable Y is said to be monotonic *relative to variable X in a causal model M if and only if the function $Y_x(u)$ is monotonic in x for all u. Equivalently, Y is monotonic relative to X if and only if*

$$y'_x \wedge y_{x'} = \textit{false}. \tag{9.20}$$

Monotonicity expresses the assumption that a change from X = false to X = true cannot, under any circumstance, make Y change from true to false.[7] In epidemiology, this assumption is often expressed as "no prevention," that is, no individual in the population can be helped by exposure to the risk factor.

Theorem 9.2.14 (Identifiability under Exogeneity and Monotonicity)
If X is exogenous and Y is monotonic relative to X, then the probabilities PN, PS, *and* PNS *are all identifiable and are given by* (9.11)–(9.12), *with*

$$\text{PNS} = P(y \mid x) - P(y \mid x'). \tag{9.21}$$

[7] Our analysis remains invariant to complementing x or y (or both); hence, the general condition of monotonicity should read: Either $y'_x \wedge y_{x'}$ = false or $y'_{x'} \wedge y_x$ = false. For simplicity, however, we will adhere to the definition in (9.20). Note: monotonicity implies that (5.30) entails (9.7).

The r.h.s. of (9.21) is called "risk difference" in epidemiology, and is also misnomered "attributable risk" (Hennekens and Buring 1987, p. 87).

From (9.11) we see that the probability of necessity is identifiable and given by the *excess risk ratio*

$$\text{PN} = \frac{P(y \mid x) - P(y \mid x')}{P(y \mid x)}, \tag{9.22}$$

often misnomered as the "attributable fraction" (Schlesselman 1982), "attributable-rate percent" (Hennekens and Buring 1987, p. 88), or "attributable proportion" (Cole 1997). Taken literally, the ratio presented in (9.22) has nothing to do with attribution, since it is made up of statistical terms and not of causal or counterfactual relationships. However, the assumptions of exogeneity and monotonicity together enable us to translate the notion of attribution embedded in the definition of PN (equation (9.1)) into a ratio of purely statistical associations. This suggests that exogeneity and monotonicity were tacitly assumed by the many authors who proposed or derived (9.22) as a measure for the "fraction of exposed cases that are attributable to the exposure."

Robins and Greenland (1989) analyzed the identification of PN under the assumption of stochastic monotonicity (i.e., $P(Y_x(u) = y) > P(Y_{x'}(u) = y)$) and showed that this assumption is too weak to permit such identification; in fact, it yields the same bounds as in (9.13). This indicates that stochastic monotonicity imposes no constraints whatsoever on the functional mechanisms that mediate between X and Y.

The expression for PS (equation (9.12)) is likewise quite revealing,

$$\text{PS} = \frac{P(y \mid x) - P(y \mid x')}{1 - P(y \mid x')}, \tag{9.23}$$

since it coincides with what epidemiologists call the "relative difference" (Shep 1958), which is used to measure the *susceptibility* of a population to a risk factor x. Susceptibility is defined as the proportion of persons who possess "an underlying factor sufficient to make a person contract a disease following exposure" (Khoury et al. 1989). PS offers a formal counterfactual interpretation of susceptibility, which sharpens this definition and renders susceptibility amenable to systematic analysis.

Khoury et al. (1989) recognized that susceptibility in general is not identifiable and derived (9.23) by making three assumptions: no-confounding, monotonicity,[8] and independence (i.e., assuming that susceptibility to exposure is independent of susceptibility to background not involving exposure). This last assumption is often criticized as untenable, and Theorem 9.2.14 assures us that independence is in fact unnecessary; (9.23) attains its validity through exogeneity and monotonicity alone.

Equation (9.23) also coincides with what Cheng (1997) calls "causal power," namely, the effect of x on y after suppressing "all other causes of y." The counterfactual definition of PS, $P(y_x \mid x', y')$, suggests another interpretation of this quantity. It measures the

[8] Monotonicity is not mentioned in Khoury et al. (1989), but it must have been assumed implicitly to make their derivations valid.

probability that setting x would produce y in a situation where x and y are in fact absent. Conditioning on y' amounts to selecting (or hypothesizing) only those worlds in which "all other causes of y" are indeed suppressed.

It is important to note, however, that the simple relationships among the three notions of causation (equations (9.11)–(9.12)) hold only under the assumption of exogeneity; the weaker relationship of (9.5) prevails in the general, nonexogenous case. Additionally, all these notions of causation are defined in terms of the global relationships $Y_x(u)$ and $Y_{x'}(u)$, which are too crude to fully characterize the many nuances of causation; the detailed structure of the causal model leading from X to Y is often needed to explicate more refined notions, such as "actual cause" (see Chapter 10).

Proof of Theorem 9.2.14

Writing $y_{x'} \vee y'_{x'} = \text{true}$, we have

$$y_x = y_x \wedge (y_{x'} \vee y'_{x'}) = (y_x \wedge y_{x'}) \vee (y_x \wedge y'_{x'}) \tag{9.24}$$

and

$$y_{x'} = y_{x'} \wedge (y_x \vee y'_x) = (y_{x'} \wedge y_x) \vee (y_{x'} \wedge y'_x) = y_{x'} \wedge y_x, \tag{9.25}$$

since monotonicity entails $y_{x'} \wedge y'_x = \text{false}$. Substituting (9.25) into (9.24) yields

$$y_x = y_{x'} \vee (y_x \wedge y'_{x'}). \tag{9.26}$$

Taking the probability of (9.26) and using the disjointness of $y_{x'}$ and $y'_{x'}$, we obtain

$$P(y_x) = P(y_{x'}) + P(y_x, y'_{x'})$$

or

$$P(y_x, y'_{x'}) = P(y_x) - P(y_{x'}). \tag{9.27}$$

Equation (9.27), together with the assumption of exogeneity (equation (9.8)) establishes equation (9.21). □

9.2.4 Identifiability under Monotonicity and Nonexogeneity

The relations established in Theorems 9.2.10–9.2.14 were based on the assumption of exogeneity. In this section, we relax this assumption and consider cases where the effect of X on Y is confounded, that is, when $P(y_x) \neq P(y \mid x)$. In such cases $P(y_x)$ may still be estimated by auxiliary means (e.g., through adjustment of certain covariates or through experimental studies), and the question is whether this added information can render the probability of causation identifiable. The answer is affirmative.

Theorem 9.2.15

If Y is monotonic relative to X, then PNS, PN, *and* PS *are identifiable whenever the causal effects $P(y_x)$ and $P(y_{x'})$ are identifiable*:

$$\text{PNS} = P(y_x, y'_{x'}) = P(y_x) - P(y_{x'}), \tag{9.28}$$

$$\text{PN} = P(y'_{x'} \mid x, y) = \frac{P(y) - P(y_{x'})}{P(x, y)}, \tag{9.29}$$

$$\text{PS} = P(y_x \mid x', y') = \frac{P(y_x) - P(y)}{P(x', y')}. \tag{9.30}$$

In order to appreciate the difference between equations (9.29) and (9.22), we can expand $P(y)$ and write

$$\begin{aligned}\text{PN} &= \frac{P(y \mid x)P(x) + P(y \mid x')P(x') - P(y_{x'})}{P(y \mid x)P(x)} \\ &= \frac{P(y \mid x) - P(y \mid x')}{P(y \mid x)} + \frac{P(y \mid x') - P(y_{x'})}{P(x, y)}.\end{aligned} \tag{9.31}$$

The first term on the r.h.s. of (9.31) is the familiar excess risk ratio (as in (9.22)) and represents the value of PN under exogeneity. The second term represents the *correction* needed to account for X's nonexogeneity, that is, $P(y_{x'}) \neq P(y \mid x')$.

Equations (9.28)–(9.30) thus provide more refined measures of causation, which can be used in situations where the causal effect $P(y_x)$ can be identified through auxiliary means (see Example 4, Section 9.3.4). It can also be shown that expressions in (9.28)–(9.30) provide lower bounds for PNS, PN, and PS in the general, nonmonotonic case (J. Tian, personal communication).

Remarkably, since PS and PN must be nonnegative, (9.29)–(9.30) provide a simple necessary test for the assumption of monotonicity:

$$P(y_x) \geq P(y) \geq P(y_{x'}), \tag{9.32}$$

which tightens the standard inequalities (from $x' \wedge y \implies y_{x'}$ and $x \wedge y' \implies y'_x$)

$$P(y_{x'}) \geq P(x', y), \qquad P(y'_x) \geq P(x, y'). \tag{9.33}$$

J. Tian has shown that these inequalities are in fact sharp: every combination of experimental and nonexperimental data that satisfies these inequalities can be generated from some causal model in which Y is monotonic in X. That the commonly made assumption of "no prevention" is not entirely exempt from empirical scrutiny should come as a relief to many epidemiologists. Alternatively, if the no-prevention assumption is theoretically unassailable, then (9.32) can be used for testing the compatibility of the experimental and nonexperimental data, that is, whether subjects used in clinical trials are representative of the target population as characterized by the joint distribution $P(x, y)$.

Proof of Theorem 9.2.15

Equation (9.28) was established in (9.27). To prove (9.30), we write

$$P(y_x \mid x', y') = \frac{P(y_x, x', y')}{P(x', y')} = \frac{P(y_x, x', y'_{x'})}{P(x', y')}, \tag{9.34}$$

because $x' \wedge y' = x' \wedge y'_{x'}$ (by consistency). To calculate the numerator of (9.34), we conjoin (9.26) with x' to obtain

$$x' \wedge y_x = (x' \wedge y_{x'}) \vee (y_x \wedge y'_{x'} \wedge x').$$

We then take the probability on both sides, which gives (since $y_{x'}$ and $y'_{x'}$ are disjoint)

$$\begin{aligned} P(y_x, y'_{x'}, x') &= P(x', y_x) - P(x', y_{x'}) \\ &= P(x', y_x) - P(x', y) \\ &= P(y_x) - P(x, y_x) - P(x', y) \\ &= P(y_x) - P(x, y) - P(x', y) \\ &= P(y_x) - P(y). \end{aligned}$$

Substituting into (9.34), we finally obtain

$$P(y_x \mid x', y') = \frac{P(y_x) - P(y)}{P(x', y')},$$

which establishes (9.30). Equation (9.29) follows via identical steps. □

One common class of models that permits the identification of $P(y_x)$ under conditions of nonexogeneity was exemplified in Chapter 3. It was shown in Section 3.2 (equation (3.13)) that, for every two variables X and Y in a positive Markovian model M, the causal effect $P(y_x)$ is identifiable and is given by

$$P(y_x) = \sum_{pa_X} P(y \mid pa_X, x) P(pa_X), \tag{9.35}$$

where pa_X are (realizations of) the *parents* of X in the causal graph associated with M. Thus, we can combine (9.35) with Theorem 9.2.15 to obtain a concrete condition for the identification of the probability of causation.

Corollary 9.2.16
For any positive Markovian model M, if the function $Y_x(u)$ is monotonic then the probabilities of causation PNS, PS, *and* PN *are identifiable and are given by* (9.28)–(9.30), *with $P(y_x)$ as given in* (9.35).

A broader identification condition can be obtained through the use of the back-door and front-door criteria (Section 3.3), which are applicable to semi-Markovian models. These were further generalized in Galles and Pearl (1995) (see Section 4.3.1) and lead to the following corollary.

Corollary 9.2.17
Let **GP** *be the class of semi-Markovian models that satisfy the graphical criterion of Theorem 4.3.1. If $Y_x(u)$ is monotonic, then the probabilities of causation* PNS, PS, *and*

PN *are identifiable in* **GP** *and are given by* (9.28)–(9.30), *with* $P(y_x)$ *determined by the topology of* $G(M)$ *through the algorithm of Section 4.3.3.*

9.3 EXAMPLES AND APPLICATIONS

9.3.1 Example 1: Betting against a Fair Coin

We must bet heads or tails on the outcome of a fair coin toss; we win a dollar if we guess correctly and lose if we don't. Suppose we bet heads and win a dollar, without glancing at the actual outcome of the coin. Was our bet a necessary cause (or a sufficient cause, or both) for winning?

This example is isomorphic to the clinical trial discussed in Section 1.4.4 (Figure 1.6). Let x stand for "we bet on heads," y for "we win a dollar," and u for "the coin turned up heads." The functional relationship between y, x, and u is

$$y = (x \wedge u) \vee (x' \wedge u'), \tag{9.36}$$

which is not monotonic but nevertheless permits us to compute the probabilities of causation from the basic definitions of (9.1)–(9.3). To exemplify,

$$\text{PN} = P(y'_{x'} \mid x, y) = P(y'_{x'} \mid u) = 1,$$

because $x \wedge y \implies u$ and $Y_{x'}(u) =$ false. In words, knowing the current bet (x) and current win (y) permits us to infer that the coin outcome must have been a head (u), from which we can further deduce that betting tails (x') instead of heads would have resulted in a loss. Similarly,

$$\text{PS} = P(y_x \mid x', y') = P(y_x \mid u) = 1$$

(because $x' \wedge y' \implies u$) and

$$\begin{aligned}\text{PNS} &= P(y_x, y'_{x'}) \\ &= P(y_x, y'_{x'} \mid u)P(u) + P(y_x, y'_{x'} \mid u')P(u') \\ &= 1(0.5) + 0(0.5) = 0.5.\end{aligned}$$

We see that betting heads has 50% chance of being a necessary and sufficient cause of winning. Still, once we win, we can be 100% sure that our bet was necessary for our win, and once we lose (say, on betting tails) we can be 100% sure that betting heads would have been sufficient for producing a win. The empirical content of such counterfactuals is discussed in Section 7.2.2.

It is easy to verify that these counterfactual quantities cannot be computed from the joint probability of X and Y without knowledge of the functional relationship in (9.36), which tells us the (deterministic) policy by which a win or a loss is decided (Section 1.4.4). This can be seen, for instance, from the conditional probabilities and causal effects associated with this example,

$$P(y \mid x) = P(y \mid x') = P(y_x) = P(y_{x'}) = P(y) = \tfrac{1}{2},$$

because identical probabilities would be generated by a random payoff policy in which y is functionally independent of x – say, by a bookie who watches the coin and ignores our bet. In such a random policy, the probabilities of causation PN, PS, and PNS are all zero. Thus, according to our definition of identifiability (Definition 3.2.3), if two models agree on P and do not agree on a quantity Q, then Q is not identifiable. Indeed, the bounds delineated in Theorem 9.2.10 (equation (9.9)) read $0 \leq \text{PNS} \leq \frac{1}{2}$, meaning that the three probabilities of causation cannot be determined from statistical data on X and Y alone, not even in a controlled experiment; knowledge of the functional mechanism is required, as in (9.36).

It is interesting to note that whether the coin is tossed before or after the bet has no bearing on the probabilities of causation as just defined. This stands in contrast with some theories of probabilistic causality (e.g. Good 1961), which attempt to avoid deterministic mechanisms by conditioning all probabilities on "the state of the world just before" the occurrence of the cause in question (x). When applied to our betting story, the intention is to condition all probabilities on the state of the coin (u), but this is not fulfilled if the coin is tossed after the bet is placed. Attempts to enrich the conditioning set with events occurring after the cause in question have led back to deterministic relationships involving counterfactual variables (see Cartwright 1989, Eells 1991, and the discussion in Section 7.5.4).

One may argue, of course, that if the coin is tossed *after* the bet then it is not at all clear what our winnings would be had we bet differently; merely uttering our bet could conceivably affect the trajectory of the coin (Dawid 1997). This objection can be diffused by placing x and u in two remote locations and tossing the coin a split second after the bet is placed but before any light ray could arrive from the betting room to the coin-tossing room. In such a hypothetical situation, the counterfactual statement "our winning would be different had we bet differently" is rather compelling, even though the conditioning event (u) occurs after the cause in question (x). We conclude that temporal descriptions such as "the state of the world just before x" cannot be used to properly identify the appropriate set of conditioning events (u) in a problem; a deterministic model of the mechanisms involved is needed for formulating the notion of "probability of causation."

9.3.2 Example 2: The Firing Squad

Consider again the firing squad of Section 7.1.2 (see Figure 9.1); A and B are riflemen, C is the squad's captain (who is waiting for the court order, U), and T is a condemned prisoner. Let u be the proposition that the court has ordered an execution, x the proposition stating that A pulled the trigger, and y that T is dead. We assume again that $P(u) = \frac{1}{2}$, that A and B are perfectly accurate marksmen who are alert and law-abiding, and that T is not likely to die from fright or other extraneous causes. We wish to compute the probability that x was a necessary (or sufficient, or both) cause for y (i.e., we wish to calculate PN, PS, and PNS).

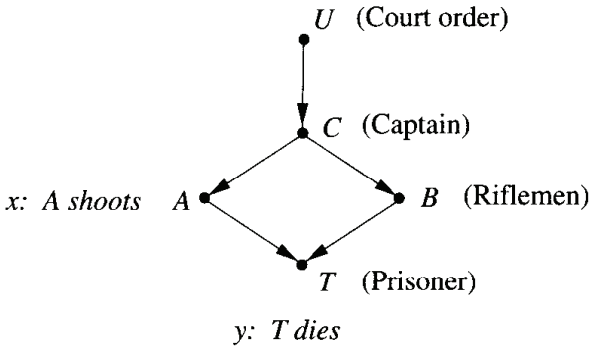

Figure 9.1 Causal relationships in the two-man firing-squad example.

Definitions 9.2.1–9.2.3 permit us to compute these probabilities directly from the given causal model, since all functions and all probabilities are specified, with the truth value of each variable tracing that of U. Accordingly, we can write[9]

$$\begin{aligned} P(y_x) &= P(Y_x(u) = \text{true})P(u) + P(Y_x(u') = \text{true})P(u') \\ &= \tfrac{1}{2}(1+1) = 1. \end{aligned} \tag{9.37}$$

Similarly, we have

$$\begin{aligned} P(y_{x'}) &= P(Y_{x'}(u) = \text{true})P(u) + P(Y_{x'}(u') = \text{true})P(u') \\ &= \tfrac{1}{2}(1+0) = \tfrac{1}{2}. \end{aligned} \tag{9.38}$$

In order to compute PNS, we must evaluate the probability of the joint event $y_{x'} \wedge y_x$. Given that these two events are jointly true only when $U = \text{true}$, we have

$$\begin{aligned} \text{PNS} &= P(y_x, y_{x'}) \\ &= P(y_x, y_{x'} \mid u)P(u) + P(y_x, y_{x'} \mid u')P(u') \\ &= \tfrac{1}{2}(1+0) = \tfrac{1}{2}. \end{aligned} \tag{9.39}$$

The calculation of PS and PN is likewise simplified by the fact that each of the conditioning events, $x \wedge y$ for PN and $x' \wedge y'$ for PS, is true in only one state of U. We thus have

$$\text{PN} = P(y'_{x'} \mid x, y) = P(y'_{x'} \mid u) = 0,$$

reflecting that, once the court orders an execution (u), T will die (y) from the shot of rifleman B, even if A refrains from shooting (x'). Indeed, upon learning of T's death, we can categorically state that rifleman A's shot was *not* a necessary cause of the death.

Similarly,

$$\text{PS} = P(y_x \mid x', y') = P(y_x \mid u') = 1,$$

[9] Recall that $P(Y_x(u') = \text{true})$ involves the submodel M_x, in which X is set to "true" independently of U. Thus, although under condition u' the captain has not given a signal, the potential outcome $Y_x(u')$ calls for hypothesizing that rifleman A pulls the trigger (x) unlawfully.

Table 9.1

	Exposure	
	High (x)	Low (x')
Deaths (y)	30	16
Survivals (y')	69,130	59,010

matching our intuition that a shot fired by an expert marksman would be sufficient for causing the death of T, regardless of the court decision.

Note that Theorems 9.2.10 and 9.2.11 are not applicable to this example because x is not exogenous; events x and y have a common cause (the captain's signal), which renders $P(y \mid x') = 0 \neq P(y_{x'}) = \frac{1}{2}$. However, the monotonicity of Y (in x) permits us to compute PNS, PS, and PN from the joint distribution $P(x, y)$ and the causal effects (using (9.28)–(9.30)), instead of consulting the functional model. Indeed, writing

$$P(x, y) = P(x', y') = \tfrac{1}{2} \tag{9.40}$$

and

$$P(x, y') = P(x', y) = 0, \tag{9.41}$$

we obtain

$$\text{PN} = \frac{P(y) - P(y_{x'})}{P(x, y)} = \frac{\frac{1}{2} - \frac{1}{2}}{\frac{1}{2}} = 0 \tag{9.42}$$

and

$$\text{PS} = \frac{P(y_x) - P(y)}{P(x', y')} = \frac{1 - \frac{1}{2}}{\frac{1}{2}} = 1, \tag{9.43}$$

as expected.

9.3.3 Example 3: The Effect of Radiation on Leukemia

Consider the following data (Table 9.1, adapted[10] from Finkelstein and Levin 1990) comparing leukemia deaths in children in southern Utah with high and low exposure to radiation from the fallout of nuclear tests in Nevada. Given these data, we wish to estimate the probabilities that high exposure to radiation was a necessary (or sufficient, or both) cause of death due to leukemia.

[10] The data in Finkelstein and Levin (1990) are given in "person-year" units. For the purpose of illustration we have converted the data to absolute numbers (of deaths and nondeaths) assuming a ten-year observation period.

Assuming monotonicity – that exposure to nuclear radiation had no remedial effect on any individual in the study – the process can be modeled by a simple disjunctive mechanism represented by the equation

$$y = f(x, u, q) = (x \wedge q) \vee u, \tag{9.44}$$

where u represents "all other causes" of y and where q represents all "enabling" mechanisms that must be present for x to trigger y. Assuming that q and u are both unobserved, the question we ask is under what conditions we can identify the probabilities of causation (PNS, PN, and PS) from the joint distribution of X and Y.

Since (9.44) is monotonic in x, Theorem 9.2.14 states that all three quantities would be identifiable provided X is exogenous; that is, x should be independent of q and u. Under this assumption, (9.21)–(9.23) further permit us to compute the probabilities of causation from frequency data. Taking fractions to represent probabilities, the data in Table 9.1 imply the following numerical results:

$$\text{PNS} = P(y \mid x) - P(y \mid x') = \frac{30}{30 + 69{,}130} - \frac{16}{16 + 59{,}010} = 0.0001625, \tag{9.45}$$

$$\text{PN} = \frac{\text{PNS}}{P(y \mid x)} = \frac{\text{PNS}}{30/(30 + 69{,}130)} = 0.37535, \tag{9.46}$$

$$\text{PS} = \frac{\text{PNS}}{1 - P(y \mid x')} = \frac{\text{PNS}}{1 - 16/(16 + 59{,}010)} = 0.0001625. \tag{9.47}$$

Statistically, these figures mean that:

1. There is a 1.625 in ten thousand chance that a randomly chosen child would both die of leukemia if exposed and survive if not exposed;
2. There is a 37.544% chance that an exposed child who died from leukemia would have survived had he or she not been exposed;
3. There is a 1.625 in ten thousand chance that any unexposed surviving child would have died of leukemia had he or she been exposed.

Glymour (1998) analyzed this example with the aim of identifying the probability $P(q)$ (Cheng's "causal power"), which coincides with PS (see Lemma 9.2.8). Glymour concluded that $P(q)$ is identifiable and is given by (9.23), provided that x, u, and q are mutually independent. Our analysis shows that Glymour's result can be generalized in several ways. First, since Y is monotonic in X, the validity of (9.23) is assured even when q and u are dependent, because exogeneity merely requires independence between x and $\{u, q\}$ jointly. This is important in epidemiological settings, because an individual's susceptibility to nuclear radiation is likely to be associated with susceptibility to other potential causes of leukemia (e.g., natural kinds of radiation).

Second, Theorem 9.2.11 assures us that the relationships between PN, PS, and PNS (equations (9.11)–(9.12)), which Glymour derives for independent q and u, should remain valid even when u and q are dependent.

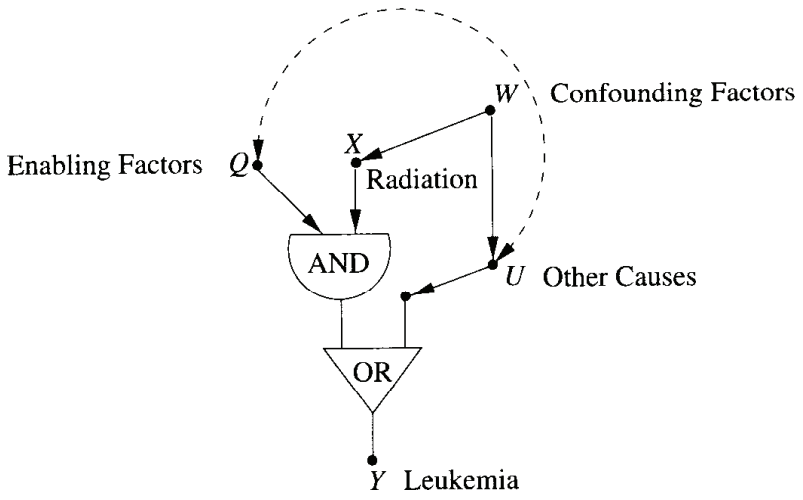

Figure 9.2 Causal relationships in the radiation–leukemia example, where W represents confounding factors.

Finally, Theorem 9.2.15 assures us that PN and PS are identifiable even when x is not independent of $\{u, q\}$, provided only that the mechanism of (9.44) is embedded in a larger causal structure that permits the identification of $P(y_x)$ and $P(y_{x'})$. For example, assume that exposure to nuclear radiation (x) is suspect of being associated with terrain and altitude, which are also factors in determining exposure to cosmic radiation. A model reflecting such consideration is depicted in Figure 9.2, where W represents factors affecting both X and U. A natural way to correct for possible confounding bias in the causal effect of X on Y would be to adjust for W, that is, to calculate $P(y_x)$ and $P(y_{x'})$ using the standard adjustment formula (equation (3.19))

$$P(y_x) = \sum_w P(y \mid x, w)P(w), \qquad P(y_{x'}) = \sum_w P(y \mid x', w)P(w) \tag{9.48}$$

(instead of $P(y \mid x)$ and $P(y \mid x')$), where the summation runs over levels of W. This adjustment formula, which follows from (9.35), is correct regardless of the mechanisms mediating X and Y, provided only that W represents *all* common factors affecting X and Y (see Section 3.3.1).

Theorem 9.2.15 instructs us to evaluate PN and PS by substituting (9.48) into (9.29) and (9.30), respectively, and it assures us that the resulting expressions constitute consistent estimates of PN and PS. This consistency is guaranteed jointly by the assumption of monotonicity and by the (assumed) topology of the causal graph.

Note that monotonicity as defined in (9.20) is a global property of all pathways between x and y. The causal model may include several nonmonotonic mechanisms along these pathways without affecting the validity of (9.20). However, arguments for the validity of monotonicity must be based on substantive information, since it is not testable in general. For example, Robins and Greenland (1989) argued that exposure to nuclear radiation may conceivably be of benefit to some individuals because such radiation is routinely used clinically in treating cancer patients. The inequalities in (9.32) constitute a statistical test of monotonicity (albeit a weak one) that is based on both experimental and observational studies.

Table 9.2

	Experimental		Nonexperimental	
	x	x'	x	x'
Deaths (y)	16	14	2	28
Survivals (y')	984	986	998	972

9.3.4 Example 4: Legal Responsibility from Experimental and Nonexperimental Data

A lawsuit is filed against the manufacturer of drug x, charging that the drug is likely to have caused the death of Mr. A, who took the drug to relieve symptom S associated with disease D.

The manufacturer claims that experimental data on patients with symptom S show conclusively that drug x may cause only minor increase in death rates. However, the plaintiff argues that the experimental study is of little relevance to this case because it represents the effect of the drug on *all* patients, not on patients like Mr. A who actually died while using drug x. Moreover, argues the plaintiff, Mr. A is unique in that he used the drug on his own volition, unlike subjects in the experimental study who took the drug to comply with experimental protocols. To support this argument, the plaintiff furnishes nonexperimental data indicating that most patients who chose drug x would have been alive were it not for the drug. The manufacturer counterargues by stating that: (1) counterfactual speculations regarding whether patients would or would not have died are purely metaphysical and should be avoided (Dawid 1997); and (2) nonexperimental data should be dismissed a priori on the grounds that such data may be highly confounded by extraneous factors. The court must now decide, based on both the experimental and nonexperimental studies, what the probability is that drug x was in fact the cause of Mr. A's death.

The (hypothetical) data associated with the two studies are shown in Table 9.2. The experimental data provide the estimates

$$P(y_x) = 16/1000 = 0.016, \tag{9.49}$$

$$P(y_{x'}) = 14/1000 = 0.014; \tag{9.50}$$

the nonexperimental data provide the estimates

$$P(y) = 30/2000 = 0.015, \tag{9.51}$$

$$P(y, x) = 2/2000 = 0.001. \tag{9.52}$$

Assuming that drug x can only cause (can never prevent) death, Theorem 9.2.15 is applicable and (9.29) yields

$$\text{PN} = \frac{P(y) - P(y_{x'})}{P(y, x)} = \frac{0.015 - 0.014}{0.001} = 1.00. \tag{9.53}$$

Thus, the plaintiff was correct; barring sampling errors, the data provide us with 100% assurance that drug x was in fact responsible for the death of Mr. A. Note that a straight-

forward use of the experimental excess risk ratio would yield a much lower (and incorrect) result:

$$\frac{P(y_x) - P(y_{x'})}{P(y_x)} = \frac{0.016 - 0.014}{0.016} = 0.125. \tag{9.54}$$

Evidently, what the experimental study does not reveal is that, given a choice, terminal patients avoid drug x. Indeed, if there were any terminal patients who would choose x (given the choice), then the control group (x') would have included some such patients (due to randomization) and so the proportion of deaths among the control group $P(y_{x'})$ would have been higher than $P(x', y)$, the population proportion of terminal patients avoiding x. However, the equality $P(y_{x'}) = P(y, x')$ tells us that no such patients were included in the control group; hence (by randomization) no such patients exist in the population at large and therefore none of the patients who freely chose drug x was a terminal case; all were susceptible to x.

The numbers in Table 9.2 were obviously contrived to represent an extreme case and so facilitate a qualitative explanation of the validity of (9.29). Nevertheless, it is instructive to note that a combination of experimental and nonexperimental studies may unravel what experimental studies alone will not reveal and, in addition, that such combination may provide a necessary test for the assumption of no-prevention, as outlined in Section 9.2.4 (equation (9.32)). For example, if the frequencies in Table 9.2 were slightly different, they could easily yield a negative value for PN in (9.53) and thus indicate violation of the fundamental inequalities of (9.32)–(9.33). Such violation might be due either to nonmonotonicity or to incompatibility of the experimental and nonexperimental groups.

This last point may warrant a word of explanation, lest the reader wonder why two data sets – taken from two separate groups under different experimental conditions – should constrain one another. The explanation is that certain quantities in the two subpopulations are expected to remain invariant to all these differences, provided that the two subpopulations were sampled properly from the population at large. These invariant quantities are simply the causal effects probabilities, $P(y_{x'})$ and $P(y_x)$. Although these counterfactual probabilities were not measured in the observational group, they must (by definition) nevertheless be the same as those measured in the experimental group. The invariance of these quantities is the basic axiom of controlled experimentation, without which *no* inference would be possible from experimental studies to general behavior of the population. The invariance of these quantities, together with monotonicity, implies the inequalities of (9.32)–(9.33).

9.3.5 Summary of Results

We now summarize the results from Sections 9.2 and 9.3 that should be of value to practicing epidemiologists and policy makers. These results are shown in Table 9.3, which lists the best estimand of PN (for a nonexperimental event) under various assumptions and various types of data – the stronger the assumptions, the more informative the estimates.

We see that the excess risk ratio (ERR), which epidemiologists commonly equate with the probability of causation, is a valid measure of PN only when two assumptions

Table 9.3. ***PN as a Function of Assumptions and Available Data***

Assumptions			Data Available		
Exogeneity	Monotonicity	Additional	Experimental	Observational	Combined
+	+		ERR	ERR	ERR
+	−		bounds	bounds	bounds
−	+	covariate control	—	corrected ERR	corrected ERR
−	+		—	—	corrected ERR
−	−		—	—	bounds

Note: ERR stands for the excess risk ratio, $1 - P(y \mid x')/P(y' \mid x')$; corrected ERR is given in (9.31).

can be ascertained: exogeneity (i.e., no confounding) and monotonicity (i.e., no prevention). When monotonicity does not hold, ERR provides merely a lower bound for PN, as shown in (9.13). (The upper bound is usually unity.) The nonentries (—) in the right-hand side of Table 9.3 represent vacuous bounds (i.e., $0 \leq \text{PN} \leq 1$). In the presence of confounding, ERR must be corrected by the additive term $[P(y \mid x') - P(y_{x'})]/P(x, y)$, as stated in (9.31). In other words, when confounding bias (of the causal effect) is positive, PN is higher than ERR by the amount of this additive term. Clearly, owing to the division by $P(x, y)$, the PN bias can be many times higher than the causal effect bias $P(y \mid x') - P(y_{x'})$. However, confounding results only from association between exposure and other factors that affect the outcome; one need not be concerned with associations between such factors and susceptibility to exposure (see Figure 9.2).

The last row in Table 9.3, corresponding to no assumptions whatsoever, leads to vacuous bounds for PN, unless we have combined data. This does not mean, however, that justifiable assumptions *other* than monotonicity and exogeneity could not be helpful in rendering PN identifiable. The use of such assumptions is explored in the next section.

9.4 IDENTIFICATION IN NONMONOTONIC MODELS

In this section we discuss the identification of probabilities of causation without making the assumption of monotonicity. We will assume that we are given a causal model M in which all functional relationships are known, but since the background variables U are not observed, their distribution is not known and the model specification is not complete.

Our first step would be to study under what conditions the function $P(u)$ can be identified, thus rendering the entire model identifiable. If M is Markovian, then the problem can be analyzed by considering each parents–child family separately. Consider any arbitrary equation in M,

$$\begin{aligned} y &= f(pa_Y, u_Y) \\ &= f(x_1, x_2, \ldots, x_k, u_1, \ldots, u_m), \end{aligned} \tag{9.55}$$

where $U_Y = \{U_1, \ldots, U_m\}$ is the set of background (possibly dependent) variables that appear in the equation for Y. In general, the domain of U_Y can be arbitrary, discrete, or continuous, since these variables represent unobserved factors that were omitted from the model. However, since the observed variables are binary, there is only a finite number $(2^{(2^k)})$ of functions from PA_Y to Y and, for any point $U_Y = u$, only one of those functions is realized. This defines a partition of the domain of U_Y into a set S of equivalence classes, where each equivalence class $s \in S$ induces the same function $f^{(s)}$ from PA_Y to Y (see Section 8.2.2). Thus, as u varies over its domain, a set S of such functions is realized, and we can regard S as a new background variable whose values correspond to the set $\{f^{(s)} : s \in S\}$ of functions from PA_Y to Y that are realizable in U_Y. The number of such functions will usually be smaller than $2^{(2^k)}$.[11]

For example, consider the model described in Figure 9.2. As the background variables (Q, U) vary over their respective domains, the relation between X and Y spans three distinct functions:

$$f^{(1)} : Y = \text{true}, \quad f^{(2)} : Y = \text{false}, \quad \text{and} \quad f^{(3)} : Y = X.$$

The fourth possible function, $Y \neq X$, is never realized because $f_Y(\cdot)$ is monotonic. The cells (q, u) and (q', u) induce the same function between X and Y; hence they belong to the same equivalence class.

If we are given the distribution $P(u_Y)$ then we can compute the distribution $P(s)$, and this will determine the conditional probabilities $P(y \mid pa_Y)$ by summing $P(s)$ over all those functions $f^{(s)}$ that map pa_Y into the value true,

$$P(y \mid pa_Y) = \sum_{s: f^{(s)}(pa_Y)=\text{true}} P(s). \tag{9.56}$$

To ensure model identifiability, it is sufficient that we can invert the process and determine $P(s)$ from $P(y \mid pa_Y)$. If we let the set of conditional probabilities $P(y \mid pa_Y)$ be represented by a vector $\vec{p}$ (of dimensionality 2^k) and $P(s)$ by a vector $\vec{q}$, then (9.56) defines a linear relation between $\vec{p}$ and $\vec{q}$ that can be represented as a matrix multiplication (as in (8.13)),

$$\vec{p} = \boldsymbol{R}\vec{q}, \tag{9.57}$$

where $\boldsymbol{R}$ is a $2^k \times |S|$ matrix whose entries are either 0 or 1. Thus, a sufficient condition for identification is simply that $\boldsymbol{R}$, together with the normalizing equation $\sum_j \vec{q}_j = 1$, be invertible.

In general, $\boldsymbol{R}$ will *not* be invertible because the dimensionality of $\vec{q}$ can be much larger than that of $\vec{p}$. However, in many cases, such as the "noisy OR" mechanism

$$Y = U_0 \bigvee_{i=1,\ldots,k} (X_i \wedge U_i), \tag{9.58}$$

[11] Balke and Pearl (1994a,b) called these S variables "response variables," as in Section 8.2.2; Heckerman and Shachter (1995) called them "mapping variables."

symmetry permits $\vec{q}$ to be identified from $P(y \mid pa_Y)$ even when the exogenous variables $U_0, U_1, \ldots, U_k$ are not independent. This can be seen by noting that every point u for which $U_0 = \text{false}$ defines a unique function $f^{(s)}$ because, if T is the set of indices i for which U_i is true, the relationship between PA_Y and Y becomes

$$Y = U_0 \bigvee_{i \in T} X_i \tag{9.59}$$

and, for $U_0 = \text{false}$, this equation defines a distinct function for each T. The number of induced functions is $2^k + 1$, which (subtracting 1 for normalization) is exactly the number of distinct realizations of PA_Y. Moreover, it is easy to show that the matrix connecting $\vec{p}$ and $\vec{q}$ is invertible. We thus conclude that the probability of every counterfactual sentence can be identified in any Markovian model composed of noisy OR mechanisms, regardless of whether the background variables in each family are mutually independent. The same holds, of course, for noisy AND mechanisms or any combination thereof (including negating mechanisms), provided that each family consists of one type of mechanism.

To generalize this results to mechanisms other than noisy OR and noisy AND, we note that – although $f_Y(\cdot)$ in this example was monotonic (in each X_i) – it was the *redundancy* of $f_Y(\cdot)$ and not its monotonicity that ensured identifiability. The following is an example of a monotonic function for which the $\boldsymbol{R}$ matrix is not invertible:

$$Y = (X_1 \wedge U_1) \vee (X_2 \wedge U_1) \vee (X_1 \wedge X_2 \wedge U_3).$$

This function represents a noisy OR gate for $U_3 = \text{false}$; it becomes a noisy AND gate for $U_3 = \text{true}$ and $U_1 = U_2 = \text{false}$. The number of equivalence classes induced is six, which would require five independent equations to determine their probabilities; the data $P(y \mid pa_Y)$ provide only four such equations.

In contrast, the mechanism governed by the following function, although nonmonotonic, is invertible:

$$Y = \text{XOR}(X_1, \text{XOR}(U_2, \ldots, \text{XOR}(U_{k-1}, \text{XOR}(X_k, U_k)))),$$

where XOR$(\cdot)$ stands for exclusive OR. This equation induces only two functions from PA_Y to Y:

$$Y = \begin{cases} \text{XOR}(X_1, \ldots, X_k) & \text{if } \text{XOR}(U_1, \ldots, U_k) = \text{false}, \\ \neg\text{XOR}(X_1, \ldots, X_k) & \text{if } \text{XOR}(U_1, \ldots, U_k) = \text{true}. \end{cases}$$

A single conditional probability, say $P(y \mid x_1, \ldots, x_k)$, would therefore suffice for computing the one parameter needed for identification: $P[\text{XOR}(U_1, \ldots, U_k) = \text{true}]$.

We summarize these considerations with a theorem.

Definition 9.4.1 (Local Invertibility)
A model M is said to be locally invertible *if, for every variable $V_i \in V$, the set of $2^k + 1$ equations*

$$P(y \mid pa_i) = \sum_{s:\, f^{(s)}(pa_i) = true} q_i(s), \tag{9.60}$$

$$\sum_s q_i(s) = 1 \tag{9.61}$$

has a unique solution for $q_i(s)$, *where each* $f_i^{(s)}(pa_i)$ *corresponds to the function* $f_i(pa_i, u_i)$ *induced by* u_i *in equivalence class* s.

Theorem 9.4.2

Given a Markovian model $M = \langle U, V, \{f_i\}\rangle$ *in which the functions* $\{f_i\}$ *are known and the exogenous variables* U *are unobserved, if* M *is locally invertible then the probability of every counterfactual sentence is identifiable from the joint probability* $P(v)$.

Proof

If (9.60) has a unique solution for $q_i(s)$, then we can replace U with S and obtain an equivalent model as follows:

$$M' = \langle S, V, \{f_i'\}\rangle, \quad \text{where } f_i' = f_i^{(s)}(pa_i).$$

The model M', together with $q_i(s)$, completely specifies a probabilistic causal model $\langle M', P(s)\rangle$ (owing to the Markov property), from which probabilities of counterfactuals are derivable by definition. □

Theorem 9.4.2 provides a sufficient condition for identifying probabilities of causation, but of course it does not exhaust the spectrum of assumptions that are helpful in achieving identification. In many cases we might be justified in hypothesizing additional structure on the model – for example, that the U variables entering each family are themselves independent. In such cases, additional constraints are imposed on the probabilities $P(s)$, and (9.60) may be solved even when the cardinality of S far exceeds the number of conditional probabilities $P(y \mid pa_Y)$.

9.5 CONCLUSIONS

This chapter has explicated and analyzed the interplay between the necessary and sufficient components of causation. Using counterfactual interpretations that rest on structural model semantics, we demonstrated how simple techniques of computing probabilities of counterfactuals can be used in computing probabilities of causes, deciding questions of identification, uncovering conditions under which probabilities of causes can be estimated from statistical data, and devising tests for assumptions that are routinely made (often unwittingly) by analysts and investigators.

On the practical side, we have offered several useful tools (partly summarized in Table 9.3) for epidemiologists and health scientists. This chapter formulates and calls attention to subtle assumptions that must be ascertained before statistical measures such as excess risk ratio can be used to represent causal quantities such as attributable risk or probability of causes (Theorem 9.2.14). It shows how data from both experimental and nonexperimental studies can be combined to yield information that neither study alone can reveal (Theorem 9.2.15 and Section 9.3.4). Finally, it provides tests for the commonly

made assumption of "no prevention" and for the often asked question of whether a clinical study is representative of its target population (equation (9.32)).

On the conceptual side, we have seen that both the probability of necessity (PN) and probability of sufficiency (PS) play a role in our understanding of causation and that each component has its logic and computational rules. Although the counterfactual concept of necessary cause (i.e., that an outcome would not have occurred "but for" the action) is predominant in legal settings (Robertson 1997) and in ordinary discourse, the sufficiency component of causation has a definite influence on causal thoughts.

The importance of the sufficiency component can be uncovered in examples where the necessary component is either dormant or ensured. Why do we consider striking a match to be a more adequate explanation (of a fire) than the presence of oxygen? Recasting the question in the language of PN and PS, we note that, since both explanations are necessary for the fire, each will command a PN of unity. (In fact, the PN is actually higher for the oxygen if we allow for alternative ways of igniting a spark). Thus, it must be the sufficiency component that endows the match with greater explanatory power than the oxygen. If the probabilities associated with striking a match and the presence of oxygen are denoted p_m and p_o, respectively, then the PS measures associated with these explanations evaluate to PS(match) $= p_o$ and PS(oxygen) $= p_m$, clearly favoring the match when $p_o \gg p_m$. Thus, a robot instructed to explain why a fire broke out has no choice but to consider both PN and PS in its deliberations.

Should PS enter legal considerations in criminal and tort law? I believe that it should – as does Good (1993) – because attention to sufficiency implies attention to the consequences of one's action. The person who lighted the match ought to have anticipated the presence of oxygen, whereas the person who supplied – or could (but did not) remove – the oxygen is not generally expected to have anticipated match-striking ceremonies.

However, what weight should the law assign to the necessary versus the sufficient component of causation? This question obviously lies beyond the scope of our investigation, and it is not at all clear who would be qualified to tackle the issue or whether our legal system would be prepared to implement the recommendation. I am hopeful, however, that whoever undertakes to consider such questions will find the analysis in this chapter to be of some use. The next chapter combines aspects of necessity and sufficiency in explicating a more refined notion: "actual cause."

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CHAPTER TEN

The Actual Cause

And now remains
That we find out the cause of this effect,
Or rather say, the cause of this defect,
For this effect defective comes by cause.
Shakespeare (Hamlet II.ii.100–4)

Preface

This chapter offers a formal explication of the notion of "actual cause," an event recognized as responsible for the production of a given outcome in a specific scenario, as in: "Socrates drinking hemlock was the actual cause of Socrates death." Human intuition is extremely keen in detecting and ascertaining this type of causation and hence is considered the key to constructing explanations (Section 7.2.3) and the ultimate criterion (known as "cause in fact") for determining legal responsibility.

Yet despite its ubiquity in natural thoughts, actual causation is not an easy concept to formulate. A typical example (introduced by Wright 1988) considers two fires advancing toward a house. If fire A burned the house before fire B, we (and many juries nationwide) would surely consider fire A "the actual cause" for the damage, though either fire alone is sufficient (and neither one was necessary) for burning the house. Clearly, actual causation requires information beyond that of necessity and sufficiency; the actual process mediating between the cause and the effect must enter into consideration. But what precisely is a "process" in the language of structural models? What aspects of causal processes define actual causation? How do we piece together evidence about the uncertain aspects of a scenario and so compute probabilities of actual causation?

In this chapter we propose a plausible account of actual causation that can be formulated in structural model semantics. The account is based on the notion of *sustenance,* to be defined in Section 10.2, which combines aspects of necessity and sufficiency to measure the capacity of the cause to maintain the effect despite certain *structural* changes in the model. We show by examples how this account avoids problems associated with the counterfactual dependence account of Lewis (1986) and how it can be used both in generating explanations of specific scenarios and in computing the probabilities that such explanations are in fact correct.

10.1 INTRODUCTION: THE INSUFFICIENCY OF NECESSARY CAUSATION

10.1.1 Singular Causes Revisited

Statements of the type "a car accident was the cause of Joe's death," made relative to a specific scenario, are classified as "singular," "single-event," or "token-level" causal

statements. Statements of the type "car accidents cause deaths," when made relative to a type of events or a class of individuals, are classified as "generic" or "type-level" causal claims (see Section 7.5.4). We will call the cause in a single-event statement an *actual cause* and the one in a type-level statement a *general cause.*

The relationship between type and token causal claims has been controversial in the philosophical literature (Woodward 1990; Hitchcock 1995), and priority questions such as "which comes first?" or "can one level be reduced to the other?" (Cartwright 1989; Eells 1991; Hausman 1998) have diverted attention from the more fundamental question: "What tangible claims do type and token statements make about our world, and how is causal knowledge organized so as to substantiate such claims?" The debate has led to theories that view type and token claims as two distinct species of causal relations (as in Good 1961, 1962), each requiring its own philosophical account (see e.g. Sober 1985; Eells 1991, chap. 6) – "not an altogether happy predicament" (Hitchcock 1997). In contrast, the structural account treats type and token claims as instances of the same species, differing only in the details of the scenario-specific information that is brought to bear on the question. As such, the structural account offers a formal basis for studying the anatomy of the two levels of claims, what information is needed to support each level, and why philosophers have found their relationships so hard to disentangle.

The basic building blocks of the structural account are the functions $\{f_i\}$, which represent lawlike mechanisms and supply information for both type-level and token-level claims. These functions are type-level in the sense of representing generic, counterfactual relationships among variables that are applicable to every hypothetical scenario, not just ones that were realized. At the same time, any specific instantiation of those relationships represents a token-level claim. The ingredients that distinguish one scenario from another are represented in the background variables U. When all such factors are known, $U = u$, we have a "world" on our hands (Definition 7.1.8) – an ideal, full description of a specific scenario in which all relevant details are spelled out and nothing is left to chance or guessing. Causal claims made at the world level would be extreme cases of token causal claims. In general, however, we do not possess the detailed knowledge necessary for specifying a single world $U = u$, and we use a probability $P(u)$ to summarize our ignorance of those details. This takes us to the level of probabilistic causal models $\langle M, P(u)\rangle$ (Definition 7.1.6). Causal claims made on the basis of such models, with no reference to the actual scenario, would be classified as type-level claims. Causal effects assertions, such as $P(Y_x = y) = p$, are examples of such claims, for they express the general tendency of x to bring about y, as judged over all potential scenarios.[1] In most cases, however, we possess partial information about the scenario at hand – for example, that Joe died, that he was in a car accident, and perhaps that he drove a sports car and suffered a head injury. The totality of such episode-specific information is called "evidence" (e) and can be used to update $P(u)$ into $P(u \mid e)$. Causal claims derived from the model $\langle M, P(u \mid e)\rangle$ represent token claims of varying shades, depending on the specificity of e.

[1] Occasionally, causal effect assertions can even be made on the basis of an incomplete probabilistic model, where only $G(M)$ and $P(v)$ are given – this is the issue of identification (Chapter 3). But no token-level statement can be made on such basis alone without some knowledge of $\{f_i\}$ or $P(u)$ (assuming, of course, that x and y are known to have occurred).

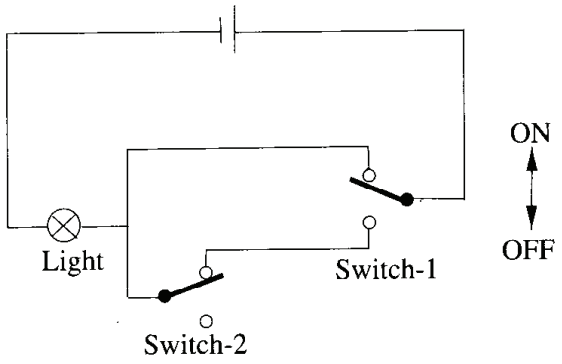

Figure 10.1 Switch 1 (and not switch 2) is perceived to be causing the light, though neither is necessary.

Thus, the distinction between type and token claims is a matter of degree in the structural account. The more episode-specific evidence we gather, the closer we come to the ideals of token claims and actual causes. The notions of PS and PN (the focus of Chapter 9) represent intermediate points along this spectrum. Probable sufficiency (PS) is close to a type-level claim because the actual scenario is not taken into account and is, in fact, excluded from consideration. Probable necessity (PN) makes some reference to the actual scenario, albeit a rudimentary one (i.e., that x and y are true). In this section we will attempt to come closer to the notion of actual cause by taking additional information into consideration.

10.1.2 Preemption and the Role of Structural Information

In Section 9.2, we alluded to the fact that both PN and PS are global (i.e. input–output) features of a causal model, depending only on the function $Y_x(u)$ and not on the structure of the process mediating between the cause (x) and the effect (y). That such structure plays a role in causal explanation is seen in the following example.

Consider an electrical circuit consisting of a light bulb and two switches, as shown in Figure 10.1. From the user's viewpoint, the light responds symmetrically to the two switches; either switch is sufficient to turn the light on. Internally, however, when switch 1 is on it not only activates the light but also disconnects switch 2 from the circuit, rendering it inoperative. Consequently, with both switches on, we would not hesitate to proclaim switch 1 as the "actual cause" of the current flowing in the light bulb, knowing as we do that switch 2 can have no effect whatsoever on the electric pathway in this particular state of affairs. There is nothing in PN and PS that could possibly account for this asymmetry; each is based on the response function $Y_x(u)$ and is therefore oblivious to the internal workings of the circuit.

This example is representative of a class of counterexamples, involving *preemption,* that were brought up against Lewis's counterfactual account of causation. It illustrates how an event (e.g., switch 1 being on) can be considered a cause although the effect persists in its absence. Lewis's (1986) answer to such counterexamples was to modify the counterfactual criterion and let x be a cause of y as long as there exists a *counterfactual dependence chain* of intermediate variables between x to y; that is, the output of every link in the chain is counterfactually dependent on its input. Such a chain does not exist for switch 2 because, given the current state of affairs (i.e., both switches being on), no

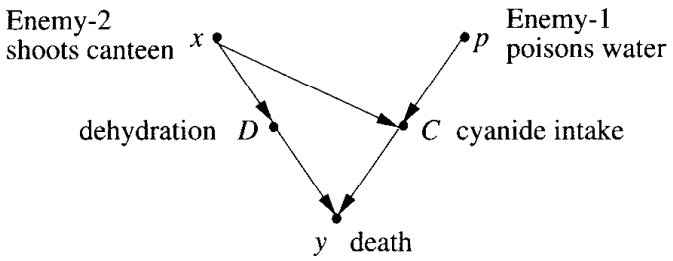

Figure 10.2 Causal relationships in the desert traveler example.

part of the circuit would be affected (electrically) by turning switch 2 on or off. This can be shown more clearly in the following example.

Example 10.1.1 (The Desert Traveler – after P. Suppes) A desert traveler T has two enemies. Enemy 1 poisons T's canteen, and enemy 2, unaware of enemy 1's action, shoots and empties the canteen. A week later, T is found dead and the two enemies confess to action and intention. A jury must decide whose action was the *actual cause* of T's death.

Let x and p be (respectively) the propositions "enemy 2 shot" and "enemy 1 poisoned the water," and let y denote "T is dead." In addition to these events we will also use the intermediate variable C (connoting cyanide) and D (connoting dehydration), as shown in Figure 10.2. The functions $f_i(pa_i, u)$ are not shown explicitly in Figure 10.2, but they are presumed to determine the value of each child variable from those of its parent variables in the graph, in accordance with the usual understanding of the story:[2]

$$\begin{aligned} c &= px', \\ d &= x, \\ y &= c \vee d. \end{aligned} \tag{10.1}$$

When we substitute c and d into the expression for y, we obtain a simple disjunction

$$y = x \vee px' \equiv x \vee p, \tag{10.2}$$

which is deceiving in its symmetry.

Here we see in vivid symbols the role played by structural information. Although it is true that $x \vee x'p$ is logically equivalent to $x \vee p$, the two are not structurally equivalent; $x \vee p$ is completely symmetric relative to exchanging x and p, whereas $x \vee x'p$ tells us that, when x is true, p has no effect whatsoever – not only on y, but also on any of the intermediate conditions that could potentially affect y. It is this asymmetry that makes us proclaim x and not p to be the cause of death.

According to Lewis, the difference between x and p lies in the nature of the chains that connect each of them to y. From x, there exists a causal chain $x \rightarrow d \rightarrow y$ such that every element is counterfactually dependent on its antecedent. Such a chain does not exist from p to y because, when x is true, the chain $p \rightarrow c \rightarrow y$ is *preempted* (at c);

[2] For simplicity, we drop the "$\wedge$" symbol in the rest of this chapter.

that is, c is "stuck" at false regardless of p. Put another way, although x does not satisfy the counterfactual test for causing y, one of its consequences (d) does; given that x and p are true, y would be false were it not for d.

Lewis's chain criterion retains the connection between causation and counterfactuals, but it is rather ad hoc; after all, why should the existence of a counterfactual dependence chain be taken as a defining test for a concept as crucial as "actual cause," by which we decide the guilt or innocence of defendants in a court of law? The basic counterfactual criterion does embody a pragmatic rationale; we would not wish to punish a person for a damage that could not have been avoided, and we would like to encourage people to watch for circumstances where their actions could make a substantial difference. However, once the counterfactual dependence between the action and the consequence is destroyed by the presence of another cause, what good is it to insist on intermediate counterfactual dependencies along a chain that connects them?

10.1.3 Overdetermination and Quasi-Dependence

Another problem with Lewis's chain is its failure to capture cases of simultaneous disjunctive causes. For example, consider the firing squad in Figure 9.1, and assume that riflemen A and B shot together and killed the prisoner. Our intuition regards each of the riflemen as a *contributory* actual cause of the death, though neither rifleman passes the counterfactual test and neither supports a counterfactual dependence chain in the presence of the other.

This example is representative of a condition called *overdetermination*, which presents a tough challenge to the counterfactual account. Lewis answered this challenge by offering yet another repair of the counterfactual criterion. He proposed that chains of counterfactual dependence should be regarded as intrinsic to the process (e.g., the flight of the bullet from A to D) and that the disappearance of dependence due to peculiar surroundings (e.g., the flight of the bullet from B to D) should not be considered an intrinsic loss of dependence; we should still count such a process as *quasi-dependent* "if only the surroundings were different " (Lewis 1986, p. 206).

Hall (1998) observed that the notion of quasi-dependence raises difficult questions: "First, what exactly is a process? Second, what does it mean to say that one process is 'just like' another process in its intrinsic character? Third, how exactly do we 'measure the variety of the surroundings'?" We will propose an answer to these questions using an object called a *causal beam* (Section 10.3.1), which can be regarded as a structural–semantic explication of the notion of a "process." We will return to chains and beams and to questions of preemption and overdetermination in Section 10.2, after a short excursion into Mackie's approach, which also deals with the problem of actual causation – though from a different perspective.

10.1.4 Mackie's INUS Condition

The problems we encountered in the previous section are typical of many attempts by philosophers to give a satisfactory logical explication to the notion of single-event causation (here, "actual causation"). These attempts seem to have started with Mill's observation that no cause is truly sufficient or necessary for its effect (Mill 1843, p. 398). The

numerous accounts subsequently proposed – based on more elaborate combinations of sufficiency and necessity conditions – all suffer from insurmountable difficulties (Sosa and Tooley 1993, pp. 1–8). Mackie's treatment (1965) appears to be the earliest attempt to offer a semiformal explication of "actual causation" within this logical framework; his solution, known as the INUS condition, became extremely popular.

The INUS condition states that an event C is perceived to be the cause of event E if C is "an *insufficient* but *necessary* part of a condition which is itself *unnecessary* but *sufficient* for the result" (Mackie 1965).[3] Although attempts to give INUS precise formulation (including some by Mackie 1980) have not resulted in a coherent proposal (Sosa and Tooley 1993, pp. 1–8), the basic idea behind INUS is appealing: If we can think of $\{S_1, S_2, S_3, \ldots\}$ as a collection of every minimally sufficient set of conditions (for E), then event C is an INUS condition for E if it is a conjunct of some S_i. Furthermore, C is considered a *cause* of E if C is an INUS condition for E and if, under the circumstances, C was sufficient for one of those S_i. Thus, for example, if E can be written in disjunctive normal form as

$$E = AB \vee CD,$$

then C is an INUS condition by virtue of being a member of a disjunct, CD, which is minimal and sufficient for E. Thus C would be considered a cause of E if D were present on the occasion in question.[4]

This basic intuition is shared by researchers from many disciplines. Legal scholars, for example, have advocated a relation called NESS (Wright 1988), standing for "necessary element of sufficient set," which is a rephrasing of Mackie's INUS condition in a simpler mnemonic. In epidemiology, Rothman (1976) proposed a similar criterion for recognizing when an exposure is said to cause a disease: "We say that the exposure E causes disease if a sufficient cause that contains E is the first sufficient cause to be completed" (Rothman and Greenland 1998, p. 53). Hoover (1990, p. 218) related the INUS condition to causality in econometrics: "Any variable that causes another in Simon's sense may be regarded as an INUS condition for that other variable."

However, the language of logical necessity and sufficiency is inadequate for explicating these intuitions (Kim 1971). Similar conclusions are implicit in the analysis of Cartwright (1989, pp. 25–34), who starts out enchanted with INUS's intuition and ends up having to correct INUS's mistakes.

The basic limitation of the logical account stems from the lack of a syntactic distinction between formulas that represent stable mechanisms (or "dispositional relations," to use Mackie's terminology) and those that represent circumstantial conditions. The simplest manifestation of this limitation can be seen in contraposition: "A implies B"

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[3] The two negations and the two "buts" in this acronym make INUS one of the least helpful mnemonics in the philosophical literature. Simplified, it should read: "a necessary element in a sufficient set of conditions, NESS" (Wright 1988).

[4] Mackie (1965) also required that every disjunct of E that does not contain C as a conjunct be absent, but this would render Mackie's definition identical to the counterfactual test of Lewis. I use a broader definition here to allow for simultaneous causes and overdetermination; see Mackie (1980, pp. 43–7).

is logically equivalent to "not B implies not A," yet this inversion is not supported by causal implications; from "disease causes a symptom" we cannot infer that eliminating a symptom will cause the disappearance of the disease. The failure of contraposition further entails problems with transduction (inference through common causes): if a disease D causes two symptoms, A and B, then curing symptom A would entail (in the logical account) the disappearance of symptom B.

Another set of problems stems from syntax sensitivity. Suppose we apply Mackie's INUS condition to the firing squad story of Figure 9.1. If we write the conditions for the prisoner's death as:

$$D = A \vee B,$$

then A satisfies the INUS criterion and we can plausibly conclude that A was a cause of D. However, substituting $A = C$, which is explicit in our model, we obtain

$$D = C \vee B,$$

and suddenly A no longer appears as a conjunct in the expression for D. Shall we conclude that A was not a cause of D? We can, of course, avoid this disappearance by forbidding substitutions and insisting that A remain in the disjunction together with B and C. But then a worse problems ensues: in circumstances where the captain gives a signal (C) and both riflemen fail to shoot, the prisoner will still be deemed dead. In short, the structural information conveying the flow of influences in the story cannot be encoded in standard logical syntax.

Finally, let us consider the desert traveler example, where the traveler's death was expressed in (10.2) as

$$y = x \vee x'p.$$

This expression is not in minimal disjunctive normal form because it can be rewritten as

$$y = x \vee p,$$

from which one would obtain the counterintuitive result that x and p are equal partners in causing y. If, on the other hand, we permit nonminimal expressions like $y = x \vee x'p$ then we might as well permit the equivalent expression $y = xp' \vee p$, from which we would absurdly conclude that not poisoning the water (p') would be a cause for our traveler's misfortune, provided someone shoots the canteen (x).

We return now to structural analysis, in which such syntactic problems do not arise. Dispositional information is conveyed through structural or counterfactual expressions (e.g., $v_i = f_i(pa_i, u)$) in which u is generic, whereas circumstantial information is conveyed through propositional expressions (e.g., $X(u) = x$)) that refer to one specific world $U = u$. Structural models do not permit arbitrary transformations and substitutions, even when truth values are preserved. For example, substituting the expression for c in $y = d \vee c$ would not be permitted if c (cyanide intake) is understood to be governed by a separate mechanism, independent of that which governs y.

Using structural analysis, we will now propose a formal setting that captures the intuitions of Mackie and Lewis. Our analysis will be based on an aspect of causation called

sustenance, which combines elements of sufficiency and necessity and also takes structural information into account.

10.2 PRODUCTION, DEPENDENCE, AND SUSTENANCE

The probabilistic concept of causal sufficiency, PS (Definition 9.2.2), suggests a way of rescuing the counterfactual account of causation. Consider again the symmetric overdetermination in the firing-squad example. The shot of each rifleman features a PS value of unity (see (9.43)), because each shot would cause the prisoner's death in a state u' in which the prisoner is alive. This high PS value supports our intuition that each shot is an actual cause of death, despite a low PN value (PN = 0). Thus, it seems plausible to argue that our intuition gives some consideration to sufficiency, and that we could formulate an adequate criterion for actual causation using the right mixture of PN and PS components.

Similar expectations are expressed in Hall (1998). In analyzing problems faced by the counterfactual approach, Hall made the observation that there are two concepts of causation, only one of which is captured by the counterfactual account, and that failure to capture the second concept may well explain its clashes with intuition. Hall calls the first concept "dependence" and the second "production." In the firing-squad example, intuition considers each shot to be an equal "producer" of death. In contrast, the counterfactual account tests for "dependence" only, and it fails because the state of the prisoner does not "depend" on either shot alone.

The notions of dependence and production closely parallel those of necessity and sufficiency, respectively. Thus, our formulation of PS could well provide the formal basis for Hall's notion of production and serve as a step toward the formalization of actual causation. However, for this program to succeed, a basic hurdle must first be overcome: productive causation is oblivious to scenario-specific information (Pearl 1999), as can be seen from the following considerations.

The ***dependence*** aspect of causation appeals to the necessity of a cause x in maintaining the effect y in the face of certain contingencies, which otherwise will negate y (Definition 9.2.1):

$$X(u) = x, \quad Y(u) = y, \quad Y_{x'}(u) = y'. \tag{10.3}$$

The ***production*** aspect, on the other hand, appeals to the capacity of a cause (x) to bring about the effect (y) in a situation (u') where both are absent (Definition 9.2.2):

$$X(u') = x', \quad Y(u') = y', \quad Y_x(u') = y. \tag{10.4}$$

Comparing these two definitions, we note a peculiar feature of production: To test production, we must step outside our world momentarily, imagine a new world u' with x and y absent, apply x, and see if y sets in. Therefore, the sentence "x produced y" can be true only in worlds u' where x and y are false, and thus it appears (a) that nothing could possibly explain (by consideration of production) any events that did materialize in the actual world and (b) that evidence gathered about the actual world u could not be brought to bear on the hypothetical world u' in which production is defined.

To overcome this hurdle, we resort to an aspect of causation called sustenance, which enriches the notion of dependence with features of production while remaining in a world

enemy 1 into the actual cause of death, contrary to intuition and contrary to the actual scenario (which excludes cyanide intake). The notion of "causal beam" (Pearl 1998b) is devised to make the choice of W minimally disruptive to the actual scenario.[5]

10.3 CAUSAL BEAMS AND SUSTENANCE-BASED CAUSATION

10.3.1 Causal Beams: Definitions and Implications

We start by considering a causal model M, as defined in Section 7.1, and selecting a subset S of *sustaining* parent variables for each family and each u. Recall that the arguments of the functions $\{f_i\}$ in a causal model were assumed to be minimal in some sense, since we have pruned from each f_i all redundant arguments and retained only those called pa_i that render $f_i(pa_i, u)$ nontrivial (Definition 7.1.1). However, in that definition we were concerned with nontriviality relative to all possible u; further pruning is feasible when we are situated at a particular state $U = u$.

To illustrate, consider the function $f_i = ax_1 + bux_2$. Here $PA_i = \{X_1, X_2\}$, because there is always some value of u that would make f_i sensitive to changes in either x_1 or x_2. However, given that we are in a state for which $u = 0$, we can safely consider X_2 to be a trivial argument, replace f_i with $f_i^0 = ax_1$, and consider X_1 as the only *essential* argument of f_i'. We shall call f_i^0 the *projection* of f_i on $u = 0$; more generally, we will consider the projection of the entire model M by replacing every function in $\{f_i\}$ with its projection relative to a specific u and a specific value of its nonessential part. This leads to a new model, which we call *causal beam.*

Definition 10.3.1 (Causal Beam)
For model $M = \langle U, V, \{f_i\}\rangle$ *and state* $U = u$, *a* causal beam *is a new model* $M_u = \langle u, V, \{f_i^u\}\rangle$ *in which the set of functions* f_i^u *is constructed from* $\{f_i\}$ *as follows.*

1. *For each variable* $V_i \in V$, *partition* PA_i *into two subsets,* $PA_i = S \cup \bar{S}$, *where* S *(connoting "sustaining") is any subset of* PA_i *satisfying*[6]

$$f_i(S(u), \bar{s}, u) = f_i(S(u), \bar{s'}, u) \text{ for all } \bar{s'}. \tag{10.6}$$

   In words, S *is any set of* PA_i *sufficient to entail the actual value of* $V_i(u)$, *regardless of how we set the other members of* PA_i.

2. *For each variable* $V_i \in V$, *find a subset* W *of* $\bar{S}$ *for which there exists some realization* $W = w$ *that renders the function* $f_i(s, \bar{S}_w(u), u)$ *nontrivial in* s; *that is,*

$$f_i(s', \bar{S}_w(u), u) \neq V_i(u) \text{ for some } s'.$$

[5] Halpern and Pearl (1999) permit the choice of any set W such that its complement, $Z = V - W$, is sustained by x; that is, $Z_{xw}(u) = Z(u)$ for all w.

[6] Pearl (1998b) required that S be minimal, but this restriction is unnecessary for our purposes (though all our examples will invoke minimally sufficient sets). As usual, we use lowercase letters (e.g., $s, \bar{s}$) to denote specific realizations of the corresponding variables (e.g., $S, \bar{S}$) and use $S_x(u)$ to denote the realization of S under $U = u$ and $do(X = x)$. Of course, each parent set PA_i, would have a distinct partition $PA_i = S_i \cup \bar{S_i}$, but we drop the i index for clarity.

Here, $\bar{S}$ should not intersect the sustaining set of any other variable V_j, $j \neq i$. (Likewise, setting $W = w$ should not contradict any such setting elsewhere.)

3. *Replace $f_i(s, \bar{s}, u)$ by its projection $f_i^u(s)$, which is given by*

$$f_i^u(s) = f_i(s, \bar{S}_w(u), u). \tag{10.7}$$

Thus the new parent set of V_i becomes $PA_i^u = S$, and every f^u function is responsive to its new parent set S.

Definition 10.3.2 (Natural Beam)
A causal beam M_u is said to be natural *if condition 2 of Definition 10.3.1 is satisfied with $W = \emptyset$ for all $V_i \in V$.*

In words, a natural beam is formed by "freezing" all variables outside the sustaining set at their actual values, $\bar{S}(u)$, thus yielding the projection $f_i^u(s) = f_i(s, \bar{S}(u), u)$.

Definition 10.3.3 (Actual Cause)
We say that event $X = x$ was an actual cause *of $Y = y$ in a state u (abbreviated "x caused y") if and only if there exists a natural beam M_u such that*

$$Y_x = y \text{ in } M_u \tag{10.8}$$

and

$$Y_{x'} \neq y \text{ in } M_u \quad \text{for some } x' \neq x. \tag{10.9}$$

Note that (10.8) is equivalent to

$$Y_x(u) = y, \tag{10.10}$$

which is implied by $X(u) = x$ and $Y(u) = y$. But (10.9) ensures that, after "freezing the trivial surroundings" represented by $\bar{S}$, $Y = y$ would not be sustained by some value x' of X.

Definition 10.3.4 (Contributory Cause)
We say that x is a contributory cause *of y in a state u if and only if there exists a causal beam, but no natural beam, that satisfies* (10.8) *and* (10.9).

In summary, the causal beam can be interpreted as a theory that provides a sufficient and nontrivial explanation for each actual event $V_i(u) = v_i$ under a hypothetical freezing of some variables ($\bar{S}$) by the $do(\cdot)$ operator. Using this new theory, we subject the event $X = x$ to a counterfactual test and check whether Y would change if X were not x. If a change occurs in Y when freezing takes place at the actual values of $\bar{S}$ (i.e., $W = \emptyset$), we say that "x was an actual cause of y." If changes occur only under a freeze state that is removed from the actual state (i.e., $W \neq \emptyset$), we say that "x was a contributory cause of y."

Remark: Although W was chosen to make V_i responsive to S, this does not guarantee that $S(u)$ is necessary and sufficient for $V_i(u)$ because local responsiveness

does not preclude the existence of another state $s'' \neq S(u)$ for which $f_i^u(s'') = V_i(u)$. Thus, (10.8) does not guarantee that x is both necessary and sufficient for y. That is the reason for the final counterfactual test in (10.9). It would be too restrictive to require that w render f^u nontrivial for every s of S; such a W may not exist. If (10.8)–(10.9) are satisfied, then $W = w$ represents some hypothetical modification of the world model under which x is both sufficient and necessary for y.

Remarks on Multivariate Events: Although Definitions 10.3.3 and 10.3.4 apply to univariate as well as multivariate causes and effects, some refinements are in order when X and Y consist of sets of variables.[7] If the effect considered, E, is any Boolean function of a set $Y = \{Y_1, \ldots, Y_k\}$ of variables, then (10.8) should apply to every member Y_i of Y and (10.9) should be modified to read $Y_{x'} \Longrightarrow \neg E$ instead of $Y_{x'} \neq y$. Additionally, if X consists of several variables then it is reasonable to demand that X be minimal – in other words, to demand that no subset of those variables passes the test of (10.8)–(10.9). This requirement strips X from irrelevant, overspecified details. For example, if drinking poison qualifies as the actual cause of Joe's death then, awkwardly, drinking poison and sneezing would also pass the test of (10.8)–(10.9) and qualify as the cause of Joe's death. Minimality removes "sneezing" from the causal event $X = x$.

Incorporating Probabilities and Evidence

Suppose that the state u is uncertain and that the uncertainty is characterized by the probability $P(u)$. If e is the evidence available in the case, then the probability that x caused y can be obtained by summing up the weight of evidence $P(u \mid e)$ over all states u in which the assertion "x caused y" is true.

Definition 10.3.5 (Probability of Actual Causation)
Let U_{xy} be the set of states in which the assertion "x is an actual cause of y" is true (Definition 10.3.2), and let U_e be the set of states compatible with the evidence e. The probability that x caused y in light of evidence e, denoted $P(\text{caused}(x, y \mid e))$, is given by the expression

$$P(\text{caused}(x, y \mid e)) = \frac{P(U_{xy} \cap U_e)}{P(U_e)}. \tag{10.11}$$

10.3.2 Examples: From Disjunction to General Formulas

Overdetermination and Contributory Causes

Contributory causation is typified by cases where two actions concur to bring about an event yet either action, operating alone, would still have brought about the event. In such cases the model consists of just one mechanism, which connects the effect E to the two

[7] These were formulated by Joseph Halpern in the context of the definition presented in Halpern and Pearl (1999).

actions through a simple disjunction: $E = A_1 \vee A_2$. There exists no natural beam to qualify either A_1 or A_2 as an actual cause of E. If we fix either A_1 or A_2 at its current value (namely, true), then E will become a trivial function of the other action. However, if we deviate from the current state of affairs and set A_2 to false (i.e., forming a beam with $W = \{A_2\}$ and setting W to false), then E would then become responsive to A_1 and so pass the counterfactual test of (10.9).

This example illustrates the sense in which the beam criterion encapsulates Lewis's notion of quasi-dependence. Event E can be considered quasi-dependent on A_1 if we agree to test such dependence in a hypothetical submodel created by the $do(A_2 = \text{false})$ operator. In Section 10.2 we argued that such a hypothetical test – though it conflicts with the current scenario u – is implicitly written into the charter of every causal model. A causal beam may thus be considered a formal explication of Lewis's notion of a quasi-dependent process, and the combined sets W represent the "peculiar surroundings" of the process that (when properly modified) renders $X = x$ necessary for $Y = y$.

Disjunctive Normal Form

Consider a single mechanism characterized by the Boolean function

$$y = f(x, z, r, h, t, u) = xz \vee rh \vee t,$$

where (for simplicity) the variables X, Z, R, H, T are assumed to be causally independent of each other (i.e., none is a descendant of another in the causal graph $G(M)$). We next illustrate conditions under which x would qualify as a contributory or an actual cause for y.

First, consider a state $U = u$ where all variables are true:

$$X(u) = Z(u) = R(u) = H(u) = T(u) = Y(u) = \text{true}.$$

In this state, every disjunct represents a minimal set of sustaining variables. In particular, taking $S = \{X, Z\}$, we find that the projection $f^u(x, z) = f(x, z, R(u), H(u), T(u))$ becomes trivially true. Thus, there is no natural beam M_u, and x could not be the actual cause of y. Feasible causal beams can be obtained by using $w = \{r', t'\}$ or $w = \{h', t'\}$, where primes denote complementation. Each of these two choices yields the projection $f^u(x, z) = xz$. Clearly, M_u meets the conditions of (10.8) and (10.9), thus certifying x as a contributory cause of y.

Using the same argument, it is easy to see that, at a state u' for which

$$X(u') = Z(u') = \text{true} \quad \text{and} \quad R(u') = T(u') = \text{false},$$

a natural beam exists; that is, a nontrivial projection $f^{u'}(x, z) = xz$ is realized by setting the redundant ($\bar{S}$) variables R, H, and T to their actual values in u'. Hence, x qualifies as an actual cause of y.

This example illustrates how Mackie's intuition for the INUS condition can be explicated in the structural framework. It also illustrates the precise roles played by structural (or "dispositional") knowledge (e.g., $f_i(pa_i, u)$) and circumstantial knowledge ($X(u) = \text{true}$), which were not clearly distinguished by the strictly logical account.

The next example illustrates how the INUS condition generalizes to arbitrary Boolean functions, especially those having several minimal disjunctive normal forms.

Single Mechanism in General Boolean Form

Consider the function

$$y = f(x, z, h, u) = xz' \vee x'z \vee xh', \tag{10.12}$$

which has the equivalent form

$$y = f(x, z, h, u) = xz' \vee x'z \vee zh'. \tag{10.13}$$

Assume, as before, that (a) we consider a state u in which X, Z, and H are true and (b) we inquire as to whether the event $x : X =$ true caused the event $y : Y =$ false. In this state, the only sustaining set is $S = \{X, Z, R\}$, because no choice of two variables (valued at this u) would entail $Y =$ false regardless of the third. Since $\bar{S}$ is empty, the choice of beam is unique: $M_u = M$, for which $y = f^u(x, z, h) = xz' \vee x'z \vee xh'$. This M_u passes the counterfactual test of (10.9), because $f^u(x', z, h) =$ true; we therefore conclude that x was an actual cause of y. Similarly, we can see that the event $H =$ true was an actual cause of $Y =$ false. This follows directly from the counterfactual test

$$Y_h(u) = \text{false} \quad \text{and} \quad Y_{h'}(u) = \text{true}.$$

Because Definitions 10.3.3 and 10.3.4 rest on semantical considerations, identical conclusions would be obtained from any logically equivalent form of f (not necessarily in minimal disjunctive form) – as long as f represents a single mechanism. In simple, single-mechanism models, the beam criterion can therefore be considered the semantical basis behind the INUS intuition. The structure-sensitive aspects of the beam criterion will surface in the next two examples, where models of several layers are considered.

10.3.3 Beams, Preemption, and the Probability of Single-Event Causation

In this section we apply the beam criterion to a probabilistic version of the desert traveler example. This will illustrate (i) how structural information is utilized in problems involving preemption and (ii) how we can compute the probability that one event "was the actual cause of another," given a set of observations.

Consider a modification of the desert traveler example in which we do not know whether the traveler managed to drink any of the poisoned water before the canteen was emptied. To model this uncertainty, we add a bivalued variable U that indicates whether poison was drunk ($u = 0$) or not ($u = 1$). Since U affects both D and C, we obtain the structure shown in Figure 10.3. To complete the specification of the model, we need to assign functions $f_i(pa_i, u)$ to the families in the diagram and a probability distribution $P(u)$. To formally complete the model, we introduce the dummy background variables U_X and U_P, which represent the factors behind the enemies' actions.

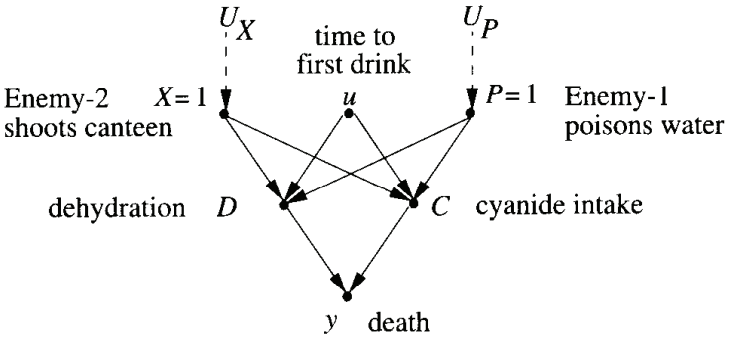


Figure 10.3 Causal relationships for the probabilistic desert traveler.

The usual understanding of the story yields the following functional relationships:

$$c = p(u' \vee x'),$$

$$d = x(u \vee p'),$$

$$y = c \vee d,$$

together with the evidential information

$$X(u_X) = 1, \qquad P(u_P) = 1.$$

(We assume that T will not survive with an empty canteen (x) even after drinking unpoisoned water before the shot ($p'u'$).)

In order to construct the causal beam M_u, we examine each of the three functions and form their respective projections on u. For example, for $u = 1$ we obtain the functions shown in (10.1), for which the (minimal) sustaining parent sets are: X (for C), X (for D), and D (for Y). The projected functions become

$$\begin{aligned} c &= x', \\ d &= x, \\ y &= d, \end{aligned} \tag{10.14}$$

and the beam model $M_{u=1}$ is natural; its structure is depicted in Figure 10.4. To test whether x (or p) was the cause of y, we apply (10.8)–(10.9) and obtain

$$\begin{aligned} &Y_x = 1 \text{ and } Y_{x'} = 0 \text{ in } M_{u=1}, \\ &Y_p = 1 \text{ and } Y_{p'} = 1 \text{ in } M_{u=1}. \end{aligned} \tag{10.15}$$

Thus, enemy 2 shooting at the container (x) is classified as the actual cause of T's death (y), whereas enemy 1 poisoning the water (p) was not the actual cause of y.

Next, consider the state $u = 0$, which denotes the event that our traveler reached for a drink before enemy 2 shot at the canteen. The graph corresponding to $M_{u=0}$ is shown in Figure 10.5 and gives

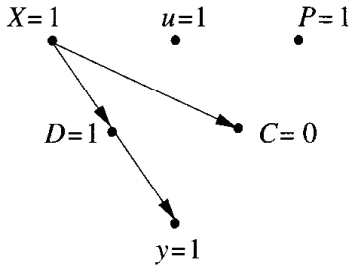


Figure 10.4 Natural causal beam representing the state $u = 1$.

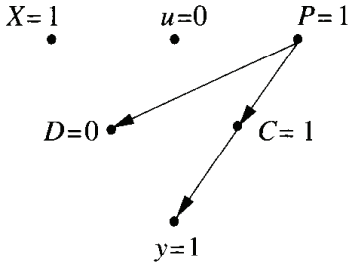


Figure 10.5 Natural causal beam representing the state $u = 0$.

$$\begin{aligned} &Y_x = 1 \text{ and } Y_{x'} = 1 \text{ in } M_{u=0}, \\ &Y_p = 1 \text{ and } Y_{p'} = 0 \text{ in } M_{u=0}. \end{aligned} \tag{10.16}$$

Thus, in this state of affairs we classify enemy 1's action to be the actual cause of T's death, while enemy 2's action is not considered the cause of death.

If we do not know which state prevailed, $u = 1$ or $u = 0$, then we must settle for the *probability* that x caused y. Likewise, if we observe some evidence e reflecting on the probability $P(u)$, such evidence would yield (see (10.11))

$$P(\text{caused}(x, y \mid e)) = P(u = 1 \mid e)$$

and

$$P(\text{caused}(p, y \mid e)) = P(u = 0 \mid e).$$

For example, a forensic report confirming "no cyanide in the body" would rule out state $u = 0$ in favor of $u = 1$, and the probability of x being the cause of y becomes 100%. More elaborate probabilistic models are analyzed in Pearl (1999).

10.3.4 Path-Switching Causation

Example 10.3.6 Let x be the state of a two-position switch. In position 1 ($x = 1$), the switch turns on a lamp ($z = 1$), and turns off a flashlight ($w = 0$). In position 0 ($x = 0$), the switch turns on the flashlight ($w = 1$) and turns off the lamp ($z = 0$). Let $Y = 1$ be the proposition that the room is lighted.

The causal beams M_u and $M_{u'}$ associated with the states in which the switch is in position 1 and 2 (respectively) are shown in the graphs of Figure 10.6. Once again, M_u entails

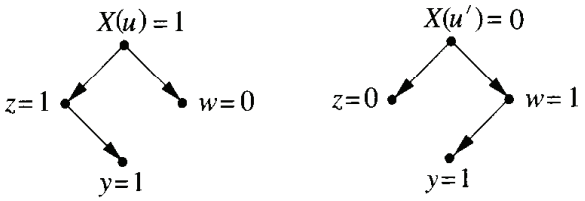


Figure 10.6 Natural beams that represent path switching in Example 10.3.6.

$Y_x = 1$ and $Y_{x'} = 0$. Likewise $M_{u'}$ entails $Y_x = 1$ and $Y_{x'} = 0$. Thus "switch in position 1" and "switch in position 2" are *both* considered actual causes for "room is lighted," although neither is a necessary cause.

This example further highlights the subtlety of the notion of "actual cause"; changing X from 1 to 0 merely changes the course of the causal pathway while keeping its source and destination the same. Should the current switch position ($X = 1$) be considered the actual cause of (or an "explanation of") the light in the room? Although $X = 1$ enables the passage of electric current through the lamp and is in fact the only mechanism currently sustaining light, one may argue that it does not deserve the title "cause" in ordinary conversation. It would be odd to say, for instance, that $X = 1$ was the cause of spoiling an attempted burglary. However, recalling that causal explanations earn their value in the abnormal circumstances created by structural contingencies, the possibility of a malfunctioning flashlight should enter our mind whenever we designate it as a separate mechanism in the model. Keeping this contingency in mind, it should not be too odd to name the switch position as a cause of spoiling the burglary.

10.3.5 Temporal Preemption

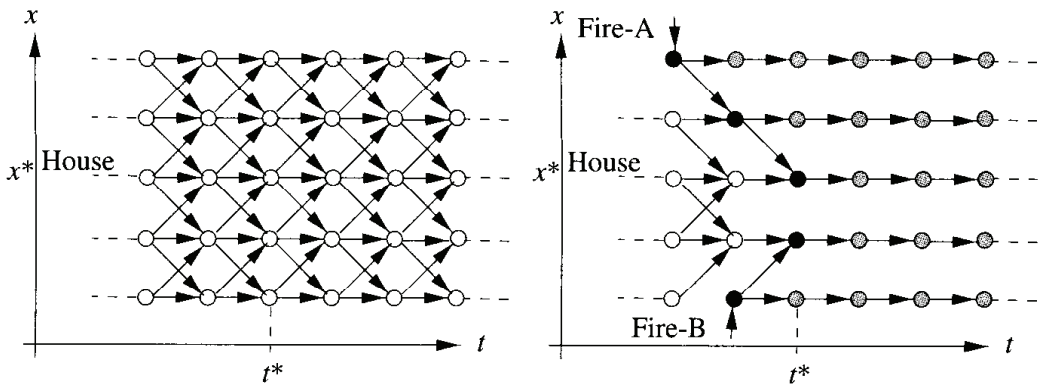
Consider the example mentioned in the preface of this chapter, in which two fires are advancing toward a house. If fire A burned the house before fire B then we would consider fire A "the actual cause" for the damage, even though fire B would have done the same were it not for A. If we simply write the structural model as

$$H = A \vee B,$$

where H stands for "house burns down," then the beam method would classify each fire as an equally contributory cause, which is counterintuitive – fire B is not regarded as having made any contribution to H.

This example is similar to yet differs from the desert traveler; here, the way in which one cause preempts the other is more subtle in that the second cause becomes ineffective only because the effect has already happened. Hall (1998) regards this sort of preemption as equivalent to ordinary preemption, and he models it by a causal diagram in which H, once activated, inhibits its own parents. Such inhibitory feedback loops lead to irreversible behavior, contrary to the unique-solution assumption of Definition 7.1.1.

A more direct way of expressing the fact that a house, once burned, will remain burned even when the causes of fire disappear is to resort to dynamic causal models (as in Figure 3.3), in which variables are time-indexed. Indeed, it is impossible to capture temporal relationships such as "arriving first" by using the static causal models defined in Section 7.1; instead, dynamic models must be invoked.



(a) (b)

Figure 10.7 (a) Causal diagram associated with the dynamic model of (10.17). (b) Causal beam associated with starting fire A and fire B at different times, showing no connection between fire B and the state of the house at $x = x^*$.

Let the state of the fire $V(x, t)$ at location x and time t take on three values: g (for green), f (for on fire), and b (for burned). The dynamic structural equations characterizing the propagation of fire can then be written (in simplified form) as:

$$V(x,t) = \begin{cases} f & \text{if } V(x,t-1) = g \text{ and } V(x-1,t-1) = f, \\ f & \text{if } V(x,t-1) = g \text{ and } V(x+1,t-1) = f, \\ b & \text{if } V(x,t-1) = b \text{ and } V(x,t-1) = f, \\ g & \text{otherwise.} \end{cases} \tag{10.17}$$

The causal diagram associated with this model is illustrated in Figure 10.7(a), designating three parents for each variable $V(x, t)$: the previous state $V(x + 1, t - 1)$ of its northern neighbor, the previous state $V(x - 1, t - 1)$ of its southern neighbor, and the previous state $V(x, t - 1)$ at location x. The scenario emanating from starting fire A and fire B one time unit apart (corresponding to actions $do(V(x^* + 2, t^* - 2) = f)$ and $do(V(x^* - 2, t^* - 1) = f)$) is shown in Figure 10.7(b). Black and grey bullets represent, respectively, space–time regions in states f (on fire) and b (burned). This beam is both natural and unique, as can be seen from (10.17). The arrows in Figure 10.7(b) represent a natural beam constructed from the (unique) minimally sufficient sets S at each family. The state of the parent set S that this beam assigns to each variable constitutes an event that is both necessary and sufficient for the actual state of that variable (assuming variables in $\bar{S}$ are frozen at their actual values).

Applying the test of (10.9) to this beam, we find that a counterfactual dependence exists between the event $V(x^* - 2, t^* - 2) = f$ (representing the start of fire A) and the sequence $V(x^*, t)$, $t > t^*$ (representing the state of the house through time). No such dependence exists for fire B. On that basis, we classify fire A as the actual cause of the house fire. Remarkably, the common intuition of attributing causation to an event that hastens the occurrence of the effect is seen to be a corollary of the beam test in the spatiotemporal representation of the story. However, this intuition cannot serve as the

defining principle for actual causation, as suggested by Paul (1998). In our story, for example, each fire alone did not hasten (or delay, or change any property of) the following event: E = the owner of the house did not enjoy breakfast the next day. Yet we still consider fire A, not B, to be the actual cause of E, as predicted by the beam criterion.

The conceptual basis of this criterion can be illuminated by examining the construction of the minimal beam shown in Figure 10.7(b). The pivotal step in this construction lies in the space–time region (x^*, t^*), which represents the house at the arrival of fire. The variable representing the state of the house at that time, $V(x^*, t^*)$, has a two-parent sustaining set, $S = \{V(x^* + 1, t^* - 1)$ and $V(x^*, t^* - 1)\}$, with values f and g, respectively. Using (10.17), we see that the south parent $V(x^* - 1, t^* - 1)$ is redundant, because the value of $V(x^*, t^*)$ is determined (at f) by the current values of the other two parents. Hence, this parent can be excluded from the beam, rendering $V(x^*, t^*)$ dependent on fire A. Moreover, since the value of the south parent is g, that parent cannot be part of any minimally sustaining set, thus ensuring that $V(x^*, t^*)$ is independent of fire B. (We could, of course, add this parent to S, but $V(x^*, t^*)$ would remain independent of fire B.) The next variable to examine is $V(x^*, t^* + 1)$, with parents $V(x^* - 1, t^*)$, $V(x^*, t^*)$, and $V(x^* - 1, t^*)$ valued at b, f, and f, respectively. From (10.17), the value f of the middle parent is sufficient to ensure the value b for the child variable; hence this parent qualifies as a singleton sustaining set, $S = \{V(x^*, t^*)\}$, which permits us to exclude the other two parents from the beam and so render the child dependent on fire A (through S) but not on fire B. The north and south parents are not, in themselves, sufficient for sustaining the current value (b) of the child node (fires at neighboring regions can cause the house to catch fire but not to become immediately "burned"); hence we must keep the middle parent in S and, in so doing, we render all variables $V(x^*, t)$, $t > t^*$, independent of fire B.

We see that sustenance considerations lead to the intuitive results through two crucial steps: (1) permitting the exclusion (from the beam) of the south parent of every variable $V(x^*, t)$, $t > t^*$, thus maintaining the dependence of $V(x^*, t)$ on fire A; and (2) requiring the inclusion (in any beam) of the middle parent of every variable $V(x^*, t)$, $t > t^*$, thus preventing the dependence of $V(x^*, t)$ on fire B. Step (1) corresponds to selecting the intrinsic process from cause to effect and then suppressing the influence of its nonintrinsic surrounding. Step (2) prevents the growth of causal processes beyond their intrinsic boundaries.

10.4 CONCLUSIONS

We have seen that the property of sustenance (Definition 10.2.1), as embodied in the beam test (Definition 10.3.3), is the key to explicating the notion of actual causation (or "cause in fact," in legal terminology); this property should replace the "but for" test in cases involving multistage scenarios with several potential causes. Sustenance captures the capacity of the putative cause to maintain the value of the effect in the face of structural contingencies and includes the counterfactual test of necessity as a special case, with structural contingencies suppressed (i.e., $W = \emptyset$). We have argued that (a) it is the structural rather than circumstantial contingencies that convey the true meaning of

causal claims and (b) these structural contingencies should therefore serve as the basis for causal explanation. We further demonstrated how explanations based on such contingencies resolve difficulties that have plagued the counterfactual account of single-event causation – primarily difficulties associated with preemption, overdetermination, temporal preemption, and switching causation.

Sustenance, however, does not totally replace production, the second component of sufficiency – that is, the capacity of the putative cause to produce the effect in situations where the effect is absent. In the match–oxygen example (see Section 9.5), for instance, oxygen and a lit match each satisfy the sustenance test of Definition 10.3.3 (with $W = \emptyset$ and $\bar{S} = \emptyset$); hence, each factor would qualify as an actual cause of the observed fire. What makes oxygen an awkward explanation in this case is not its ineptness at sustaining fire against contingencies (the contingency set W is empty) but rather its inability to produce fire in the most common circumstance that we encounter, $U = u'$, in which a match is not struck (and a fire does not break out).

This argument still does not tell us why we should consider such hypothetical circumstances ($U = u'$) in the match–oxygen story and not, say, in any of the examples considered in this chapter, where sustenance ruled triumphantly. With all due respect to the regularity and commonality of worlds $U = u'$ in which a match is not struck, those are nevertheless contrary-to-fact worlds, since a fire did break out. Why, then, should one travel to such a would-be world when issuing an explanation for events (fire) in the actual world?

The answer, I believe, lies in the pragmatics of the explanation sought. The tacit target of explanation in the match–oxygen story is the question: "How could the fire have been prevented?" In view of this target, we have no choice but abandon the actual world (in which fire broke out) and travel to one ($U = u'$) in which agents are still capable of preventing this fire.[8]

A different pragmatics motivates the causal explanation in the switch–light story of Example 10.3.6. Here one might be more concerned with keeping the room lit, and the target question is: "How can we ensure that the room remains lit in the face of unforeseen contingencies?" Given this target, we might as well remain in the comfort of our factual world, $U = u$, and apply the criterion of sustenance rather than production.

It appears that pragmatic issues surrounding our quest for explanation are the key to deciding which facet of causation should be used, and that the mathematical formulation of this pragmatics is a key step toward the automatic generation of adequate explanations. Unfortunately, I must now leave this task for future investigation.

Acknowledgment

My interest in the topic of actual causation was kindled by Don Michie, who spent many e-mail messages trying to convince me that (1) the problem is not trivial and (2) Good's

[8] Herbert Simon has related to me that a common criterion in accident liability cases, often applied to railroad crossing accidents, is the "last clear chance" doctrine: the person liable for a collision is the one who had the last clear chance of avoiding it.

(1961, 1962) measures of causal tendency can be extended to handle individual events. He succeeded with regard to (1), and this chapter is based on a seminar given at UCLA (in the Spring of 1998) in which "actual causation" was the main topic. I thank the seminar participants, Ray Golish, Andrew Lister, Eitan Mendelowitz, Peyman Meshkat, Igor Roizen, and Jin Tian for knocking down two earlier attempts at beams and sustenance and for stimulating discussions leading to the current proposal. Discussions with Clark Glymour, Igal Kvart, Jim Woodward, Ned Hall, Herbert Simon, Gary Schwartz, and Richard Baldwin sharpened my understanding of the philosophical and legal issues involved. Subsequent collaboration with Joseph Halpern helped to polish these ideas further and led to the more general and declarative definition of actual cause reported in Halpern and Pearl (1999).

EPILOGUE

The Art and Science of Cause and Effect

A public lecture delivered November 1996 as part of the UCLA Faculty Research Lectureship Program

The topic of this lecture is causality – namely, our awareness of what causes what in the world and why it matters.

Though it is basic to human thought, causality is a notion shrouded in mystery, controversy, and caution, because scientists and philosophers have had difficulties defining when one event *truly causes* another.

We all understand that the rooster's crow does not cause the sun to rise, but even this simple fact cannot easily be translated into a mathematical equation.

Today, I would like to share with you a set of ideas which I have found very useful in studying phenomena of this kind. These ideas have led to practical tools that I hope you will find useful on your next encounter with a cause and effect.

It is hard to imagine anyone here who is *not* dealing with cause and effect.

Whether you are evaluating the impact of bilingual education programs or running an experiment on how mice distinguish food from danger or speculating about why Julius Caesar crossed the Rubicon or diagnosing a patient or predicting who will win the presidential election, you are dealing with a tangled web of cause–effect considerations.

The story that I am about to tell is aimed at helping researchers deal with the complexities of such considerations, and to clarify their meaning.

This lecture is divided into three parts.

I begin with a brief historical sketch of the difficulties that various disciplines have had with causation.

Next I outline the ideas that reduce or eliminate several of these historical difficulties.


2

Finally, in honor of my engineering background, I will show how these ideas lead to simple practical tools, which will be demonstrated in the areas of statistics and social science.

In the beginning, as far as we can tell, causality was not problematic.

The urge to ask *why* and the capacity to find causal explanations came very early in human development.

The bible, for example, tells us that just a few hours after tasting from the tree of knowledge, Adam is already an expert in causal arguments.

When God asks: "Did you eat from that tree?"

This is what Adam replies: "The woman whom you gave to be with me, She handed me the fruit from the tree; and I ate."

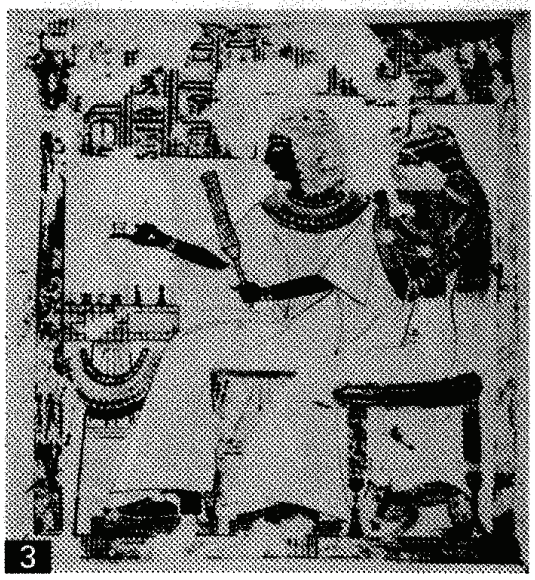
Eve is just as skillful: "The serpent deceived me, and I ate."

The thing to notice about this story is that God did not ask for explanation, only for the facts – it was Adam who felt the need to explain. The message is clear: causal explanation is a man-made concept.

Another interesting point about the story: explanations are used exclusively for passing responsibilities.

Indeed, for thousands of years explanations had no other function. Therefore, only Gods, people, and animals could cause things to happen, not objects, events, or physical processes.

Natural events entered into causal explanations much later because, in the ancient world, events were simply *predetermined*.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Storms and earthquakes were *controlled* by the angry gods [slide 2] and could not in themselves assume causal responsibility for the consequences.

Even an erratic and unpredictable event such as the role of a die [3] was not considered a *chance* event but rather a divine message demanding proper interpretation.

One such message gave the prophet Jonah the scare of his life when he was identified as God's renegade and was thrown *overboard* [4].

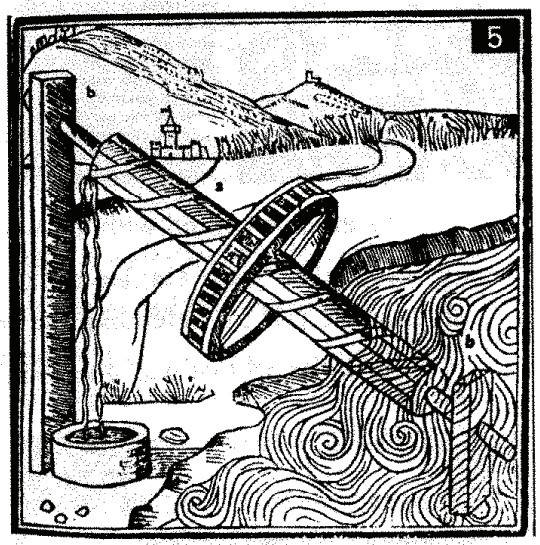
Quoting from the book of Jonah: "And the sailors said: 'Come and let us cast lots to find out who is to blame for this ordeal.' So they cast lots and the lot fell on Jonah."

Obviously, on this luxury Phoenician cruiser, "casting lots" was used not for recreation but for communication – a one-way modem for processing messages of vital importance.

In summary, the agents of causal forces in the ancient world were either deities, who cause things to happen for a purpose, or human beings and animals, who possess free will, for which they are punished and rewarded.

This notion of causation was naive, but clear and unproblematic.

The problems began, as usual, with engineering; when machines had to be constructed to do useful jobs [5].



As engineers grew ambitious, they decided that the earth, too, can be moved [6], but not with a single lever.

Systems consisting of many pulleys and wheels [7], one driving another, were needed for projects of such magnitude.

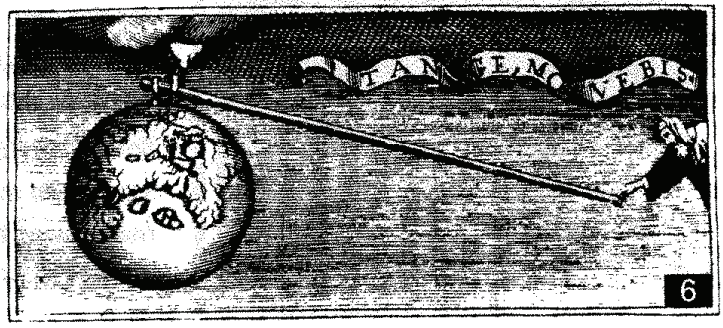
And, once people started building multistage systems, an interesting thing happened to causality – *physical objects began acquiring causal character.*

When a system like that broke down, it was futile to blame God or the operator – instead, a broken rope or a rusty pulley were more useful explanations, simply because these could be replaced easily and make the system work.

At that point in history, Gods and humans ceased to be the sole agents of causal forces – lifeless objects and processes became partners in responsibility.

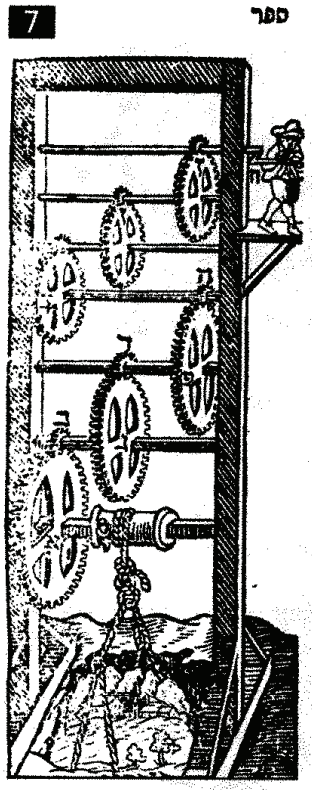
A wheel turned and stopped *because* the wheel preceding it turned and stopped – the human operator became secondary.

Not surprisingly, these new agents of causation *took on* some of the characteristics of their predecessors – Gods and humans.



Natural objects became not only carriers of credit and blame but also carriers of force, will, and even purpose.

Aristotle regarded explanation in terms of a *purpose* to be the only complete and satisfactory explanation for why a thing is what it is.



7

He even called it a *final cause* – namely, the final aim of scientific inquiry.

From that point on, causality served a dual role: *causes* were the targets of credit and blame on one hand and the carriers of physical flow of control on the other.

This duality survived in relative tranquility [8] until about the time of the Renaissance, when it encountered conceptual difficulties.

What happened can be seen on the title page [9] of Recordes's book "The Castle of Knowledge," the first science book in English, published in 1575.

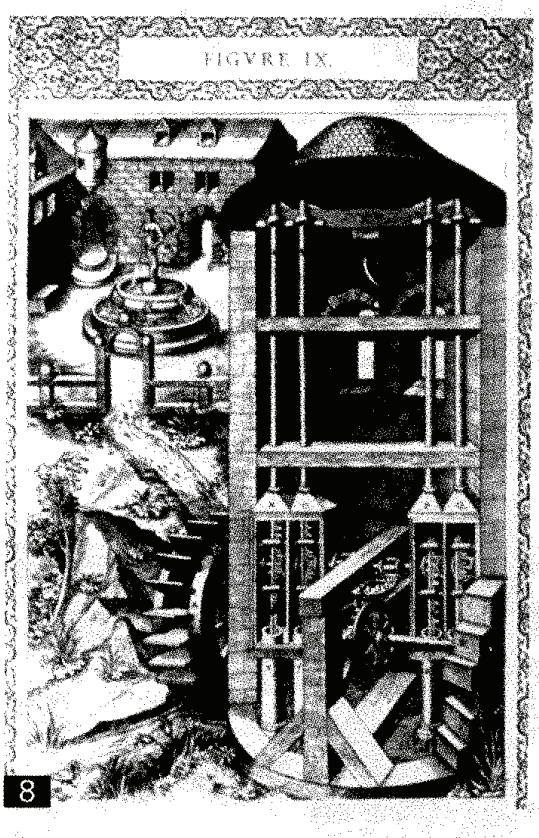
The wheel of fortune is turned, not by the wisdom of God, but by the ignorance of man.

And, as God's role as the final cause was taken over by human knowledge, the whole notion of causal explanation came under attack.

The erosion started with the work of Galileo [10].

Most of us know Galileo as the man who was brought before by the inquisition and imprisoned [11] for defending the heliocentric theory of the world.

But while all that was going on, Galileo also managed to quietly engineer the most profound revolution that science has ever known.



8

This revolution, expounded in his 1638 book "Discorsi" [12], published in Leyden, far from Rome, consists of two maxims:

One, description first, explanation second – that is, the "how" precedes the "why"; and

Two, description is carried out in the language of mathematics; namely, equations.

Ask not, said Galileo, whether an object falls because it is pulled from below or pushed from above.

Ask how well you can predict the time it takes for the object to travel a certain distance, and how that time will vary from object to object and as the angle of the track changes.

Moreover, said Galileo, do not attempt to answer such questions in the qualitative and slippery nuances of human language; say it in the form of mathematical equations [13].

9

It is hard for us to appreciate today how strange that idea sounded in 1638, barely 50 years after the introduction of algebraic notation by Vieta. To proclaim algebra the *universal* language of science would sound today like proclaiming Esperanto the language of economics.

Why would Nature agree to speak algebra? Of all languages?

But you can't argue with success.

The distance traveled by an object turned out indeed to be proportional to the square of the time.

Even more successful than predicting outcomes of experiments were the computational aspects of algebraic equations.

They enabled engineers, for the first time in history, to ask "how to" questions in addition to "what if" questions.

In addition to asking: "What if we narrow the beam, will it carry the load?", they began to ask more difficult questions: "How to shape the beam so that it *will* carry the load?" [14]

This was made possible by the availability of methods for solving equations.

The algebraic machinery does not discriminate among variables; instead of predicting behavior in terms of parameters, we can turn things around and solve for the parameters in terms of the desired behavior.

Let us concentrate now on Galileo's first maxim – "description first, explanation second" – because that idea was taken very seriously by the scientists and changed the character of science from speculative to empirical.

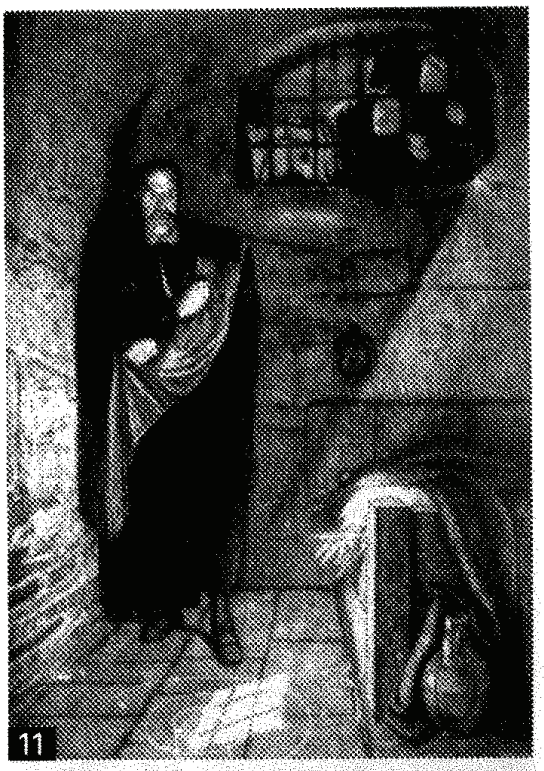

10

Physics became flooded with empirical laws that were extremely useful.

Snell's law [15], Hooke's law, Ohm's law, and Joule's law are examples of purely empirical generalizations that were discovered and used much before they were explained by more fundamental principles.

Philosophers, however, were reluctant to give up the idea of causal explanation and continued to search for the origin and justification of those successful Galilean equations.

For example, Descartes ascribed cause to *eternal truth*.



11

Liebniz made cause a *self-evident logical law.*

Finally, about one hundred years after Galileo, a Scottish philosopher by the name of David Hume [16] carried Galileo's first maxim to an extreme [17].

Hume argued convincingly that the *why* is not merely second to the *how,* but that the *why* is totally superfluous as it is subsumed by the *how.*

On page 156 of Hume's "Treatise of Human Nature" [18], we find the paragraph that shook up causation so thoroughly that it has not recovered to this day.

I always get a kick reading it: "Thus we remember to have seen that species of object we call *flame,* and to have felt that species of sensation we call *heat.* We likewise call to mind their constant conjunction in all past instances. Without any farther ceremony, we call the one *cause* and the other *effect,* and infer the existence of the one from that of the other."

Thus, causal connections according to Hume are the product of observations. Causation is a learnable habit of the mind, almost as fictional as optical illusions and as transitory as Pavlov's conditioning.

DISCORSI
E
DIMOSTRAZIONI
MATEMATICHE,
intorno à due nuoue ſcienze
Attenenti alla
MECANICA & i MOVIMENTI LOCALI;
del Signor
GALILEO GALILEI LINCEO,
Filoſofo e Matematico primario del Sereniſſimo
Grand Duca di Toſcana.
Con vna Appendice del centro di grauità d' alcuni Solidi.

IN LEIDA,
Appreſſo gli Elſeuirii. M. D. C. XXXVIII.

12

It is hard to believe that Hume was not aware of the difficulties inherent in his proposed recipe.

He knew quite well that the rooster crow *stands* in constant conjunction to the sunrise, yet it does not *cause* the sun to rise.

He knew that the barometer reading *stands* in constant conjunction to the rain but does not *cause* the rain.

Today these difficulties fall under the rubric of *spurious correlations,* namely "correlations that do not imply causation."

Now, taking Hume's dictum that all knowledge comes from experience encoded in the mind as correlation, and our observation that correlation does not imply causation, we are led into our first riddle of causation: How do people *ever* acquire knowledge of *causation*?

We saw in the rooster example that regularity of succession is not sufficient; what *would* be sufficient?

What patterns of experience would justify calling a connection "causal"?

Moreover: What patterns of experience *convince* people that a connection is "causal"?

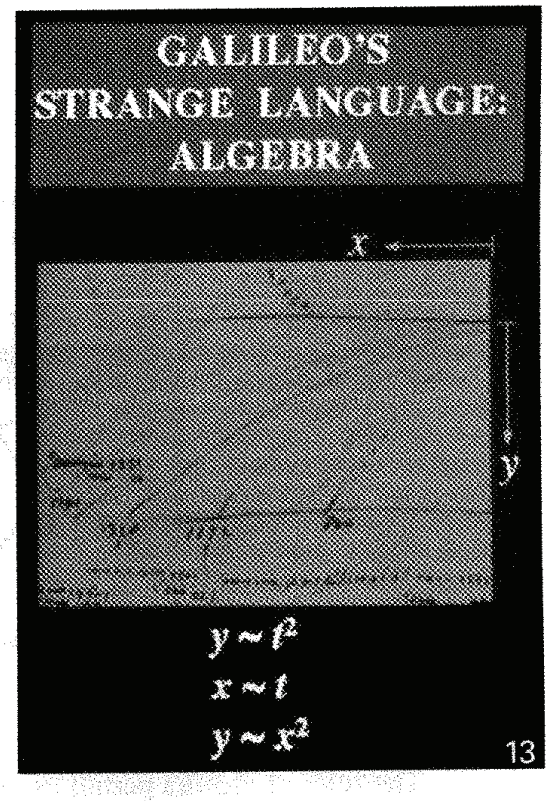
If the first riddle concerns the *learning* of causal connection, the second concerns its usage: What *difference* does it make if I told you that a certain connection is or is not causal?

Continuing our example, what difference does it make if I told you that the rooster does cause the sun to rise?

This may sound trivial.

The obvious answer is that knowing "what causes what" makes a big difference in how we act.

If the rooster's crow causes the sun to rise, we could make the night shorter by waking up our rooster earlier and making him crow – say, by telling him the latest rooster joke.



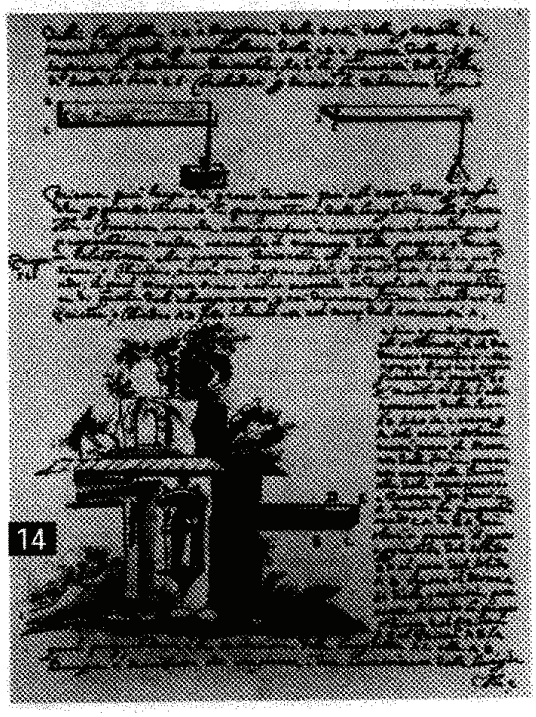
But this riddle is *not* as trivial as it seems.

If causal information has an empirical meaning beyond regularity of succession, then that information should show up in the laws of physics.

But it does not!

The philosopher Bertrand Russell made this argument [19] in 1913:

"All philosophers," says Russell, "imagine that causation is one of the fundamental axioms of science, yet oddly enough, in advanced sciences, the word 'cause' never occurs.... The law of causality, I believe, is a relic of bygone age, surviving, like the monarchy, only because it is erroneously supposed to do no harm."

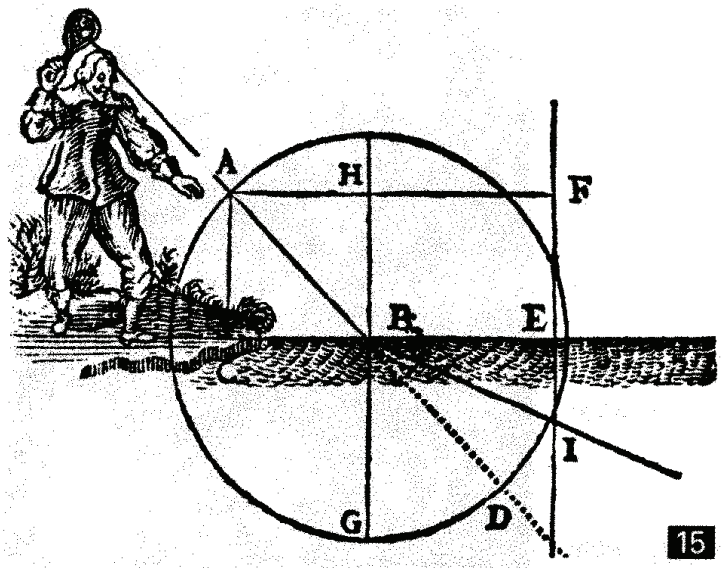


Another philosopher, Patrick Suppes, who argued for the importance of causality, noted that:

"There is scarcely an issue of 'Physical Review' that does not contain at least one article using either 'cause' or 'causality' in its title."

What we conclude from this exchange is that physicists talk, write, and think one way and formulate physics in another.

Such bilingual activity would be forgiven if causality was used merely as a convenient communication device – a shorthand for expressing complex patterns of physical relationships that would otherwise take many equations to write.



15

After all! Science is full of abbreviations: We use "multiply x by 5" instead of "add x to itself 5 times"; we say "density" instead of "the ratio of weight to volume."

Why pick on causality?

"Because causality is different," Lord Russell would argue, "It could not possibly be an abbreviation, because the laws of physics are all symmetrical, going both ways, while causal relations are unidirectional, going from cause to effect."

Take, for instance, Newton's law:

$$f = ma.$$

The rules of algebra permit us to write this law in a wild variety of syntactic forms, all meaning the same thing – that if we know any two of the three quantities, the third is determined.

Yet, in ordinary discourse we say that force causes acceleration – not that acceleration causes force, and we feel very strongly about this distinction.


16

Likewise, we say that the ratio f/a helps us *determine* the mass, not that it *causes* the mass.

Such distinctions are not supported by the equations of physics, and this leads us to ask whether the whole causal vocabulary is purely metaphysical, "surviving, like the monarchy ...".

Fortunately, very few physicists paid attention to Russell's enigma. They continued to write equations in the office and talk cause–effect in the *cafeteria*; with astonishing success they smashed the atom, invented the transistor and the laser.

The same is true for engineering.

But in another arena the tension could not go unnoticed, because in that arena the demand for distinguishing causal from other relationships was very explicit.

This arena is statistics.

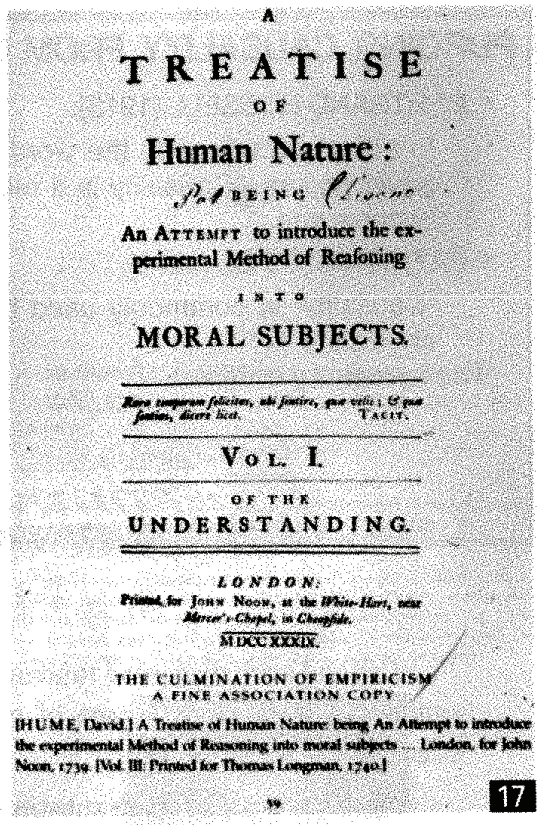
The story begins with the discovery of correlation, about one hundred years ago.

Francis Galton [20], inventor of fingerprinting and cousin of Charles Darwin, quite understandably set out to prove that talent and virtue run in families.

Galton's investigations drove him to consider various ways of measuring how properties of one class of individuals or objects are related to those of another class.

In 1888, he measured the length of a person's forearm and the size of that person's head and asked to what degree can one of these quantities predict the other [21].

He stumbled upon the following discovery: If you plot one quantity against the other and scale the two axes properly, then the slope of the best-fit line has some nice mathematical properties. The slope is 1 only when one quantity can predict the other precisely; it is zero whenever the prediction is no better than a random guess, and, most remarkably, the slope is the same no matter if you plot X against Y or Y against X.


A

TREATISE

OF

Human Nature:

BEING

An ATTEMPT to introduce the experimental Method of Reasoning

INTO

MORAL SUBJECTS.

Rara temporum felicitate, ubi sentire, quæ velis; & quæ sentias, dicere licet. TACIT.

VOL. I.

OF THE

UNDERSTANDING.

LONDON:

Printed for JOHN NOON, at the White-Hart, near Mercer's-Chapel, in Cheapside.

MDCCXXXIX.

THE CULMINATION OF EMPIRICISM
A FINE ASSOCIATION COPY

[HUME, David.] A Treatise of Human Nature: being An Attempt to introduce the experimental Method of Reasoning into moral subjects ... London, for John Noon, 1739. [Vol. III: Printed for Thomas Longman, 1740.]

17

"It is easy to see," said Galton, "that co-relation must be the consequence of the variations of the two organs being partly due to common causes."

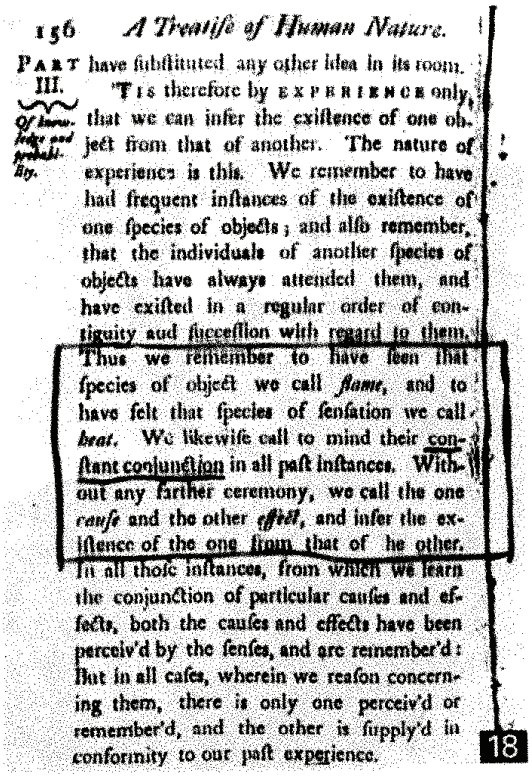
Here we have, for the first time, an objective measure of how two variables are "related" to each other, based strictly on the data, clear of human judgment or opinion.

Galton's discovery dazzled one of his disciples, Karl Pearson [22], now considered to be one of the founders of modern statistics.

Pearson was 30 years old at the time, an accomplished physicist and philosopher about to turn lawyer, and this is how he describes, 45 years later [23], his initial reaction to Galton's discovery:

"I felt like a buccaneer of Drake's days ....

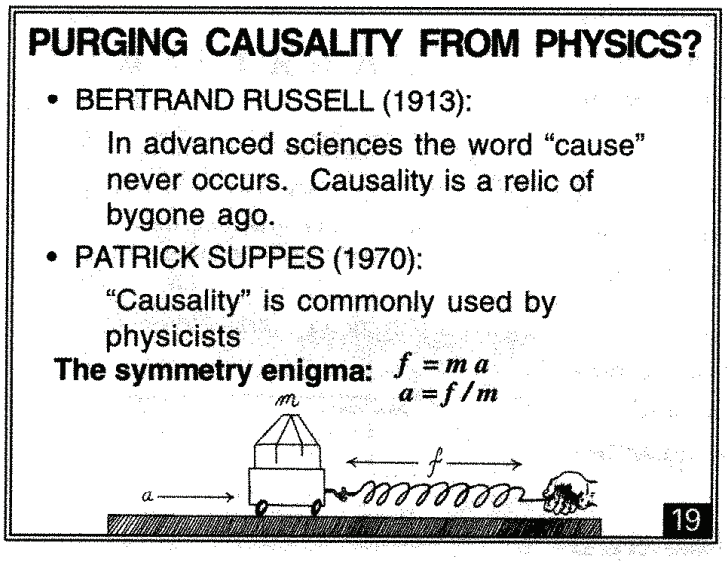
"I interpreted ... Galton to mean that there was a category broader than causation, namely correlation, of which causation was only the limit, and that this new conception of correlation brought psychology, anthropology, medicine, and sociology in large parts into the field of mathematical treatment."


156 *A Treatiſe of Human Nature.*

PART III. Of knowledge and probability.

have ſubſtituted any other idea in its room. 'Tis therefore by EXPERIENCE only, that we can infer the exiſtence of one object from that of another. The nature of experience is this. We remember to have had frequent inſtances of the exiſtence of one ſpecies of objects; and alſo remember, that the individuals of another ſpecies of objects have always attended them, and have exiſted in a regular order of contiguity and ſucceſſion with regard to them. Thus we remember to have ſeen that ſpecies of object we call *flame*, and to have felt that ſpecies of ſenſation we call *heat*. We likewiſe call to mind their conſtant conjunction in all paſt inſtances. Without any farther ceremony, we call the one *cauſe* and the other *effect*, and infer the exiſtence of the one from that of the other. In all thoſe inſtances, from which we learn the conjunction of particular cauſes and effects, both the cauſes and effects have been perceiv'd by the ſenſes, and are remember'd: But in all caſes, wherein we reaſon concerning them, there is only one perceiv'd or remember'd, and the other is ſupply'd in conformity to our paſt experience.

18



Now, Pearson has been described as a man "with the kind of drive and determination that took Hannibal over the Alps and Marco Polo to China."

When Pearson felt like a buccaneer, you can be sure he gets his bounty.

The year 1911 saw the publication of the third edition of his book "The Grammar of Science." It contained a new chapter titled "Contingency and Correlation – The Insufficiency of Causation," and this is what Pearson says in that chapter:

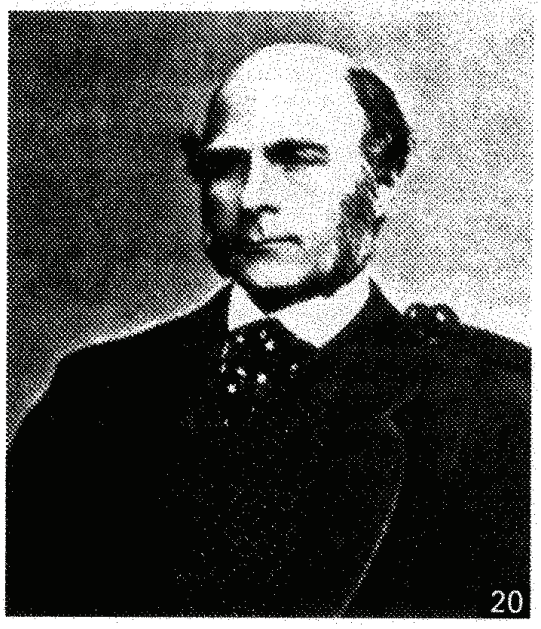
"Beyond such discarded fundamentals as 'matter' and 'force' lies still another fetish amidst the inscrutable arcana of modern science, namely, the category of cause and effect."

And what does Pearson substitute for the archaic category of cause and effect? You wouldn't believe your ears: *contingency tables* [24].

"Such a table is termed a contingency table, and the ultimate scientific statement of description of the relation between two things can always be thrown back upon such a contingency table....

"Once the reader realizes the nature of such a table, he will have grasped the essence of the conception of association between cause and effect."

Thus, Pearson categorically denies the need for an independent concept of causal relation beyond correlation.



He held this view throughout his life and, accordingly, did not mention causation in *any* of his technical papers.

His crusade against animistic concepts such as "will" and "force" was so fierce and his rejection of determinism so absolute that he *exterminated* causation from statistics before it had a chance to take root.

It took another 25 years and another strong-willed person, Sir Ronald Fisher [25], for statisticians to formulate the randomized experiment – the only scientifically proven method of testing causal relations from data, and to this day, the one and only causal concept permitted in mainstream statistics.

And that is roughly where things stand today.

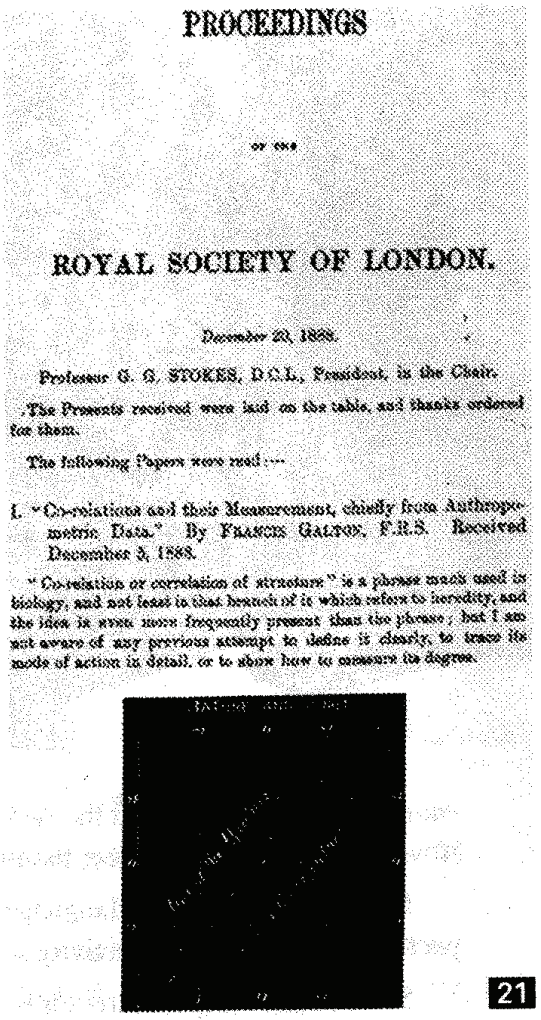
If we count the number of doctoral theses, research papers, or textbooks pages written on causation, we get the impression that Pearson still rules statistics.

The "Encyclopedia of Statistical Science" devotes twelve pages to correlation but only two pages to causation – and spends one of those pages demonstrating that "correlation does not imply causation."

Let us hear what modern statisticians say about causality.

Philip Dawid, the current editor of "Biometrika" (the journal founded by Pearson), admits: "Causal inference is one of the most important, most subtle, and most neglected of all the problems of statistics."

Terry Speed, former president of the Biometric Society (whom you might remember as an expert witness at the O. J. Simpson murder trial), declares: "Considerations of causality should be treated as they have always been treated in statistics: preferably not at all but, if necessary, then with very great care."



PROCEEDINGS

OF THE

ROYAL SOCIETY OF LONDON.

December 20, 1888.

Professor G. G. STOKES, D.C.L., President, in the Chair.

The Presents received were laid on the table, and thanks ordered for them.

The following Papers were read:—

I. "Co-relations and their Measurement, chiefly from Anthropometric Data." By FRANCIS GALTON, F.R.S. Received December 3, 1888.

"Co-relation or correlation of structure" is a phrase much used in biology, and not least in that branch of it which refers to heredity, and the idea is even more frequently present than the phrase; but I am not aware of any previous attempt to define it clearly, to trace its mode of action in detail, or to show how to measure its degree.

21

Sir David Cox and Nanny Wermuth, in a book published just a few months ago, apologize as follows: "We did not in this book use the words *causal* or *causality*.... Our reason for caution is that it is rare that firm conclusions about causality can be drawn from one study."

This position of caution and avoidance has paralyzed many fields that look to statistics for guidance, especially economics and social science.

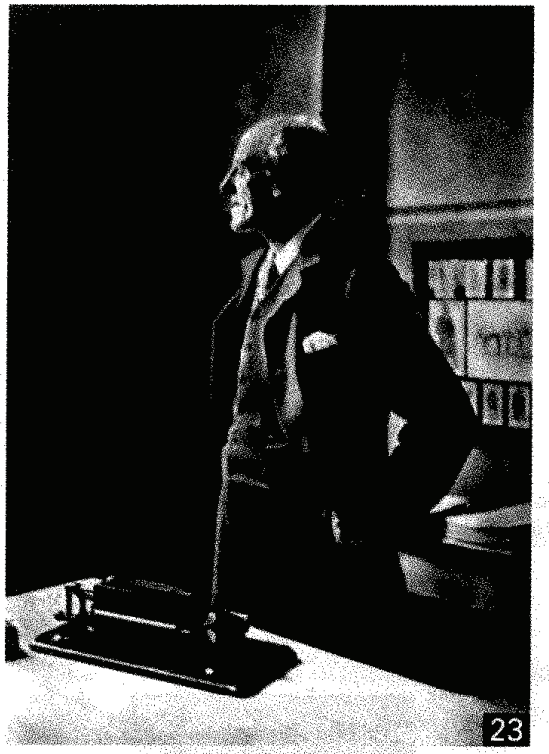
A leading social scientist stated in 1987: "It would be very healthy if more researchers abandon thinking of and using terms such as cause and effect."

Can this state of affairs be the work of just one person? Even a buccaneer like Pearson?

I doubt it.

But how else can we explain why statistics, the field that has given the world such powerful concepts as the testing of hypothesis and the

22



design of experiment, would give up so early on causation?

One obvious explanation is, of course, that causation is much harder to measure than correlation.

Correlations can be estimated directly in a single uncontrolled study, while causal conclusions require controlled experiments.

But this is too simplistic; statisticians are not easily deterred by difficulties, and children manage to learn cause effect relations *without* running controlled experiments.

The answer, I believe lies deeper, and it has to do with the official language of statistics – namely, the language of probability.

This may come as a surprise to some of you but the word *cause* is not in the vocabulary of probability theory; we cannot express in the language of probabilities the sentence, *mud does not cause rain* – all we can say is that the two are mutually correlated or dependent – meaning that if we find one, we can expect the other.

Naturally, if we lack a language to express a certain concept explicitly, we can't expect to develop scientific activity around that concept.

Scientific development requires that knowledge be transferred reliably from one study to another and, as Galileo showed 350 years ago, such transference requires the precision and computational benefits of a formal language.

I will soon come back to discuss the importance of language and notation, but first I wish to conclude this historical survey with a tale from another field in which causation has had its share of difficulty.

This time it is computer science – the science of symbols – a field that is relatively new yet one that has placed a tremendous emphasis on language and notation and therefore may offer a useful perspective on the problem.

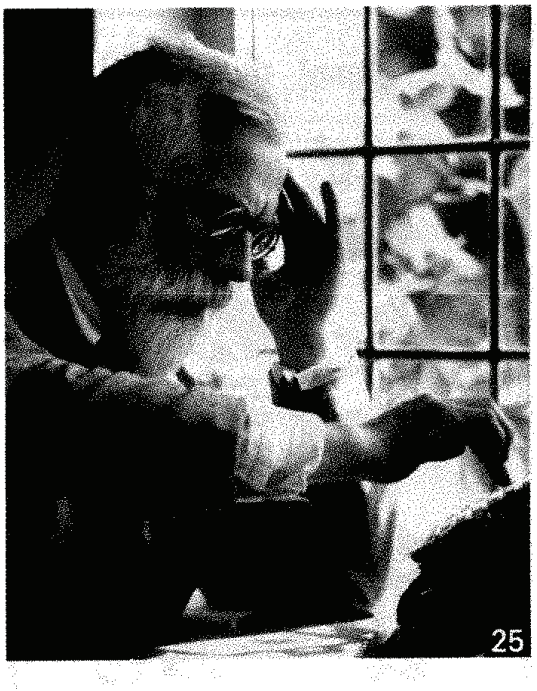
When researchers began to encode causal relationships using computers, the two riddles of causation were awakened with renewed vigor.

24 CONTINGENCY AND CORRELATION 159

B_1 occurs n_{p1}, B_2 occurs n_{p2} times, and so on. We thus are able to obtain a general distribution of B's for each class of A that we can form, and were we to go through the whole population, N, of A's in this manner we should obtain a table of the following kind :—

TYPE OF A OBSERVED

TYPE OF B OBSERVED	A_1.	A_2.	A_3.	...	...	A_p.	...	...	...	Total.
B_1	n_{11}	n_{21}	n_{31}	...	...	n_{p1}	...	...	...	n_{s1}
B_2	n_{12}	n_{22}	n_{32}	...	...	n_{p2}	...	...	...	n_{s2}
B_3	n_{13}	n_{23}	n_{33}	...	...	n_{p3}	...	...	...	n_{s3}
...	...	...	...	...	...	...	...	...	...	...
...	...	...	...	...	...	...	...	...	...	...
B_s	n_{1s}	n_{2s}	n_{3s}	...	...	n_{ps}	...	...	...	n_{ss}
...	...	...	...	...	...	...	...	...	...	...
...	...	...	...	...	...	...	...	...	...	...
Total	$n_{1.}$	$n_{2.}$	$n_{3.}$	...	...	$n_{p.}$	...	...	...	N


25

Put yourself in the shoes of this robot [26] who is trying to make sense of what is going on in a kitchen or a laboratory.

Conceptually, the robot's problems are the same as those faced by an economist seeking to model the national debt or an epidemiologist attempting to understand the spread of a disease.

Our robot, economist, and epidemiologist all need to track down cause–effect relations from the environment, using limited actions and noisy observations.

This puts them right at Hume's first riddle of causation: *how*?

The second riddle of causation also plays a role in the robot's world.

Assume we wish to take a shortcut and teach our robot all we know about cause and effect in this room [27].

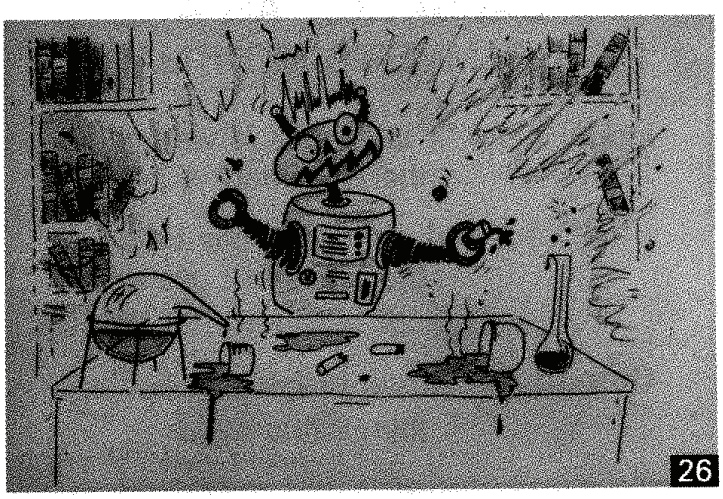
How should the robot organize and make use of this information?

Thus, the two philosophical riddles of causation are now translated into concrete and practical questions:

How should a robot acquire causal information through interaction with its environment? How should a robot process causal information received from its creator–programmer?

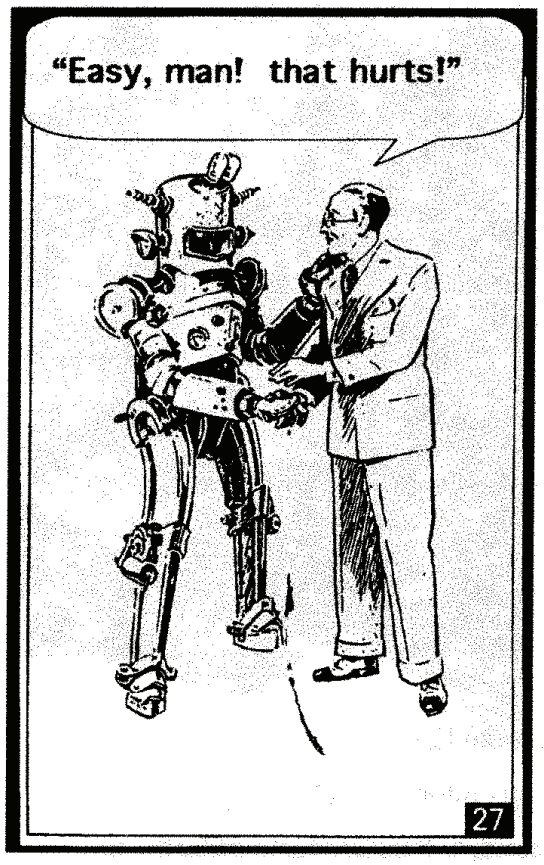
Again, the second riddle is not as trivial as it might seem. Lord Russell's warning that causal relations and physical equations are incompatible now surfaces as an apparent flaw in logic.

For example, when given the information, "If the grass is wet, then it rained" and "If we break this bottle, the grass will get wet," the computer will conclude "If we break this bottle, then it rained" [28].


26

The swiftness and specificity with which such programming bugs surface have made Artificial Intelligence programs an ideal laboratory for studying the fine print of causation.

This brings us to the second part of the lecture: how the second riddle of causation can be solved by combining equations with graphs, and how this solution makes the first riddle less formidable.



The overriding ideas in this solution are:

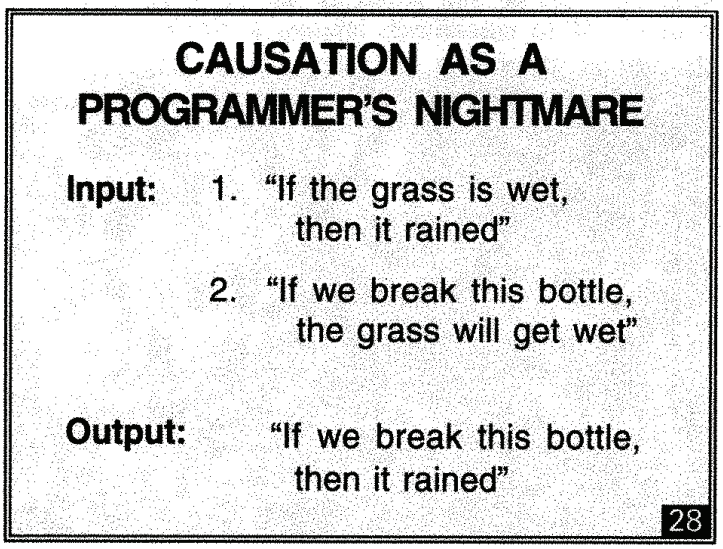
First – treating causation as a summary of behavior under interventions; and

Second – using equations and graphs as a mathematical language within which causal thoughts can be represented and manipulated.

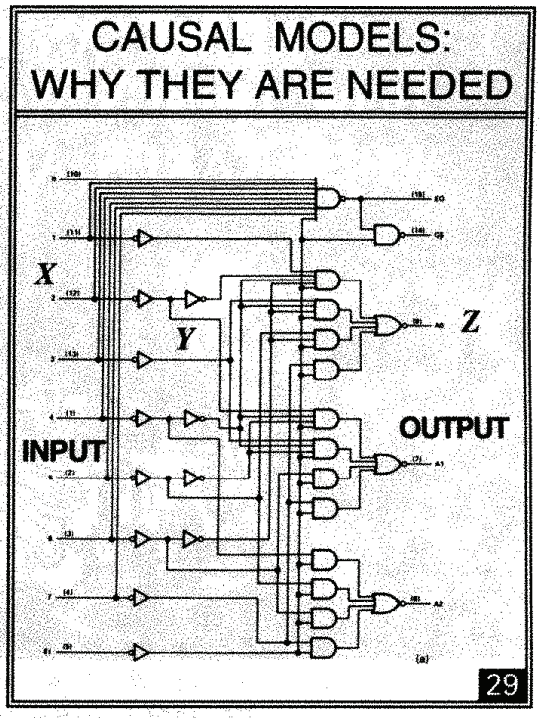
And to put the two together, we need a *third* concept: Treating interventions as a surgery over equations.

Let us start with an area that uses causation extensively and never had any trouble with it: engineering.

Here is an engineering drawing [29] of a circuit diagram that shows cause–effect relations among the signals in the circuit. The circuit consists of *and* gates and *or* gates, each performing some logical function between input and output. Let us examine this diagram closely, since its simplicity and familiarity are very deceiving. This diagram is, in fact, one of the greatest marvels of science. It is capable of conveying more information than millions of algebraic equations or probability functions or logical expressions. What makes this diagram so much more powerful is the ability to predict not merely how the circuit behaves under normal conditions but also how the circuit will behave under millions of *abnormal* conditions. For example, given this circuit diagram, we can easily tell what the output will be if some input changes from 0 to 1. This is normal and can easily be expressed by a simple input–output equation. Now comes the abnormal part. We can also tell what the output will be when we set Y to 0 (zero), or tie it to X, or change this *and* gate to an *or* gate, or when we perform any of the millions of combinations of these operations. The designer of this circuit did not anticipate or even consider such weird interventions, yet, miraculously, we can predict their consequences. How? Where does this representational power come from?



It comes from what early economists called *autonomy*. Namely, the gates in this diagram represent independent mechanisms – it is easy to change one without changing the other. The diagram takes advantage of this independence and



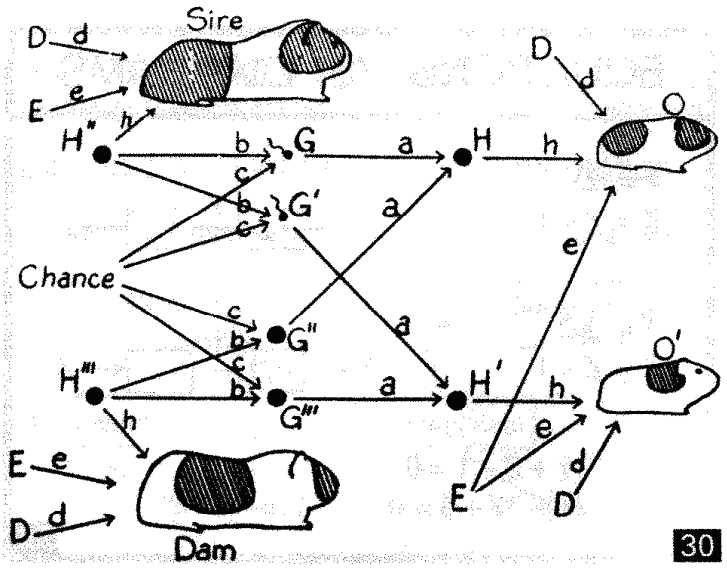
describes the normal functioning of the circuit *using precisely those building blocks that will remain unaltered under intervention.*

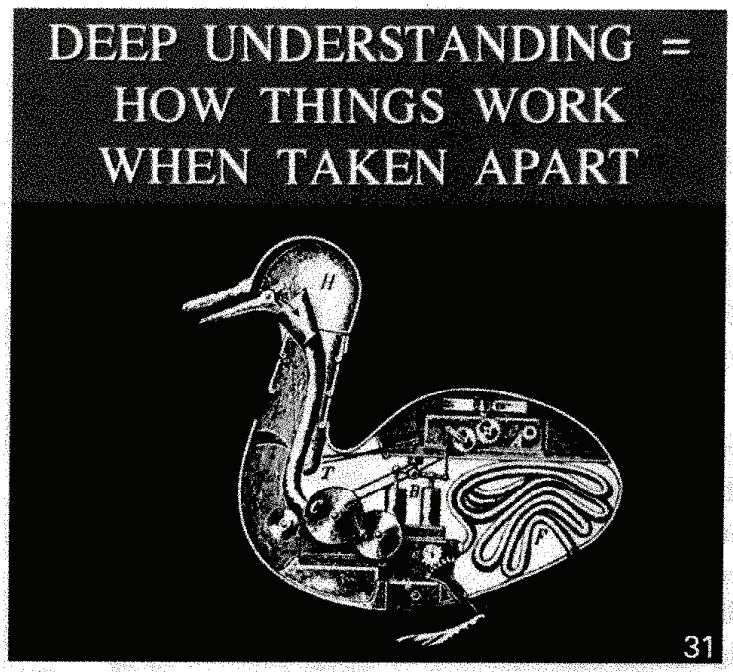
My colleagues from Boelter Hall are surely wondering why I stand here before you blathering about an engineering triviality as if it were the eighth wonder of the world. I have three reasons for doing this. First, I will try to show that there is a lot of unexploited wisdom in practices that engineers take for granted.

Second, I am trying to remind economists and social scientists of the benefits of this diagrammatic method. They have been using a similar method on and off for over 75 years, called structural equations modeling and path diagrams, but in recent years they have allowed algebraic convenience to suppress the diagrammatic representation, together with its benefits. Finally, these diagrams capture, in my opinion, the very essence of causation – the ability to predict the consequences of abnormal eventualities and new manipulations. In S. Wright's diagram [30], for example, it is possible to predict what coat pattern the guinea-pig litter is likely to have if we change environmental factors, shown here by as input (E), or even genetic factors, shown as intermediate nodes between parents and offsprings (H). Such predictions cannot be made on the basis of algebraic or correlational analysis.

Viewing causality this way explains why scientists pursue causal explanations with such zeal and why attaining a causal model is accompanied with a sense of gaining "deep understanding" and "being in control."

Deep understanding [31] means knowing not merely how things behaved yesterday but also how things will behave under new hypothetical circumstances, control being one such circumstance. Interestingly, when we have such understanding we feel "in control" even if we have no practical way of controlling things. For example, we have no practical way to control celestial motion, and still the theory of gravitation gives us a feeling of understanding and control, because it provides a blueprint for hypothetical control. We can predict the effect on tidal waves of unexpected new events – say, the moon being hit by a meteor or the gravitational constant suddenly diminishing by a

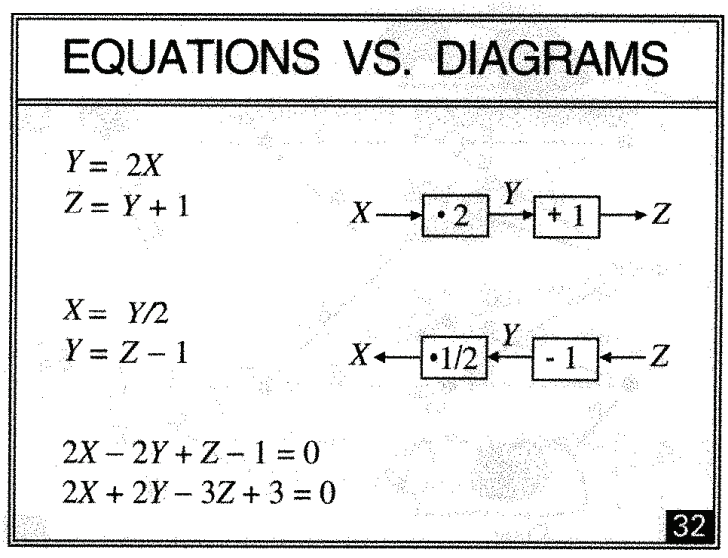




factor of 2 – and, just as important, the gravitational theory gives us the assurance that ordinary manipulation of earthly things will *not* control tidal waves. It is not surprising that causal models are viewed as the litmus test for distinguishing deliberate reasoning from reactive or instinctive response. Birds and monkeys may possibly be trained to perform complex tasks such as fixing a broken wire, but that requires trial-and-error training. Deliberate reasoners, on the other hand, can anticipate the consequences of new manipulations *without ever trying* those manipulations.

Let us magnify [32] a portion of the circuit diagram so that we can understand why the diagram can predict outcomes that equations can not. Let us also switch from logical gates to linear equations (to make everyone here more comfortable), and assume we are dealing with a system containing just two components: a multiplier and an adder. The *multiplier* takes the input and multiplies it by a factor of 2; the *adder* takes its input and adds a 1 to it. The equations describing these two components are given here on the left.

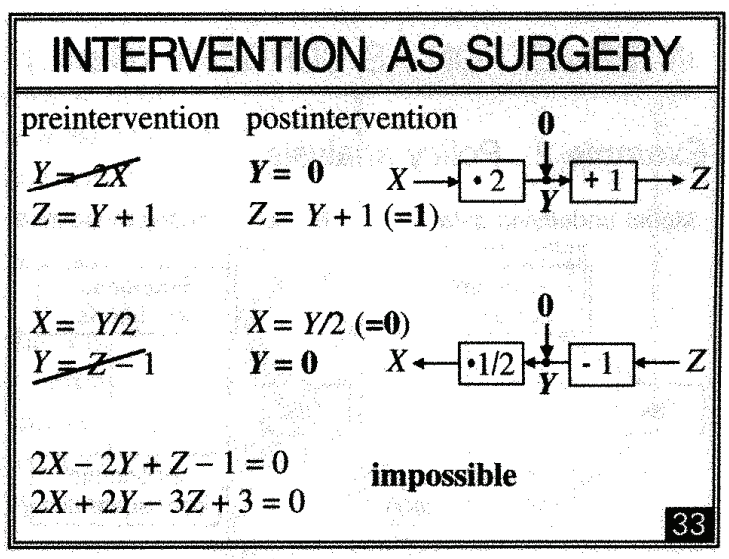
But are these equations *equivalent* to the diagram on the right? Obviously not! If they were, then let us switch the variables around, and the resulting two equations should be equivalent to the circuit shown below. But these two circuits are different. The top one tells us that if we physically manipulate Y it will affect Z, while the bottom one shows that manipulating Y will affect X and will have no effect on Z. Moreover, performing some additional algebraic operations on our equations, we can obtain two new equations, shown at the bottom, which point to no structure *at all*; they simply represent two constraints on three variables without telling us how they influence each other.



Let us examine more closely the mental process by which we determine the effect of physically manipulating Y – say, setting Y to 0 [33].

Clearly, when we set Y to 0, the relation between X and Y is no longer given by the multiplier – a

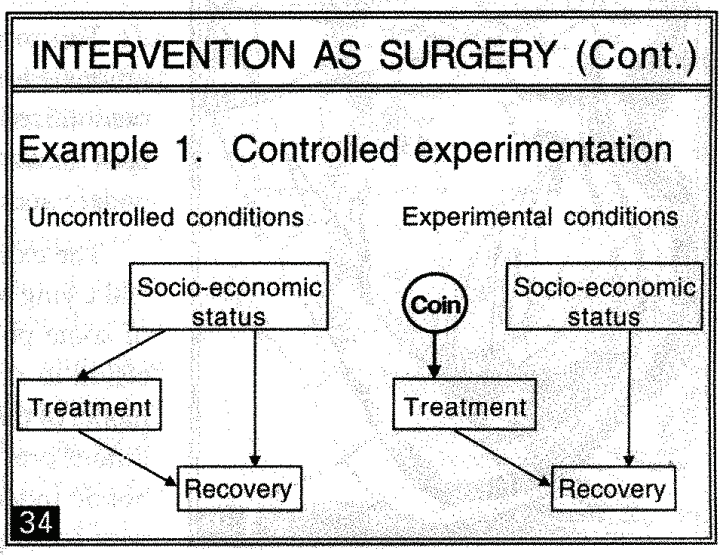
new mechanism now controls Y, in which X has no say. In the equational representation, this amounts to replacing the equation $Y = 2X$ by a new equation $Y = 0$ and solving a new set of equations, which gives $Z = 1$. If we perform this surgery on the lower pair of equations, representing the lower model, we get of course a different solution. The second equation will need to be replaced, which will yield $X = 0$ and leave Z unconstrained.



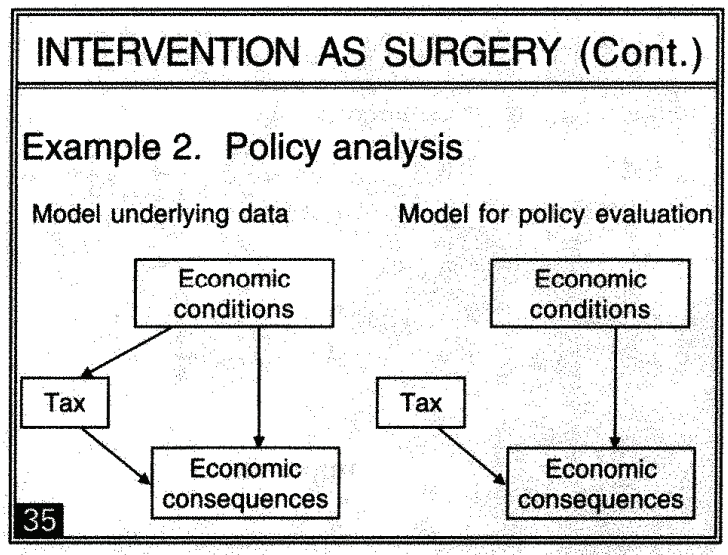
We now see how this model of intervention leads to a formal definition of causation: "Y is a cause of Z if we can change Z by manipulating Y, namely, if after surgically removing the equation for Y, the solution for Z will depend on the new value we substitute for Y." We also see how vital the diagram is in this process. *The diagram tells us which equation is to be deleted when we manipulate Y.* That information is totally washed out when we transform the equations into algebraically equivalent form, as shown at the bottom of the screen. From this pair of equations alone, it is impossible to predict the result of setting Y to 0, because we do not know what surgery to perform – there is no such thing as "the equation for Y."

In summary, *intervention amounts to a surgery on equations* (guided by a diagram) and *causation means predicting the consequences of such a surgery.*

This is a universal theme that goes beyond physical systems. In fact, the idea of modeling interventions by "wiping out" equations was first proposed in 1960 by an *economist,* Herman Wold, but his teachings have all but disappeared from the economics literature. History books attribute this mysterious disappearance to Wold's personality, but I tend to believe that the reason goes deeper: Early econometricians were very careful mathematicians; they fought hard to keep their algebra clean and formal, and they could not agree to have it contaminated by gimmicks such as diagrams. And as we see on the screen, the surgery operation makes no mathematical sense without the diagram, as it is sensitive to the way we write the equations.

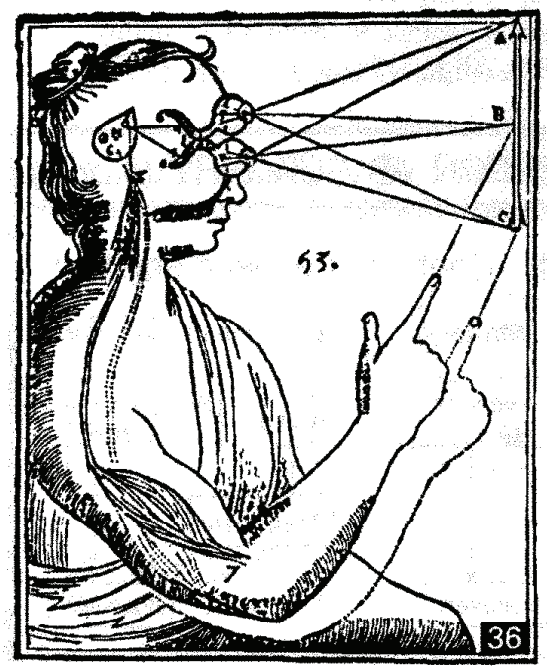


Before expounding on the properties of this new mathematical operation, let me demonstrate how useful it is for clarifying concepts in statistics and economics.



Why do we prefer controlled experiment over uncontrolled studies? Assume we wish to study the effect of some drug treatment on recovery of patients suffering from a given disorder. The mechanism governing the behavior of each patient is similar in structure to the circuit diagram we saw earlier. Recovery is a function of both the treatment and other factors, such as socioeconomic conditions, life style, diet, age, et cetera. Only one such factor is shown here [34]. Under uncontrolled conditions, the choice of treatment is up to the patients and may depend on the patients' socioeconomic backgrounds. This creates a problem, because we can't tell if changes in recovery rates are due to treatment or to those background factors. What we wish to do is compare patients of like backgrounds, and that is precisely what Fisher's *randomized experiment* accomplishes. How? It actually consists of two parts, randomization and *intervention*.

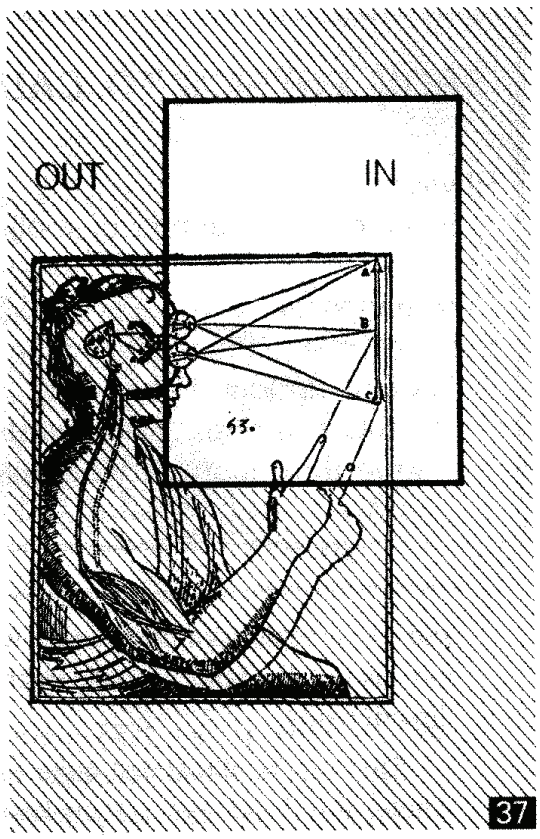
Intervention means that we change the natural behavior of the individual: we separate subjects into two groups, called treatment and control, and we convince the subjects to obey the experimental policy. We assign treatment to some patients who, under normal circumstances, will not seek treatment, and we give placebo to patients who otherwise would receive treatment. That, in our new vocabulary, means *surgery* – we are severing one functional link and replacing it with another. Fisher's great insight was that connecting the new link to a random coin flip *guarantees* that the link we wish to break is actually broken. The reason is that a random coin is assumed to be unaffected by anything we can measure on a macroscopic level – including, of course, a patient's socioeconomic background.



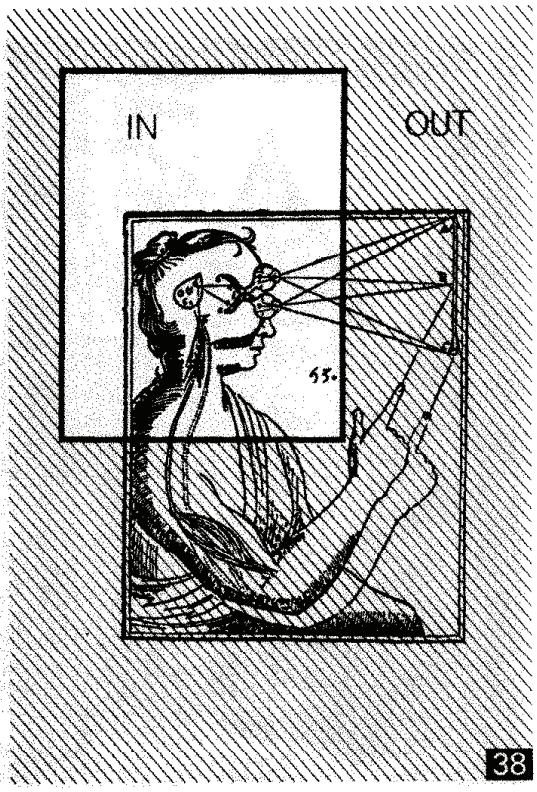
This picture provides a meaningful and formal rationale for the universally accepted procedure of randomized trials. In contrast, our next example uses the surgery idea to point out inadequacies in widely accepted procedures.

The example [35] involves a government official trying to evaluate the economic consequences of some policy – say, taxation. A deliberate decision to raise or lower taxes is a surgery on the model of the economy because it modifies the conditions prevailing while the model was built. Economic models are built on the basis of data taken over some period of time, and during this period

of time taxes were lowered and raised in response to some economic conditions or political pressure. However, when we *evaluate* a policy, we wish to compare alternative policies under the *same* economic conditions – namely, we wish to sever this link that, in the past, has tied policies to those conditions. In this setup, it is of course impossible to connect our policy to a coin toss and run a controlled experiment; we do not have the time for that, and we might ruin the economy before the experiment is over. Nevertheless the analysis that we *should conduct* is to infer the behavior of this mutilated model from data governed by a nonmutilated model.



I said *should conduct* because you will not find such analysis in any economics textbook. As I mentioned earlier, the surgery idea of Herman Wold was stamped out of the economics literature in the 1970s, and all discussions on policy analysis that I could find assume that the mutilated model prevails throughout. That taxation is under government control at the time of evaluation is assumed to be sufficient for treating taxation as an exogenous variable throughout, when in fact taxation is an endogenous variable during the model-building phase and turns exogenous only when evaluated. Of course, I am not claiming that reinstating the surgery model would enable the government to balance its budget overnight, but it is certainly something worth trying.



Let us now examine how the surgery interpretation resolves Russell's enigma concerning the clash between the directionality of causal relations and the symmetry of physical equations. The equations of physics are indeed symmetrical, but when we compare the phrases "A causes B" versus "B causes A," we are not talking about a single set of equations. Rather, we are comparing two world models, represented by two different sets of equations: one in which the equation for A is surgically removed; the other where the equation for B is removed. Russell would probably stop us at this point and ask: "How can you talk about *two* world models when in fact there is only one world model, given by all the equations of physics put together?" The answer is: *yes*. If you wish to

FROM PHYSICS TO CAUSALITY

Physics:
Symmetric equations of motion

Causal models:
Symmetric equations of motion
Circumsciption (in vs. out)
Locality (autonomy of mechanisms)
Intervention = surgery on mechanisms

39

include the entire universe in the model, causality disappears because interventions disappear – the manipulator and the manipulated loose their distinction. However, scientists rarely consider the entirety of the universe as an object of investigation. In most cases the scientist carves a piece from the universe and proclaims that piece *in* – namely, the *focus* of investigation. The rest of the universe is then considered *out* or *background* and is summarized by what we call *boundary conditions*. This choice of *ins* and *outs* creates asymmetry in the way we look at things, and it is this asymmetry that permits us to talk about "outside intervention" and hence about causality and cause–effect directionality.

This can be illustrated quite nicely using Descartes' classical drawing [36]. As a whole, this hand–eye system knows nothing about causation. It is merely a messy plasma of particles and photons trying their very best to obey Schroedinger's equation, which is symmetric.

However, carve a chunk from it – say, the object part [37] – and we can talk about the motion of the hand *causing* this light ray to change angle.

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Carve it another way, focusing on the brain part [38], and lo and behold it is now the light ray that causes the hand to move – precisely the opposite direction. The lesson is that it is the way we carve up the universe that determines the directionality we associate with cause and effect. Such carving is tacitly assumed in every scientific investigation. In artificial intelligence it was called "circumscription" by J. McCarthy. In economics, circumscription amounts to deciding which variables are deemed endogenous and which exogenous, *in* the model or *external to* the model.

Let us summarize the essential differences between equational and causal models [39]. Both use a set of symmetric equations to describe normal conditions. The causal model, however, contains three additional ingredients: (i) a distinction between the *in* and the *out*; (ii) an assumption that each equation corresponds to an independent mechanism and hence must be preserved as a

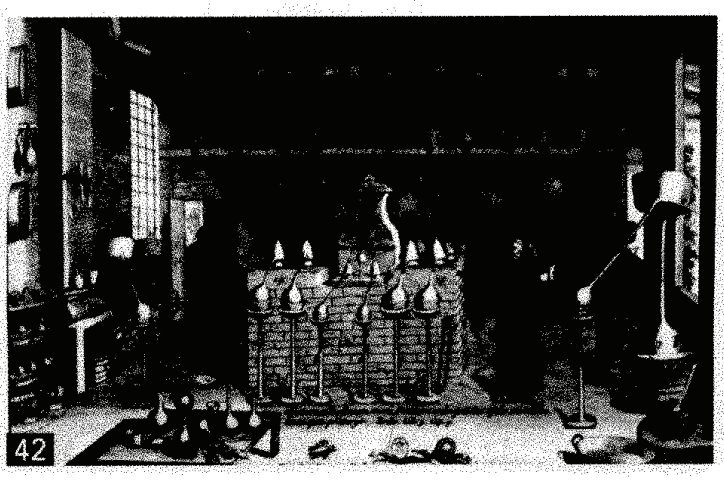
separate mathematical sentence; and (iii) interventions that are interpreted as surgeries over those mechanism. This brings us closer to realizing the dream of making causality a friendly part of physics. But one ingredient is missing: *the algebra.* We discussed earlier how important the computational facility of algebra was to scientists and engineers in the Galilean era. Can we expect such algebraic facility to serve causality as well? Let me rephrase it differently: Scientific activity, as we know it, consists of two basic components:

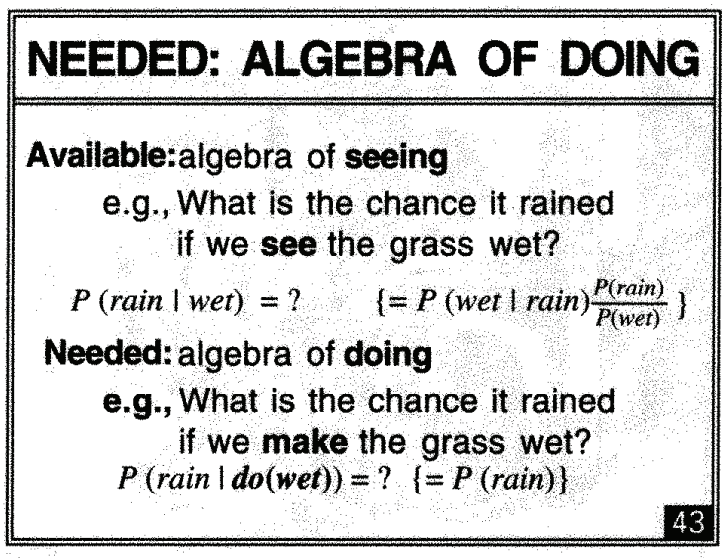
Observations [40] and interventions [41].

The combination of the two is what we call a *laboratory* [42], a place where we control some of the conditions and observe others. It so happened that standard algebras have served the observational component very well but thus far have not benefitted the interventional component. This is true for the algebra of equations, Boolean algebra, and probability calculus – all are geared to serve observational sentences but not interventional sentences.

Take, for example, probability theory. If we wish to find the chance it rained, given that we see the grass wet, we can express our question in a formal sentence written like that: $P(\text{Rain} \mid \text{Wet})$, to be read: the probability of Rain, given Wet [43]. The vertical bar stands for the phrase: "given that we see." Not only can we express this question in a formal sentence, we can also use the machinery of probability theory and transform the sentence into other expressions. In our example, the sentence on the left can be transformed to the one on the right, if we find it more convenient or informative.

But suppose we ask a different question: "What is the chance it rained if we *make* the grass wet?" We cannot even express our query in the syntax of probability, because the vertical bar is already taken to mean "given that I see." We can invent a new symbol *do,* and each time we see a *do* after the bar we read it *given that we do* – but this does not help us compute the answer to our question, because the rules of probability do not apply to this new reading. We know intuitively what the answer should be: $P(\text{Rain})$, because making the grass





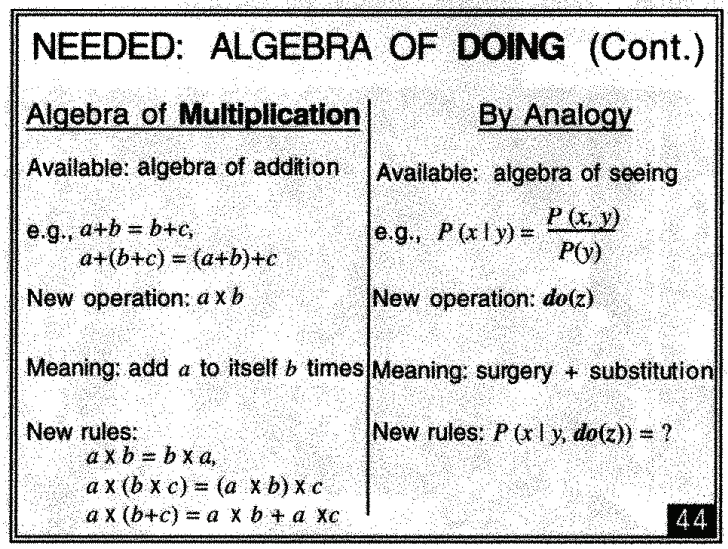
wet does not change the chance of rain. But can this intuitive answer, and others like it, be derived mechanically, so as to comfort our thoughts when intuition fails?

The answer is *yes,* and it takes a new algebra. First, we assign a symbol to the new operator "given that I do." Second, we find the rules for manipulating sentences containing this new symbol. We do that by a process analogous to the way mathematicians found the rules of standard algebra.

Imagine that you are a mathematician in the sixteenth century, you are now an expert in the algebra of *addition,* and you feel an urgent need to introduce a new operator, *multiplication,* because you are tired of adding a number to itself all day long [44]. The first thing you do is assign the new operator a symbol: *multiply.* Then you go down to the meaning of the operator, from which you can deduce its rules of transformations. For example: the commutative law of multiplication can be deduced that way, the associative law, and so on. We now learn all this in high school.

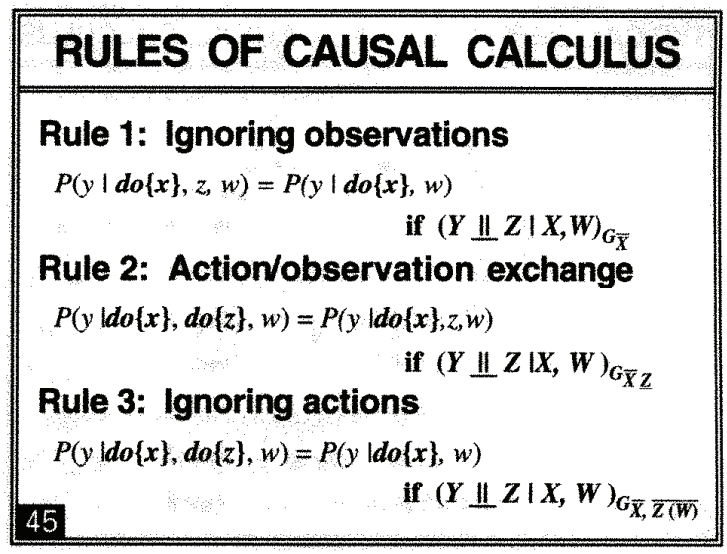
In exactly the same fashion, we can deduce the rules that govern our new symbol: $do(\cdot)$. We have an algebra for seeing – namely, probability theory. We have a new operator, with a brand new outfit and a very clear meaning, given to us by the surgery procedure. The door is open for deduction and the result is given in the next slide [45].

Please do not get alarmed, I do not expect you to read these equations right now, but I think you can still get the flavor of this new calculus. It consists of three rules that permit us to transform expressions involving actions and observations into other expressions of this type. The first allows us to ignore an irrelevant observation, the third to ignore an irrelevant action, the second allows us to exchange an action with an observation of the same fact. What are those symbols on the right? They are the "green lights" that the diagram gives us whenever the transformation is legal. We will see them in action on our next example.



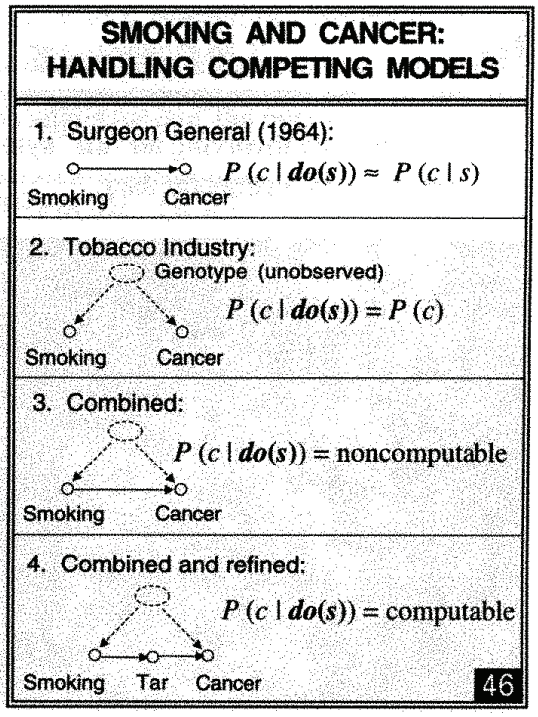
This brings us to part three of the lecture, where I will demonstrate how the ideas presented thus far can be used to solve new problems of practical importance.

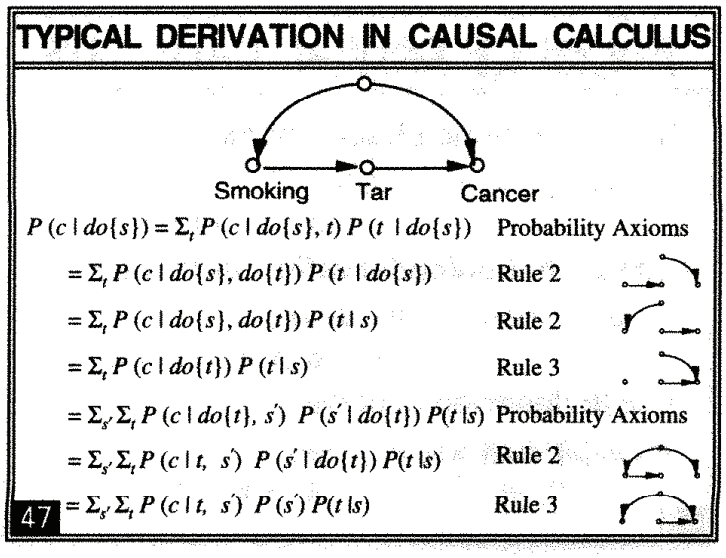
Consider the century-old debate concerning the effect of smoking on lung cancer [46]. In 1964, the Surgeon General issued a report linking cigarette smoking to death, cancer, and most particularly lung cancer. The report was based on nonexperimental studies in which a strong correlation was found between smoking and lung cancer, and the claim was that the correlation found is causal: If we ban smoking, then the rate of cancer cases will be roughly the same as the one we find today among nonsmokers in the population.



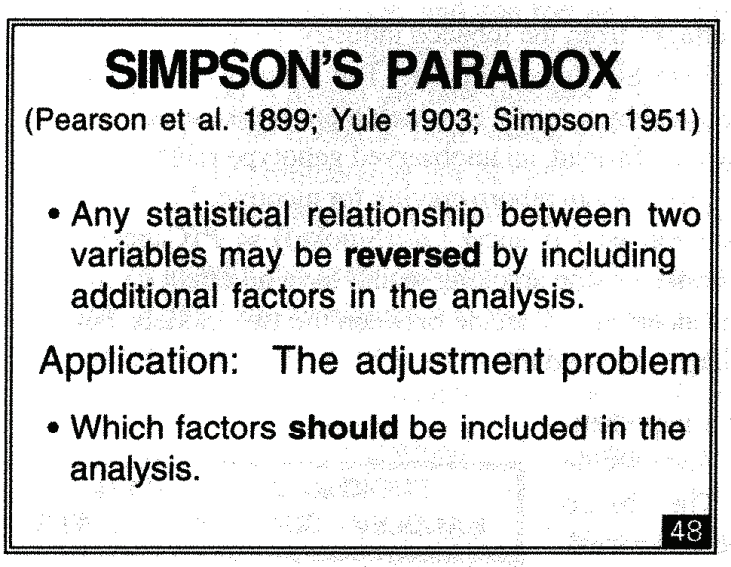
These studies came under severe attacks from the tobacco industry, backed by some very prominent statisticians, among them Sir Ronald Fisher. The claim was that the observed correlations can also be explained by a model in which there is no causal connection between smoking and lung cancer. Instead, an unobserved genotype might exist that simultaneously causes cancer and produces an inborn craving for nicotine. Formally, this claim would be written in our notation as: $P(\text{Cancer} \mid do(\text{Smoke})) = P(\text{Cancer})$, meaning that making the population smoke or stop smoking would have no effect on the rate of cancer cases. Controlled experiments could decide between the two models, but these are impossible (and now also illegal) to conduct.

This is all history. Now we enter a hypothetical era where representatives of both sides decide to meet and iron out their differences. The tobacco industry concedes that there might be some weak causal link between smoking and cancer and representatives of the health group concede that there might be some weak links to genetic factors. Accordingly, they draw this combined model, and the question boils down to assessing, from the data, the strengths of the various links. They submit the query to a statistician and the answer comes back immediately: *impossible*. Meaning: there is no way to estimate the strength from the data, because any data whatsoever can perfectly fit either one of these two extreme models. So they give up and decide to continue the political battle as usual. Before parting, a suggestion comes up: perhaps we can resolve our differences if we measure some auxiliary factors. For example, since the

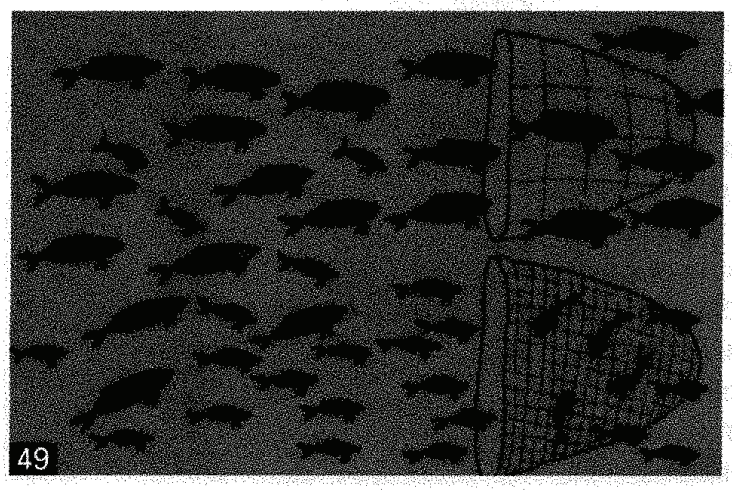




causal link model is based on the understanding that smoking affects lung cancer through the accumulation of tar deposits in the lungs, perhaps we can measure the amount of tar deposits in the lungs of sampled individuals, and this might provide the necessary information for quantifying the links. Both sides agree that this is a reasonable suggestion, so they submit a new query to the statistician: Can we find the effect of smoking on cancer assuming that an intermediate measurement of tar deposits is available? The statistician comes back with good news: *it is computable* and, moreover, the solution is given in closed mathematical form. *How*?

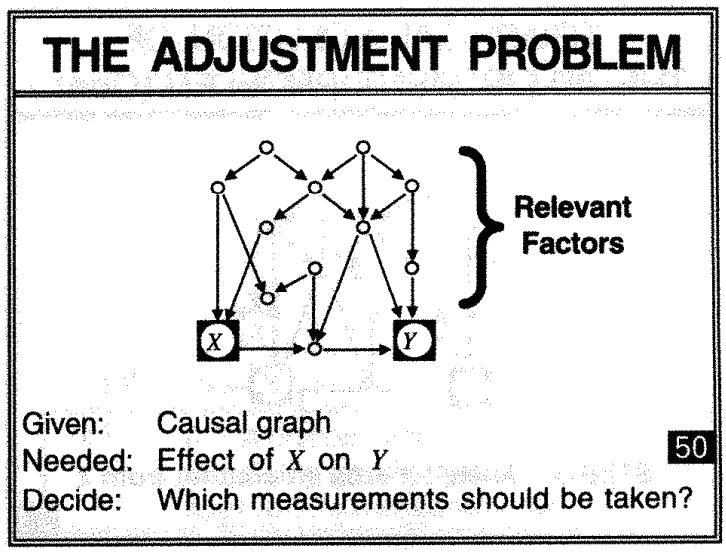


The statistician receives the problem and treats it as a problem in High School *algebra*: We need to compute P(Cancer), under hypothetical action, from nonexperimental data – namely, from expressions involving *no actions*. Or: We need to eliminate the "*do*" symbol from the initial expression. The elimination proceeds like ordinary solution of algebraic equation – in each stage [47], a new rule is applied, licensed by some subgraph of the diagram, eventually leading to a formula involving no "*do*" symbols, which denotes an expression that is computable from nonexperimental data.

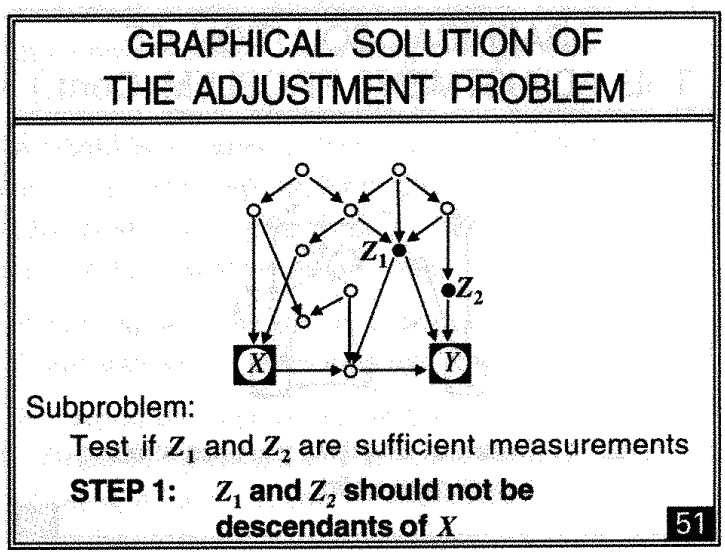


You are probably wondering whether this derivation solves the smoking–cancer debate. The answer is *no*. Even if we could get the data on tar deposits, our model is quite simplistic, as it is based on certain assumptions that both parties might not agree to – for instance, that there is no direct link between smoking and lung cancer mediated by tar deposits. The model would need to be refined

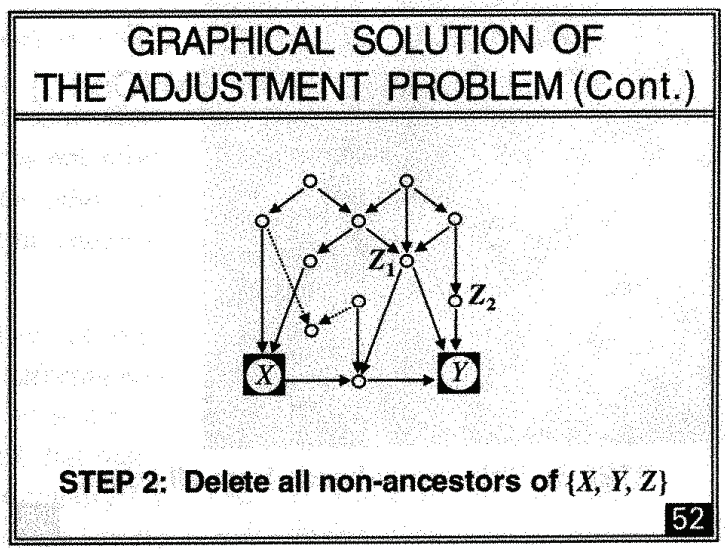
then, and we might end up with a graph containing twenty variables or more. There is no need to panic when someone tells us: "you did not take this or that factor into account." On the contrary, the graph welcomes such new ideas, because it is so easy to add factors and measurements into the model. Simple tests are now available that permit an investigator to merely glance at the graph and decide if we can compute the effect of one variable on another.



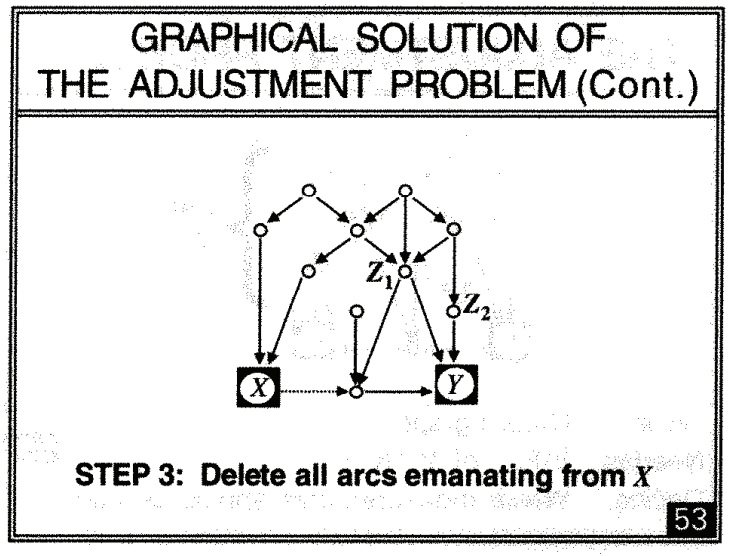
Our next example illustrates how a long-standing problem is solved by purely graphical means – proven by the new algebra. The problem is called *the adjustment problem* or "the covariate selection problem" and represents the practical side of Simpson's paradox [48].



Simpson's paradox, first noticed by Karl Pearson in 1899, concerns the disturbing observation that every statistical relationship between two variables may be *reversed* by including additional factors in the analysis. For example, you might run a study and find that students who smoke get higher grades; however, if you adjust for *age*, the opposite is true in every *age group*, that is, smoking predicts lower grades. If you further adjust for *parent income*, you find that smoking predicts higher grades again, in every *age–income* group, and so on.

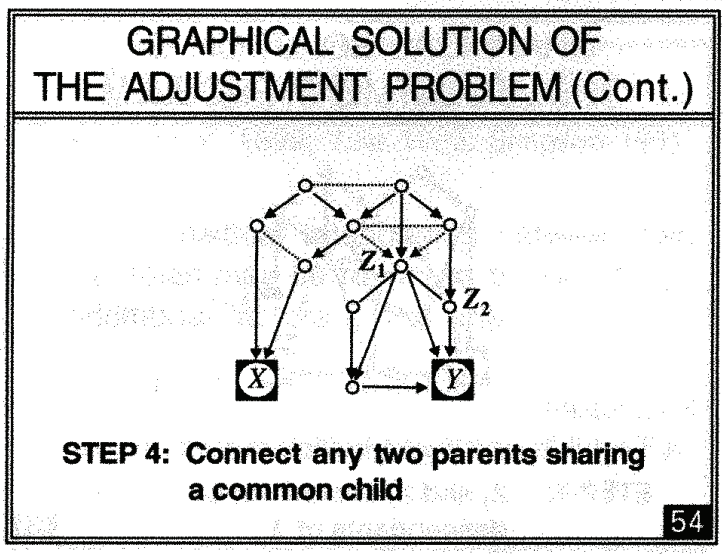


Equally disturbing is the fact that no one has been able to tell us which factors *should* be included in the analysis. Such factors can now be identified by simple graphical means. The classical case demonstrating Simpson's paradox took place in 1975, when UC-Berkeley

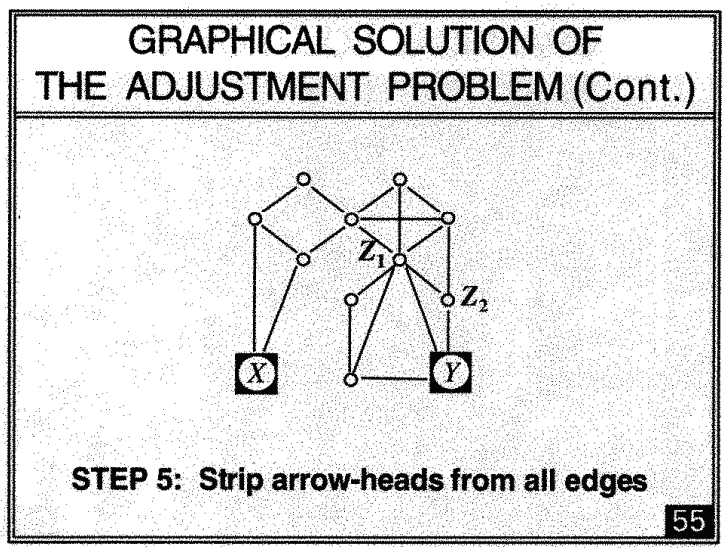


was investigated for sex bias in graduate admission. In this study, overall data showed a higher rate of admission among male applicants; but, broken down by departments, data showed a slight bias in favor of admitting female applicants. The explanation is simple: female applicants tended to apply to more competitive departments than males, and in these departments, the rate of admission was low for both males and females.

To illustrate this point, imagine a fishing boat with two different nets, a large mesh and a small net [49]. A school of fish swim toward the boat and seek to pass it. The female fish try for the small-mesh challenge, while the male fish try for the easy route. The males go through and only females are caught. Judging by the final catch, preference toward females is clearly evident. However, if analyzed separately, each individual net would surely trap males more easily than females.

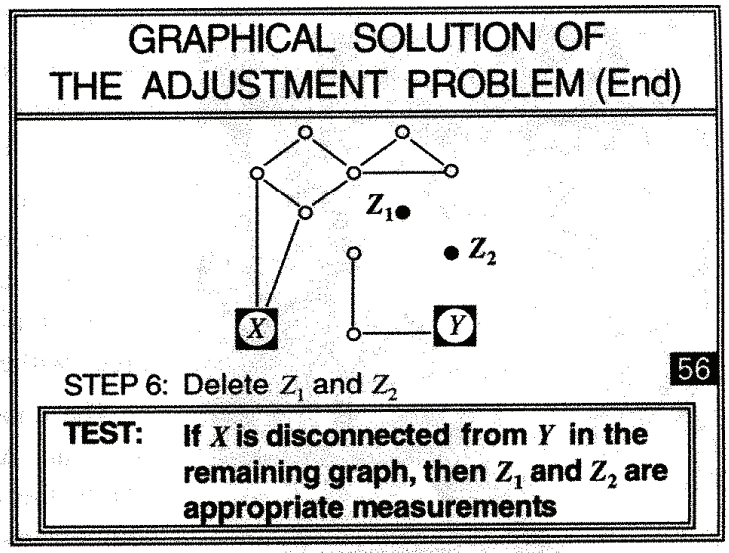


Another example involves a controversy called "reverse regression," which occupied the social science literature in the 1970s. Should we, in salary discrimination cases, compare salaries of equally qualified men and women or instead compare qualifications of equally paid men and women?



Remarkably, the two choices led to opposite conclusions. It turned out that men earned a higher salary than equally qualified women and, *simultaneously,* men were more qualified than equally paid women. The moral is that all conclusions are extremely sensitive to which variables we choose to hold constant when we are comparing,

and that is why the adjustment problem is so critical in the analysis of observational studies.



Consider an observational study where we wish to find the effect of X on Y, for example, treatment on response [50]. We can think of many factors that are relevant to the problem; some are affected by the treatment, some are affecting the treatment, and some are affecting both treatment and response. Some of these factors may be unmeasurable, such as genetic trait or life style; others are measurable, such as gender, age, and salary level. Our problem is to select a subset of these factors for measurement and adjustment so that, if we compare subjects under the same value of those measurements and average, we get the right result.

Let us follow together the steps that would be required to test if two candidate measurements, Z_1 and Z_2, would be sufficient [51]. The steps are rather simple, and can be performed manually even on large graphs. However, to give you the feel of their mechanizability, I will go through them rather quickly. Here we go [52–56].

At the end of these manipulations, we end up with the answer to our question: "*If* X is disconnected from Y, then Z_1 and Z_2 are appropriate measurements."

I now wish to summarize briefly the central message of this lecture. It is true that testing for cause and effect is difficult. Discovering causes of effects is even more difficult. But causality is not *mystical* or *metaphysical*. It can be understood in terms of simple processes, and it can be expressed in a friendly mathematical language, ready for computer analysis.

What I have presented to you today is a sort of pocket calculator, an *abacus* [57], to help us investigate certain problems of cause and effect with mathematical precision. This does not solve all the problems of causality, but the power of *symbols* and mathematics should not be underestimated [58].

Many scientific discoveries have been delayed over the centuries for the lack of a mathematical language that can amplify ideas and let scientists communicate results. I am convinced that many discoveries have been delayed in our century for lack of a mathematical language that can handle

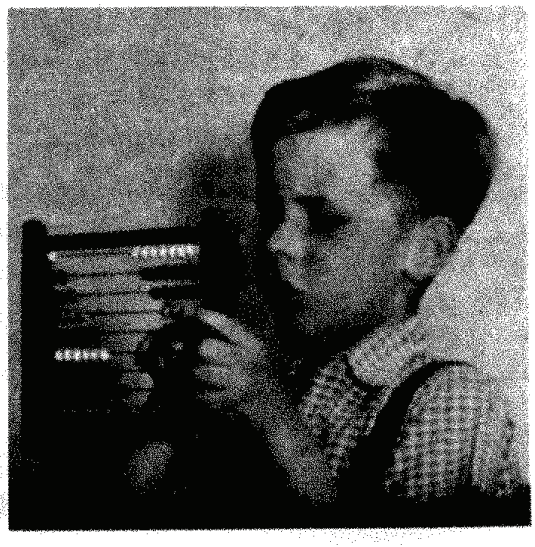
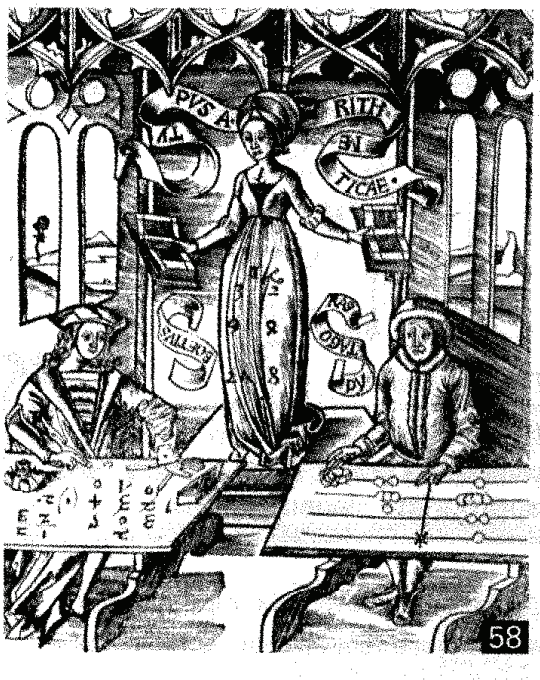


Fig. 155 Little Johnny and his "calculating machine." 57



causation. For example, I am sure that Karl Pearson could have thought up the idea of *randomized experiment* in 1901 if he had allowed causal diagrams into his mathematics.

But the really challenging problems are still ahead: We still do not have a causal understanding of *poverty* and *cancer* and *intolerance,* and only the accumulation of data and the insight of great minds will eventually lead to such understanding.

The data is all over the place, the insight is yours, and now an abacus is at your disposal, too. I hope the combination amplifies each of these components.

Thank you.

Acknowledgments

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Name Index

Subject Index

CAUSALITY CORRECTIONS IMPLEMENTED IN 2nd PRINTING

Updated 9/26/00

page v *insert* "TO RUTH" centered in middle of page

page xv *insert* in second paragraph "David Galles" after "Dechter"

page 2 *replace* "2000" with "2004" in 2nd paragraph, line 8 of 1.1.2

page 3 *insert* ", $\Rightarrow$" after "($\neg$)" in footnote 1
replace "and *not*" with "*not*, and *implies*," in footnote 1

page 19 *append* (continue italics) to end of Theorem 1.2.7, "(*We exclude* X_i *when speaking of its "nondescendants*".)"

page 30 *replace* in line 7 from top "mutually" with "jointly"
insert "parental" before "Markov" in first line of 2nd paragraph after Theorem 1.4.1
append to end of footnote 16 "but I am not aware of any nonparametric version."

page 52 *insert* "stable" after "IC*, that takes a" in 2nd paragraph after Theorem 2.6.2,
replace "sampled" with "stable" in Input line of IC* Algorithm.
append "(with respect to some latent structure)." to same line

page 68 *replace* in line 1 after Eq. (3.2), "mutually" with "jointly"

page 72 *replace* "(1990, 1999)" with "(1990, 2001)" on line 6

page 89 *replace* in paragraph starting "Indeed, if condition...". Should be "conditions require" not "condition require"

page 126 *insert* "and Robins (1997)" after "Pearl and Robins (1995)", line 2.

page 130 *replace* the "P" with "E" in the formula (second line of section 4.5.4.) Should read "$E(Y|\hat{x}, \widehat{pa}_{Y\setminus X})$"
replace "we can compute the difference" with "we should replace the controlled difference" in last line of page

page 131 *replace* from top of page through the end of section 4.5.4 with the following:

$$P(\text{admission}|\widehat{\text{male}}, \widehat{\text{dept}}) - P(\text{admission}|\widehat{\text{female}}, \widehat{\text{dept}})$$

with some average of this difference over all departments. This average should measure the increase in admission rate in a hypothetical experiment in which we instruct all female candidates to retain their department preferences but change their gender identification (on the application form) from female to male.

In general, the average direct effect is defined as the expected change in Y induced by changing X from x to x' while keeping the other parents of Y constant at whatever value they obtain under $do(x)$. This hypothetical change is what law makers instruct us to consider in race or sex discrimination cases: "The central question in any employment-discrimination case is whether the employer would have taken the same action had the employee been of a different race (age, sex, religion, national origin etc.) and everything else had been the same." (In Carson versus Bethlehem Steel Corp., 70 FEP Cases 921, 7th Cir. (1996)).

The formal expression for this hypothetical change involves probabilities of (nested) counterfactuals (see Section 7.1 for semantics and computation) that cannot be written in terms of the $do(x)$ operator.[9] Therefore, the average direct effect cannot in general be identified, even from data obtained under randomized control of all variables. However, if certain assumptions of "no confounding" are deemed valid,[10] then the average direct effect can be reduced to

$$\Delta_{x,x'}(Y) = \sum_{pa_{Y\setminus X}} [E(Y|\widehat{x'}, \widehat{pa}_{Y\setminus X}) - E(Y|\hat{x}, \widehat{pa}_{Y\setminus X})]P(pa_{Y\setminus X}|\hat{x}), \quad \textbf{(4.11)}$$

and the techniques developed in Section 4.4 for identifying control-specific plans, $P(y|\hat{x}_1, \hat{x}_2, \ldots, \hat{x}_n)$, become applicable.

[9] Using the counterfactual notation of Section 7.1, the general expression for the average direct effect is

$$\Delta_{x,x'}(Y) = E(Y_{x'Z_x}) - E(Y_x)$$

where $Z = pa_{Y\setminus X}$. The subscript $x'Z_x$ represents the operation of setting X to x' and, simultaneously, setting Z to whatever value it would have obtained under the setting $X = x$. This general expression reduces to (4.11) if $Z_x \perp\!\!\!\perp Y_{x'z}$ holds for all z. Likewise, the average *indirect* effect is defi ned as $E(Y_{xZ_{x'}}) - E(Y_x)$.

[10] See details in Technical Report R-273 posted on www.cs.ucla.edu/∼judea.

page 164 *replace* ... "$do(x, y, w)$" with "$do(x, z, w)$" in line 8 after Definition 5.4.3.

page 165 *replace* last two sentences of section 5.4.2 with:
The expressions corresponding to these policies are $P(y|do(x),\ do(z))$ and $P(y|do(x))$, and this pair of distributions fully represents the policy implications of indirect effects. Similar conclusions have been expressed by Robins and Greenland (1992). (But see Chapter 4, footnote 9, page 131.)

page 177 *delete* "tormented" in paragraph 3, line 2

page 184 *append* to end of Definition 6.2.1 (continue italics - except 'unbiased'): "*If* (6.10) *holds, we say that* $P(y|x)$ *is* unbiased."

page 236 *replace* "& $(X \rightarrow Y|$" with "& $(X \not\rightarrow Y|$" in first formula of Theorem 7.3.8

page 240 *replace* the last sentence in the last paragraph of section 7.4.1 with:
However, this effectiveness is partly acquired by limiting the counterfactual antecedent to conjunction of elementary propositions. Disjunctive hypotheticals, such as "if Bizet and Verdi were compatriots," usually lead to multiple solutions and hence to nonunique probability assignments.

page 246 *insert* in footnote 26 after "(see Section 5.4.3)." "Epidemiologists refer to (7.46) as "no-confounding" (see (6.10))."

page 255 *replace* in the 2nd line "pregnant" with "nonpregnant"

page 259 *insert* close parentheses after "(Sections 3.2 and 7.1", line 2 of Preface

page 284 *replace* "Michie in press" with "Michie 1999" in the last line of paragraph 4

page 329 *replace* "(1999)" with "(2000)" in last line of page

page 332 *replace* in paragraph starting "Even an erratic and ...". Change "role" to "roll"

page 354 line 2 from bottom, *replace* "mediated by tar deposits" with "unmediated by tar deposits"

page 361 *update* Dawid 1997 citation. *replace* "To appear ..." with "Also [with discussion] in *Journal of the American Statistical Association* 95:407–48, 2000."

page 363 *append* to Halpern (1998) citation, "Also, *Journal of Artificial Intelligence Research* 12:317–37, 2000."
update Halpern and Pearl (1999) citation. *replace* "(1999)" with "(2000)", *replace* "Actual causality." with "Causes and explanations.", and *append* "www.cs.ucla.edu/∼judea/"

page 364 *update* Hoover 1999 citation. *replace* "(1999)" with "(2001)"

page 365 *update* Kuroki citation. *append* “29: 105–17.” after “*Journal of the Japan Statistical Society*”.

page 366 *update* Michie citation. *replace* “(in press)” with “(1999)” and *insert* “pp. 60-86” before “vol. 15”

page 368 *update* Pearl 1999 citation. *remove* “To appear in” and replace “121” with “121:93–149.”

page 369 *insert* in Robins 1997 citation, “M. Berkane (Ed.),” before “*Latent Variable Modeling...*”

page 370 *add* to Shipley 1997 citation, “Also in *Structural Equation Modelling*, 7:206–18, 2000.”

page 381 *insert* “27–8”, after “(examples) price and demand” and before “215-17”
replace “245” with “245–7”, at end of “(exogeneity) controversies regarding...245”
combine “explanation” and “explanations” to read “explanation, 25, 58, 221–3, 285, 308–9”

page 382 *insert* “131” after “indirect effects,” and before “165”

ADDENDUM TO CORRECTIONS IMPLEMENTED IN 2nd PRINTING

Updated 12/14/00

page 28 *replace* "income (Z)" with "income (I)" in the caption of Figure 1.5

page 48 *replace* in line before Definition 2.4.1, "when one of the coins becomes slightly biased." with "when the coins become slightly biased."

page 51 *append* to line 7, Rule R_4 to read:
Orient a–b into $a \to b$ whenever there are two chains a–$c \to d$ and $c \to d \to b$ such that c and b are nonadjacent and a and d are adjacent.

page 231 *replace* Definition 7.3.4 and 2 lines following to read:

Definition 7.3.4 (Recursiveness)
Let X and Y be singleton variables in a model, and let $X \to Y$ stand for the inequality $Y_{xw}(u) \neq Y_w(u)$ for some values of x, w, and u. A model M is recursive if, for any sequence $X_1, X_2, \ldots, X_k$, we have

$$X_1 \to X_2,\ X_2 \to X_3, \ldots, X_{k-1} \to X_k \Rightarrow X_k \not\to X_1 \qquad \textbf{(7.24)}$$

Clearly, any model M for which the causal diagram $G(M)$ is acyclic must be recursive.

page 382 *change* "Markov (assumptions underlying, 30)" to "Markov (assumption, 30, 69)"

page 382 *append* "69" after "causal, 30" in "Markov condition (causal, 30)"

page 384 *add* as subentry after "structural model, 27, 44, 202" "Markovian, 30, 69".

PRINTING CORRECTIONS TO BE IMPLEMENTED BY CAMBRIDGE

Updated 3/13/07

page 52 *replace* in line 17-18 "protection" with "projection"

page 73 *replace* equation between (3.11) and (3.12)

$$\begin{aligned} P(pa_i|do(x_i')) &= P(pa_i); \\ \frac{P(s_i, pa_i|do(x_i'))}{P(s_i', pa_i|do(x_i'))} &= \frac{P(s_i, pa_i)}{P(s_i', pa_i)}. \end{aligned}$$

should be:

$$\begin{aligned} P(pa_i|do(x_i')) &= P(pa_i); \\ \frac{P(s_i, pa_i, x_i'|do(x_i'))}{P(s_i', pa_i, x_i'|do(x_i'))} &= \frac{P(s_i, pa_i, x_i')}{P(s_i', pa_i, x_i')}. \end{aligned}$$

page 82 *replace* at end of paragraph 2: "since there is no back-door path from X to Z, we simply have" with "since there is no unblocked back-door path from X to Z in Figure 3.5, we simply have"

page 82 *replace* in Definition 3.3.3 (Front-Door): "(ii) there is no back-door path from X to Z; and" to "there is no unblocked back-door path from X to Z; and"

page 103 *replace* last paragraph on page 103 (including footnote 15) with:

To place this result in the context of our analysis in this chapter, we note that one class of semi-Markovian models satisfying assumption (3.62) corresponds to graphs in which all arrowheads pointing to X_k originate from observed variables. Indeed, in such models, the parents $PA_k = L_k, X_{k-1}$ of variable X_k satisfy the back-door condition of Definition 3.3.1,

$$(X_k \perp\!\!\!\perp Y | PA_k)_{G_{\underline{X}_k}},$$

which implies (3.62).[15] This class of models falls under Theorem 3.2.5, which states that all causal effects in this class are identifiable and are given by the truncated factorization formula of (3.14); the formula coincides with (3.63) after marginalizing over the uncontrolled covariates.

[15] Condition (3.62) is too restrictive and lacks intuitive basis; a graphical, more general condition leading to (3.63) is formulated in (4.5), Theorem 4.4.1, read: $P(y|g = x)$ is identifi able and is given by (3.63) if every action-avoiding back-door path from X_k to Y is blocked by some subset l_k of non-descendants of X_k. (by "action-avoiding" we mean a path containing no arrow entering an X variable later than X_k) see ⟨http://bayes.cs.ucla.edu/BOOK-2K/yudkowsky.html⟩.

page 174 *replace* in 4th line from end of page: “is not a statement about C being a positive causal factor for E, properly written” with “is not a statement about C having a positive influence on E, properly written”

page 195 *replace* ”Figure 6.1” with ”Figure 6.3” in 5th line before end of 3rd paragraph