Evolutionary Developmental Psychopathology

by

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Chapter 1

Introduction

Genealogical Actors in Ecological Roles

> Surely the way to encourage people to think about their lives and to improve them is not to replace one set of coercive determinants with another, and surely the way to think about responsible action is not to juggle inner and outer, ultimate and proximate causes, and hope that reasons and responsibility will miraculously squeeze through some narrow space where causes collide in persons.
>
> (Oyama, 1985, p. 16)

> People, like all other organisms, are not evolved to maximise health, wealth, happiness or any other trait – but to have descendants, which is the continuation of life.
>
> (Chisholm, 1999, p. 48)

How can psychiatric nosology[1] generate an epistemic benefit, and can a scientific taxonomy of mental disorders ever be entirely coextensive with a clinical taxonomy of such disorders? I shall argue that useful taxonomic concepts for a science of psychopathology are those representing projectable categories, and that such categories delineate *natural kinds*, or non-arbitrary aspects of the world. I shall also argue that because our attitude towards the treatment of disorders or problems of any kind necessarily involves a complex psycho-social cost-benefit analysis, clinical taxonomy will always reflect a nonepistemic agenda that is itself mutable according to the strictures of prevailing norms and resources. These considerations imply that the search for a single psychiatric taxonomy based on the natural and human sciences and capable of accommodating the needs of both clinicians and researchers could be futile, and that a clear acknowledgement of the differing ends of psychiatric treatment and research into psychopathology should be a starting point in the classification of mental disorders.

[1] *Nosology* is the branch of medicine concerned with the classification and description of diseases.

Recent attempts to promote the extension of evolutionary theorising to human psychology and behaviour have awakened renewed interest in a field variously called *Darwinian psychiatry* (McGuire & Troisi, 1998), *evolutionary psychopathology* (Baron-Cohen, 1997), or *evolutionary psychiatry* (Stevens & Price, 1996). According to some of its most prominent practitioners this discipline 'introduces a broad and much needed deductive framework; it facilitates the functional analysis of behaviour; it identifies important differences between ultimate causes and proximate mechanisms, [and] it promotes a reassessment of current views about aetiology and pathogenesis' (McGuire, et al., 1992, p. 89). However, drawing as it does on the concerns of human sociobiology (Wilson, 1975; 1978), much of the work in evolutionary psychopathology has concentrated on the study of adaptive *behaviours* 'such as acquiring a mate, sexual intercourse, having offspring, parent-offspring bonding, stranger anxiety' and other 'general behaviour profiles and patterns of human behaviour… set by the species' genome [which], within limits, unfold in predictable ways' (McGuire, et al., 1992, p. 90).

Although it is certainly correct that 'human physiology is importantly influenced by selective forces' (Sterelny, 1992, p. 156), which is all that human sociobiology requires as a basic justification, there is a serious epistemic asymmetry between animal sociobiology and human sociobiology owing to the fact that humans are long-lived and unavailable for scientific manipulation in the form of controlled breeding experiments. Another problem in considering particular human behaviours as adaptive is the human capacity to replicate learned behaviour through cultural means. Although our culture and social institutions may reflect aspects of our evolved psychological mechanisms (Boyer, 1994; Sperber, 1996), our behaviour is certainly

> …the result of perceptual inputs, our learning history, and very complex interactions between distinct psychological mechanisms… very little human behaviour is the result of a specialised capacity, built by genes that have proliferated in virtue of their ability to build the device that produces the behaviour. In us, if functionalism is right, there is *nothing like* a one-one correlation between behaviours and mechanisms (Sterelny, 1992, p. 168).

Crawford argues for the distinction between *innate adaptation*, the genetically encoded design for the development of proximate mechanisms, and *operational adaptation*, the phenotypic psychological processes actually producing the behaviour (Crawford, 1993). Inasmuch as the environment in which the phenotype develops differs significantly from the environment of evolutionary adaptedness an operational adaptation may be typified by entirely novel features, and may contribute to behaviours having little bearing on lifetime reproductive success (LRS). Consequently, as Sterelny suggests 'we need from sociobiology an evolutionary psychology, not an evolutionary theory of human behaviour' (1992, p. 170). Two of the field's early advocates, Leda Cosmides and John Tooby, argue that to embrace evolutionary psychology

> …means shedding certain concepts and prejudices inherited from parochial parent traditions: the obsessive search for a cognitive architecture that is general purpose and initially content-free; the excessive reliance on results derived from artificial "intellectual" tasks; the idea that the field's scope is limited to the study of "higher" mental processes; and a long list of false dichotomies reflecting premodern biological thought – evolved/learned, evolved/developed, innate/learned, genetic environmental, biological/social, biological/cultural, emotion/cognition, animal/human. Most importantly, cognitive scientists will have to abandon the functional agnosticism that is endemic to the field (Cosmides & Tooby, 1994, p. 42).

Evolutionary psychology eschews what it regards as the behavioural determinism of sociobiology, but it does, however, retain a commitment to a modified genetic determinism (of mechanisms rather than behaviour) which may itself obscure a full appreciation of human psychological plasticity and the intricacies of development. To borrow a phrase from David Hull (1987) we need to remember that human beings are *genealogical actors in ecological roles*, and a large portion of this work constitutes a consideration of ways in which we should perceive the contribution of genes and ecology to our evolved psychology. How then should we conceive of 'evolutionary psychology'? What concepts and debates characterise this field? How does it relate to other disciplines? What does it have to say about psychiatric classification and mental illness?

To provide a coherent framework within which to analyse conceptual disputes in psychiatry it is an indispensable prerequisite to evaluate competing perspectives on human evolution (and evolutionary biology in general) and perspectives in the history and philosophy of science. Although this can often seem a highly circuitous route to an understanding of mental illness, recent work in these areas does allow us to clarify and refine some of the concepts and theories that provide the foundation for the profoundly antagonistic debates that impede the exploration of human nature. Consequently, chapter two 'The Separation of Contradictory Things' considers the origins and consequences of the arbitrary allocation of causal co-determinants to mutually incompatible schemes of explanation and advocates the developmental systems approach to evolution and the causal homeostatic theory of natural kinds as frameworks capable of avoiding damaging dichotomies. Chapter three 'The Problem of Classification in Psychiatry' provides an overview of the recent history of biological psychiatry and examines the failure of the principal neurochemical hypotheses of mental disorders it has produced. Psychiatric classification is examined from a number of perspectives and a distinction is drawn between arbitrary concepts and projectable categories as the foundation for explanation and induction. Chapter four 'Evolution and Human Nature' examines the development of sociobiology and evolutionary psychology. Chapter five 'The Society of Mind' commends the modular view of psychological faculties within the developmental systems perspective, and finally chapter six 'Evolutionary Developmental Psychopathology' demonstrates how the ideas advocated within this work can provide novel insights into the nature of mental disorders. These insights allow us to re-organize research findings into an alternative scheme (or schemes) of investigation and classification.

Chapter 2

The Separation of Contradictory Things

> Since the genome represents only a part of the entire developmental ensemble, it cannot by itself contain or cause the form that results. But then, neither can its surroundings. As is frequently the case in these matters, people in some way know this perfectly well and say so. The reason they often end up belying their own good sense seems to be their tendency to view a lack of variation (within the organism if focus is on individual nature and within the species if focus is on species nature) as evidence of inherent, necessary qualities.
>
> (Oyama, 1985, pp. 19-20)

The word 'dichotomy' is derived from the Greek *dikhotomia*, which means literally 'cutting in two'. In this chapter I will discuss the arbitrary separation of variables and argue that the allocation of causal co-determinants to opposing explanatory schemata undermines our understanding of the natural world and human nature. The pervasive influence of three pivotal dichotomies on scientific enquiry and on therapeutic intervention: those of mind *versus* body, cognition *versus* emotion and nature *versus* nurture, will be a recurrent theme throughout this work. Although scholars in the natural and human sciences usually disavow belief in distinct material and immaterial substances contemporary debates are phrased largely in terms that would have been familiar to the Greek philosophers, and which still divide human characteristics into *divine* or *transcendent attributes*[2], in modern terminology the surrogate terms include 'rational', 'cognitive', 'discursive', 'autonomous' and 'unrestricted', and *animal* or *corporeal attributes*, the surrogates being terms such as 'emotional', 'instinctive', 'determined', 'immutable', and 'bounded'. The three dichotomies are all inspired by this essential dualism and each term evokes one or more of the properties associated with each category. Viewed in these terms many contemporary scientific, political, and cultural debates often have an unacknowledged quasi-

[2] It can be extremely enlightening to keep in mind some synonyms for *divine*: heavenly, sublime, ineffable, numinous, supernatural, supramundane; and for *animal*: brutish, bestial, subhuman, mindless, unthinking, intemperate, sensual.

theological dimension, and it is this dimension that is responsible for some of the greatest impediments to the understanding of human nature.

I will argue that we should attempt to employ a rigorously mechanistic approach to the natural world. This does not imply a commitment to unrestrained and unrealistic reductionism, or to the arbitrary exclusion of phenomena that are clearly characteristic of the human condition, such as emotional experience or the moral sentiments – traits that are often considered to fall outside the domain of scientific enquiry. This standpoint can be achieved through a synthesis of two key perspectives: the *developmental systems approach* to evolution by natural selection, and the *causal homeostatic theory of natural kinds*.

Divining the Essence: Cleaving Mind from Body

The doctrine of dualism, which holds that there are two distinct substances, one corporeal and earthly, and the other incorporeal and transcendent, has a long history in Western philosophical and theological thought. The Greek philosopher Plato (428-347 BC), perhaps the most influential of all philosophers, ancient or modern, held that the *soul* (or *divine mind*) as the source of reason, thought, and intellect, was the essential property setting humankind apart from animals. In *The Phaedo* Plato writes of the body that

> …it fills us full of lusts, and fears, and fancies of all kinds, and endless foolery, and in fact, as men say, takes away all power of thinking from us at all… It has been proved to us by experience that if we would have true knowledge of anything we must be quit of the body – the soul in herself must behold things in themselves: and then we shall attain the wisdom we desire…(quoted in Russell, 1961, p. 151).[3]

In the same discourse Plato employs the famous metaphor ‘depicting intellect as the charioteer who holds the reins, with emotion and will as the horses that draw the chariot. This triarchic model of the human psyche, comprising, intellect, emotion, and will, is perhaps the most easily recognizable aspect of phi-

[3] The text of *The Phaedo* is available on the Internet at http://plato.evansville.edu/texts/jowett/phaedo.htm.

losophy's legacy to psychology' (Jensen, 1998, p. 4) Plato's pupil, Aristotle, later reduced the triarchic division of the psyche to two main functions, which he termed the *dianoetic*[4], or what we would now call the cognitive functions, and the *orectic*, which included the emotions, will, and moral sense.

Because mind and body were held to be separate, the problem of the interaction between the two became one of the most intractable questions in philosophy. The father of modern philosophy, René Descartes (1596-1650), believed that the 'thinking substance', or mind, interacted with the 'extended substance', or body, by way of the pineal gland. He saw this as the likely organ of interaction because it is the only part of the brain that is not divided into two hemispheres. Descartes argued that thinking was the essence of humankind, and that the foundation for all true knowledge could be summarised in the aphorism '*Cogito, ergo sum*' or 'I think, therefore I am'. Although the properties of extended substances could be analysed in terms of the laws of physics, thinking substances could be understood only in terms of the laws of thinking. Descartes offers no coherent explanation of how extended substances and thinking substances could interact, but he contends that all conflicts are conflicts between the soul and the body (Gaukroger, 1995, p. 402).

In the *Discourse on the Method of Rightly Conducting the Reason, and Seeking Truth in the Sciences* (1637), Descartes explains how by 'deducing effects from their causes, and by showing from what elements and in what manner nature must produce them' the recent triumph of the scientific explanation of the circulation of the blood had been achieved, and cautions:

> …lest those who are ignorant of the force of mathematical demonstrations and who are not accustomed to distinguish true reasons from mere verisimilitudes, should venture without examination, to deny what has been said, I wish it to be considered that the motion which I have now explained follows as necessarily from the very arrangement of the parts, which may be observed in the heart by the eye alone, and from the heat which may be felt with the fingers, and from the nature of the blood as learned from experience, as does the motion of a clock from the power, the situa-

[4] *Dianoetic*: rational, discursory, analytic, synthetic.

> tion, and shape of its counterweights and wheels (*Discourse on the Method*, Part V).[5]

But lest anyone should think that the laws of mechanics could explain the nature of humankind, Descartes goes on to argue that though the mechanical properties of extended substances such as human bodies could be regarded as no different to those of an ape or 'any other irrational animal', there would remain 'two most certain tests whereby to know that they were not therefore really men'. These tests are the ability to use language and the ability to reason – abilities that could only be dependent on the properties of a reasonable soul that

> …could by no means be educed from the power of matter, as the other things of which I had spoken, but that it must be expressly created; and that it is not sufficient that it be lodged in the human body exactly like a pilot in a ship, unless perhaps to move its members, but that it is necessary for it to be joined and united more closely to the body, in order to have sensations and appetites similar to ours, and thus constitute a real man. I here entered, in conclusion, upon the subject of the soul at considerable length, because it is of the greatest moment: for after the error of those who deny the existence of God, an error which I think I have already sufficiently refuted, there is none that is more powerful in leading feeble minds astray from the straight path of virtue than the supposition that the soul of the brutes is of the same nature with our own; and consequently that after this life we have nothing to hope for or fear, more than flies and ants; in place of which, when we know how far they differ we much better comprehend the reasons which establish that the soul is of a nature wholly independent of the body, and that consequently it is not liable to die with the latter and, finally, because no other causes are observed capable of destroying it, we are naturally led thence to judge that it is immortal (*Discourse on the Method*, Part V).

In proposing this substantial union of mind and body, Descartes is effectively arguing the case for the notion of the embodied mind – a mind which has features distinct from disembodied mind or from bodies, but he retains a commitment to the idea of an indivisible and immaterial soul as the essence of human nature. As Stephen Gaukroger points out:

[5] Available on the internet at http://human-nature.com/reason/descartes/part5.html.

> The behaviour of a human being… can never be explained reductively. A human being has the faculties of judgement and will, and – something which is a precondition of these – consciousness of her own mental states, whereas an automaton does not. The key point is that human sensations are quite unlike animal sensations, and the reason for this is now clear: it is not that human corporeal faculties are significantly different from animal ones, but that human corporeal faculties are largely regulated by and subordinate to the mind, and their content takes on a distinctively different kind of quality as a result (Gaukroger, 1995, pp. 392-3).

At the beginning of his book the *Passions of the Soul* (1649) Descartes writes that he approaches the subject matter not as 'an orator, nor as a moral philosopher, but as a physicist' (quoted in Gaukroger, 1995, p. 399), by which he means to point out that he seeks to establish some degree of certainty, and that he means to distance himself from the views of the Stoics, who saw passion as a *pathological* phenomenon. The passions must be interpreted in terms of the substantial union, as Gaukroger points out

> Descartes begins… the *Passions* by noting that whether something is called an action or a passion depends simply on whether it is considered with respect to the mind or the body, so the crucial thing is to start with the difference between the soul and the body… We are then provided with a division of the soul into two: actions and passions. Actions comprise volitions which either terminate in the soul, as "when we will to love God", or in the body, as when we move our legs by willing to walk. They also include those perceptions which have their origin in the soul, as when we reflect upon our own existence. Perceptions which have their origin in the body, on the other hand, are passions (Gaukroger, 1995, p. 401).

In his *Meditations on First Philosophy* (1641) Descartes refers to the passions as *confusi status mentis*, confused states of mind, or 'confused ideas' (Jáuregui, 1995, p. 4). These passions are

> Functions of the soul which depend on its union with the body. Perceptions which do not derive from the soul itself can be caused either by external bodies acting on us, or from natural appetites of the body, such as hunger, which we sense through bodily organs, or they can be felt "as in the soul itself", in which case no immediate cause is evident. These last are the "passions of the soul" to which Descartes' account is devoted, and he is concerned with

> their phenomenology rather than their causes; for while we may be deceived about their causes.. we cannot be deceived about their existence or specific nature. They are defined as being "caused, maintained, and strengthened by a movement of the spirits", and take the form of "excitations of the soul", as do volitions; but, unlike volitions, they do not have their source in the soul (Gaukroger, 1995, p. 401).

Through his influence on the development of both science and philosophy mind-body dualism has become known as Cartesian dualism, and the problem of mind-body interaction as Descartes' problem.

In psychiatry 'organic' disorders were those with a known physical cause, and the 'functional' disorders such as schizophrenia were located in the mind and could not be attributed to any known brain pathology, though it was usually held that some underlying pathology would be uncovered eventually (Rose, Lewontin & Kamin, 1990, p. 198). In the case of the functional disorders, then, the commitment to a description in terms of behavioural or psychological factors was merely heuristic, and those employed in biological psychiatry have generally endeavoured to eliminate the role of psychological elements in the pathophysiology of these disorders. This commitment to explanation in terms of exclusive psychological or non-psychological determinants has its origin in the traditions of Western dualism.

A second division in early psychiatry, which still persists in modern classification, is that between the psychoses and the neuroses. The latter are viewed as purely psychological disorders originating in dysfunctions of the psyche or the emotions. The category of neurosis does not actually appear in the most recent *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1994), but the phenomena covered by this term still appear grouped as *anxiety disorders*. These include Generalized Anxiety Disorder, Panic Disorder, Phobias, Obsessive Compulsive Disorder, Separation Anxiety Disorder, Posttraumatic Stress Disorder, and Multiple Personality Disorder. A third major category is that of *personality disorders* which, curiously, are not judged to be mental illnesses but appear in psychiatric nosology anyway, prin-

cipally because those diagnosed as such often engage in anti-social behaviour which is deemed pathological.

The Primacy of Mind

A belief in the *primacy of mind* is a ubiquitous element in the history of ideas. The ‘mind’ or ‘soul’ is not only primary as an explanation of human nature, but is the only conceivable explanation, as nothing so subtle and sublime as reason and morality could emerge from matter and motion. In his book *Darwin’s Dangerous Idea* (1995) Daniel Dennett explains that Judeo-Christian and Islamic cosmogony are established on the assumption that the genesis of all creation is dependent on the action of ‘a “cogitative Being”’ (Dennett, 1995, p. 28). In modern times the idea that complex functional design in nature is the result of the actions of another mind or other minds motivates not only Creationists, such as those in the Intelligent Design Movement, but intellectuals in schools of thought and disciplines as disparate as behaviourism, connectionism, sociology, cognitive science, neuroscience, and even evolutionary biology, who cannot view the faculties of the human mind as the product of selection. Many, if not most, of the intellectuals involved in these disciplines are materialists who would attribute most of the design of the natural world to the action of selection, but in the case of the human mind strict adherence to this foundational principle of biological science wavers. Distinctive human attributes are attributed not to the actions of a creator, or to the action of physical forces, but to influence of other human minds, either individually, or collectively in the form of culture. This unwillingness to embrace a mechanistic explanation of every aspect of the natural world is thus as pervasive in science as in popular culture and tradition. I contend that the failure to adopt a mechanistic approach to human nature is the principal source of conceptual confusion and faulty hypotheses. I shall argue that a mechanistic approach does not deny a role for minds, culture, or morality, but does deny these phenomena the role of sufficient and exclusive determinants of human faculties, a role that is also denied to genetic and other biological factors.

Cognition and Emotion: Cleaving Thought from Salience

In modern behavioural science the perennial Western philosophical agenda is of such importance that in his book *The Mind's New Science* (1985), a history of modern cognitive science, Howard Gardner writes that 'it is virtually unthinkable that cognitive science would exist, let assume its current form, had there not been a philosophical tradition dating back to the time of the Greeks' (1985b, p. 7). Given the ambiguous position of the emotions within this philosophical tradition, it is perhaps unremarkable that Gardner identifies the exclusion of affective factors or emotions as one of the five features of paramount importance 'generally associated with cognitive scientific efforts' (1985b, p. 6). Although the commitment to substance dualism played no role, cognitive science was originally conceived effectively as the science of the soul, or rather the science of mankind's most distinctive attribute, reason.

In discussing cognitive science's de-emphasis on affect, context, culture, and history Gardner notes that

> Though mainstream cognitive scientists do not necessarily bear any animus against the affective realm, against the context that surrounds any action or thought, or against historical or cultural analyses, in practice they attempt to factor out these elements to the maximum extent possible. So even do anthropologists when wearing their cognitive science hats… Critics of cognitivism have responded in two principal ways. Some critics hold that factors like affect, history, or context will never be explicable by science: they are inherently humanistic or aesthetic dimensions, destined to fall within the province of other disciplines or practices. Since these factors are central to human experience, any science that attempts to exclude them is doomed from the start. Other critics agree that some or all of these features are of the essence in human experience, but do not feel that they are insusceptible to scientific explanation. Their quarrel with an antiseptic cognitive science is that it is wrong to bracket these dimensions artificially. Instead, cognitive scientists should from the first put their noses to the grindstone and incorporate such dimensions fully into their models of thought and behaviour (Gardner, 1985b, pp. 41-42).

This antipathy to affect has also permeated other allied disciplines such as psychiatry, neurology and neuroscience. In his book *Mind-Body Deceptions: The*

Psychosomatics of Everyday Life (1997) Steven Dubovsky describes how subjective and objective approaches have conflicted and interacted in the history of psychiatry. Though the word 'psychiatry' means 'mind cure' in Greek, it was coined in 1808 by an anatomist, Johann Weil, who saw the new field as a branch of neurology. The term psychosomatic, which is redolent of Descartes' interactionism, was coined in 1818, by German physiologist Johann Heinroth, but it fell into disuse until American psychoanalyst Felix Deutsch described seven illnesses (peptic ulcer, thyrotoxicosis, rheumatoid arthritis, asthma, hypertension, neurodermatitis, and ulcerative colitis) that seemed to be strongly influenced, if not caused, by psychological factors. Dubovsky's book charts the resurgence of psychosomatic medicine, but even its title is redolent of an interactionist perspective. Dubovsky observes that

> Descriptive psychiatry retained the scientific method by abandoning the subjective non-observable realms; psychoanalysis retained the vast tapestry of human experience by abandoning the objectivity of the scientific method. Once psychoanalysis abandoned the biological for the emotional, however, its practitioners entered a realm that was particularly vulnerable to the distortion of passion and prejudices. Had it been possible to retain an attitude of objectivity while still utilizing intuition and introspection, the analytic strategy might have penetrated the psyche with a precision comparable to biological methods. But such was not the case. The politics of medicine, the very human motivations and needs of psychoanalysts, and the power of the unconscious as it emerged in the analyst-patient relationship – all these factors made this hope of "objective subjectivity" as much as an illusion as was the hope that a purely biological approach could explain the totality of the mind (Dubovsky, 1997, p. 31).

In his book *The Emotional Brain* neuroscientist Joseph LeDoux remarks that that 'by the early 1980s, very little research on the brain mechanisms of emotion was being conducted' (1998, p. 73), a situation he attributes to the combined influence of cognitive science and to an early theory of the emotions known as the limbic system theory. The latter theory was associated with psychiatrist Paul MacLean who held that the hippocampus, as a primitive structure, was likely to be the seat of the emotions. Subsequent research showed the hippocampus to be computationally complex, and to be implicated primarily in 'one of the most important cognitive systems of the brain, the temporal lobe memory system

(LeDoux, 1998, p. 200). In surveying the attitude of cognitive scientists to the study of emotion LeDoux notes:

> In his seminal 1968 textbook, *Cognitive Psychology*, Ulric Neisser states that the field is not about the dynamic factors (like emotions) that motivate behaviour. Jerry Fodor, in *The Language of Thought*, a groundbreaking book in the philosophy of cognitive science, describes emotions as mental states that fall outside the domain of cognitive explanation. And Barbara von Eckardt, in a book titled *What is Cognitive Science?* says that most cognitive scientists do not consider the study of emotions to be part of the field. (LeDoux, 1998, pp. 34-35)

LeDoux believes there are a number of key points justifying the belief that emotion and cognition are best thought of as interacting mental functions mediated by separate (or perhaps it would be better to say *distinct*), but functionally united, brain systems:

> Brain damage can disrupt the ability to interpret the emotional significance of stimuli without any loss in the capacity to perceive the same stimuli as objects.
>
> The emotional meaning of a stimulus can begin to be appraised by the brain without any before the perceptual systems have fully processed the stimulus.
>
> The brain mechanisms through which memories of the emotional significance of stimuli are registered, stored, and retrieved are different from the mechanisms through which cognitive memories of the same stimuli are processed.
>
> The systems that perform emotional appraisals are directly connected with systems involved in the control of emotional responses.
>
> The linkage of appraisal mechanisms with response control systems means that when the appraisal mechanism detects a significant event, the programming and often the execution of a set of appropriate responses will occur (LeDoux, 1998, pp. 69-70).

In summary, LeDoux believes that emotion is not merely a collection of thoughts about a situation, it is not simply reasoning, and it cannot be understood just by asking people what went on in their minds when they had an emotion. The basic emotions (or what are often called *affect programs*) are functions involved in

survival, but since different emotions are involved in different survival systems, each may involve different brain systems that evolved for different reasons. As LeDoux puts it

> Although we often talk about the brain as if it has a function, the brain itself actually has no function. It is a collection of systems, sometimes called modules, each with different functions. There is no equation by which the combination of functions of all the different systems mixed together equals an additional function called brain function (LeDoux, 1998, p. 105).

Our intention should be to describe functional systems that have developed over our evolutionary history because of their ability to promote survival and reproduction. The identification of these systems is unlikely to be enhanced by any attempt to describe them in terms of artificial distinctions between 'substances' derived from the Western philosophical tradition, or to assign their description to disciplines established on the basis of arbitrary divisions of the natural world. The emotional and cognitive 'modules' within the brain are not discrete interactants, but elements of complete functional systems. Indeed, the salience of any information in terms of its value (potential impact on survival and reproduction) cannot be assessed unless it is processed by a system that has access to both its emotional and cognitive content, though much of such processing may remain below the level of conscious awareness.

In a proposal remarkably illustrative of the dualisms afflicting neuroscience and psychology Jaak and Jules Panksepp recently postulated a striking dichotomy between 'genetically dedicated circuits' for emotions and a second system composed of 'general-purpose computational space' (2000, p. 108). The former are phylogenetically ancient subcortical structures, or neurochemical operating systems, which have homologies in many species, and reflect fitness concerns; the latter is subserved by plastic neocortex. The research program of a new discipline proposed by the Panksepps, called neuroevolutionary psychobiology, aims to elucidate the way in which human abilities emerge from developmental interactions between these two mechanisms. Although the explanation in terms of mechanical Darwinian processes is allowed to advance as far as the 'lower' regions of the brain an area of 'higher' cortex is reserved as the repository of

reason and the medium of cultural inscription. This tactic of drawing a line beyond which scientific explanations cannot proceed will be encountered many times in subsequent chapters. It unites an astonishing array of approaches which appear to share little similarity on the surface.

Nature and Nurture: Cleaving Genes and Organisms from Environment

Susan Oyama argues that the conventional view of evolution involves two mistaken ideas, the first being the 'idea that traits are "transmitted" in heredity, [which in turn] rests on notions of genetic programming that are ultimate quite preformationist'. The second is the idea termed 'developmental dualism', which 'holds that there are two kinds of developmental process, one controlled primarily from the inside and another more open to external forces' (Oyama, 2000a, p. 21). I concur with Oyama that this approach inspires investigators to 'carve the world up into innate and acquired portions, no matter how vociferously… [they] declare the distinction to be obsolete (2000a, p. 21).

Developmental systems theory undermines the basis of both 'genetic determinism' in the biological sciences and 'environmental determinism' in the social sciences by forcing us to recognise that traits are constructed during development and are consequently neither structured by internal 'genetic programs' nor by external environmental processes. Indeed,

> For differential reproduction to alter a gene pool… all that is needed is reliable genotype-phenotype correlations; and these, in turn, require not genetic "programs" for development but a reliable succession of organism environment complexes – of developmental systems that repeatedly reconstitute themselves. (Oyama, 2000a, p. 27)

In order to overcome the idea that traits are transmitted and that evolution consists of changes in gene frequencies we need to understand that traits are constructed by developmental systems, that nurture is 'as crucial to typical characteristics as atypical ones', and that nature and nurture are joint determiners of form and function. Any element of the developmental system which we arbitrarily apportion to nature ('genetic programs') or nurture (environment) can be the

source of variation, and evolution is, therefore 'the derivational history of developmental systems' (Oyama, 2000a, p. 49). The interactants in developmental systems include the genome (whose parts interact), cell structure (including organelles such as mitochondria which have their own DNA), the extracellular environment, parental reproductive systems, self-stimulation, physical environment, conspecifics, and climate (Oyama, 2000a, pp. 73-74). All of these elements have informational status identical to that of information within the genome, hence Oyama's reference to her view of developmental systems theory as *the ontogeny of information*.

Perhaps a simple way to think of developmental systems is in terms of 'emergence'. In biology this concept is used to describe phenomena that cannot be explained in terms of their component parts. In one of the most famous examples the biologist Thomas Huxley (1825-1895), observed that the distinctive properties of water (what might be referred to as its 'aquosity') could not be detected in or deduced from our knowledge of the properties of hydrogen or oxygen atoms (Mayr, 1982, p. 63). The molecule haemoglobin, as the transporter of oxygen, is an indispensable component of our circulatory system. The actual three dimensional structure of this molecule is determined by the electrostatic forces between its constituent atoms, and not by information contained in the genome. Genes code for proteins, though even the three dimensional structure of these proteins is not determined by instructions in the genome. Because genes are not causal determinants, even at the molecular level, we should say that they are selected to remain in the genome because they participate in certain outcomes, and that these outcomes are selected for. Genes do not include instructions for building proteins, organisms, or behaviours, and we should say that molecules such as haemoglobin emerge and are causally co-determined by information in the genome and information in the environment. Of course, at the molecular level, the functioning of the developmental system is extremely reliable because the factors involved are stable. This is not necessarily the case at more complex levels of explanation, where the diverse range of co-determinants allows great variability in outcomes.

Oyama (2000a) outlines eight key ideas and methodological strategies of developmental systems theory. One of these is *parity of reasoning*, or placing the 'poverty of the gene' on a par with the 'poverty of the stimulus' in the explanation of traits. This springs from the commitment to grant informational status to all elements of developmental systems. When considering all of the factors involved in an outcome we should be conscious of the need to reveal 'hidden inequalities and questionable assumptions' such as the reason for assigning causal priority to the genes. Those assigning such priority will often assign all elements other than genes the role of 'elicitor', 'trigger', or 'substrate'. The concept of *interpenetration* allows us to acknowledge the developmental and evolutionary interdependence of organism and environment. There is no independent transmission of the information from which traits are constructed. The concept of the developmental system also encourages us to acknowledge and explore many contributions to the phenotype, and not simply to search for 'genetic determinants'. An understanding that a novel feature can emerge as a result of change in any developmental variable opens up many diverse, but complementary, routes of investigation. The fact that the components of the developmental system range from the microscopic to the macroscopic and from the biological to the social allows us to integrate multiple levels of explanation, and to seek 'natural kinds' appropriate to each level of investigation. Our notion of 'heredity' is also extended as the developmental systems approach compels us to be conscious of the fact that many components of the system are 'transmitted', or rather that the presence of the components of the system allow a trait to be reconstructed reliably in ontogeny. Once we have removed the status of genes as unique causal entities we appreciate that organisms are not constructed as a result of 'blueprints' or 'programs'; we are conscious of the non-hierarchical and distributed nature of the regulation of traits, which in turn allows us to think in terms of 'continuous construction and transformation' rather than transmission. Finally, the developmental systems approach promotes 'theoretical extension and unification', as our theories must encompass a broader range of information (Oyama, 2000a, pp. 2-7). Developmental systems theory holds the promise of a non-reductive integration of what are often characterised as competing disciplines, such as biology, psychology, and sociology, and as such it is comple-

mentary to the causal homeostatic theory of natural kinds, which is discussed in the following chapter.

The preformationist, or 'genetic program' perspective on development is particularly prevalent in psychiatry. For example, Simon Barondes, an eminent neurobiologist and professor of psychiatry at the University of California, San Francisco, describes the distinction between 'genotype' and 'phenotype' in the following terms 'genotype refers to an individual's specific gene variants, whereas phenotype refers to their observable expression' (Barondes, 1999, p. 22). This definition is clearly based on the notion that genes carry the instructions for an organism and the environment provides the substrate or backdrop against which the developmental program unfurls. In fact the word 'phenotype' in biology refers to all of the observable characteristics of the organism resulting from the interaction of its genotype and the environment, not to the observable expression of genes, though perhaps the etymology of the word is suggestive of its preformationist roots[6].

Even those who are well-disposed towards developmental systems theory seem to find it difficult to abandon the idea of genes as the repositories of codes and programs. The neurobiologists Jaak and Jules Panksepp claim to have assimilated the prescriptions of developmental systems theory, as embodied in the work of Oyama (2000b) and Griffiths (1997), but throughout their recent paper describing the new discipline of neuroevolutionary psychobiology (which is offered as an alternative to evolutionary psychology) they refer constantly to 'genetically dedicated circuits' and 'genetically dictated adaptations' (Panksepp & Panksepp, 2000), which suggests that they are not aware that the theory explicitly opposes the notion of genes as privileged causal entities. When their misconception of this approach was pointed out to them (Pitchford, 2001), the Panksepps responded

> We also do not support the notion that the genetic material contains pre-determined outcomes… As Pitchford may have detected, we do disagree with certain variants of developmental per-

[6] *Phenotype* n. Early 20th century. From German *Phänotypus*, literally 'type that shows', from Greek *phainein.*

> spectives as advocated by some members of the philosophical community, who seem to relegate DNA to less of an "informational" molecule than most biologists are prone to agree… Although we fully subscribe to the importance of developmental landscapes in moulding higher mind/brain capacities, we do not agree with the full revolutionary fervour of the "Ontogeny of Information" critique of genetic influences. The genes are more influential in the construction of organisms than the classic Oyama type of view seems to accept. We would be surprised if Pitchford would disagree. Surely, we all now agree that genes can do nothing without supportive environments. However, a remarkable amount of organismic competence naturally unfolds from the genome and the resulting internal milieu as long as a minimally supportive external environment is present (Panksepp & Panksepp, 2001, p. 66)

The Panksepps earnestly advocate developmental systems theory whilst simultaneously holding onto the idea that characteristics of the organism reside in the genome and develop given a minimal environmental substrate. This is a vision of *growth*, rather than of development. Once again a line is drawn, and though the Panksepps' model permits genes to determine most aspects of the organism, the 'higher mind/brain' is moulded by the 'developmental landscape'. This is an extraordinary demonstration of the power of the preformationist vision of genes and genomes to persist even when a commitment to an interactionist perspective is made explicit. The Panksepps' formulation perpetuates the very 'nature versus nurture' dichotomy that developmental systems theory aims to transcend.

Perhaps the most insidious consequence of the genetic blueprint idea is the expectation that phenotypic characteristics should be innate, meaning 'hereditarily determined', 'preformed', or 'arising independently of environment or experience' (Lehrman, 1953). Describing a trait as 'innate' confuses at least four properties that can vary independently:

> (1) that it is found in an individual because of their ancestry rather than their current environment; (2) that its growth does not depend on that environment for anything but basic sustenance; (3) that it is present at birth or early in development; and (4) that it is part of the "nature" of the species… The result of this mismatch between concept and reality is that when theorists discover that one element of the innateness concept applies to a trait, they are liable to

> assume that the other elements must also apply' (Griffiths, 1997, p. 104).

Our faculties are the product of developmental systems, and consequently they are neither innate nor structured by the environment.

One recent example of the extent to which confusion over innateness confounds contemporary debates about the nature of our psychological faculties is found in a volume on connectionism entitled *Rethinking Innateness: A Connectionist Perspective on Development* (Elman, et al., 1996). The authors set out to explain how highly constrained and universal forms and behaviours emerge from interactions at all levels but are not contained in the genes in any domain-specific way. Although they claim allegiance to an 'obvious' interactionist perspective in which neither genes nor environment determine outcomes they insist that evolved 'modules' or domain-specific psychological adaptations are untenable because these structures must be ensured by and contained within the genome. Hence, their commitment to an interactionist perspective is intended only to be applicable to one very small part of the natural world, the human brain. Accordingly, these authors regret a 'widespread willingness to believe in single genes for complex outcomes' (Elman, et al., 1996, p. 41). Of course this is simply hyperbole; domain-specific outcomes can be the result of developmental systems in which genes play an indispensable role, even if the modules are not 'contained in the genes', and no such outcome need be influenced by a single gene. All evolved traits emerge in development under the influence of genes, but none of these outcomes is determined by the genes themselves. Moreover, what these authors really seem to oppose is not the idea of domain-specific psychological modules but the idea of innate representations or 'prior knowledge'. They say that 'we are prepared to call many universally recurring patterns of behaviour – in languages, for example – innate, even though we find them specified nowhere in the genome' (Elman, et al., 1996, p. 46). From a developmental systems perspective it is true that 'the interesting question is not whether or not the brain is modular (it clearly is), but how and why it gets to be that way' and that 'there is a huge difference between starting modular and becoming modular' (Elman, et al., 1996, p. 101). However, because Elman et al.

cannot reconcile their view of development with their preformationist conception of genes and their distaste for innate representations they end up with a scheme that seeks to reject the involvement of genes completely, and holds explanations incorporating genes to be ideologically pernicious and irresponsible.

> Interest in innate ideas and innate constraints on cognition has reached another high-water mark. This is evident in popular books on "human instincts" (e.g., Pinker, 1994), but it is also evident in books that argue for racial differences in intelligence (Herrnstein and Murray, 1994). Of course, these two approaches to innateness are not the same. One can obviously argue for the innate basis of characteristics shared by all human beings while rejecting the notion that individual or subgroup differences are immutable. But this neat division runs into difficulty as we move from behavioural description to the elucidation of an underlying mechanism. The problem is that genetic differences and genetic commonalities come from the same source. If we ascribe a complex and highly specific ability to some direct genetic base, then we have opened the door to genetic variation and the disturbing socio-political implications that ensue (Elman, et al., 1996, p. 391).

After almost four hundred pages on connectionist modelling and brain development we find that, behind an insistence on the developmental emergence of modules and of the rejection of the *tabula rasa,* lies the misconception that genes really do create *only* immutable characteristics and that, accordingly, the brain (being highly mutable) is best viewed as an organ that becomes modular without the involvement of genes in any specific modular outcome. For Elman and colleagues any other result would be an 'unhappy conclusion' capable of doing 'damage to future generations of children' (1996, p. 391). This confusion about innateness begins with a commitment to an appreciation of interactionism but ends with an abiological view of the human brain in which the appearance of modules is better attributed to creation rather than development. In essence, these authors are discussing not the architecture of the mind, but the structure of the soul.

One of the authors of *Rethinking Innateness* has since take the conclusions of this volume even further arguing that because 'behaviours are not simply triggered from genetically determined mechanisms', insights relevant to evolutionary claims cannot be made from the study of adult brains, though the study of

the brains of children could be relevant (Karmiloff-Smith, 2000, p. 147). However, it is precisely because genes participate in outcomes that evolutionary models of the psychological functioning of children and adults are valid. Karmiloff-Smith tells us,

> Nativists would argue for mosaic development. It is under tight genetic control, fast, involves the independent development of different parts of the system and is fine under optimal conditions. However, more or less everything must be specified in advance and there are upper bounds on complexity. Some species do indeed follow mosaic development and some parts of all development are mosaic in nature, that is, their epigenesis (their genetically determined development) is indeed deterministic (Karmiloff-Smith, 2000, p. 153)

What appears to be a developmental model, and is described as an interactionist perspective, ultimately draws on a belief in the preformationist ideas that are initially ruled out as 'obviously wrong'. Karmiloff-Smith and her colleagues evidently believe that there are genes for characteristics, and that organisms evolve, but there is one special area where these concepts simply do not apply: the higher reaches of the human mind. If this is correct, Elman et al. provide no coherent reasons as to why we should think so, and their belief that genes are only relevant if they specify everything in advance is simply false.

Darwinian Fundamentals and Darwinian Fundamentalism

Amongst the most prominent of those attempting to restore the primacy of mind is the evolutionary biologist Stephen Jay Gould, one of the early critics of sociobiology (Allen, et al., 1975; 1976; 1977), and now of evolutionary psychology (Gould, 1991; 1997a; 1997b). As Gould is influential in many fields outside biology the viewpoint he promotes is of particular interest and relevance. Gould's critique is based on a number of contributions to evolutionary theory that have led him to believe (at least sometimes) that neo-Darwinism 'as a general proposition, is effectively dead, despite its persistence as textbook orthodoxy' (1980, p. 120).

In a recently published critique of evolutionary psychology amongst the developments said to represent ‘the invigoration of modern evolutionary biology with exciting nonselectionist and nonadaptationist [are] data from the three central disciplines of population genetics, developmental biology and palaeontology’ (Gould, 2000, p. 86). The first of these ‘nonadaptationist’ ideas is Motoo Kimura’s neutral theory of evolution (Kimura, 1983). This theory deals with the random substitution of nucleotides, and represents change at the molecular level which has no effect on the structure of the protein coded for, and therefore has no phenotypic effect. Because such neutral evolution has no phenotypic effect it is irrelevant as far as a consideration of adaptationism is concerned (Dawkins, 1982, p. 32). Gould describes it as ‘an elegant, mathematical account of the large role that neutral, and therefore nonadaptive, changes play in the evolution of nucleotides, or individual units of DNA programmes’ (2000, p. 89), which is correct, but he fails to point out that this has no bearing on his critique of evolutionary psychology specifically, or evolution by natural selection generally.

The second example of alleged ‘exciting nonselectionist and nonadaptationist data’ is the case of *Homeobox* genes taken from developmental biology. These genes contain a special DNA sequence called the *homeobox* that codes for a 60-amino-acid sequence called the *homeodomain*. The homeodomain forms part of a gene product known as a transcription factor. These transcription factors bind to specific sites on DNA and regulate gene expression. Some of these homeobox genes specify a region of the body where a structure will form, and a subset of homeobox genes called *Hox* genes keep the segments along the anterioposterior axis from being the same (Stearns & Hoekstra, 2000, p. 299). The *Hox* genes are highly conserved and regulate the basic body plan in species as diverse as fruit flies, nematode worms and humans. They provide a powerful demonstration of how the ‘morphological diversity of at least all animals with three tissue layers, and possibly of all multicellular animals, consists of variations within a framework provided by conserved genes’ (Stearns & Hoekstra, 2000, p. 301). For developmental biologists these genuinely exciting discoveries are neither nonselectionist nor nonadaptationist. On the contrary, they demonstrate how natural selection can result in incredible diversity even within the

constraints established by historical contingency. For Gould though ‘if organisms of such different function, and ecology must build bodies along the same basic pathways, then limitation of possibilities rather than adaptive honing to perfection becomes a dominant theme in evolution’ (2000, p. 90). Once again, however, these discoveries have no bearing on the validity of the enquiry into the nature of human psychological adaptations, rather they should gives us encouragement that the study of homologies will yield interesting information about the structure of the human brain.

Gould’s third example, drawn from the field of palaeontology, is his and Niles Eldredge’s theory of punctuated equilibrium (Eldredge & Gould, 1972), described here merely as ‘the extended stability of most species, and the branching off of new species in geological moments… the pattern known as punctuated equilibrium’ (Gould, 2000, p. 90). However, Jerry A. Coyne and Brian Charlesworth of the Department of Ecology and Evolution at the University of Chicago describe this ‘theory’ in the following terms

> Punctuated equilibrium originally attracted great attention because it invoked distinctly non-Darwinian mechanisms for stasis and change. These mechanisms were said to decouple macroevolution from microevolution, leading to Gould's pronouncement that "if Mayr's characterization of the synthetic theory [of evolution] is accurate, then that theory, as a general proposition, is effectively dead, despite its persistence as textbook orthodoxy”. Yet many evolutionists saw no obvious contradiction between punctuated pattern and Darwinian process: Stasis can result from stabilizing selection (for example, long periods of environmental stability); rapid evolution can result from selection-driven responses to sudden environmental change or invasion of new habitats; and the association of morphological change with speciation can result from the fact that both are promoted by adaptation to new environments)... If a scientific theory is to be of any value as a tool for exploring the real world, it must have some stability as a set of propositions open to empirical test. Punctuated equilibrium has undergone so many transformations that it is hard to distinguish its core of truth from the “statement that morphological evolution sometimes occurs episodically” (Coyne & Charlesworth, 1997, pp. 340-1).

At one point in the debate over punctuated equilibrium Gould wrote ‘I envisage a potential saltational origin for the essential features of key adaptations. Why

may we not imagine that gill arch bones of an ancestral agnathan moved forward in one step to surround the mouth and form proto-jaws?' (1980, p. 127), though he no longer describes the theory in those terms. However, it is this idea of a 'sudden leap' or saltation that has become influential in academic disciplines beyond biology. The philosopher Jerry Fodor, for example, uses this idea in his critique of evolutionary psychology to claim that 'it is entirely possible that quite small neurological reorganizations could have effected wild psychological discontinuities… ("saltations" as one says) in cognitive capacities in the transition from the ancestral apes to us'. Fodor clearly believes saltationism to be a viable and revolutionary non-Darwinian explanation (just as Gould originally implied) and he concludes 'If that's right there is no reason at all to believe that our cognition was shaped by the gradual action of Darwinian selection' (Fodor, 2000, p. 88). Significantly, Daniel Dennett has explained said that he first learned of the famous critique of adaptationism by Gould and Lewontin (1979) from Jerry Fodor who '…let me in on what the *cognoscenti* all knew: Gould and Lewontin's article had shown adaptationism "to be completely bankrupt"' (Dennett, 1995, p. 240).

Gould also refers to three concepts that 'work as pluralistic correctives to both the poverty and limited explanatory power of the ultra-Darwinian research programme' (2000, p. 96). In addition to punctuated equilibrium ('morphological evolution sometimes occurs episodically'[7]), a concept of no consequence for the viability of evolutionary psychology as a research program, Gould refers to 'contingency and chance in the history of life', which also has no bearing on the fact that complex features of organisms are adaptations, since we should hardly think that there are no evolved human psychological mechanisms because the dinosaurs were wiped out by a catastrophe. These two ideas are said to challenge the gradualism and extrapolationism of neo-Darwinism. Gould's third 'corrective to traditional theory… stresses the limits faced by *any* set of general principles in our quest to explain the actual patterns of life's history' (2000, p.

[7] In Chapter X of *On the Origin of Species* (1859) 'On the Geological Succession of Organic Beings' Darwin writes 'species of different genera and classes have not changed at the same rate, or in the same degree'.

96), but as evolutionary psychology attempts no such explanations this argument is also specious.

Gould's final argument rests on the 'internal error of adaptationism'. This is 'the failure to recognise that even the strictest operation of pure natural selection builds organisms full of non-adaptive parts and behaviours' (2000, p. 103). Gould explains that

> Many, if not most, universal behaviours are probably spandrels, often co-opted later in human history for important secondary functions… Natural selection made the human brain big, but most of our mental properties and potentials may be spandrels – that is, nonadaptive side consequences of building a device with such structural complexity… The human brain must be bursting with spandrels that establish central components of what we call human nature but that arose as nonadaptations and therefore fall outside the compass of evolutionary psychology or any other ultra-Darwinian theory (Gould, 2000, p. 104)

Gould claims not to disagree with biology's emphasis on natural selection but believes 'that we have become overzealous about the power and range of selection by trying to attribute every significant form and behavior to its direct action (1984). Obviously, we should not be interested in 'attributing' anything at all to natural selection. We need to look at the evidence that 'a function is served with sufficient precision, economy, efficiency, etc. to rule out pure chance as an explanation' (Williams, 1966, p. 10).

In their original paper on 'spandrels' Gould and Lewontin (1979) make entirely prosaic observations concerning the ubiquity of phyletic constraints, and argue that the evidence for Aztec cannibalism, the chin, and papillary ridges as adaptations is not strong. In a second paper 'Exaptation: A Crucial Tool for an Evolutionary Psychology' (1991) Gould describes useful characters that did not arise by the action of natural selection (spandrels) as a type of exaptation, or coopted nonaptation, if they come to serve a useful function, but he also describes features that did arise by the action of natural selection as exaptations if they have subsequently been moulded by natural selection for another role:

> Co-opted characters may have been built by natural selection for a different function (e.g., the proto-wing, initially evolved as an adaptation for thermoregulation and later coopted for flight, according to the standard classical conjecture), or may have arisen for no adaptive purpose at all (e.g., as a sequel or consequence of another adaptation, in what Darwin called "correlation of growth"). In either case, co-opted structures will probably undergo some secondary modification-counting as superimposed, true adaptation-for the newly seized function. (The feather, for example, will need some redesign for efficient flight-as we can scarcely imagine that a structure evolved for thermoregulation would be accidentally and optimally suited for something so different as aerial locomotion.) But such secondary tinkering does not alter the primary status of such a structure as coopted rather than adapted (Gould, 1991, p. 47).

Gould seems to be arguing that the proto-wing may or may not be an adaptation, but the wing itself, though it is moulded by natural selection from a proto-wing, is a coopted structure, i.e., an exaptation. As Griffiths and Sterelny point out ‘Gould and Vrba think that a trait is an adaptation only for the purpose for which it was first selected. But what justifies this special status for the first of many selection pressures? The importance of the concept of adaptation in biology is that it explains the existence of many traits of the organisms we see around us. This explanation is not just a matter of how traits first arose, but of why they persisted and why they are still here today’ (Sterelny & Griffiths, 1999, p. 219). The only complex functional characteristic claimed as an exaptation is language, and this is done by argument from authority – the authority in question being Noam Chomsky, who is said to have ‘long advocated a position corresponding to the claim that language is an exaptation of brain structure’ (Gould, 1991, p. 61). Gould has also previously described language as a ‘spandrel’ of the human brain (1987). However, Chomsky actually claims that he has not ‘expressed views on the lack of a role for natural selection in… the origin of language’, on the contrary he believes ‘that natural selection is operative in this case’. (personal communication, 1999).

To complicate matters further Gould claims that exaptations are ‘neither rare nor arcane, but dominant features of evolution - though previously unappreciated in the context of the overly adaptationsist neo-Darwinian theory,’ (1991, p.

43) even though he also insists that 'we reluctantly permit *stare decisis*[8] in retaining "adaptation" for characters built by natural selection for their current use' (1991, p. 47). So when Gould claims that in the human brain ‘exaptations must greatly exceed adaptations by orders of magnitude’ (1991, p. 57), the statement is ambiguous as he has already conceded that many ‘exaptations’ are simply ‘adaptations’ in the normal parlance of biology. As noted above, Gould also refers to the probability that the brain is ‘bursting with spandrels’ (2000, p. 104), but admits that spandrels are often ‘coopted’, and as coopted structures ‘probably undergo some secondary modification – counting as superimposed, true adaptation – for the newly seized function’ (1991, p. 47) many of these ‘spandrels’ are probably adaptations. Apparently Gould is actually saying that the brain is bursting with adaptations, which is a conclusion entirely compatible with the viewpoint of evolutionary psychology. The only remaining ‘exaptations’ are spandrels (byproducts) and adaptations that become useful in a new role without being explicitly moulded for current use (Gould, 1991; Gould & Vrba, 1982). It is difficult to imagine how an unmodified spandrel, papillary ridges, for example, would come to serve some complex function, or how an unmodified adaptation, such as the heart, could possibly take on a new complex function. This useless terminology therefore places adaptations and byproducts in the same category, and Gould regrets that tradition dictates otherwise. Ultimately Gould’s argument seems not be to about adaptationism at all, or with the claim that human psychological attributes are adaptations, although that is how it is phrased, but rather about the *origins* of variation on which selection can act. Gould makes this (fairly) clear in a paper called ‘The Exaptive Excellence of Spandrels as a Prototype’ published in the *Proceedings of the National Academy of Sciences* in 1997 in which he writes

> …in analyzing the evolutionary basis of features now crucial to the functional success of organisms, we must learn to appreciate the range of potential reasons for the *origin* of such traits. The biases of strict Darwinism often narrow our focus to adaptive bases for all aspects of a feature’s evolutionary history — so that the primary mechanism of natural selection may be viewed as a direct *causal basis* for the entire sequence, whatever shifts of function may occur. However, and perhaps ironically, we must recognize that

[8] This is a legal term meaning ‘to stand by things already decided’.

> complexities of structure and development clearly impose a set of attendant sequelae upon any adaptive change. These sequelae — spandrels in the terminology of this paper — arise nonadaptively as architectural byproducts but may regulate, and even dominate, the later history of a lineage as a result of their capacity for *cooptation* to subsequent (and evolutionarily crucial) utility. (Or they may continue as nonadaptive spandrels and still remain important as features central to our understanding and analysis of organic form in evolution.)
>
> A failure to appreciate the central role of spandrels, and the general importance of nonadaptation in the *origin* of evolutionary novelties, has been the principal impediment in efforts to construct a proper evolutionary theory for the biological basis of universal traits in *Homo sapiens* — or what our vernacular language calls "human nature." Promoters of the importance of spandrels, and of nonadaptation in general, are not trying to derail the effort to establish a true "evolutionary psychology" on genuine Darwinian principles ... or even to overthrow the centrality of adaptation in evolutionary theory. We wish, rather, to enrich evolutionary theory by a proper appreciation of the interaction between structural channeling (including the nonadaptive origin of spandrels as a central theme) and functional adaptation (as conventionally analyzed in studies of natural selection) for generating the totality and historically contingent complexity of organic form and behavior (Gould, 1997d, p. 10755, emphasis added).

In an exchange in the *New York Review of Books* Gould takes Steven Pinker to task for attributing complex design to the action of natural selection, explaining that he and Lewontin proposed the term 'spandrel' to 'make a distinction between nonadaptive origin and possible later utility' and to 'expose one of the great fallacies so commonly made in evolutionary argument: the misuse of *a current utility* to infer an adaptive *orig*in'. (Gould, 1997c, emphasis in the original). Gould explains:

> He [Pinker] argues that when an ancestral spandrel becomes modified for an adaptive purpose in a descendant species, then natural selection is the agent of modification. Sure —and I have said so, prominently, in all my papers on the subject. But s*o what*? The origin of the spandrel remains nonadaptive as an automatic architectural byproduct. The secondary modification for utility is, well, secondary — and therefore not a criticism of the claim for nonadaptive origin of the original feature (Gould, 1997c).[9]

[9] Available online at http://www.nybooks.com/nyrev/WWWarchdisplay.cgi?19971009055E1.

However, the discussion of origins is simply a distraction. The issue is whether human psychological faculties are the product of natural selection, not whether natural selection accounts for the origin of all the raw material and all aspects of design, including the features that Gould and Lewontin identify as spandrels. Gould's whole argument seems to be that 'cooptable potentials' are 'inherent in structures built for other reasons' (1991, p. 59). In other words evolution is 'descent with modification', but this conventional Darwinian position is presented as non- or anti-adaptationist in order to restore a 'mind first' approach to human psychology, and to label evolutionary psychology as 'hyperadaptationist'. 'Hyperadaptationism' in the way Gould uses it refers to the claim that selection accounts for the origins of all the design features of organisms, including potentially co-optable spandrels, but evolutionary psychology rests on no such claim. As Gould's critique is directed at accounts of origins rather than of outcomes it is not pertinent to the question of whether or not any components of human psychology display 'eminently workable design'. After considering Gould's arguments we should agree with him enthusiastically that 'words and taxonomies often exert a tyranny over thoughts' (1997c).

The Immortal Merit of Darwin

In 1909 a volume edited by A. C. Seward entitled *Darwin and Modern Science* was published to celebrate the centenary of the birth of Charles Darwin and the fiftieth anniversary of the publication of On the Origin of Species. In a chapter called 'Darwin as an Anthropologist' Ernst Haeckel, professor of zoology at the University of Jena, observed

> To appreciate fully the immortal merit of Darwin in connection with anthropology, we must remember that not only did his chief work, "The Origin of Species", which opened up a new era in natural history in 1859, sustain the most virulent and widespread opposition for a lengthy period, but even thirty years later, when its principles were generally recognised and adopted, the application of them to man was energetically contested by many high scientific authorities. Even Alfred Russel Wallace, who discovered the principle of natural selection independently in 1858, did not concede that it was applicable to the higher mental and moral qualities of man. Dr Wallace still holds a spiritualist and dualist view of the nature of

> man, contending that he is composed of a material frame (descended from the apes) and an immortal immaterial soul (infused by a higher power). This dual conception, moreover, is still predominant in the wide circles of modern theology and metaphysics, and has the general and influential adherence of the more conservative classes of society.
>
> In strict contradiction to this mystical dualism, which is generally connected with teleology and vitalism, Darwin always maintained the complete unity of human nature, and showed convincingly that the psychological side of man was developed, in the same way as the body, from the less advanced soul of the anthropoid ape, and, at a still more remote period, from the cerebral functions of the older vertebrates. The eighth chapter of the "Origin of Species", which is devoted to instinct, contains weighty evidence that the instincts of animals are subject, like all other vital processes, to the general laws of historic development. The special instincts of particular species were formed by adaptation, and the modifications thus acquired were handed on to posterity by heredity; in their formation and preservation natural selection plays the same part as in the transformation of every other physiological function (Haeckel, 1909, electronic edition[10]).

Daniel Dennett argues that 'before Darwin, a "Mind-first" view of the universe reigned unchallenged' (1995, p. 33). Perhaps we could say that since Darwin much of intellectual life has been dominated by the desire to restore a 'mind-first' view of the world.

In this chapter I have discussed the insidious role of covert quasi-theological concepts on contemporary debate and enquiry. In the next chapter I will examine the current status of psychiatric classification before moving on to an overview of the development of sociobiology and evolutionary psychology.

[10] The electronic version of this volume is available for download at http://human-nature.com/darwin/ebooks.html.

Chapter 3

The Problem of Classification in Psychiatry

> Biological thinking gave psychiatry at the end of the twentieth century the capacity to be as science-driven as the rest of medicine. But this promise has remained unfulfilled, a result of psychiatry's enmeshment in popular values, in corporate culture, and in a boggy swamp of diagnostic scientism.
>
> (Shorter, 1997, p. 288)

The Development of Modern Psychiatry

The dominant mode of analysis in contemporary psychiatry is based on what is known variously as the medical, biomedical, biological, or disease model. This model consists of four stages: the description of the clinical syndrome, the identification of pathology, the study of the natural history of the syndrome, and finally the determination of the aetiology (Tyrer & Steinberg, 1993, pp. 7-8). The last three stages covering the effect, development, and cause of the disease are generally subsumed under the term *pathogenesis*, and signs or symptoms considered diagnostic of a particular disease are described as *pathognomonic*[11]. Within the domain of psychiatry, however, the attempt to uncover pathognomonic features of mental illness proceeds at the level of psychology, even at the level of everyday folk psychology, whereas the assessment of pathogenesis generally proceeds at the biological level, being the domain of genetic, physiological, and anatomical investigations. The core problem of psychiatry is to explain how the identification of pathognomonic features at the psycho-behavioural level illuminates underlying biological pathology and vice versa. Although specific biological malfunctions may produce specific patters of psycho-behavioural malfunctioning, we do not yet have a taxonomy of human psychological functions, nor do we have categories of mentally disorder sufficiently specific to allow for investigation to proceed systematically. Indeed, our current schemes of classification in psychiatry do not even identify specific, pathogno-

[11] From Greek *pathognōmonikos*, literally 'that is a judge of disease', from *pathos* 'disease' + *gnōmōn* 'judge'.

monic, features of mental disorders. It is not surprising that biological investigations based on these categories have failed to uncover the aetiology and pathophysiology of any mental disorder.

Because of our failure to produce a model capable of integrating social, psychological, and biological investigations the dichotomies of nature and nurture, mind and body, and emotion and reason remain largely unperturbed in the field of mental health. Indeed, explanation of any phenomena in terms of one of these factors is perceived to rule out an explanation in terms of any of the others. Thus, there are endless debates and controversies surrounding claims as to whether this or that disorder, trait or behaviour can be described as genetic, psychological, or cultural. Our investigations are structured according to whichever theoretical structure prevails in any of the arbitrarily delineated 'disciplines' around which our universities and research institutes are organised. Currently those working in social science departments are prone to favour psychological and cultural explanations, those in psychiatry and biomedical departments are likely to favour genetic or biological explanations. In clinical work psychiatrists, psychologists and psychotherapists usually identify themselves according to their allegiance to some school or tradition based on one of the major models which are broadly-speaking, the biomedical, the psychodynamic, the behavioural, the cognitive and the social. One striking illustration of the supremacy of the nature-nurture dichotomy is the recent upsurge of interest in interdisciplinarity, and in interactionist models, which are claimed to pay due regard to the contribution of genes and environment. However, these models often clearly regard 'natural' and 'nurtural' factors as separate interactants a formulation that actually guarantees a dichotomous approach.

Through its history the science of 'mind healing' has only been able to offer palliatives rather than cures, and the nature of the palliatives offered has been guided by the dominant tradition amongst psychiatrists. According to Valenstein (1998), psychiatry in the post-war era can be divided in to two phases, one from roughly 1945-1960, which was characterised by an emphasis on psychoanalysis ('blaming the mother'), and the period from 1960 onwards which has seen a growing emphasis on neurotransmitters ('blaming the brain'). Allan Hobson and

Jonathan Leonard describe the same period as witnessing the pendulum swing ‘from the brainless mind of Freud to the mindless brain of biomedicine’ (Hobson & Leonard, 2001, p. 12).

Currently in both popular culture and psychiatric practice neurotransmitters are seen as the basis of character traits and disorders. Depression, for example, is often referred to as a disease caused by insufficient serotonin in the brain, whereas schizophrenia is believed to be caused by an excess of dopamine. Various psychopharmacological substances are said to correct these imbalances. In fact, there is almost no empirical support for these assertions. This predilection for single factor explanations of complex phenomena moved the editor of the journal *Psychological Medicine* to observe, perhaps with some understatement, that ‘unfortunately, biological psychiatry has not always been able to avoid the problem identified by Dr Johnson in one of his colleagues – “that fellow seems to me to possess but one idea, and that is a wrong one”' (Cowen, 1998).

In the nineteenth century Heinrich Laehr (1852) and the founder of modern neurochemistry J. W. L. Thudichum (1884), had speculated that mental disorders were caused by chemical changes in the brain, but these ideas had little impact. The rapid post-war move away from the introspectionism of psychoanalysis to the objectivity of biological psychiatry occurred because of key discoveries made in psychopharmacology in the 1940s and the 1950s. The most important of these were Albert Hofmann’s discovery of LSD and its hallucinogenic effects in 1943; the discovery by Jean Delay and Pierre Deniker in 1952 that chlorpromazine could alleviate the symptoms of schizophrenia; Nathan Kline’s discovery in 1956 that the monoamine oxydase inhibitor iproniazid could alleviate depression; Roland Kuhn’s discovery in the 1950s that the tricyclic antidepressant imipramine could elevate mood; John Cade’s discovery in the 1940s that lithium could alleviate the symptoms of manic depression, and the discovery by Frank Berger and William Bradley in 1946 that the minor tranquillisers could alleviate anxiety (for a detailed discussion see Valenstein, 1998, pp. 9-57).

In 1953 Sir John Gaddum reported that LSD blocked the effect of serotonin on the uterus of experimental animals and, following the discovery by Betty Twarog that serotonin was present in the brain (Twarog & Page, 1953), Gaddum hypothesised that LSD's hallucinogenic effects were caused by its antagonistic effect on brain serotonin, although he was careful to point out that ergometrine and Dibenamine also block serotonin without producing psychotic states, and that mescaline, which is comparable to LSD in its psychotogenic effects does not block serotonin (Valenstein, 1998, p. 80). The following year Gaddum speculated that serotonin might be essential for sanity, and that mental states could be modified through the action of psychotropic and psychopharmacological substances on neurotransmitters (Valenstein, 1998, p. 15).

The first report that a psychotherapeutic (rather than a psychotropic) drug could alter the activity of a neurotransmitter was made in 1955 by Bernard 'Steve' Brodie who found that reserpine (used as a treatment for hypertension) reduced the amount of serotonin in the brain. This work was inspired by that of Sir John Gaddum, but had a much greater impact because Brodie's laboratory at the National Institutes of Health was considered at the forefront of research in neuropharmacology and was at that time also hosting Arvid Carlsson who, on his return to Sweden, demonstrated with his colleague Nils-Ake Hillarp that reserpine also reduced brain noradrenaline and dopamine. Thus the three major biogenic amines, serotonin, dopamine, and noradrenaline, were all shown to be reduced in the brain by the administration of reserpine (Snyder, 1986; Valenstein, 1998, p. 70). Although only about 15 percent of those treated for hypertension with reserpine were found to develop symptoms of depression (Barondes, 1999, p. 132) the impact of this work spawned the 'biogenic amine theory of depression' which is still with us today in slightly modified form. The theory has persisted despite the fact that as early as 1959 Erik Jacobsen had shown that two agonists of the biogenic amines, caffeine and amphetamine, were not effective as antidepressants. Jacobsen believed that noradrenaline was key to the elevation of mood and his theory became known as the 'catecholamine theory of depression' as noradrenaline, along with dopamine and adrenalin, is one of the catecholamines, whereas serotonin, central to the biogenic amine theory of depression, is classified as an indoleamine. The relative contribution of these sub-

stances to depression has still not been resolved. In a recent review of research in this area Ronald Duman of Yale University School of Medicine concluded

> These studies have focussed largely on level of monoamines and their receptors and have led to several theories of depression, including the monoamine depletion and receptor sensitivity hypotheses. However, this work has not led to a unifying hypothesis of antidepressant action. Nor can the pathophysiology of depression be explained simply by dysregulation of 5-HT [serotonin] and/or NE [noradrenaline/norepinephrine] neurotransmission. (Duman, 1999, p. 333)

And yet in a book written for a popular audience called *Understanding Depression* Donald F. Klein, professor of psychiatry at Columbia University, and Paul Wender, professor of psychiatry at the University of Utah, write:

> As psychiatrists who have been involved in research with psychiatric patients for almost thirty years, we have been increasingly impressed by the evidence that many severe psychiatric disorders are *diseases*. They are often hereditary, arising from physiological malfunctions (especially in brain chemistry), and their symptoms can be lessened or eliminated by treatment with medication… A striking gap has grown between what is known by clinicians and researchers and what is known by the public, even the psychologically sophisticated public (Klein & Wender, 1993, p. vi, emphasis in the original).

In 1954 the American journal *Science* published a paper by D. W. Woolley and E. Shaw in which they noted that the affects of serotonin on smooth muscle were blocked by LSD, harmaline, yohimbine and a number of other drugs. They argued that schizophrenia and other mental disorders could be a result of a deficiency of serotonin. However, in 1959 Oleh Hornykiewicz demonstrated that patients who had died from Parkinson's disease had brain dopamine levels only 20 percent of normal, and as antipsychotic substances were also known to produce parkinsonlike symptoms, this suggested that they worked by blocking the action of dopamine, and that, consequently, schizophrenia could be a result of an excess of dopamine. The specific suggestion that antipsychotics might work by blocking dopamine receptors was made by J. M. Van Rossum in 1966, despite the fact that it was known that these drugs also inhibited serotonin and

noradrenaline. William Byne of Mount Sinai School of Medicine and his colleagues concluded recently that

> Although the original dopamine hypothesis guided research for three decades, recently a variety of limitations have become apparent. Studies of dopamine metabolites and receptors in postmortem brain and of dopamine metabolites in cerebrospinal fluid (CSF) and plasma have failed to consistently support the hypothesis. Moreover, a substantial proportion of schizophrenics are resistant to treatment with drugs that block dopamine activity… Conversely, the full spectrum of symptoms associated with schizophrenia is not exacerbated by drugs that augment dopaminergic activity (Byne, et al., 1999, p. 236).

The use of lithium treatment for manic depressive disorder (now known as bipolar disorder) has been somewhat more successful as one of the palliative therapies offered by contemporary psychiatry. It is often said that 60-70 percent of patients improve with lithium treatment, though around 20 percent improve with placebo. It is clear, however, that patients have received significant help from treatment with lithium and the anticonvulsant drugs, although no plausible hypothesis as to the action of these drugs has been formulated (Valenstein, 1998, p. 91). The lack of a theoretical model of bipolar disorder probably explains the lack of research work in this area, and indeed Robert Berman of Yale University School of Medicine and his colleagues have remarked that ‘given the severe morbidity of bipolar illness and striking paucity of clinical trials, this subtype of affective illness should become a prime agenda for future research’ (Berman, et al., 1999, p. 424). This seems an astonishing admission coming almost sixty years after the introduction of lithium treatment (Boland & Keller, 1999, p. 292), though it is not surprising that a scheme of investigation based on the neurochemical individuation of traits has been unable to accommodate a disorder characterised by the oscillation between two different states, depression and elation. Overall, it is clear that there are in fact no simple neurotransmitter-illness relationships and as research proceeds it becomes excruciatingly clear that ‘the more that is learned about neurotransmitters and psychopharmacology, the more complex the picture grows: there are more kinds of neurotransmitters, more kinds of receptors, more interdependence’ (Luhrmann, 2000, p. 54).

J. Allan Hobson, professor of psychiatry at Harvard Medical School and director of the Laboratory of Neurophysiology at the Massachusetts Mental Health Center, still claims that 'the antipsychotic drugs that began emerging in the 1950s (the so-called "neuroleptics") were quite specific. They did not simply "dope up" the recipient until he or she became quite compliant. Rather, they targeted particular diseases' (Hobson & Leonard, 2001, p. 13). As we have seen specific targeting is exactly what the antipsychotic drugs did not do. Ironically, Hobson and Leonard remark that 'the brain science knowledge of many practicing psychiatrists remains mostly informal or anecdotal' (2001, p. 72). The conundrum is to explain why these mono-causal neurochemical hypotheses persist in spite of a transparent lack of merit. As part of his explanation for the persistence of unworthy hypotheses Valenstein cites the lack of 'time, inclination, or background to critically examine the evidence' (1998, p. 165) on the part of mental health professionals, and the influence of powerful special interest groups, especially drug companies as contributing factors.

It is certainly true that the influence of the drug companies is pervasive. The journal *Nature Medicine* is holding an opinion poll on the case of David Healy who accepted a joint faculty position at the Centre for Addiction and Mental Health in Toronto, Canada, and the Department of Psychiatry at the University of Toronto, 'only to have the roles declined to him on the basis of a single lecture he gave critical of the drug industry' (Birmingham, 2001)[12]. Healy's lecture contains much of the standard (i.e., relatively uncontroversial) history of drug therapies and discoveries as described above, and his views are also a matter of record. In a review of the book *Deconstructing Psychopathology* (written for the prestigious journal *Psychological* Medicine) for example, Healy explains:

> They [the authors] take issue with, and make much of, a traditional target – psychiatry's power to detain patients on the basis of a supposed dangerousness – but the power invested in prescription-only arrangements is missed. This recent development obviously leads to a much more widespread potential for abuse than any potentially abusive removal of liberties under the Mental Health Act – detention is a rare event compared with prescription. Depriving the people of free and open access to psychotropic

[12] *Nature Medicine* has made the whole of Healy's lecture, including the slides, available on their world wide web site http://www.nature.com/nm/voting/lecture.html.

> drugs, which people essentially "believe" in much more than they do in those who prescribe them or the theories prescribers hold, must necessarily introduce massive distortions into the discourse about psychopathology. Dismantling this privilege would arguably in rather short order dismantle the hierarchies of expertise and authority that have presided over the construction of *DSM-III, DSM-IV* and *ICD-10*. If the pharmaceutical industry could sell directly to the people, how bothered would they be with *DSM-IV*?… I would imagine that the authors would find many professionals – and indeed the higher up the hierarchy they go the more likely they are to find them (the book review editor of this journal would be a good bet) – who would happily concede that the entire edifice of psychiatry depends at least as much if not more on the potential of certain views and practices to sustain livelihoods than by any correspondence that these views or practices have with "the truth". (Healy, 1998, p. 745).

The Guardian (9th July, 2001) also includes an appeal by a group of psychiatrists to the president of their Royal College about the influence of the drug companies' marketing which 'distorts the mental health agenda to the point where pills are seen as the answer to all ills'. More significantly 34 percent of the primary authors of papers in prestigious journals such as *Nature, Science, Lancet* and the *New England Journal of Medicine* have been found to have financial interests in the work they have published (Valenstein, 1998, p. 199). Sheldon Krimsky (2001) of Tufts University also recently reported that of 1400 journals listed in the Science Citation Index (which were chosen for impact factor) less than 1 percent reported any conflict of interest. The editor of the *New England Journal of Medicine* (which in 1984 became the first of the major medical journals to require authors of original research articles to disclose any financial ties with companies) has argued that science is being compromised by the growing influence of industry money, owing to the difficulty of finding reviewers without links to the drug companies. In one recent case the authors of a paper had such extensive ties to the manufacturers of antidepressants that there was insufficient space to list them. The *Journal* had to resort to providing additional material on its web site (Angell, 2000).

This shouldn't be taken to imply that the current state of affairs in biological psychiatry is sustained for the benefit of the drug companies. There are many factors contributing to contemporary nosological chaos including the influence of

the dualist traditions of Western philosophical thought, the genuine efficacy of some psychopharmacological substances in palliative therapy, which leads much scientific research astray (often with perfectly good intentions), and the lack of a coherent alternative to current models of mental illness. As Thomas Kuhn has pointed out there is little chance of a change of paradigms unless there are coherent alternatives (or at least one alternative) on offer (Kuhn, 1962, p. 94). As yet, no scientific alternative to current approaches has been clearly articulated, though psychiatry is not in short supply of critics who think the entire endeavour is misguided. Amongst the most influential views of mental illness articulated in recent views are those of: Thomas Szasz (1961), who sees it as a myth, Ronald Laing and David Cooper, who characterise it as a sane reaction to an insane world (Cooper, 1967; Laing, 1965; Laing, 1967; Laing & Esterson, 1964); Erving Goffman (1968), whose work on asylums led him to view mental illness as a role forced on the individual and Thomas Scheff (1967; 1975; 1984) who attributes it to social processes.

Overall, I concur with Valenstein's assessment

> We are currently in a position where it is clear that none of our theories is right, but we do not know what to replace them with. In the meantime, there are a number of groups that have their own reasons for promoting the theories and glossing over their serious deficiencies, rather than admitting that we really do not know what causes mental disorders or why drugs are sometimes helpful… it is indeed amazing how little biochemical theories of mental disorders have changed over the last half-century… Is this conservatism the result of having been fortunate in getting the theories essentially right at the outset? No, but it reflects two facts: First, a theory that is wrong is considered preferable to admitting our ignorance. Second, the tendency of pharmaceutical companies to develop drugs that are similar to those being successfully marketed seemingly provides support for existing theories without really testing them (Valenstein, 1998, pp. 94-96)

I will demonstrate, however, that Valenstein is wrong in claiming that 'there are few rewards waiting for the person who claims that "the emperor really is nude" or who claims that we do not know what causes depression or why an antidepressant sometimes helps to relieve this condition' (Valenstein, 1998, p. 102). An acknowledgement of the parlous state of affairs prevailing in psychiatry is an

essential prerequisite for progress in both the scientific and the clinical domains. Once we can recognise that the transition from a discipline based on psychoanalysis to one based on the neurochemical individuation of traits and disorders was motivated more by optimism engendered by some success in pharmacotherapy than by solid empirical judgement we can begin to ask what branches of the sciences can best inform our theorising about psychopathology. Those who are overly wedded to current notions of psychopathology should bear in mind the lessons of history. As Edward Shorter argues 'the demise of psychoanalysis was in large measure a result of its own lack of flexibility, its resistance to incorporating new findings from the neurosciences. And this reluctance was directly related to the analysts' fear of being proven wrong' (1997, p. 311).

Classification in Psychiatry

The earliest classification system, the legacy of which is still with us today, was the division of the psychoses by Emil Kraepelin (1856-1926) into the *affective psychoses* and *dementia praecox*, a condition later renamed *schizophrenia* by Eugen Bleuler (1857-1939). This system of classification became known as the Kraepelinian binary system. Only thirty years after the system was established Ernst Kretschmer (1888-1964) argued that it should be replaced by a unitary system in which the psychoses could be viewed as extreme accentuations of normal characteristics, an idea endorsed most recently by psychiatrist Tim Crow (1998) and behaviour geneticist Robert Plomin, who claims that 'there may be no disorders as such, just the extremes of quantitative dimensions' (Plomin, 2001). As Crow explains, in keeping with what we have learned so far, the idea that there are two or more psychoses is undermined by the 'failure to establish 1) that there are pathognomonic features associated with the proposed categories, (2) defined boundaries between categories, or (3) aetiologic agents that are specific to any of the categories' (Crow, 1998). The idea of a single psychosis, however, seems even more unlikely, precisely for the reasons Crow gives.

The United States census provided the first stimulus to the systematic categorization of mental disorders. In 1849 the census included the category 'idiocy/insanity' and in 1880 this was replaced by seven categories of mental ill-

ness: mania, melancholia, monomania, paresis, dementia, dipsomania and epilepsy. On realizing the inadequacy of its efforts the Bureau of the Census assigned the task of delineating variants of mental disorder to the American Medico-Psychological Association, which later developed into the American Psychiatric Association. The Association's first manual the *Statistical Manual for the Use of Institutions for the Insane* was finally published in 1918, and included twenty-two diagnostic categories, most of which we would now recognise as physical disorders. The main purpose of the manual was to facilitate the keeping of accurate records in mental institutions (Valenstein, 1998, pp. 155-156).

The standard system of classification now employed in much of clinical practice and research in the United States and throughout the world is the updated version of the early manual devised by the American Psychiatric Association and now published under the title *Diagnostic and Statistical Manual of Mental Disorders* (1952; 1968; 1980; 1987; 1994). The latest version, *DSM-IV* is said to be 'fully compatible with… *ICD-10*' (American Psychiatric Association, 1994, p. xxi), which is the *International Classification of Diseases and Related Health Problems* published by the World Health Organization. However, Andrews and colleagues have found that the percentage of people positive on either classification who are positive on both ranges from 33 percent to 87 percent for eleven disorders studied, with the average concordance being 68 percent (Andrews, Slade & Peters, 1999). This seems as good an indication as one could require that clinicians around the world are not necessarily speaking about the same phenomena when using current classifications of mental disorder. This approach does still have its adherents, however. Hobson and Leonard recall how, during a visit by the Dalai Lama, Lewis Judd, then Director of the National Institute of Mental Health, was heard to say 'that there were 1800 discrete diagnostic conditions defining mental illness'. As Hobson and Leonard explain

> This official classification system makes it tempting to pigeonhole patients and prescribe psychiatric drugs by rote… Of course, most experienced psychiatrists realize that mental ills defy this sort of pigeonholing and respond poorly to such cavalier treatment. Even so, *DSM-IV's* authoritative status and detailed nature tends to promote the idea that rote diagnosis and pill-pushing are acceptable (Hobson & Leonard, 2001, p. 125).

‘Schizophrenia’ as an Exemplar of DSM Categorisation

‘Schizophrenia’ has been described by one critic as ‘the sacred symbol of psychiatry’ (Szasz, 1976) as it is often regarded as the prototypical example of a genuine mental disorder. However, as Valenstein concludes ‘schizophrenics are a very heterogeneous group and most if not all mental health professionals think that it is likely the diagnosis covers several separate disorders with different aetiologies’ (1998, p. 115). It is tempting to think that this is just the idiosyncratic of one neuroscientist, but in fact it does represent the consensus in the field, even though many clinicians may not be aware of this. In 1999 Oxford University Press published an authoritative guide to the current state of research in the neurobiology of mental illness authored by over 130 distinguished individuals. In their introduction to the section on the neurochemistry of schizophrenia William Byne, Eileen Kemether, Liesl Jones, Vahram Haroutian, and Kenneth L. Davis, who are all based in the respected department of psychiatry at the Mount Sinai School of Medicine in New York, write:

> Schizophrenia involves impairments in a variety of functional systems. The exact constellation of symptoms varies tremendously from one patient to the next and no single one is pathognomonic of illness. In addition to the heterogeneity of symptoms, schizophrenia is heterogeneous in other respects including age of onset, clinical course, neuroanatomical correlates, and responsiveness to particular pharmacological agents. There are also differences in genetic loading… Given the heterogeneity of schizophrenia, it is unlikely that all cases share a common aetiology. Instead, it is more likely that impairments resulting from a variety of different neurological insults are collectively classified as schizophrenia in our current nosology. Because these insults could affect different aspects of brain function as well as different brain regions, neuronal types, and neurotransmitter systems, we should not expect any singular hypothesis to account fully for either the full range of schizophrenic symptoms or every case of schizophrenia (Byne, et al., 1999, p. 236).

In other words the diagnostic category has no validity, and its presence in psychiatric nosology is detrimental to scientific research and clinical practice, because it subsumes groups of people who have quite different functional impairments, and these impairments are probably attributable to quite different

causes. As a rough analogy we might group together all people suffering problems of vision (though this would be far more specific than the *DSM* category of schizophrenia) as a prelude to further investigation of functional impairment, genetic influences, clinical course and outcome, epidemiology, and so on. It would come as no surprise that a single model would be incapable of describing the data collected. Some with vision problems have no eyes, others have damage to the visual cortex, still others are suffering from infections and other impairments attributable to environmental factors. And yet the *DSM-IV* classification of 'schizophrenia' refers to 'characteristic symptoms' and claims that 'structural abnormalities in the brain have consistently been demonstrated in individuals with schizophrenia as a group' (American Psychiatric Association, 1994, p. 280). The manual goes on to claim that there is a 'typical' age of onset, that 'the essential features of the condition are the same in children' and that prevalence rates are 'similar throughout the world' (1994, pp. 281-2). None of these claims is accurate, but they help to convey the impression that 'schizophrenia' is a recognisable and relatively homogeneous entity. Needless to say, there is no reference to the dopamine hypothesis of schizophrenia as the flaws in this model have always been apparent, and in fact the only reference to treatment with antipsychotics addresses the serious motor abnormalities that result from this treatment, such as tardive dyskinesia and neuroleptic malignant syndrome (1994, p. 280). In the section on differential diagnosis psychiatrists are advised to differentiate between schizophrenia and general medical conditions which 'can present with psychotic symptoms' (1994, p. 283), even though it seems plain that valuable data on the nature of the functional impairments implicated in psychosis could be obtained by grouping together those displaying the same specific symptom.

Although research scientists in neurobiology are much more keenly aware of the problems with current nosology, the situation in clinical practice is somewhat different, and clinicians are often keen to endorse current models. Peter Tyrer and Derek Steinberg, both British psychiatrists, write in their book *Models of Mental Disorder* that 'it is remarkable that the symptoms of schizophrenia are virtually the same in all cultures and all races; people are not the same but ill-

nesses are' (Tyrer & Steinberg, 1993, p. 18). With regard to the possibility of establishing schizophrenia as a medical condition they conclude,

> It has recently been confirmed that major tranquillizers are effective in schizophrenia because they block the effects of a naturally occurring amine dopamine, on certain sites (receptors) in the brain. There is also evidence that patients with schizophrenia have a structural abnormality in the brain (temporal lobe) which differentiates them from those with other mental disorders. If this is confirmed the second stage of the disease model, identification of pathology, will soon be complete (Tyrer & Steinberg, 1993, p. 20-21).

Mary Boyle, author of the classic critique of the concept of 'schizophrenia', *Schizophrenia: A Scientific Delusion,* would refer to this passage as an example of the 'we're getting there' argument (Boyle, 1990, p. vii). Boyle discusses a number of arguments often employed to support the construct of 'schizophrenia', including *the confusion of observation of and inference argument*, in which those who deny the validity of the concept are judged to be denying the existence of genuine behaviours or symptoms covered by the syndrome, such as hallucinations and delusions; *the necessity-of-classification argument*, in which it is argued that psychiatrists are simply following the method of the natural sciences by producing systems of classification, even though the systems of classification produced are not predictive, nor based on consistent observations; *the 'it might be true' argument* which relies on the fact that tentative syndromes in medicine have previously been demonstrated to be valid; *the defence by comparison argument* in which 'schizophrenia' is regarded as similar to other constructs in science and medicine (e.g., 'electricity' or 'diabetes') which are not yet fully understood; *the usefulness of 'schizophrenia' argument* in which it is argued that the construct helps to predict outcome and response to intervention, even though, as we have seen, there are no regular outcomes or patterns of response to therapy; and finally *the patterns by multivariate analysis argument* in which certain 'schizophrenic' behaviours subjected to factor analysis are said to cluster together above chance levels, even though the technique is dependent on subjective judgements and the samples are highly pre-selected. (Boyle, 1990, pp. 161-177). Though the ingenuity of these arguments is admirable, it is regrettable that so much time and effort has been spent on constructing dubious

defences of current nosology rather than in exploring the foundations of viable alternatives.

DSM Classification

Good classification in any discipline should have heuristic value and 'predict a maximum number of unknown characters' (Fink, 1979, p. 371) in order to allow robust extrapolation from observed to unobserved instances. The current version of *DSM*, *DSM-IV*, aims to be 'a helpful guide to clinicians' and 'to facilitate research and improve communication among clinicians and researchers' (American Psychiatric Association, 1994, p. xv). The definition of mental disorder used in the earlier versions of the manual, *DSM-III* and *DSM-III-R*, is retained

> …because it is as useful as any other available definition and has helped to guide decisions regarding which conditions on the boundary between normality and pathology should be included in *DSM-IV*. In *DSM-IV*, each of the mental disorders is conceptualized as a clinically significant behavioral or psychological syndrome or pattern that occurs in an individual and that is associated with present distress (e.g., a painful symptom) or disability (i.e., impairment in one or more important areas of functioning) or with a significantly increased risk of suffering death, pain, disability, or an important loss of freedom. In addition, this syndrome or pattern must not be merely an expectable and culturally sanctioned response to a particular event, for example, the death of a loved one. Whatever its original cause, it must currently be considered a manifestation of a behavioral, psychological, or biological dysfunction in the individual. Neither deviant behaviour (e.g., political, religious, or sexual) nor conflicts that are primarily between the individual and society are mental disorders unless the deviance or conflict is a symptom of a dysfunction in the individual, as described above (American Psychiatric Association, 1994, pp. xxi-xxii).

By locating the source of disorder within the individual this approach parallels that of general medicine and perhaps distracts attention from external factors (Kutchins & Kirk, 1997, pp. 31-32). As Tanya Luhrmann (2000) explains, there are good cultural reasons for locating aetiology within the body, which once again owe their origins to the dualistic traditions of Western theology and phi-

losophy. ‘We still think of the body as something unintentional, something given, something for which any individual is not responsible… If something is in the body an individual cannot be blamed; the body is always morally innocent. If something is in the mind, however, it can be controlled and mastered, and a person who fails to do so is morally at fault’ (Luhrmann, 2000, p. 8). The *DSM* definition fails to explain why disorders should be unexpected or rare (in medicine some pathogens affect a majority); why impairment should be a sign of dysfunction (problems with reading or calculation, for example, usually aren’t); or why the primary cause should be within the individual (Kutchins & Kirk, 1997, p. 32-34). This latter requirement is particularly ambiguous, as in the case of many well-defined medical disorders the primary causal agents, such as toxins, are within the environment, though of course they can have no effect unless mediated by processes within the individual. However, most would consider it inappropriate to think of ameliorating lead poisoning by administering to the individual substances capable of increasing lead tolerance. The definition also implies that suffering is a guide to dysfunction, without giving any clear guidance as to how one might distinguish function from dysfunction.

By narrowing the focus to dysfunctions causing harm the definition resembles Wakefield’s evolutionary definition of mental disorder as ‘harmful dysfunction’, in which a function is that for which a structure or process is selected for (Wakefield, 1992; 1997; 1999), but it makes no explicit reference to the principles of evolutionary theory, which underlie our understanding of biological function in general. Wakefield’s attempt to introduce evolutionary thinking into psychiatric classification is admirable as an attempt to bring psychiatric definitions in line with those used in biology, but it is likely to be abortive because whether dysfunctions cause harm and should be treated is a matter of historical and sociopolitical contingency. There is no reason in principle why a dysfunction should not be considered particularly desirable, depending on how optimality is currently defined against the backdrop of prevailing local conditions. Inevitably, Wakefield has been taken to task for failing to disentangle the evaluative and objective elements in his formulation (Fulford, 1999; Kirmayer & Young, 1999; Sadler, 1999). It is not surprising that one of the chief architects of *DSM* nosology (since *DSM-III*) Robert Spitzer (1999), has already expressed the

opinion that the current schemes of classification would remain largely unchanged should Wakefield's suggestion be adopted. Clearly, *harm* and *dysfunction* need to be assessed separately, and if this is done our conclusions about causality and about the validity of the *DSM* approach will be considerably more radical.

One of the most significant aspects of the *DSM-IV* definition is that it 'avoids any requirement that the etiology… be identified or that the disorder be understood through the lens of some theoretical system of explanation' (Kutchins & Kirk, 1997, p. 32). Though the *DSM* is nominally atheoretical, and lacking in reference to aetiology or pathology for most of the syndromes described therein, the approach taken does imply that only disorders whose primary causal factors are internal to the individual and capable of causing harmful dysfunction are the legitimate objects of attention, not only in terms of clinical intervention, but also for scientific research. *DSM* can be regarded as an arbitrary or nominalistic scheme of classification because of its inattention to causality, and Paul Muscari has described this 'nominalist turn' in psychiatry as 'raising serious doubt as to whether the traditional concept of 'mental disorder', or for that matter any other psychopathological designation, can truly posses either existential or practical import'. Muscari refers to the *DSM* vision of mental disorder as 'an indexical cluster of properties and events rather than a distinct psychological impairment' (1981, p. 553). Unless we know that the phenomena grouped together in a clinical syndrome systematically co-vary because they are causally related to an underlying unitary process our taxonomy is unlikely to serve as a suitable basis for induction and explanation in a science of psychopathology. In fact, there are no good reasons at all to believe that an avowedly atheoretical scheme of classification, designed to minimise disagreement among professionals with differing responsibilities and emphases, which is built upon the vague terms of clinical phenomenology, and aimed at ameliorating individual and social distress, could significantly enhance our understanding of human psychological functioning. As Poland and colleagues observe,

> It appears *unlikely* that the domain of psychopathology is best conceived of in terms of syndromes with unity or that natural kinds

> will be discovered at the level of clinical phenomenology. There is simply no reason to suppose that the features of clinical phenomenology that catch our attention and are the source of great human distress are also features upon which a science of psychopathology should directly focus when searching for regularities and natural kinds. Human interests and saliencies tend to carve out an unnatural domain from the point of view of nomological structure. Hence the relations between the scientific understanding of psychopathology and clinical responsiveness to it may be less direct than is commonly supposed. In insisting that classification be exclusively focused on clinical phenomenology, *DSM* not only undermines productive research but also undermines the development of effective relations between clinical practice and scientific understanding (Poland, Von Eckardt & Spaulding, 1994, p. 254).

Whilst Poland and colleagues are surely right in claiming that the lack of methodology capable of identifying natural kinds inhibits both scientific research and the possibility of action at an appropriate level of intervention, we should acknowledge that the conflicting aims of research and clinical practice may be irreconcilable simply because the clinician's primary concern will always be with the reduction of harm as it is currently perceived, and not with a concern to correct dysfunction. Kandel recalls that when he entered clinical training in the summer of 1960 an interest in people and an interest in research were regarded as mutually incompatible:[13] 'Reading, they argued, interfered with a resident's ability to listen to patients and therefore biased his or her perception of the patients' life histories. One famous and much quoted remark was that 'there are those who care about people and there are those who care about research' (Kandel, 1998, p. 458).

A Sociological Perspective on Psychiatric Classification

Tanya Luhrmann's idea of the distinction between the 'blameless body' and the 'morally culpable mind' helps us to interpret some of the political machinations surrounding the development of the *Diagnostic and Statistical Manual of Mental Disorders*. As Kutchins and Kirk note 'mental disorders are no longer created by

[13] George Heninger notes that 'as recently as 30 years ago, there was strong opinion in American psychiatry, incorporated into institutional procedures and organization, that clinical questions in psychiatry could not be investigated with the scientific method' (1999, p. 90).

any one small group', but by many pressure groups located inside of the American Psychiatric Association and in the wider culture, and hence 'we get the best view of this process when there are public disputes regarding particular diagnostic categories' (1997, p. 17). Amongst the disputes analysed by Kutchins and Kirk are those over homosexuality, which appears in *DSM-II* but was removed from *DSM-III* after a campaign by gay activists, including gay psychiatrists; Posttraumatic Stress Disorder, which was included in *DSM-III* after a call by war veterans to have their plight acknowledged, and the proposed Masochistic Personality Disorder, which was finally included in *DSM-III-R* in 1987 (reframed and renamed as Self-Defeating Personality Disorder), but which was excluded from *DSM-IV* in 1994 after a campaign by feminists who saw it as a tool to pathologise the struggle of women under patriarchy. The quite different motives of these campaigns are quite striking. In the case of homosexuals and women the desire was not to have their lives pathologised, but in the case of war veterans the motive was to secure appropriate medical intervention and to have their plight officially acknowledged. A pressure group called the Vietnam Veterans Working Group was established to gain allies for their proposed diagnostic category of Catastrophic Stress Disorder, and the new category renamed 'Posttraumatic Stress Disorder' appeared in *DSM-III* in 1980 (Kutchins & Kirk, 1997, pp. 100-125). Edward Shorter summarises the impact of the disputes over homosexuality, PTSD and Self-Defeating Personality Disorder

> In the years after 1971, the Vietnam veterans represented a powerful interest group. They believed that their difficulties in reentering American society were psychiatric in nature and could only be explained as a result of the trauma of war. In language that anticipated the "struggle for recognition" of numerous later illness attributions, such as repressed memory syndrome, the veterans and their psychiatrists argued that "delayed massive trauma" could produce subsequent "guilt, rage, the feeling of being scapegoated, psychic numbing and alienation"… Once it became known how easily the APA's Nomenclature Committee had given way on homosexuality it was clear that the psychiatrists could be rolled… It was not the result of further study but of political pressure that self-defeating personality was dropped… These matters could all be pathologized and depathologized at the will of the majority, or following campaigns of insistent pressure groups. The underlying failure to let science point the way emphasised the extent to which *DSM-III* and its successors, designed to lead psy-

> chiatry from the swamp of psychoanalysis, was in fact guiding it in to the wilderness (Shorter, 1997, p. 304-305).

There is little wonder that Kutchins and Kirk describe *DSM* mental illness as a 'construct', that is a 'shared idea supported by general agreement' (1997, p. 23). I have considerable sympathy with their designation of *DSM* as 'compendium of constructs' (1997, p. 24). This does not imply, of course, that many people are not suffering functional impairment, or that medical intervention cannot be ameliorative or curative, only that the current nosology does not advance these aims. Our clinical and scientific objectives will not be attained until we recognize that the promotion of diagnostic reliability in terms of these constructs is not a desirable aim in itself.

Concepts, Categories, and Theories

If we take it that the history and current status of psychiatric classification provide little scope for confidence how are we to proceed? The evidence from developmental psychology suggests that people do not use concepts to record a summary of properties in past instances, but instead group instances according to their possession of theoretically significant properties in their causal-explanatory theory of the domain (Gopnik & Meltzoff, 1997; Keil, 1989). That is, they have a theory-driven approach and not a probabilistic approach to concept formation. Concept acquisition in science is thus a continuation of normal human conceptual development in which the intension and extension of concepts is amended in the light of empirical evidence. Psychiatric classification, however, is based on phenomenological entities composed of clusters of seemingly correlated properties deemed significant by clinicians. Although it is recognised that classification should be revised as a result of discoveries pertaining to causality, in practice the human tendency toward essentialism and the emphasis on reducing harm via clinical intervention has resulted in premature confidence in the existing taxonomy of disorders.

In the next section I consider a way to describe and identify natural kinds, and how to define 'function' in order that we might understand the causes of dys-

function. This in turn should enable us to determine whether the causes of mental disorders are indeed internal or external to the individual, and illustrate further the underlying antagonism between the concerns of scientists and clinicians.

What are 'Natural Kinds'?

Paul Griffiths argues that 'the existence of natural kinds… provides the ontological element of a solution to the problem of induction. The epistemic element of a solution requires a way of identifying natural kinds' (1997, p. 174). According to the causal theory of reference the world is not 'a welter of sensation which can be parsed with equal plausibility in an indefinite number of ways' (Gardner, 1985b, p. 351), rather 'natural kinds' (Putnam, 1975) or 'rigid designators' (Kripke, 1972; 1980) allow extrapolation from observed to unobserved instances, and therefore serve as a basis for explanation and induction, because all instances possess an underlying microstructural essence. Thus the microstructural properties of natural kinds account for their projectability and utility in the natural sciences. Unfortunately for this conception of natural kinds, John Dupré has explained that this vision could not apply to biological taxa, which are defined on the basis of common descent and not on the basis of a microstructural essence, a problem he claimed as 'fatal to the theory' of natural kinds. (1981, p. 66). However, the formulation of natural kinds as in part a schema to be filled in by empirical investigation need not be linked inextricably to essentialism, or to the notion of natural kinds as the objects of the universal deterministic laws of nature, in which they form the nodes around which theories in the fundamental sciences are constructed (Griffiths, 1997). In his famous paper on the disunity of science as a working hypothesis Jerry Fodor argues that the special sciences can make 'interesting generalizations (e.g., counterfactual supporting generalizations)… about events whose physical descriptions have nothing in common' (1974, p. 103), an insight reinforced by the causal homeostatic theory of natural kinds. As we have seen this is already a basic principle in scientific investigation. To take Thomas Huxley's example, referred to in the previous chapter, water is a natural kind, as all water molecules share a common microstructural essence, but the property of 'aquosity' cannot be identified at the mo-

lecular level. It is clear that even in the physical sciences, where natural kinds as originally envisaged are to be found in abundance, it is still necessary to have multiple levels of analysis, and these levels cannot be collapsed into each other. In the biological, psychological, and sociological domains of enquiry the need for multiple levels of explanation and for varieties of natural kinds that do not depend on a shared microstructural essence is even more apparent.

The Causal Homeostatic Theory of Natural Kinds

Stoljar and Gold have recently characterised the field of mental science as divided between those subscribing to the *biological neuroscience thesis* and those subscribing to the *cognitive neuroscience thesis*. The first group includes theorists such as Crick (1994) who hold that the mind can be understood in terms of its neural substrate. Those constituting the latter group hold that a vast number of disciplines, including biology and psychology, will contribute to an understanding of the mind, a view described as ‘common sense if anything is’ (Stoljar & Gold, 1998, p. 130). Can this commonsense view be articulated more clearly? In the causal homeostatic theory of natural kinds a category brings together a set of objects with correlated properties, and such a category has causal homeostasis if the ‘set of correlations has some underlying explanation that makes it projectable’ (Griffiths, 1997, p. 188). As a corrective to the revival of essentialism inspired by the semantic naturalism of Kripke and Putnam, which was based primarily on a notion of natural kinds drawn from the physical sciences in which microstructural essences are indeed prominent entities, Richard Boyd has argued that

> Kinds, properties, relations, etc. are natural if they reflect important features of the causal structure of the world… Naturalness in this sense is not a property of kinds, but also of properties (solubility in water), magnitude (mass), and relations (exert a force on)…Theoretical considerations determine which complex predicates formulated from natural kind, property or relation terms we should consider projectable; the role of the terms themselves is to refer to causally significant features of the world… Finally… both explanations and scientifically important laws and generalizations may be merely statistical or reflect trends rather than exceptionless regularities, and finding such generalizations or explana-

> tions is no less dependent upon theory-determined identification of causally important kinds, properties or relations than is the identification of exceptionless laws. Indeed to decide otherwise would be to exclude the paradigm of natural kinds – those of biology (Boyd, 1984, pp. 9-11, quoted in Keil, 1989, pp. 42-43)

Though a microstructural essence is one type of causal homeostatic mechanism, there may be many alternatives in other domains of enquiry, and in other disciplines though 'when there are several legitimate taxonomies of a domain, each must have some underlying causal homeostatic mechanism' (Griffiths, 1997, p. 190). Within biology the causal homeostatic mechanism making 'species' into projectable categories is not underlying essences but descent from a common ancestor. Within scientific research generally 'the use of a concept for explanation and induction commits its user to the *project* of having a category with causal homeostasis' (Griffiths, 1997, p. 193). Accordingly,

> Projects for the reduction of special sciences to more "fundamental" sciences have been abandoned, and the "unity of science" has dwindled to a single reality studied in many different theoretical frameworks. This has led to what Richard Boyd has called "the enthusiasm for natural kinds" (Boyd, 1991). Categories from any special science that enter into the generalizations of that science are now commonly regarded as natural kinds… They are ways of classifying the world that correspond to some structure inherent in the subject matter being classified. The "naturalness" of such schemes of classification is not undermined by the fact that there are many of them (Griffiths, 1997, pp. 5-6).

Hence quarks, plutonium, G-proteins, kangaroos, and inflation are all natural kinds at the appropriate level of explanation and induction because rather than representing nominal or arbitrary concepts these concepts represent projectable categories, that is categories which correctly pick out features of the world that cluster together because of some underlying causal homeostatic mechanism. Griffiths has suggested that we should acknowledge that there are at least four levels of explanation in biology alone (each having its particular natural kinds), which are postulated to have the following relationship with levels of explanation in psychology:

<table>
<tr><td>Population Dynamic Level

Traits classified solely by relative fitness functions. Explanation by consequence laws - laws specifying the consequences of variation (Sterelny, 1992, p. 164).</td><td></td></tr>
<tr><td>General Ecological Level

Traits classified by the adaptive problem they solve. Explanation involves source laws - laws explaining variation in fitness (Sterelny, 1992, p. 164).</td><td>Ecological Level
(Level of Task Description)

What does the trait do for the organism?</td></tr>
<tr><td>Natural Historical Level

Traits classified by homology. Explanation by historical narratives.</td><td rowspan="2">Computational Level

How is information processed to accomplish the task?</td></tr>
<tr><td>Anatomical Level

Traits classified by their physical capacities.</td></tr>
<tr><td></td><td>Implementation Level

How are computations physically implemented?</td></tr>
</table>

Table 1: Levels of explanation in biology and psychology. Adapted from Griffiths (1997, p. 221)

We should also acknowledge that there are other problems in trying to interpret phenomena in terms of the microstructure of the systems in which they are instantiated, and this is because higher level properties of complex systems may be multiply realisable in lower level ones (Botterill & Carruthers, 1999, p. 186). A commitment to explanation solely in terms of essential attributes would leave us without access to some of the lawful regularity inherent in higher level processes. However, we should be aware that a commitment to classifying psychological processes functionally, that is in terms of what they do, does not imply that neuroscience and other more fundamental sciences are irrelevant in trying to understand how cognitive-emotional processes function. The concept of *multiple realizability* does not warrant a completely autonomous cognitive science

because knowledge of the organization of the brain, and of the mechanisms operative in its evolution, should enhance our understanding the organization of cognitive-affective systems and may enable us to determine whether there are alternative possible realizations of any given psychological process (Bechtel & Mundale, 1999).

Though functional classifications (rather than genealogical classifications) in biology allow for sophisticated cross-species analyses, and even for comparisons in other disciplines between animate and inanimate systems, we should not forget that the optimal scheme of classification in biology is that based on homology, the core concept of comparative biology. Homologous features in organisms are those characteristics shared by organisms because of descent from a common ancestor. When considering aspects of our own psychology we should remain aware that it is certain that elements of that psychology rely on phylogenetically ancient (though not necessarily unmodified) mechanisms. The serotonergic systems thought to be key to understanding motivation, for example, were essentially in place when the brain first appeared, over 500 million years ago, and serotonin receptors themselves have an evolutionary history going back at least 800 million years (Allman, 1999, pp. 20-21). One of the most surprising discoveries in developmental biology, which dramatically underpins the importance of phylogeny in understanding aspects of human development, was the finding in 1984 that homeobox genes (discussed briefly in the previous chapter) control the development of spatial organization in the fruit fly *Drosophila* and have homologues in animals as different as nematodes and humans (Holland, 1999).

An absolutely key property of homologous structures, which are linked by virtue of descent from a common ancestor, is that they share many arbitrary features that cannot be accounted for by any other means. This property ensures that cladistic (genealogical or historical) taxonomies are ‘maximally predictive’ (Fink, 1979). Any evolutionary psychology or evolutionary psychopathology *must* retain a commitment to the comparative and phylogenetic perspectives. Further progress could rest on an acknowledgement that proper functional taxonomies represent only one level of analysis and that ‘psychology and other human sci-

ences could benefit from the realisation that homologies are legitimate objects of study, and that these studies may be as profitable than studies of functional or analogous categories' (Griffiths, 1997, p. 14). In the case of psychopathology notions of function and dysfunction will only be captured satisfactorily in terms of the descent of mechanisms in particular lineages and the optimal functioning of these mechanisms in evolutionarily significant environments. Whilst an analysis of functional categories will enhance our understanding we should be ever mindful that functional kinds are 'either coextensive with cladistic kinds or with disjunctions of cladistic kinds' and that 'if functional classifications are to be of value in biology, it must be because of their superior generality – the fact that they unite disjunctions of cladistic homologues' (Griffiths, 1997, p. 216).

Natural Kinds, Realism, and Social Constructionism

As Boyd (1991) has pointed out, his view of natural kinds can be detached from a commitment to realism. Scientific concepts are designed to pick out theoretically significant (i.e., projectable) categories and, rather than claim that these concepts correspond to features of the actual structure of the real world, we can say that our theories embrace the relevant empirical information and enable us to construct projectable concepts. Thus Kitcher's 'Kantian Realist' can say that 'the natural kinds would be the extension of the predicates that figured in our explanatory schemata and were counted as projectable in the limit, as our practices developed to embrace more and more phenomena' (Kitcher, 1993, p. 172; quoted in Griffiths, 1997, p. 175). Science can proceed without resolving the perpetual debate over whether theories produce ever more accurate depictions of an objective reality, are models affording greater probability of predicting events, or are better characterised as sociolinguistic constructs which 'appear to be about one thing, nature and her lawful operations, [but] are really about another, man and his ideological manipulations' (Richards, 1987, p. 556). Concepts, including scientific concepts, can be used for many epistemic and non-epistemic purposes, but science must be based on projectable categories and not arbitrary concepts.

Most importantly for a synthesis of the natural and social science perspectives we should be aware that just as the epistemic role of concepts can be severed from a commitment to realism, the separate epistemic and nonepistemic roles of concepts can be discerned via a reconciliation of the causal homeostatic theory of natural kinds and non-trivial versions of social constructionism in the form of the disavowed action and reinforcement versions of the social role model. In a development of particular importance for a science of psychopathology, and for the practice of psychiatry, Griffiths argues that the theory view of concepts, unlike the older causal theory of meaning, can achieve a rapprochement not only between realism and empiricism but between realism and all of its rivals, including social constructionism (1997, p. 175).

Psychiatry and the Social Role Model of Social Constructionism: The Case of Multiple Personality Disorder

In the case of the diagnostic category of Multiple Personality Disorder[14] Ian Hacking has noted that

> Throughout the history of psychiatry, that is, since 1800, there have been two competing ways to classify mental illness. One model organizes the field according to symptom clusters; disorders are sorted according to how they look. Another organizes according to underlying causes; disorders are sorted according to theories about them. Because of the enormous variety of doctrine among American psychiatrists, it seemed expedient to create a merely symptomatic classification. The idea was that people of different schools could agree on the symptoms even if disagreeing on causes or treatment. From the very beginning American *DS*Ms have tried to be purely symptomatic. That is one reason for their limited relevance to the question of whether multiple personality is real. A mere collection of symptoms may leave us with the sense that the symptoms may have different causes (Hacking, 1995, p. 12).

Hacking argues that Multiple Personality Disorder arises as a result of ‘a very general phenomenon: the looping effect of human kinds [(Hacking, 1994)].

[14] *Multiple Personality Disorder*, first included in DSM-III (American Psychiatric Association, 1980), appears in DSM-IV as 300.14 *Dissociative Identity Disorder* [DID] (American Psychiatric Association, 1994, pp. 484-487).

People classified in a certain way tend to conform to or grow into the ways that they are described… multiple personality is an almost too perfect illustration of this feedback effect' (1995, p. 21).

The manifestation of symptoms congruent with the diagnostic category of Multiple Personality Disorder (hereafter MPD/DID) ensures that individuals in need of help and support have access to facilities deemed appropriate for those suffering from mental disorders, but the category itself has its origins in a reflexive mechanism of *dynamic nominalism* (Griffiths, 1997, p. 146). Although no child multiples were known in 1984 Philip Coons stated that 'the onset of multiple personality is early in childhood, and is often associated with physical and sexual abuse' (Coons, 1984, p. 53, quoted in Hacking, 1995, p. 85), and by 1989 Frank Putnam's leading clinical textbook on MPD/DID claimed that 'MPD appears to be a psychobiological response to a relatively specific set of experiences occurring within a circumscribed developmental window' (F. Putnam, 1989, p.45 quoted in Hacking, 1995, p.85). Abundant prototypical cases of MPD/DID appeared as descriptions of the disorder and it's putative causes became widely known; an example of how a 'seemingly innocent theory on causation… becomes formative and regulatory' (Hacking, 1995, p. 95). By 1996 advocates of MPD/DID could claim (truthfully) that 'no reason exists to doubt the connection between DID and childhood trauma' (Gleaves, 1996, p. 42), but the question as to the nature of that connection remains. In fact MPD/DID seems to have its origins in a process which, according to *DSM-IV*, disqualifies it from consideration as a disorder being 'merely an expectable and culturally sanctioned response to a particular event' (American Psychiatric Association, 1994, p. xxi). It is perhaps telling that in a recent survey of board-certified American psychiatrists only one quarter expressed the belief that dissociative amnesia and dissociative identity disorder were supported by strong evidence of scientific validity (Pope, et al., 1999). The authors contributing to a comprehensive survey of the neurobiology of mental disorders stretching to almost one thousand pages referred to in the previous chapter could say of dissociation only that

> The association between different dissociative states and post-traumatic responses remains to be established. At present, dissociative phenomena remain poorly understood, and they may be pathophysiologically heterogeneous. The term *dissociation* itself is unfortunately vague and refers to such a breadth of phenomena that different measurement instruments may be assessing different constructs. The term is used to describe general traits of dissociative tendencies; acute peritraumatic dissociative symptoms such as severe depersonalization or dissociative amnesia; and severe disruptions of normal consciousness as seen in fugue states and dissociative identity disorder. Reliable clarification of phenomenologic models will be especially important to advancing pathophysiologic and clinical study of dissociation… Some forms of dissociation might… be best described as a manifestation of severe anxiety in vulnerable individuals (Marshall & Klein, 1999, pp. 446-447).

Other investigators have claimed that MPD/DID is a 'context bounded, goal-directed, social behavior geared to the expectations of significant others' (Spanos, 1994, p. 143) or an 'adaptive deception of self and others' (Beahrs, 1994, p. 223), and even those committed to the validity of the diagnosis have acknowledged that in some murder cases in which MPD/DID has been cited by defendants as a mitigating factor the explanation of the behaviour is either iatrogenesis or malingering (Coons, 1991). As the controversy over the validity of the diagnosis has grown researchers have attempted to bolster the reality of MPD/DID as a mental disorder (as described in *DSM-IV*) by means of cross cultural comparisons and neuroscientific research. This is a sensible approach, but the results have not been convincing. By administering the Dissociative Experiences Scale to 994 subjects in Turkey, Akyuez and colleagues diagnosed four people as suffering from DID (indicating a prevalence of 0.4 percent) and concluded that these results 'suggest that dissociative identity disorder cannot be considered simply an iatrogenic artifact, a culture-bound syndrome, or a phenomenon induced by media influences' (Akyuez, et al., 1999, p. 151). It should be made clear, however, that the study did not actually identify individuals exhibiting multiple personalities, but simply those who rated highly on a measure of dissociation devised by those committed to the diagnosis of MPD/DID. Much psychiatric research is devoted to establishing the reliability of such measures (or 'instruments' as they are usually called), but without some indication of causality there is no reason to believe that the properties identified are correlated

owing to some mechanism of biological or medical relevance. To take an example from a simpler and unrelated domain: how successful would we be in categorising apparent defects in the operation of a computer in the absence of any knowledge of its functional components? As our examination of psychiatric classification has shown five entirely different phenomena could be attributed to five different errors, or five similar phenomena to one single cause, when in fact all of the different phenomena could be caused by a single hardware fault, or all of the similar phenomena could be caused by five different software faults. We could certainly train individuals to group computer 'pathologies' reliably according to some scheme of classification, and hence gain some indication of prevalence, but this would still leave us without projectable categories (i.e., natural kinds), and without knowledge likely to enhance our understanding of the components mediating the phenomena under investigation.

MPD/DID research also received a boost in the middle of 1999 when Tsai and colleagues reported a functional magnetic resonance imaging study of one individual undergoing a personality switch. This showed a bilateral reduction of hippocampal volume and 'changes in hippocampal and medial temporal activity correlated with the switch, suggesting that personality switch may result from changes in hippocampal and temporal function' (Tsai, et al., 1999, p. 119). But surely, unless we are to believe in supernatural phenomena, all changes in psychological functioning must be underpinned by changes in brain functioning. Any observable changes in brain activity might just as easily be correlated with dissembling or confabulation as with pathological processes underlying dissociation. Tsai and colleagues established only that in this one *single* case the pattern of activation recorded during a putative personality switch differed from that observed while a personality switch was being imagined. The reduction of hippocampal volume is simply consistent with previous studies of the long-term effect[15] of glucocorticoids released during prolonged stress (Sapolsky, 1992).

[15] As opposed to the short-term effect of glucocorticoids which enhance hippocampal functioning (and hence long-term declarative memory for emotionally arousing events) through their effects on the basolateral nucleus of the amygdala (Roozendaal, et al., 1999).

As Tom Fahy, a psychiatrist at the Maudsley Hospital in London, says of MPD/DID 'It's silly to argue whether it exists or not... you can't deny that MPD patients exist. The question is, how has this patient got into such a bizarre mental state?' (quoted in Adler, 1999, p. 28). European researchers generally are more critical of MPD/DID and according to Brugger they tend to 'ascribe the fact that a disproportional number of cases of MPD/DID are reported in the US to the uneasy coexistence of secular and fundamentalist trends which currently splits the American nation so deeply' (Brugger, 1998, p. 283).

Jensen and Hoagwood point out that 'culture and context shape all aspects of mental illness: a given person's subjective experience is culturally shaped, as is the phenomenon or 'disorder' itself, as are the classifications systems by which different groups of persons are classified' (1997, p. 233). The causal homeostatic theory of natural kinds has to accommodate the fact that there are often nonepistemic dynamics at work in concept formation. Concepts are not used solely for explanation or induction but 'to further the interests of individuals or groups, and to promote programs of political action' (Griffiths, 1997, p. 7). In some cases 'the causal homeostatic mechanism for a category might be the existence of the concept of that category and the broader sociolinguistic practices in which the concept is used' (Griffiths, 1997, p. 197). To ask the question 'Is Multiple Personality Disorder real?' when we really intend to ask if the underlying cause or causes are primarily biological reduces our chances of identifying a projectable category at an appropriate (socio-psychological rather than psychobiological) level of explanation. However, though research scientists in biology, psychology and the social sciences may wish to acknowledge a category of 'socially constructed conditions', that is, 'legitimate reasons for adopting the sick role', the question remains as to whether clinicians would wish to do so, given the likelihood that this would undermine their effectiveness, or perhaps even the possibility, that individuals would seek their help.

Socio-psychological natural kinds such as MPD/DID represent disclaimed actions designed (not necessarily consciously) to mimic the passivity (that is, lack of responsiveness to long-term planning) typical of the basic emotional responses, or affect programs, of which there are approximately six or seven:

surprise, anger, fear, disgust[16], sadness, joy and contempt, each with its particular category of elicitor (Darwin, 1998). The passivity of these affect programs arises because they are mediated by relatively autonomous structures (identified in the previous chapters as 'modules') for information storage and processing designed by natural selection (Griffiths, 1997, pp 230-1). Though the number of these basic emotions is in dispute they are considered to be biological responses displaying 'automatic appraisal, commonalities in antecedent events, presence in other primates, quick onset, brief duration, unbidden occurrence, and distinctive physiology' (Ekman, 1994, p. 18). In performing disclaimed actions

> …people display the behaviour that they have learned is socially appropriate in that situation. Neither the individual nor society, however, acknowledges that this is what is happening. Instead, they represent the behaviour as a natural and inevitable response to the circumstances and outside the control of the individual' (Griffiths, 1997, p. 141)

Other examples of disclaimed actions probably include the recent phenomena 'road rage', 'air rage', and 'running amok' (as in the mass murders at Columbine High School or Dunblane Primary School), which are claimed to be natural and uncontrollable responses to environmental or social pressures, but which tend to follow fairly well-defined 'scripts' reinforced by media coverage such as news reports and Hollywood films.

We generally refer to those suffering from genuine illnesses as patients, but the word *patient* itself is derived from the Latin *pati*; which means *I suffer*, and *illness* has usually been seen as 'an involuntary affliction that justifies the sick role and immunizes the patient against charges of exploitive parasitism. Because the sick person has involuntarily impaired functioning, it is only reasonable to exempt him from normal responsibilities' (Klein, 1999, p. 421). Our covert socially constructed disorders, such as MPD/DID allow individuals to adopt the sick role by manifesting signs of a condition regarded as an illness, and

[16] *Disgust* here refers to a basic emotion of *strong revulsion* (evoked by such biologically-relevant things as rotting flesh, parasites, and faeces) produced as the output of something akin to a 'poison-detector module'.

such illnesses gain biological credibility by mimicking the passivity of the basic affect programs on which they are probably constructed. An interesting anthropological example comparable to this Western diagnostic category comes from Philip Newman's study of the Gururumba people who experience a state of 'being a wild pig', which they define as an illness caused by being bitten by the ghost of a recently deceased tribe member. The condition is largely restricted to young males under financial pressure as a result of recent marriage, who can win special dispensations on manifesting the symptoms of this disorder. These symptoms include petty theft and indiscriminate attacks on bystanders (Newman, 1964). As there are no non-domesticated pigs within the Gururumba environment 'the wildness of a pig does not consist of its living outside the realm of human control, but consists of its breaking away from a set of imposed conditions. It is this quality that is at the base of the analogy, for this is also one of the important characteristics of a wild man' (Newman, 1964, pp. 1-2).

Although covert social pretenses are interpreted as being natural and involuntary, they actually conform to local socio-cultural norms and expectations. They need not be simple pretenses, as the subject may be unaware that they are conforming to a sub-conscious schema. In other cases a pattern of operant conditioning in early childhood may produce conformity 'without explicit representation of conformity as a goal' (Griffiths, 1997, p. 10). The role of cultural models in producing emotional behaviours that conform to these models may therefore be diachronic: 'they act during the agent's development by structuring the patterns of reinforcement in the cultural environment so as to produce automatic behaviours that conform to cultural norms'. This contrasts with the 'synchronic' or 'strategic' responses typical of the disavowed actions that we see in 'road rage', 'running amok' and other conditions in which it is likely that the actions can be controlled and amended by the perpetrator (Griffiths, 1997, p. 149).

1. Trivial Constructionism A concept exists because of sociolinguistic activity involving the concept.	
2/3. Substantial Constructionism The category corresponding to a concept exists (its members have something in common) because of sociolinguistic activity involving the concept.	**2. Overt Construction** The nature of the category is, or can be, known to those who use the concept without disrupting the process by which the category is constructed.
	3. Covert Construction Knowledge of the nature of the category by those who use the concept would disrupt the process by which the category is constructed. This category incorporates the reinforcement version and the disclaimed action version of the social role model of social constructionism.

Table 2: Three kinds of social construction, adapted from Griffiths (1997, p. 147).

The social role model of social constructionism incorporates not only the disclaimed actions or social pretenses of covert constructionism, but also overtly constructed categories, such as being a banker or member of parliament, which cover social roles that can be acknowledged as socially constructed without any implications for their validity. Consequently, though a scientific taxonomy could incorporate the socially constructed conditions, it is difficult to imagine that a clinical taxonomy would do so, as this would involve undermining the purposes for which the 'disorder' was constructed: to appear involuntary, and 'natural'. Even severe critics of the *DSM* approach to classification agree that a taxonomy of disorders should 'enhance the effectiveness of clinical activity, and… promote scientific research programs' (Poland, Von Eckardt & Spaulding, 1994, p. 236), but I would contend that these can be contradictory aims. Whether assistance should be rendered to those suffering from any kind of problem (medical or otherwise), or whether action should be taken against those causing social

problems, is always a matter of moral and social priorities, whatever the basis of our schemes of classification. To be epistemically productive our diagnostic categories should identify properties that are correlated because of a causal homeostatic mechanism (at whatever level that mechanism can be identified), and our scheme of classification should 'play a significant role in integrating the study of psychopathology with the empirical findings and theoretical developments in such areas as developmental psychology, cognitive science, and neuroscience' (Poland, Von Eckardt & Spaulding, 1994, p. 237), but these endeavours remain scientifically valid whether they assist or confound the aims of clinicians and social engineers.

In summary, natural kinds are projectable categories because they represent clusters of properties that are correlated owing to an underlying causal homeostatic mechanism. Causal homeostatic mechanisms are diverse and the properties that they cause to be correlated can be identified at different levels of analysis, from the domain of sub-atomic particles to the domains of psychology and sociology. Our epistemic endeavours will allow us to revise the extension of concepts as we identify categories displaying causal homeostasis, and revisions of intension will occur as we become able to predict which features must be reliably present (Griffiths, 1997, pp. 224-5).

Conclusion

In this chapter I have argued that classification in contemporary psychiatry is based on arbitrary concepts rather than projectable categories (i.e., natural kinds) and that the recognition of certain conditions as mental disorders has been the result of socio-political advocacy rather than the result of an objective evaluation of empirical evidence. Other disorders, such as MPD/DID, appear to be constructions capable of mimicking the passivity (lack of responsiveness to long-term planning) typical of the affect programs (basic emotions), and serve the social function of allowing those in distress to adopt the sick role. I have also indicated that the fortuitous discovery of drugs useful in palliative care has resulted in premature confidence about the neurochemical individuation of traits and disorders.

Twenty years ago Paul Muscari noted that ‘what is needed is a procedure for both describing and identifying mental disorder that does not exhaust analysis; that is capable of reaching beyond documenting empirical co-occurrences and predicted consequences to provide a general explanation’ (1981, p. 560). Unfortunately we are still a considerable distance from this objective, and so I would now like to move on to a consideration of evolutionary theory in general, and evolutionary psychology in particular, as a basis for the understanding of mental disorders. Accordingly, the following chapter will outline some of the most important developments in contemporary biological thought.

Chapter 4

Evolution and Human Nature

> People who by “programmed,” mean inevitable and caused only by the genes are forgetting that a computer program does not guarantee an output regardless of input. On the contrary, the more sophisticated the program, the more subtly it responds to its input. A program whose output were completely specified by (“completely controlled by”) the program itself would be of limited value.
>
> (Oyama, 1985, pp. 116-117)

> I argue that no approach to human behaviour can be simultaneously psychologically agnostic and genuinely Darwinian.
>
> (Symons, 1992, p. 139)

Prior to the advent of Darwinism organisms were ranked in *The Great Chain of Being* or *scala naturae*. This was a hierarchy in which plants and animals occupied the lowest rung, and God and the angels the highest, with humans in an intermediate position. This ranking of beings, which carries the implication of progress from one state to the next, persists even though evolution by natural selection implies only change and not a progression through successively higher states (Gaulin & McBurney, 2001, pp. 2-4). The term ‘evolution’ has so many ideological resonances that evolutionary ideas can easily be accepted or rejected for implications that are not intended. Amongst the synonyms for ‘evolution’ listed in my dictionary, for example, are *unravelling*, *ascent*, *unwinding*, *survival of the fittest*, *development*, and *perfectibility*. All of these concepts are inspired by the pre-Darwinian notion of the hierarchy of beings, rather than by evolutionary theory. Throughout this discussion it is important to remember that organisms and features arising later in evolutionary history are neither ‘higher’ nor more advanced than those occurring earlier, nor are they more ‘optimised’ This was a misconception to which even Charles Darwin (1809-1882) himself was susceptible

> As all the living forms of life are the lineal descendants of those which lived long before the Silurian epoch, we may feel certain that the ordinary succession by generation has never once been

> broken, and that no cataclysm has desolated the whole world. Hence we may look with some confidence to a secure future of equally appreciable length. And as natural selection works solely by and for the good of each being, all corporeal and mental endowments will tend to progress toward perfection (Darwin, 1859, p. 489)

Regardless of such utopianism selection can only favour whatever randomly generated heritable variants enhance *reproductive success* in a *particular environment*. The variation that can arise is itself constrained by the evolutionary history of an organism. We are concerned, not with the ideas of progression or perfectibility, but *descent with modification* within developmental constraints. Such descent with modification produces *adaptations*. These adaptations display complex functionality, precision, economy, efficiency, constancy, and arbitrary (suboptimal) features. Adaptations are *adaptive on average*, rather than invariably adaptive, *adaptive all other things being equal*, and *adaptive in the conditions in which the adaptation originally evolved* (Badcock, 2000, pp. 10-11).

In the final chapter of *On the Origin of Species*, published in 1859, Darwin wrote: ‘In the distant future I see open fields for far more important researches. Psychology will be based on a new foundation, that of the necessary acquirement of each mental power and capacity by gradation.’ (Darwin, 1859, p.488). The most eminent psychologist to follow this lead was William James, who argued in his *Principles of Psychology* (1890, Vol. II, p. 289) that human beings are more intelligent than other animals, not because they are ruled by reason, but because they have a larger repertoire of instincts. Just a few years later, in the volume *Darwin and Modern Science* (Seward, 1909) C. Lloyd Morgan argued that James could not be correct

> The true position is that man and the higher animals have fewer complete and self-sufficing instincts than those which stand lower in the scale of mental evolution, but that they have an equally large or perhaps larger mass of instinctive raw material which may furnish the stuff to be elaborated by intelligent processes. There is, perhaps, a greater abundance of the primary tissue of experi-

> ence to be refashioned and integrated by secondary modification (Morgan, 1909, electronic edition[17]).

The prevailing view of the mind both before and after Darwin was that of the *tabula rasa*, the clean slate on which impressions and experiences are inscribed, and this is still probably the most influential view in cognitive science and general psychology, as well as in anthropology and sociology. If an evolutionary substrate of behaviour or cognitive functioning is acknowledged at all it is usually as something to be overpowered by reason. C. Lloyd Morgan demonstrates that fifty years after the publication of *Origin* the ideas associated with the *scala naturae* were still firmly in place, along with the idea a higher, governing rationality or intelligence. Morgan writes: 'mental factors have contributed to organic evolution and… in man, the highest product of Evolution, they have reached a position of unquestioned supremacy' (1909, electronic edition).

In this chapter I will use a brief historical overview of the development of psychology and evolutionary biology to highlight a number of relevant theories, hypotheses and developments that we will need to consider and apply in the subsequent chapters.

Ethology in Europe and Comparative Psychology in the United States

The revival of evolutionary approaches to psychology and behaviour began in Europe with the work of the Austrian ethologist Konrad Lorenz (1903-1989), who set the agenda for much of the tone and style of contemporary work (Lorenz, 1965; 1966). One of his most influential ideas was that of *fixed action patterns*, which he believed to be genetically determined behaviours whose development was dependent only on elicitors in the natural environment of the animal. In addition to being released by a stimulus, fixed action patterns were described as: features with a constant form; requiring no learning; characteristic of a species, and impossible to change or unlearn (Cartwright, 2000, p. 7). This key idea of genetic programs dependent only on an environmental substrate, or called forth by environmental elicitors provides strong support for the idea of

[17] The electronic edition of this volume is available for download at http://human-

genes as repositories of information or as privileged causal entities, and can be found in both popular presentations, and serious scientific work, such as that by the Panksepps discussed in chapter two. Lorenz believed that aggression, including human aggression, had evolutionary roots, but as a group selectionist held that most aggression was likely to be non-fatal. In *On Aggression* Lorenz writes 'though occasionally, in territorial or rival fights, by some mishap a horn may penetrate an eye, or a tooth an artery, we have never found that the aim of aggression was the extermination of fellow-members of the species concerned' (Lorenz, 1966, p. 38). Amongst the strengths of Lorenz's ethological approach was the emphasis on the study of animals in their natural environments, an emphasis on species-typical instincts rather than variability, and the use of instincts to reconstruct phylogeny. Lorenz's student Nikolaas Tinbergen (1907-1988), brother of the pioneer of econometrics Jan Tinbergen, studied the development of individual and social behaviour patterns in groups of animals (Tinbergen, 1951; 1953; Tinbergen, et al., 1991). The most enduring of Tinbergen's contributions was set out in a 1963 paper 'On the Aims and Methods of Ethology' in which he proposed the four 'whys' of behaviour. These are 1). what are the mechanisms that cause behaviour? (i.e., what is the proximate, mechanical, causation?); 2). how does the behaviour come to develop in the individual? (i.e., what is its developmental course or ontogeny?); 3) how has the behaviour evolved? (i.e., what is the ultimate causation?), and 4). what is the function or survival value of the behaviour? (function)?' (Cartwright, 2000, p. 10). Most contemporary evolutionists would probably substitute 'fitness' for 'survival', as survival is of little consequence unless there is differential reproduction (Betzig, 1989). Kim Sterelny provides a useful illustration of the application of Lorenz's strategy in a review of Marc Hauser's *The Evolution of Communication*:

> Tinbergen famously distinguished between proximal, developmental, functional and evolutionary approaches to behaviour. We understand the communication system of an organism - for instance, the kookaburra's laugh - when we understand the adaptive design of kookaburra laughter; how those calls are realized and executed in the bird's neural circuitry; how those calls are developed and used, and how they currently contribute to the kookaburra life strategy (Sterelny, 1998, p. 308).

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nature.com/darwin/ebooks.html.

In 1949 Tinbergen moved to Oxford, where one of his students was Richard Dawkins, later to be the most articulate proponent of the gene as the unit of selection. Lorenz, Tinbergen, and Karl von Frisch (1886-1982) shared the Nobel Prize for Physiology or Medicine in 1973. In terms of the development of evolutionary psychology a prominent influence derived from the ethology of Lorenz and Tinbergen is the emphasis on the need to understand learning in evolutionary terms 'for them, it was a basic premise that learning must be understood in an evolutionary context. This means obviously that different situations call for different responses' (Ruse, 1985, p. 182). The work of the three major ethologists, together with that by primatologist Jane Goodall, demonstrated evident parallels between animal and human behaviour and did much to inspire a revival of Darwinism (Degler, 1991). However, other incompatible traditions have co-existed with the Darwinian approach.

From Philosophy to Psychology

In the early days of Western thought the Greek philosopher Socrates (469-399 BC) contended that all knowledge is essentially reminiscence. In *The Meno*[18] Socrates says 'there is no teaching, but only recollection' (quoted in Russell, 1961, p. 153). To demonstrate his theory of knowledge Socrates offers the story of a slave boy whom he questions on simple problems of arithmetic and geometry to show that the child has knowledge of which he is not aware. The child is asked to solve the following problem: if there is a square whose sides are each one inch long, how long are the sides of a square whose area is double that of the original square? The child answers incorrectly (twice as long) but is led to the correct answer by Socrates who shows how the right answer can be derived from a square built on the diagonal of the original. Socrates holds that the fact the boy could arrive at a correct answer, and be completely sure of its accuracy, demonstrates that in some sense the child must have already known the answer.

[18] The text of *The Meno* is available on the Internet at http://classics.mit.edu/Plato/meno.html.

The doctrine of innate ideas received continuing support throughout medieval times, but gained renewed vigour from its endorsement by Descartes. Cartesian philosophy emphasises the innateness, not of cognitive mechanisms or mental organs, but of propositional content, or representations, which as we saw in chapter two has been opposed vigorously in recent years by Elman and colleagues (1996). Despite the support of Plato, Descartes, and other prominent members of the rationalist tradition in philosophy such as Baruch Spinoza (1632-1677) and Gottfried Wilhelm Leibniz (1646-1716) the apparent absurdity of innate knowledge led subsequent philosophers of the British Empiricist tradition, especially John Locke (1632-1704), George Berkeley (1685-1753) and David Hume (1711-1776) to argue that all ideas and knowledge are *a posteriori*, i.e., based on and derived from experience. Most subsequent thinking on this matter is in keeping with Locke's description of the *tabula rasa* ('blank slate' or 'white paper') as it appears in his *Essay Concerning Human Understanding* (1690)

> Let us suppose the mind to be, as we say, white paper, void of all characters, without any ideas; how comes it to be furnished? Whence comes it by that vast store, which the busy and boundless fancy of man has painted on it with an almost endless variety? When has it all the materials of reason and knowledge. To answer this in one word, from experience: in all that our knowledge is founded, and from that it ultimately derives itself (book II, chapter I, section 2).

Locke thus laid the foundations of what has come to be known as the doctrine of the association of ideas or associationism which 'since the middle of the eighteenth century… has increasingly been seen as the most basic, the most fecund, and the most pervasive explanatory principle in the human mind' (Young, 1968a, p. 111)[19].

Although there were many contributors to the development of associationism one of the most important was the Reverend John Gay who, in his anonymous preface to Edmund Law's translation of Archbishop King's *Essay on the Origin of Evil*, gave associationism an important moral dimension. In this preface Gay

[19] Available online at http://human-nature.com/rmyoung/papers/paper58h.html.

> …employed Locke's conception in opposition to the innatist theory of the origin of moral sentiments and disinterested affections advocated by Frances Hutcheson. Gay applied the association of ideas to the domain of ethics and psychology and argued that the moral sense and all the passions were acquired in experience. Men seek pleasure and avoid pain, he argued, and the habitual union of these experiences with the principle of association produces our moral and emotional dispositions… Gay's dissertation was the first coherent expression of the main tenets of utilitarian ethical theory and the associationist school of psychology (Young, 1968a, p. 113).

Empiricist epistemology thus offered an account of the origin of ideas and of the moral sentiments which dispensed with the Cartesian notion of innate ideas, and provided some of the intellectual prerequisites for the acceptance of behaviourism (which became the pre-eminent school of psychology in the United States), and for the supremacy of the environment in moulding human nature.

The work by Ivan Petrovich Pavlov (1849-1936) on conditioned and unconditioned reflexes in dogs, which began in 1889, became one of the foundations of behaviourism, but the behaviourist school of psychology itself was founded by John B. Watson (1878-1958), professor of psychology at Johns Hopkins University, who coined the term 'behaviourist' in 1912. Watson did not deny the existence of subjective experiences, but did not consider them the legitimate targets of psychological research, in contrast to the dominant introspectionist approach of that time. The school of behaviourism can date its inauguration from Watson's article in the *Psychological Review* entitled 'Psychology as the Behaviourist Views It' (1913)[20] often referred to as 'the behaviourist manifesto' (Hunt, 1993, p. 263). This approach provided a great impetus to research on learning and development across the lifespan in animals and humans. In the broader context, Watson's psychology, in attributing almost all human behaviour to stimulus-response conditioning caught the popular imagination, as it seemed to offer both the prospect of creating a better world through the scientific manipulation of human nature, and a rebuttal of the hereditarian views associated with Francis Galton (1822-1911), the founder of eugenics. In his book *Behaviourism*

[20] Available on the Internet in the *Classics in the History of Psychology* collection at http://psychclassics.yorku.ca/Watson/views.htm.

(1925) Watson made what became his most widely known and often quoted statement:

> Give me a dozen healthy infants, well-formed, and my own specified world to bring them up in and I'll guarantee to take any one at random and train him to become any type of specialist I might select – doctor, lawyer, artist, merchant-chief and, yes, even beggar-man and thief, regardless of his talents, penchants, tendencies, abilities, vocations, and race of his ancestors (quoted in Hunt, 1993, p. 261).

Behaviourism ascended to become the chief school of psychology in the United States from around 1920 until the early sixties, and although it never achieved a similar position in Europe, its appeal to progressive intellectuals assured that it had widespread influence. Remarkably, throughout this time, the chief influence on psychiatry in the United States was the highly subjective psychoanalysis of Sigmund Freud (1856-1939) and colleagues, which was anathema to the behaviourists. Though only a minority of psychologists would now regard themselves as behaviourists, the influence of behaviourism remains strong in psychiatry and psychotherapy through behaviour therapy and cognitive-behaviour therapy.

Behaviourist psychology found in a natural ally in the logical positivism of the group of philosophers known as the Vienna Circle (Cartwright, 2000, p. 13). The logical positivists adopted an empirical definition of meaning based on the verification principle. A statement could be meaningful only if it was either analytic (i.e., tautological) or verifiable by observation. Behaviourism was further developed by Burrhus Frederic Skinner (1904-1990), who agreed with Watson's emphasis on observables. Skinner's principle idea was that of *operant conditioning*, a process in which actions are reinforced by punishment (negative reinforcement) or reward (positive reinforcement) to produce complex behaviours. This general-process learning theory held the characteristics of learning to be identical across species and situations. Skinner also had a popular influence through his utopian novel *Walden Two* (1948) in which the perfect society is created through the application of the principle of operant conditioning. In 1966

the historian Robert M. Young summarized the position just as the situation in experimental psychology was beginning to change;

> J. B. Watson's methodology – which became an ontology – was based explicitly on a rejection of mental substances as part of the domain of science. And some of his more polemical writings show just how seriously he took this problem above all others Similar statements could be made about the positions of Wundt, McDougall, Tolman, Lashley, Sherrington, Eccles, and other major psychologists whose work is still influencing experimental research (Young, 1966, p. 20)[21].

Within a stimulus-response framework the comparative psychologists' emphasis on domain-general methods of learning, (the *indifference hypothesis*) translated into the principle of equipotentiality. This tenet of behaviourism asserted that differences in the strength of association between stimulus and response are simply a matter the conditions of pairing (contiguity, duration etc) and are independent of the nature of the reinforcer. Decisive work showing this principle to be incorrect was published in 1966 by Garcia and Koelling who showed that rats given electric shocks paired with exposure to either a visual or a taste stimulus subsequently only avoided the visual stimulus. When the experimenters poisoned the rats and paired the poisoning with the same visual and taste stimuli, the rats avoided the latter. The aversion to the new taste occurred even though there was a time delay of one hour between the ingestion of the food and induced radiation sickness or administration of a toxin (Garcia & Koelling, 1966a; 1966b; Garcia, McGowan & Green, 1972). It was clear that learning depended on the nature of the stimulus and on the nature of the species being studied. Indeed, much of the evidence gathered by those working within the behaviourist framework supported the notion that animals displayed localized 'dispositions to learn' (Ruse, 1985, p. 183). The principles of learning could not be generalized across species. Behaviourists argued that their view of reinforcement learning was in keeping with both the materialist traditions of science and Darwinian evolution, because just as natural selection replaced divine creation, behaviourism replaced the immaterial mind with the moulding of *behaviour* by

[21] Available online at http://human-nature.com/rmyoung/papers/paper57h.html.

reinforcement. However, as Garcia insisted, Darwin's view was that the 'mind existed materially in mental organs evolved by natural selection' (Garcia, 1996).

A classic paper by Frank Beach, 'The Snark was a Boojum', published in 1950 showed that comparative psychology under the influence of behaviourism had become something rather less than comparative, and that by 1948 the vast majority of studies were conducted on single species, the Norway rat (Beach, 1950; Cartwright, 2000, p. 15). Ironically, it was the publication of a work on language by Skinner *Verbal Behaviour* (1957) that presaged the decline of behaviourism and the upsurge of the approach that was to become cognitive psychology. In a devastating review of the book the linguist Noam Chomsky (1959) demonstrated that attempts to explain language along the lines of operant conditioning were fundamentally flawed, and claimed that 'our interpretation of the world is based on representational systems that derive from the structure of the mind itself and do not mirror in any direct way the form of the external world' (quoted in Gardner, 1985b, p. 182). In Chomsky's view Skinner was simply substituting the word 'reinforced' for the mentalistic terms ordinarily employed to make sense of behaviour, in order to justify the explanation of that behaviour in terms of antecedent external events. Chomsky's positive program seeking to explain the development of language in terms of innate structures became enormously influential. Jerry Fodor explains the basis of the cognitivists' discontent with associationist explanations of human faculties with characteristic flair:

> There is simply no reason at all to believe that the ontogeny of the elaborate psychological organization that… associationism contemplates can be explained by appeal to learning principles which do what principles of associative learning did – viz., create mental copies of environmental redundancies. In particular, the constructibility *in logical principle* of arbitrarily complicated processes from elementary ones doesn't begin to imply that such processes are constructible *in ontogeny* by the operation of any learning mechanism of a kind that associationists would be prepared to live with (Fodor, 1983, p. 34)

From the standpoint of the new cognitive psychology human cognitive abilities simply couldn't be explained by the notions of the *tabula rasa* and the association of ideas. Fodor himself has argued that many of our cognitive capacities,

particularly perception and language (but not what he calls 'central systems'), are subserved by discrete functional units (or *modules*) which operate like cognitive reflexes and which have a particular course of development, a dedicated neural architecture and particular patterns of breakdown. However, Fodor asserts that cognitive modules are not the result of evolution by natural selection, but of other unspecified physical forces, a view characterised by Gary Cziko as 'providential innatism' (1995, p.131). Fodor's analysis of the innatism of Noam Chomsky leads him to conclude:

> In Descartes and Plato, as in Chomsky, the nativism is so striking that one is likely to overlook a still deeper consensus: the idea that certain of the subject's cognitive capacities should be explained by reference to consequence relations (e.g., deductive relations) that hold among the propositions that the subject knows (believes, cognises, or whatever). I say to you: "What's 2 plus 17?" and you, being good at that sort of thing say "19". Your *behaviour* is structured in the relevant sense; what sort of mental structure is the psychologists to posit in explaining your behaviour? According to the Cartesian, it is inter alia the deductive structure of number theory to which the explanation must appeal (Fodor, 1983, p. 7, emphasis in the original).

Fodor characterises Chomsky as a neo-Cartesian, or someone who believes in innate propositional content. In contrast Fodor proclaims himself the heir of the faculty psychology of Franz Joseph Gall (1758-1828) and Johan Caspar Spurzheim (1776-1832), which is better known as phrenology. Gall and Spurzheim provided the first empirical approach to the nature of psychological faculties and their localization in the brain (Young, 1968b), though their approach later fell in disrepute because of the supplementary assumptions that a well-developed faculty would expand causing the skull above it to bulge. This conveniently allowed psychological faculties to be explored by feeling the head instead of by examining the brain. Despite its faults

> One of the ways in which phrenology helped to develop a naturalistic interpretation of the mental functions of animals and men was by challenging the prevailing view of the fundamental variables in behaviour. Philosophical psychology had passed down the abstract categories of reason, memory, will, intelligence and so on. Franz Joseph Gall questioned these categories and asserted that the study of the functions of the brain depended upon the study of

> animals in their environments and of men in society. It was only by this method, Gall argued, that we could arrive at a meaningful set of categories (Young, 1966, p. 17).

I believe, contra Fodor, that Chomsky's position is not neo-Cartesian, and that he does in fact argue for an innate faculty which facilitates the acquisition of language during ontogeny. In recent years a number of commentators including Gould (1991), Pinker and Bloom (1992) Dennett (1995) and Panksepp and Panksepp (2000) have claimed that Chomsky believes in an innate Universal Grammar but does not accept that the language faculty evolved. This is not correct. Chomsky believes that the Language Acquisition Device is a genetically-determined component of our species-typical biological endowment fashioned for its current role by the action of natural selection (personal communication, 1999). Language is not innate, but its acquisition is guided and constrained by the properties of the language faculty. Far from harbouring an antipathy to the theory of evolution Chomsky holds that constraints on the plasticity of human mentality form a bulwark against the aspirations of would-be dictators and social engineers, and that he tried to convince the early critics of sociobiology of this

> I had long debates with my friends in the Science for the People group (Steve Gould, Dick Lewontin, Steve Chorover, others) in the '70s, when sociobiology was coming along. My position was that they should have welcomed the revival (after all, it was started by Kropotkin), and recognized that any meaningful left wing politics crucially depends on (at least tacit) assumptions about human nature; that's certainly the case, say, for Marxian theories of alienation, which make no sense on other grounds. My own view, from the '60s, has been that extreme environmentalism (which goes hand in hand with marginalization of evolutionary factors) is the ideology of social managers, and that there isn't much difference between the Leninist and Western liberal (in the US sense) variety; in fact, I've sometimes compared remarks by McNamara-Lenin, and other such (personal communication, 1999).

Contemporary evolutionary psychology regards itself as a fusion of perspectives from evolutionary biology and cognitive science, and within this tradition Fodor is often credited with stimulating the revival of interest in modularity. From a broader appreciation of the history of ideas, however, Fodor's conception of modules leads into an anti-evolutionary *cul-de-sac*, primarily because he has no

explanation for their existence, and therefore no theoretical framework capable of informing ideas about their design. The revival of interest in evolved psychological mechanisms is better attributed to the work of Tinbergen, Chomsky and Trivers, all of whom have initiated positive research programmes.

From Sociobiology to Evolutionary Psychology

Along with developments in psychology, linguistics and ethology outlined above the early sixties also saw the publication of what was at that time a relatively uncontroversial defence of the group selectionist — 'for the good of the species' — approach to animal behaviour with the title *Animal Dispersion in Relation to Social Behaviour* (Wynne-Edwards, 1962). Very shortly after the publication of Wynne-Edwards' book the whole theoretical landscape of biology began to change following the publication of a model of 'inclusive fitness' by William D. Hamilton (1936-2000) (1964a; 1964b). This idea, which is better known as 'kin selection', allowed the extension of 'Darwinian fitness' by taking into account changes in the representation of genes in the gene pool caused indirectly by kinship effects. Within this framework altruistic behaviour directed at kin could be described from the 'gene's eye' point of view without recourse to explanation in terms of group selection. The classic example is the help given by sterile worker ants to the reproduction of their fertile kin. Through kin selection, therefore, a characteristic is established because of its effects on the survival and reproduction of the kin of its possessor (Maynard Smith, 1993, p. 195). Between two related individuals an altruistic act increases the reproductive success of the recipient at the expense of the bestower. An altruistic gene will spread if $rb > c$, where b is the benefit to the recipient, c is the cost to the donor, and r is the *coefficient of relatedness* (Cartwright, 2000, p. 75).

The first clear exposition of the idea of the gene as the unit of selection was George Williams' *Adaptation and Natural Selection: A Critique of Some Current Evolutionary Thought* (1966). Williams wrote:

> To minimize recurrent semantic difficulties, I will formally distinguish two kinds of natural selection. The natural selection of alter-

> native alleles in a Mendelian population will henceforth be called *genic selection*. The natural selection of more inclusive entities will be called *group selection*, a term introduced by Wynne-Edwards (1962)… Genic selection should be assumed to imply the current conception of natural selection often termed *neo-Darwinian*. An *organic adaptation* would be a mechanism designed to promote the success of an individual organism, as measured by the extent to which it contributes genes to later generations of the population of which it is a member. It has the individual's *inclusive fitness* (Hamilton, 1964) as its goal (Williams, 1966, pp. 96-97, emphasis in the original).

A second important development taking place in biology (at roughly the same time that the effects of Chomsky's work on linguistics and cognitive psychology, Garcia's work on comparative psychology, and the revival and popularisation of ethology were proving fruitful) was the application to biology of the branch of mathematics known as game theory, which was devised by John von Neumann (1903-1957) and published in *Theory of Games and Economic Behaviour* (1944) in collaboration with Oskar Morgenstern. Game theory analyses situations of 'choice under conditions of uncertainty' where each player's strategy depends on the choices made by other players. An important contribution to game theory is that of the *Nash equilibrium*. This the situation in which X's choice is optimal for him given Y's choice and vice versa, though this does not guarantee the desirable outcome that could be achieved by co-operation (Ekeland, 1999). Game theory was introduced tentatively into biology by Richard Lewontin (1961), and was used by Hamilton (1967) to explain sex ratios, but it was developed most significantly by John Maynard Smith (1972), who introduced the idea of the *evolutionarily stable strategy*. In its application to biology game theory deals with the fitness of strategies employed by animals in their interactions. An evolutionarily stable strategy (ESS) is a strategy 'which, if most members of a population adopt it, cannot be bettered by an alternative strategy. It is a subtle and important idea. Another way of putting it is to say that the best strategy for an individual depends on what the majority of the population are doing' (Dawkins, 1989, p. 69). The standard example is that of the hawk strategy 'fight aggressively retreating on when seriously injured' versus the dove strategy 'threaten aggression but always retreat'. A stable ratio of hawks to doves is reached in the idealized mathematical model at 5/12 doves to 7/12 hawks. If

these strategies are dependent on genes then the ratio of genes in the gene pool will reflect the same proportions, a state known as a *stable polymorphism*. Alternatively, the mathematics remain the same if *each individual* employs the hawk and dove strategies randomly but with 7:5 bias in favour of the hawk strategy. In a more familiar example the stable sex ratio at 50:50 occurs because there is always a payoff for favouring the rarer sex, and this pushes the ratio to equilibrium. We should remember, however that 'the general conclusions which are important are that ESSs will tend to evolve, that an ESS is not the same as the optimum that could be achieved by group conspiracy' (Dawkins, 1989, p. 75).

In a series of remarkable contributions to biology in the early seventies Robert Trivers introduced the theories of *reciprocal altruism* (1971), *parental investment* (1972), and *parent-offspring conflict* (1974). The latter two theories are discussed later in this chapter. In this section I would like to examine Trivers paper 'The Evolution of Reciprocal Altruism' (1971) as it relates to the development of evolutionary psychology. Trivers elaborates the mathematics of reciprocal altruism and specifically chooses human reciprocal altruism as one of his three examples, arguing that 'it can be shown that the details of the psychological system that regulates this altruism can be explained by this model'. In particular, Trivers argues for the following characteristics as functional processes (adaptations) subserving reciprocal altruism (Trivers, 1971, pp. 48-54):

A complex regulating system – The system subserving reciprocal altruism will be sensitive and unstable because it will often pay to cheat. For reciprocal altruism to function, therefore, 'natural selection will rapidly favour a complex psychological mechanism in each individual regulating both his own altruistic and cheating tendencies and his responses to these tendencies in others'.

Friendship and the emotions of liking and disliking – The immediate emotional rewards motivating altruistic behaviour and partnerships will be the tendency to like others, to form friendships, and to act altruistically towards friends and likeable acquaintances. 'Selection will favour liking those who are themselves altruistic'.

Moralistic aggression – As cheaters will take advantage of any positive emotions motivating altruistic behaviour there will be selection for a protective mechanism. Moralistic aggression will 'counteract the tendency of the altruist, in the absence of any reciprocity, to continue to perform altruistic acts for his own emotional rewards'. It will also educate the unreciprocating individual, and in extreme cases 'select directly against the unreciprocating individual by injuring… killing, or exiling him'.

Gratitude, sympathy, and the cost/benefit ratio of an altruistic act – Gratitude regulates the 'human response to altruistic acts' and 'is sensitive to the cost/benefit ratio of such acts. In addition, sympathy 'has been selected to motivate altruistic behaviour as a function of the plight of the recipient'.

Guilt and reparative altruism – If cheating is detected then reciprocity will end, at considerable cost to the cheater, therefore 'the cheater should be selected to make up for his misdeed and to show convincing evidence that he does not plan to continue his cheating sometime in the future'. In order to motivate a reparative gesture 'guilt has been selected for in humans partly in order to motivate the cheater to compensate his misdeed and to behave reciprocally in the future, and thus to prevent the rupture of reciprocal relationships'.

Subtle cheating: the evolution of mimics – Selection will favour the mimicking of all traits subserving reciprocal altruism 'in order to influence the behaviour of others to one's own advantage'. Subtle cheating may involve *sham moralistic aggression*, *sham guilt*, *sham sympathy*, and 'the hypocrisy of pretending one is in dire circumstances in order to induce sympathy-motivated altruistic behaviour'.

Detection of the subtle cheater: trust-worthiness, trust, and suspicion – Selection will favour the detection of moralistic aggression and 'distrusting those who perform altruistic acts without the emotional basis of generosity or guilt because the altruistic tendencies of such individuals may be less reliable in the future'.

Setting up altruistic partnerships – Because ‘humans respond to acts of altruism with feelings of friendship that lead to reciprocity’ selection will favour the strategy ‘do unto others as you would have them do unto you’. Altruistic acts towards strangers and enemies may induce friendship.

Multiparty interactions – Particularly in ancestral times humans would have lived in small, close-knit, groups where ‘selection may favour learning from the altruistic and cheating experiences of others, helping others coerce cheaters, forming multiparty exchange systems, and formulating rules for regulated exchanges in such multiparty systems’.

Developmental plasticity – As the conditions under which reciprocal altruism can operate will vary widely according to ecological and social conditions, and will vary through time for the same population ‘one would expect selection to favour developmental plasticity of those traits regulating altruistic and cheating tendencies and responses to these tendencies in others’. No simple developmental system would be expected to meet the requirements to be adaptive because ‘altruistic behaviour must be dispensed with regard to many characteristics of the recipient (including his degree of relationship, emotional makeup, past behaviour, friendships, and kin relations) of other members of the group, of the situation in which the altruistic behaviour takes place, and of many other parameters’. Such a system could only function effectively through the developmental plasticity that would accommodate education about the appropriate response, especially from kin. For example, education of the sense of guilt could permit ‘those forms of cheating that local conditions make adaptive and to discourage those with more dangerous consequences’.

In Trivers’ astonishing paper we see the entire agenda that will later become the foundation of evolutionary psychology, including the emphasis on mechanisms rather than behaviour, on developmental systems, on constrained plasticity sensitive to ecological and social conditions, on change over time, and the implicit assumption that the affect of an adaptation need not necessarily be adaptive under novel conditions. In concluding, Trivers notes that mechanisms may subserve more than one function. ‘One may be suspicious, for example, not

only of individuals likely to cheat on the altruistic system, but of any individual likely to harm oneself; one may be suspicious of the known tendencies toward adultery of another male or even of these tendencies in one's own mate' (1971, p. 54). Finally, Trivers notes that the selection pressures for the psychological mechanisms subserving reciprocal altruism could have contributed to the increase in hominid brain size during the Pleistocene. This theme resurfaces in the 'Machiavellian intelligence' (Byrne & Whiten, 1988) or 'social intelligence' hypotheses (Humphrey, 1976), and the current concern with 'theory of mind' mechanisms (Baron-Cohen, Leslie & Frith, 1985). These ideas will be the focus of chapter six.

Many of these new developments in biology were brought together by Edward O. Wilson in a massive tome *Sociobiology: The New Synthesis* (1975). This publication rapidly became a classic within biology itself, even being voted the most important book on animal behaviour of all time by members of the Animal Behaviour Society in 1989 (Wilson, 2000). However, as Wilson puts it with some reserve 'the brief segment of *Sociobiology* that addresses human behaviour, comprising 30 out of the 575 total pages, was less well received (2000, p. vi). In retrospect, Chapter twenty-seven of Wilson's book 'Man: From Sociobiology to Sociology' looks relatively innocuous. Certainly, Wilson is sceptical of the notion of 'the mind of man as a virtual equipotent response machine' which is 'neither correct nor heuristic' (1975, p. 551), but after surveying the plasticity of human social organization, examples of reciprocal altruism, bonding, communication, culture and ethics he concludes that the social sciences are relatively autonomous of biology, but that they can be informed by it, and that 'scientists and humanists should consider together the possibility that the time has come for ethics to be removed temporarily from the hands of the philosophers and biologicized' (1975, p. 562). Wilson does believe, however, that psychology will be replaced, eventually, by neurobiology:

> The transitional from purely phenomenological to fundamental theory in sociology must await a full, neuronal explanation of the human brain. Only when the machinery can be torn down on paper at the level of the cell and put together again will the properties of emotion and ethical judgment come clear. Simulations can

> then be employed to estimate the full range of behavioural responses and the precision of their homeostatic controls. Stress will be evaluated in terms of neurophysiological perturbations and their relaxation times. Cognition will be translated in to circuitry. Learning and creativeness will be defined as the alterations of specific portions of the specific machinery regulated by input from the emotional centres. Having cannibalised psychology, the new neurobiology will yield an enduring set of first principles for sociology (Wilson, 1975, p.575).

In a passage that sounds decidedly anti-reactionary and could have come from a modern text of evolutionary psychology, or a volume arguing against the thesis of *The Bell Curve* (Herrnstein & Murray, 1994) that social stratification is determined largely by IQ or general intelligence, which is itself claimed to be largely hereditary, Wilson writes:

> The hereditary factors of human success are strongly polygenic and form a long list, only a few of which have been measured. IQ constitutes only one subset of the components of intelligence. Less tangible, but equally important qualities are creativity, entrepreneurship, drive, and mental stamina. Let us assume that the genes contributing to these qualities are scattered over many chromosomes. Assume further that some of the traits are uncorrelated or even negatively correlated. Under these circumstances only the most intense forms of disruptive selection could result in the formation of stable ensembles of genes. A much more likely one is the one that apparently prevails: the maintenance of large amounts of genetics diversity within societies and the loose correlation of some of the genetically determined traits with success. This scrambling process is accelerated by the continuous shift in the fortunes of individual families from one generation to the next (Wilson, 1975, p. 555).

It is only at the very end of the chapter that Wilson's grounding in the new gene selectionism seems less secure. In the penultimate paragraph he speculates about a decline in altruistic behaviour through the loss of group-selected genes. There is also a brief mention of the perils of social engineering, but here Wilson's concern seems to be that inadequate knowledge of our genetic heritage might result in our failing to appreciate that traits we consider unacceptable or largely undesirable, such as destructiveness, and traits we consider desirable, such as creativeness, may in fact be the result of pleiotropism, the control of more than one phenotypic character by the same genes. Such pleiotropism

could make it impossible to affect one trait without affecting the other. It is notable that one of the reasons that British researchers in animal behaviour resisted the term 'sociobiology' was because 'they felt that Wilson's view of "sociobiology", which embraced group selection, clashed with their own newer gene-selectionist view' (Segerstråle, 2000, p. 98). Although Wilson had been one of the people who commented prior to publication on Trivers' reciprocal altruism paper it is significant that its emphasis on plastic psychological mechanisms doesn't surface in *Sociobiology*. Wilson's concern is with adaptive behaviours, and it is this concern that predominates in the work of most sociobiologists subsequently, including that by those engaged primarily in human sociobiology.

Although Wilson's book was initially well received, even being featured on the front cover of the *New York Times*, in November of 1975 an organization called the Sociobiology Study Group, based in the Boston area, and which included people such as Richard Lewontin and Stephen Jay Gould, published a remarkably severe attack in the *New York Review of Books* linking sociobiology to 'genetic determinist' theories of past decades:

> These theories provided an important basis for the enactment of sterilization laws and restrictive immigration laws in the United States between 1910 and 1930 and also for the eugenics policies which led to the establishment of gas chambers in Nazi Germany. The latest attempt to reinvigorate these tired theories comes with the alleged creation of a new discipline, sociobiology (Allen, et al., 1975).

The Sociobiology Study Group did not deny the existence of 'genetic components to human behaviour' but thought these most likely to be found in the 'generalities of eating, excreting, and sleeping' (Allen, et al., 1975). At this stage their critique centred almost entirely on political issues, but in a chapter of the volume *Biology as a Social Weapon* published in 1977 they set out a critique of sociobiology in the format in which it is still to be found in many publications today, along with the standard objections to 'genetic determinism' and 'reductionism':

> When we examine carefully the manner in which sociobiology pretends to explain all behaviours as adaptive, it becomes obvious that the theory is so constructed that *no tests are possible*. There exists no imaginable situation that cannot be explained; it is *necessarily confirmed by every observation*. The mode of explanation involves three possible levels of the operation of natural selection: one, classical individual selection to account for obviously self-serving behaviours; two, kin selection to account for altruistic or submissive acts toward relatives; and, three, reciprocal altruism to account for altruistic behaviours directed toward unrelated persons. All that remains is to make up a "just-so" story of adaptation with the appropriate form of selection acting (Allen, et al., 1977, p. 145, emphasis in the original).

Many of the scientific objections to sociobiology and evolutionary psychology invoke the ideas of Stephen Jay Gould, which were analysed in chapter two. Many of the arguments based on these ideas raise objections to specific hypotheses, or the implications of particular concepts, and do not provide any coherent reasons for rejecting the adaptationist approach to human psychology in its entirety.

Selfish Genes and Selfish People

A much more concise, and more popular, summary of the new gene selectionism in biology was published by Richard Dawkins in 1976. In *The Selfish Gene* Dawkins explains the application of game theory to biology, omitted by Wilson from *Sociobiology*, and also corrects Wilson's misinterpretation of kin selection. 'E. O. Wilson… defines kin selection as a special case of group selection… Kin selection is most emphatically *not* a special case of group selection. It is a special consequence of gene selection' (Dawkins, 1989, p. 95).

I believe it is important to note that Dawkins describes himself as a *functional ethologist*, or someone interested primarily 'in the adaptive explanation of how a particular behaviour may have evolved' (Segerstråle, 2000, p. 74). In Dawkins' work we see a concern with modelling only the evolutionary aspect of Tinbergen's four questions, the other three dealing with the nature of the mechanisms, function, and development of behaviour are of secondary importance. A second consideration is Dawkins failure to assimilate the implications of Trivers' theory

of reciprocal altruism as it applies to human psychology. Dawkins is concerned with evolution of animal behaviour in broad generality, rather than human psychological faculties or with modelling the peculiarities of human nature. This how he comes to write:

> I shall argue that a predominant quality to be expected in a successful gene is ruthless selfishness. This gene selfishness will usually give rise to selfishness in individual behaviour. However, as we shall see, there are special circumstances in which a gene can achieve its own selfish goals best by fostering a limited form of altruism at the level of individual animals… My own feeling is that a human society based simply on the gene's law of universal ruthless selfishness would be a very nasty society in which to live… Let us try to teach generosity and altruism, because we are born selfish (Dawkins, 1989, p. 2-3).

Although the gene selectionist approach was inspired by a concern to show how altruism at the behavioural level could be explained by 'selfishness' at the genetic level, Dawkins conflates these two levels of explanation. This is probably why some people believe that the book claims that all of human behaviour is genetically constrained to be selfish (see for example Panksepp & Panksepp, 2000). Gene selectionism does not have this implication, but here Dawkins actually seems to be implying that moral plasticity is conferred by some sort of general purpose learning mechanism acting in opposition to the dictates of genes. Newer developments in the study of altruism suggest that we need not resort to such a desperate explanatory schema.

In addition to conventional reciprocal altruism as explained by Trivers individual selection can favour cooperation through the mechanism of indirect reciprocity by image scoring, even when two individuals never encounter each other again. In an article in *Nature* Martin Nowak and Karl Sigmund showed 'that the probability of knowing the "image" of the recipient must exceed the cost-to-benefit ratio of the altruistic act'. They conclude:

> Cooperation based on indirect reciprocity works in the following way, therefore: a potential donor can choose whether to accept a certain cost in order to help another individual, or to avoid this cost. In the short term, of course, avoiding the cost yields the

> higher payoff. In the long term, however, performing the altruistic act increases the image score of the donor and may therefore increase the chance of obtaining a benefit in a future encounter as a recipient. On the other hand, a discriminator who punishes low-score players by refusing them help pays for this by having his own score reduced. The overriding idea, relevant to human societies, is that information about another player does not require a direct interaction, but can be obtained indirectly either by observing the player or by talking to others. The evolution of human language as a means of such information transfer has certainly helped in the emergence of cooperation based on indirect reciprocity (Nowak & Sigmund, 1998, p. 576).

The mathematical structure of indirect reciprocity is similar to that of Hamilton's rule in his theory of inclusive fitness, but relatedness is replaced by acquaintanceship. One problem with Nowak and Sigmund's model is that it predicts long-term cycling between co-operator and defector populations rather than an evolutionarily stable strategy. However, the fact that there are always some individuals in a population that are unable to co-operate, such as the handicapped, the very young, and the sick (termed *phenotypic defectors*), allows 'persistent discriminating cooperation under a much wide range of conditions… because there is selection against both defection and unconditional altruism'. This allows 'the evolution of a society in which cheap donations are given unconditionally to everyone, whereas more costly gifts are given discriminatingly and only to those individuals who can afford to give such gifts to others' (Lotem, Fishman & Stone, 1999, p. 227).

Roberts and Sherratt (1998) have also shown that a model of reciprocal altruism based on 'testing the water' rather than in making 'co-operative leaps of faith' is a stable strategy that can invade non-altruist populations and cannot be effectively exploited. This strategy (called *raise the stakes* or 'RTS') allows costly investment to develop incrementally

> Altruism in the form of RTS should predominate over 'averaging' strategies whenever there are cheats, subtle cheats or indeed any individuals which are, at the time, unwilling or unable to reciprocate adequately. A satisfying aspect of our model is that it represents an important step towards more biologically realistic treatments of cooperation. It should help to bridge the current gulf between theoreticians and those biologists who have questioned the

> degree to which reciprocity theory contributes to our understanding of cooperative behaviour (Roberts & Sherratt, 1998, p. 178).

To support this model Roberts and Sherratt cite examples where reciprocal relationships start from small beginnings as in the 'live-and-let-live' system of trench warfare in the first World War; the tendency to form friendships; and the tendency to act preferentially towards friends. One analytical strength of the model is that it allows behaviour to be split down into smaller units (such as grooming) that can serve as the basis for the exchange of more costly acts.

The economist Herbert Gintis has pointed out a key flaw in reciprocal altruism (or what he calls weak reciprocity): it is most likely to collapse when prosocial behaviour is most needed – when the group is threatened. A strong reciprocator 'is predisposed to cooperate with others and punish non-cooperators, even when this behaviour cannot be justified in terms of self-interest, extended kinship, or reciprocal altruism' (Gintis, 2000, p. 169). In addition to abundant evidence of strong reciprocity from everyday life, empirical evidence from experimental psychology shows that individuals will often behave prosocially and punish defectors at cost to themselves even when the probability of future interaction with the defector is low or non-existent. Gintis has devised a mathematical model showing that *strong reciprocity* could have evolved where groups experience periodic extinction-threatening events. If the proportion of strong reciprocators in a group is high enough even self-interested individuals can be induced to cooperate, thus lowering the probability of group extinction.

With this research on game theoretic approaches to altruism contemporary biology based on individual selection rather than group selection is able to provide evolutionary models of what we consider distinctive human attributes. Thus, biology does not leave us with the bleak and untenable vision of human nature that some interpretations of the 'selfish gene' hypothesis suggest.

Sexual Selection, Parental Investment, and Parent-Offspring Conflict

In chapter four of *On the Origin of Species* Darwin introduces a second mechanism of selection: 'what I call sexual selection. This depends, not on a struggle for existence, but on a struggle between the males for possession of the females; the result is not death to the unsuccessful competitor, but few or no offspring' (Darwin, 1859, p. 88). Why should males struggle for 'possession' of females, or vice versa? As a general guide it is important to determine which sex acts as a *reproductive bottleneck* for the other (Clutton-Brock & Vincent, 1991).

In a classic study by Clark and Hatfield (1989) male and female confederates engaged strangers of the opposite sex in a brief conversation before asking a number of questions such as 'Would you go to bed with me tonight?' 'Would you come over to my apartment tonight?' and 'Would you go out with me tonight? Although around 50 percent of the men and the women agreed to a date, only 6 percent of the women accepted the invitation to visit the experimenter's apartment, and none would agree to sex. Of the men, 69 percent accepted the invitation to visit, and 75 percent accepted the offer of sex. Findings such as these are interpreted in the context of Robert Trivers' parental investment theory. This theory provides 'a coherent and plausible way of examining the relationship between parental investment, sexual selection and mating behaviour' (Cartwright, 2000, p. 131). Among all four thousand species of mammals females produce large gametes which undergo internal fertilization and gestation (Buss, 1999, p. 102). In addition to this investment, females make a greater parental investment in terms of lactation, nurturing, and protecting offspring. Trivers' theory makes two important predictions '(1) the sex that invests more in offspring… will be more discriminating or selective about mating; and (2) the sex that invests less in offspring will be more competitive for sexual access to the higher investing sex' (Buss, 1999, p. 103).

Darwin's suggestion that female choice could be an important influence on the nature of male traits (and vice versa) is now the inspiration for a flourishing branch of research. Two mechanisms have been identified. Fisher's *runaway sexual selection* requires only that variation in a male trait is heritable and that

variation in female preference is heritable. In the ‘good genes’ model of sexual selection females assess *honest signals* indicating the quality of a male’s genotype. This model has two variants. In the handicap models based on Zahavi’s *handicap principle* (Zahavi & Zahavi, 1996) females select males with a costly handicap, since their ability to cope with such a handicap is a demonstration of genetic quality. In the second version proposed by Hamilton and Zuk (1982) females select males displaying elaborate ornamentation since the quality of such ornaments is an indication parasite resistance, a heritable component of the immune system (for a detailed account see Cartwright, 2000, pp. 141-155)

Robert Trivers theory of parent-offspring conflict (1974) predicts that because the genetic interests of parents and offspring are not identical, offspring will be selected to manipulate their parents in order to ensure higher investment, and that, conversely, parents will be selected to manipulate their offspring. The most astonishing illustration of such conflict is provided by David Haig’s work on genetic conflicts in pregnancy (1993). Haig has argued that fetal genes would be selected to draw more resources from the mother than it would be optimal for the mother to give, an hypothesis that has received convincing empirical support. The placenta, for example, secretes allocrine hormones that decrease the sensitivity of the mother to insulin and thus make a larger supply of blood sugar available to the fetus. The mother responds by increasing the level of insulin in her bloodstream, and to counteract this effect the placenta has insulin receptors that stimulate the production of insulin-degrading enzymes. Only about 22 percent of human conceptions progress to full term and this creates a second arena for conflict between the mother and the fetus, because the fetus will have a lower quality cut off point for spontaneous abortion than the mother. The mother’s quality cut-off point should also decline as she nears the end of her reproductive life and it may be significant that the offspring of older mothers have a higher incidence of genetic defects. Initially, the maintenance of pregnancy is controlled by the maternal hormone progesterone, but in later stages it is controlled the fetal human chorionic gonadotrophin released into the maternal bloodstream, which causes the release of maternal progesterone. There is also conflict over blood supply to the placenta, with the fetus being prepared to demand a larger blood supply than is optimal for the mother. This results in hyper-

tension and, significantly, high birth weight is positively correlated with maternal blood pressure. After birth the young infant may demand more resources than the mother is prepared to provide and the presence of benzodiazepines in breast milk may be a counter to this strategy. Within the offspring there will be genetic conflict between the genes from the father and those from the mother, with paternally derived genes activating to facilitate a demand for greater resources. Evidence for this comes from Prader-Willi syndrome in which infants with two copies of the maternal chromosomal region 15q11-13 have a poor sucking response and weak cry. Conversely, infants with Angelman syndrome have two paternal copies of 15q11-13 and are active and display strong, but poorly co-ordinated, sucking. This latter effect is an instance of *genomic imprinting* in which the effects of genes differ depending on whether they are contributed by the father or the mother (Cartwright, 2000, pp. 266-269). A second important instance of genomic imprinting is provided by the case of the *Igf-2* gene which produces an insulin-like growth factor responsible for promoting embryonic development[22]. The maternally-derived *Igf-2* allele is switched off during germline transmission, but the paternal copy is switched on (Ekstrom, et al., 1995). The paternal gene has an interest in extracting more resources from the mother because, in the absence of monogamy, it is not guaranteed to appear in subsequent offspring. This genomic conflict is therefore one cost of infidelity (Pagel, 1999). This strikingly counter-intuitive picture of pregnancy and nursing derived from the perspective of genic selectionism suggests a system best viewed as a stable tug-off-war, or ongoing arms race, rather than a co-operative venture. In summary, the genetic conflicts of pregnancy are (i) conflict between genes expressed in the mother and genes expressed in the fetus/placenta (parent-offspring conflict); (ii) conflict between maternally-derived and paternally-derived genes within the fetal genome (genomic imprinting); and (iii) conflict between maternal genes that recognize themselves in offspring and the rest of the maternal genome (gestational drive) (Haig, 1996a; 1996b).

One consequence of this struggle between paternal and maternal genes during gestation could be the maintenance of some of the variance in intelligence,

[22] Research published in September, 2001 suggests that the *Igf2* gene is not imprinted in primates (Killian, Hoffman & Jirtle, 2001; Killian, et al., 2001).

though the quality of maternal nutrition is a more significant factor. In a study of 3484 children of 1683 mothers of normal birth weight born between 1959 and 1966 Matte and colleagues (2001) found that mean IQ increased with birth weight in both sexes across the range of birth weight, and that there were no confounding socio-economic factors. It is significant that these effects can be detected for babies of normal birth weight. Previous studies have shown that babies of low birth weight score significantly lower than those of normal birth weight on tests measuring language, spatial, fine motor, tactile, and attention abilities (Breslau, et al., 1996). In a study of 564 low birth weight children 22 percent were found to have been diagnosed with a psychiatric disorder, the most common being Attention Deficit Hyperactivity Disorder, and importantly males were found to be more at risk and risk was elevated by maternal smoking (Whitaker, et al., 1997). We can be sure that the latter factor was not important in the ancestral environment. The importance of birth weight, maternal care, and current practices has also been highlighted by a series of studies of the impact of low birth weight and breast-feeding versus formula feeding. A recent meta-analysis found that breast-feeding was particularly important for babies with low birth weight; that the cognitive developmental benefit increased with duration; and that breast-feeding was associated with significantly higher scores for cognitive development than was formula feeding (Anderson, Johnstone & Remley, 1999). Lucas, Morley, and Cole (1998) have found that poor early nutrition, particularly in pre-term babies, can result in long-term impairment, particularly in verbal intelligence. This study found a major sex difference in the impact of diet, with boys being severely impaired at age eight, when IQ scores are highly predictive of adult intelligence. Infants who were not breast fed were also more vulnerable. The authors conclude that this study ‘provides further support for our more general thesis that early nutrition during critical windows in early life may have "programming" effects on long term outcomes and provides some of the first evidence from a strictly randomised, blinded, and long term trial with near complete follow up that early nutrition may have persistent effects on the human brain’ (Lucas, Morley & Cole, 1998, p. 1486).

Human Behaviour is not Adaptive (Fitness Maximising)

The anthropologist Don Symons is often credited with producing the first modern work on evolutionary psychology (1979), though as I have said I believe this distinction should go to Robert Trivers. Symons argues that natural selection forges complex adaptations over many generations and that the human mind was shaped in an environment with many differences to the modern environment. For example, males possessing a psychological mechanism that promotes 'a taste for partner variety, and the ability to discriminate low- from high-risk opportunities produced more offspring on the average, than did males with different psychological characteristics' (Symons, 1992, pp. 137-8). This psychological mechanism continues to operate despite the invention of condoms and birth control. It makes no sense, therefore, to think of this behaviour as necessarily adaptive in the current environment. Over 50,000 generations of our ancestors lived hunter-gatherer lifestyles, and many of our attributes are tailored to the requirements of such a lifestyle. In more general terms, however, it makes no sense to think of human behaviour as geared towards 'generalized reproductive striving' (Symons, 1992). Nature does not produce general-purpose solutions.

> No mechanism could possibly serve the general function of promoting gene survival because there simply is no general, universally effective way of doing so. What works in one species may not work in another; what works in the infant of the species may not work in the adult; what works in the female of the species may not work in the male; what works in a given species at one time may not work at another time; what works in solving one kind of biological problem may not work in solving another. And, in every case "what works" is determined by the crucible of evolutionary time (Symons, 1992, p. 138).

Our psychological mechanisms instantiate evolutionary goals, but the behaviour subserving such goals will be highly plastic owing to the variability of human environments. This is the point made earlier by Trivers. The claim is not that a general-purpose learning device subserves adaptive behaviours but that psychological mechanisms are themselves plastic to some degree in order to allow them to function adequately in the environment in which they find themselves.

Amongst the most significant influences on the final state of these adaptations will be parents and other kin. In some environments novel features will cause some adaptations to produce unprecedented, and perhaps maladaptive, behaviours. Consequently, a heritable psychological mechanism is an adaptation if its design promoted reproductive success in a past environment; 'natural selection is not mere differential reproduction, it is "differential reproduction in consequence of… design features"' (Burian, 1983, p. 307, quoted in Symons, 1992, p. 140).

Symons refers to *Darwinian social science* (Or DSS, a term covering human sociobiology, human behavioural ecology, evolutionary biological anthropology, and Darwinian anthropology) as research aimed at uncovering whether individuals are consciously or unconsciously striving to maximise their lifetime reproductive success (LRS). According to Symons 'such research is not genuinely Darwinian and… the reproductive data DSSes have collected rarely shed light on human nature or on the selective forces that shaped that nature' (Symons, 1992, p. 146). Symons argues that to understand the prediction that human beings are fitness maximizers we need to know what aspect of evolutionary theory would be called into question by the prediction's disconfirmation. The theory of evolution offers the prediction that adaptations exist because their design contributed to lifetime reproductive success historically, 'in short, nothing in the theory of evolution by natural selection justifies an adaptation-agnostic science of adaptiveness' (Symons, 1992, p. 146). It is simply of no consequence how an adaptation is performing in the current environment, and consequently we should concentrate our efforts on the study of design, the only known explanation of which is evolution by natural selection.

Contrasting Sociobiology and Evolutionary Psychology

Is our current environment broadly comparable to the ancestral hunter-gatherer environment in which many of our distinctive adaptations were forged? As we have seen, most sociobiologists place an emphasis on the study of fitness and 'explain adaptations in part by measuring fitness and its components' (Turke, 1990, p. 312), because 'underlying mechanisms of behaviour are modified by

selection only because of, and according to their effects on behaviour' (Alexander, 1990, p. 247). Evolutionary psychologists, however, being skeptical of the extent to which 'evolved behavioural tendencies' cause human behaviour to assume the form that maximizes inclusive fitness (Symons, 1989), place an emphasis on the study of causation (Blurton Jones, 1990, p. 354). These two groups also differ on the extent to which human adaptations can be considered *domain-specific* or *domain-general*, with those subscribing to a domain general perspective retaining a commitment to the study of phenomena such as IQ and individual differences, in the tradition of behaviour genetics (MacDonald, 1991), and those taking a domain-specific perspective concentrating on universal species-typical adaptations underlying such things as the psychological mechanism subserving language and reciprocal altruism, and more recently, folk psychological physics (e.g., Baillargeon, 1986; Baillargeon, Spelke & Wasserman, 1985), biology (e.g., Atran, 1990; 1998; Medin & Atran, 1999), mathematics, or *number sense* (e.g., Butterworth, 1999; Dehaene, 1997), and psychology (e.g., Baron-Cohen, 1995; Carruthers & Smith, 1996). As Charles Crawford summarizes:

> Darwinian anthropologists and evolutionary psychologists... differ in their emphasis on (1) the importance of proximate mechanisms, (2) the relevance of current fitness, (3) the role of behaviour, and (4) the nature of proximate mechanisms in the study of adaptations. These differences lead them to place differential importance on the ancestral environment in the study of behaviour (Crawford, 1993, p. 183).

Jerome Barkow has described three ways in which sociocultural traits can affect the fitness of their participants: 'a) they can enhance fitness because the cultural trait is a direct reflection of an evolved psychological mechanism; b) they can lower fitness or be neutral for it, because cultural processes are semi-independent of biological evolution; or c) they can enhance fitness epiphenomenally, that is, in a manner having little or no connection with past genetic selection' (1990, p. 345). Because our contemporary environment (and hence our developmental systems) incorporates such novel aspects as globalism, mass media, technology, drugs, processed foods, pollutants, large group sizes, reduced interaction with kin, and many other phenomena not typical of the

hunter-gatherer environment it is highly probable that current behaviour is not always likely to be a reliable guide to ancestral behaviour, and evolutionary psychology's commitment to the study of evolved psychological mechanisms should take precedence over (though not entirely replace) the study of current fitness. Cross-cultural comparisons, particularly with those still living in environments more similar to that of our ancestors, should allow us to assess the extent to which our psychological mechanisms have diverged as a result of the impact of novel environmental factors.

The phenotype is not the result of a genetic blueprint, but the outcome of the complete developmental recipe; a recipe that now includes many elements not present in the original ancestral developmental system. Hence, an adaptation may not in fact produce the adaptive results typical of an ancestral adaptation. This emphasis on the difference between ancestral and current environment is known as *mismatch theory*. This theory provides a useful perspective on the extent to which pathology can be located 'within the individual'. Some behaviours we currently label 'pathological' may be a result of the mismatch between our ancestral and current environments. In these cases our adaptations may be functioning in the way they were designed, but the outcome may be very different to that in the ancestral environment. However, there may also be instances where a modern environment is particularly benign and the malfunction of the adaptation may not detract from current fitness, and may even contribute to it (Crawford, 1998). For this reason, Crawford suggests some new additions to our terminology, including *quasinormal behaviours*, *true pathologies*, and *pseudopathologies*.

Quasinormal behaviours are those behaviours that would have been rare or non-existent in an ancestral environment because of their fitness costs, but that have now become prominent and socially acceptable to a relatively large proportion of the members of a particular society. For example, 'the adoption of genetically unrelated children due to the dearth of "substitute" children from extended family for childless couples' (Crawford, 1998, p. 284). *True pathologies* are caused by physical assaults to major adaptations and 'would detract from fitness in virtually any environment' The causes of true pathologies are genetic

defects, physiological damage, and extreme cultural deprivation. ‘These conditions are pathological in any bit the most benign of artificial environments’. *Pseudopathologies* occur when an environmental change produces ‘conditions or behaviours that are problematic in the current environment’ but which ‘may have their basis in adaptations that contributed to ancestral fitness’.

Crawford also proposes a category of *culturally variable-functionally invariant behaviours* which are ‘behaviours that vary across time and space, but still serve their ancestral function’ (1998, p. 285). These include language learning, age grading, athletic sports, bodily adornment, community organisation, cooperative labour, courtship, division of labour, cleanliness training, gift giving, government, marriage, and penal sanctions. Crawford believes that many current behaviours fall into this category, and are therefore the product of the adaptive plasticity of psychological mechanisms. However, Crawford goes on to claim that ‘ancestral and current environments do not differ vis-à-vis any particular adaptation’. This seems to be based on the pervasiveness of a process of *ancestralization* in which ‘some aspects of a society return to ancestral form when ecological, political, or religious cultural conditions liberalize’ (1998, p. 292). This idea seems to be based on Crawford’s implausible argument that ‘to a large extent, the environment we inhabit is a creation of our own mental processes’ (1998, p. 293), which seems to be a tactic designed to marginalize sociopolitical, economic, and ecological factors in moulding the nature of our social organization and social interactions.

Mismatch and Mental Illness

Perhaps one indication that there is a severe mismatch between our current and ancestral environments is the lifetime prevalence of psychiatric (*DSM-III-R*) disorders in the United States. The figures given in one recent study are affective disorders, 14.7 percent; anxiety disorders 19.2 percent; substance abuse/dependence 35.4 percent; antisocial personality disorders 5.8 percent, and any NCS (National Comorbidity Survey) disorder 48.7 percent. Whilst these figures most certainly do not represent the lifetime prevalence of true patholo-

gies, they do give some indication of the level of stress and distress in the current environment.

In his book *Britain on the Couch* (1997) Oliver James describes ‘an epidemic of irritability and aggression, of depression and paranoia, of obsessions, panics, addictions, compulsions, relationships that are not working, careers that dissatisfy’ (1997, p. x) which he blames on the failure of advanced capitalism to ‘meet our primordial needs, evolved over millions of years, for status and emotional attachment’ (1997, p. xi). In particular he gives striking examples of how even those of high social status, such as Princess Diana, have been prone to self-obsessed rumination (James, 1997, p. 60). The optimum group size in the environment of evolutionary adaptedness may have been as small as 150 individuals (Dunbar, 1992; 1993; 1996) and one causal factor of this rising tide of distress is claimed to be the unparalleled social exposure facilitated by the mass media, which James refers to as *death by a thousand social comparisons*. James draws on a view of depression dating back to a paper published by John Price (1967) ‘The Dominance Hierarchy and the Evolution of Mental Illness’, and now known as the *social competition theory of depression*, in which depression is considered to have originally functioned as an adaptive response to the loss or absence of power and status within the social group (Gilbert, 1992; Price, 1998; Price, et al., 1994; Sloman & Price, 1987). Perhaps the modern proliferation of micro-niches such as clubs, societies, and other organizations do represent, among other things, a means to recreate the smaller, more egalitarian and more intimate groups of our evolutionary past, and as such could constitute an example of Crawford’s process of ancestralization. In his investigation of the phenomenon of *learned helplessness* in humans Seligman has found that those prone to depression tend to have explanatory styles skewed towards *permanence* (they believe bad conditions will persist), *pervasiveness* (they catastrophise or make universal inferences from specific events), and *personalization* (they internalise rather than externalise the causes of bad events) (Seligman, 1990, pp. 44-51). It may be that this difference in explanatory styles explains the failure of depression to be all-pervasive in the debilitating social conditions described by James: those with pessimistic explanatory styles being

particularly prone to the effects of contemporary social exposure and/or limited in their capacity to benefit from participation in smaller social groups.

The social competition theory of depression is one plausible application of mismatch theory to mental illness. It remains likely, of course, that some instances of depression are the result of pathological changes in systems regulating mood (Nesse, 2000, p. 18), however, as Murphy and Stich conclude:

> One of the morals to be drawn from these… hypotheses about depression is quite general. The environment in which selection pressures acted so as to leave us with our current mental endowment is not the one we live in now. This means that any mental mechanism producing harmful behavior in the modern world *may* be fulfilling its design specifications to the letter, but in an environment it was not designed for. In the disorders that result there is nothing in the mind which is malfunctioning (Murphy & Stich, 2000, p. 83).

The modern environment can also be regarded as relatively benign, with much of the stress caused by fear and anxiety, for example, being the product of evolutionary lag. Amongst those who view mismatch as explaining the prevalence of some *DSM*-type disorders are evolutionary biologist George C. Williams and psychiatrist, Randolph Nesse. In their book *Evolution and Healing* they point out that: 'most of our excessive fears are related to prepared fears of ancient dangers. Darkness, being away from home, and being the focus of group attention were once associated with dangers but now mainly cause unwanted fears' (Nesse & Williams, 1995, p. 214). Few, if any, phobias are associated with dangers in our current environment such as guns, drugs, radioactivity, or high fat meals and so perhaps we all suffer from the condition of *hypophobia*, or inadequate aversion to harmful stimuli, though few, if any, of us feel the need to have our fear levels increased by therapy (Nesse & Williams, 1995, p. 215).

Conclusion

In this chapter I have provided an overview of some of the most relevant developments in contemporary evolutionary thought. In the following chapters these

ideas will be used to develop a framework for the analysis of psychiatric disorders.

Chapter 5

The Society of Mind

> We want to explain intelligence as a combination of simpler things. This means that we must be sure to check, at every step, that none of our agents is, itself, intelligent. Otherwise, our theory would end up resembling the nineteenth-century "chessplaying machine" that was exposed by Edgar Allan Poe to actually conceal a human dwarf inside. Accordingly, whenever we find that an agent has to do anything complicated, we'll replace it with a subsociety of agents that do simpler things.
>
> (Minsky, 1988, p.23)

Following Griffiths and the other developmental systems theorists I have argued that human psychological phenotypes are constructed by developmental systems, that is, 'heterogeneously constructed through the interaction of stereotypically biological resources like genes, stereotypically cultural resources like moral norms and resources that are hard to classify in terms of that dichotomy, like experiences of play' (Griffiths, 1997, p. 159). Adaptations are configurable, interconnected, and embodied systems whose sensitivity to the exacting demands of extra-genetic inheritance helps to explain the diverse psychological make-up of different human groups living in different environments. Such adaptations are quite unlike the discrete, autonomous, informationally encapsulated, and mandatory modules envisaged by cognitive science. Overall, the concept of 'psychological adaptation' may be more adaptable to the epistemic needs of evolutionary psychologists than that of 'module' because it is rooted in a theoretical framework long verified by empirical investigation. However, in this chapter I shall attempt to merge the concepts of 'psychological adaptation' and 'module' within a perspective on evolution based on developmental systems theory and the causal homeostatic theory of natural kinds. In tandem these theories allow us to identify projectable categories, to avoid arbitrary concepts, and to avoid inappropriate reductionism. Additionally, they allow us to overcome the epistemic hazards thrown up by the three dichotomies of mind/body, cognition/emotion and nature/nurture discussed in chapter two.

The clearest statement of the task that faces evolutionary psychology comes not from a biologist or a cognitive scientist but from one of the founders of artificial intelligence, Marvin Minsky, who sets out in his book *The Society of Mind* (1988), the questions that need to be answered 'to show how minds are built from mindless stuff, from parts that are much smaller and simpler that anything we'd consider smart'. I concur with Minsky that 'unless we can explain the mind in terms of things that have no thoughts or feelings of their own, we'll only have gone around in a circle' (Minsky, 1988, p. 18). These questions are:

Function:	How do agents work?
Embodiment:	What are they made of?
Interactions:	How do they communicate?
Origins:	Where do the first agents come from?
Heredity:	Are we all born with the same agents?
Learning:	How do we make new agents and change old ones?
Character:	What are the most important kinds of agents?
Authority:	What happens when agents disagree?
Intention:	How could such networks want or wish?
Competence:	How can groups of agents do what separate agents cannot do?
Selfness:	What gives them unity or personality?
Meaning:	How could they understand anything?
Sensibility:	How could they have feelings and emotions?
Awareness:	How could they be conscious or self-aware?

As Minsky points out 'these questions all seem difficult… but once we see the mind as a society of agents, each answer will illuminate the rest' (1988, p. 18). Although it is not possible to offer answers to all of Minsky's questions the aim of evolutionary psychology should be to identify components that simply carry out mechanical processes in a routine and reliable fashion and are not invested with the properties we seek to explain. In this and the following chapter I will present a few tentative answers to Minsky's questions, and seek to apply the results to psychopathology.

In keeping with the emphasis of developmental systems theory Patrick Bateson and Paul Martin have argued that our ideas about the connection between evolution and development would benefit from a reduced emphasis on the metaphor of a genetic blueprint and a consideration of how

> The processes involved in behavioural and psychological development have certain metaphorical similarities to cooking. Both the raw ingredients and the manner in which they are combined are important. Timing also matters. In the cooking analogy, the raw ingredients represent the many genetic and environmental influences, while cooking represents the biological and psychological processes of development. Nobody expects to find all the separate ingredients as discrete identifiable components in a soufflé. Similarly, nobody should expect to find a simple correspondence between a particular gene (or a particular experience) and particular aspects of an individual's behaviour or personality (Bateson & Martin, 1999, p. 9).

John Allman makes the reasonable claim that the brain evolved as a buffer against environmental variation, and that its structure and function represent a trade-off between costs and benefits, but additionally, in a consideration of the human brain in particular, he further emphasises that 'the development of the brain to the level of complexity we enjoy – and that makes our lives so rich – depended on the establishment of the human family as a social and reproductive unit' (1999, p. 2). As nature selects for outcomes and not genes (Lehrman, 1953) we might expect much of the information responsible for structuring human psychological mechanisms to be resident in the reliable features of human family groups, rather than in the genome. As the mechanisms subserving our psychology are also somewhat jerry-built, being constructed from materials determined by earlier contingencies of evolutionary history, we should expect random drift, trade-offs, compromises and pleiotropic effects to be a prominent feature. As Griffiths puts it 'living organisms are at the end of lines of descent which pass through many different ecologies' (1997, p. 116).

The importance of both selection and developmental constraints is acknowledged by William Wimsatt's concept of *generative entrenchment* (Wimsatt & Schank, 1988). Organisms with highly conserved characters and developmental constraints arise because of the incremental nature of natural selection in which 'each slight modification is generated against the background of the existing developmental system… The removal of ancient elements of the developmental system would be likely to remove things that later modifications have made use of and so to disrupt the growth of those modifications'. Because of this 'elements of the developmental system therefore tend to become increasingly gen-

eratively entrenched as more is built on top of them' (Sterelny & Griffiths, 1999, pp. 233-234). We should also remember, as Bateson and Martin point out in a usefully succinct phrase, 'inheritance does not mean genes' (1999, p. 46), and as illuminated with characteristic insight by Paul Griffiths

> …evolutionary psychologists do not go far enough in integrating intrinsic and environmental factors into a single "developmental system" and hence are unable to entirely escape the biology/culture divide… The developmental system, of an organism is the entire set of factors which are reliably present in each generation of that lineage of organisms and whose interaction reconstructs the typical life cycle of the lineage… Tooby and Cosmides recognize these facts by defining the developmental programs as the entire zygotic machinery passed from one generation to the next (Tooby & Cosmides, 1992, p. 78). But they are unwilling to extend the program any further. The program unfolds against the background of an environment whose contents it anticipates. This is very different from a developmental systems conception, in which the elements of the environment necessary for the construction of the life cycle are part of what the organism inherit. The social interactions that induce normal psychosocial development in the rhesus monkey are as much part of its developmental system as the endoplasmic reticulum of its maternal gamete. The nuclear genetic material, the zygotic machinery, and the social environment are all "inherited". They are all passed on from the last generation to the next and interact to reconstruct the life cycle (Griffiths, 1997, pp. 127-129).

The environment does not simply select 'from built-in options' as Gazzaniga suggests (1994, p. 3), rather any of the inherited components of the developmental system can 'mutate' producing novel phenotypic characteristics. In contrast to the perspective encouraged by the genetic blueprint or genetic program metaphors, the developmental systems approach allows for phenotypic variability and for constant, stable outcomes, provided that all of the resources required by the developmental system are available (Griffiths, 1997, p. 186). The developmental system is, in fact, 'the real source of stability across generations' (Griffiths, 1997, p. 61).

Griffiths' Taxonomy of the Emotions, or Why Hierarchies Matter

In addition to the modular affect programs, and the socially constructed emotions referred to in chapter three, Griffiths postulates a category of higher cognitive emotions, which are also known as the 'strategic emotions', the 'moral sentiments', or the 'social emotions'. These emotions, such as guilt, envy, and jealousy, do not share the passivity (lack of responsiveness to long-term planning) of the affect programs and indeed 'seem more integrated with cognitive activity leading to planned, long-term actions' (Griffiths, 1997, p. 100). They are also culturally variable. In common with Robert Frank (1988), Griffiths views these emotions as having a strategic role, and the key to understanding them is the distinction between local irrationality and global rationality. Unlike the 'tactical' affect programs the social emotions or moral sentiments are designed to be solutions to the *commitment problem*. As Matt Ridley points out 'they are a way of settling the conflict between short-term expediency and long-term prudence in favour of the latter' (1996, p. 133). Advantageous social interactions can be encouraged through commitment to 'irrational' behavior, such as being vengeful or loyal, provided that the commitment is detectable, through reputation, or honest signalling via physical and behavioural clues; such clues may be pancultural or culture specific (Griffiths, 1997, p. 120). As Frank explains, 'the idea rests on a simple paradox, namely, that in many situations the conscious pursuit of self-interest is incompatible with its attainment' (1988, p. ix).

In a series of experiments Frank and his colleagues demonstrated that in a total of 61 pairwise interactions 75.2 percent of co-operators and 60 percent of defectors were correctly identified. Clearly, if it is 'possible (if necessary after an extended period of acquaintance) to learn something about the likelihood that a person will behave opportunistically... then predispositions to eschew self-interest will emerge and prosper under the terms of the commitment model' (Frank, 1988, p. 144). In a review of the evidence on fairness in social transactions (in which Frank defines a fair transaction as 'one in which the surplus is divided (approximately) equally' (1988, p. 165), and where the surplus is the difference between the buyer's and seller's reservation prices) Frank concludes that people will suffer a penalty rather than accept an unfair bargain (1988, pp.

164-184), a find confirmed by Herbert Gintis' work on strong reciprocity referred to in the previous chapter. Unlike self-interest models, in which individuals act with perfect rationality to secure their own interests in every transaction, the commitment model of the moral sentiments makes it possible to predict that people will often reject a beneficial transaction if they perceive an unfair division of the surplus. In the long-term, however, the behavioral dispositions associated with the moral sentiments or social emotions such as guilt, envy, anger, disgust and shame, ensure that these states 'act as internal guarantors of alliances' (Griffiths, 1997, p. 128) by enforcing 'commitment to strategies that would otherwise be disrupted by the calculations of self-interest' (Griffiths, 1997, p. 118).

Were our psychology to be based only on passive modules (i.e., modules displaying minimal cultural variability and lack of responsiveness to long-term planning), designed by natural selection to produce rapid and reliable responses to basic survival needs, we would be inflexible prisoners of the moment. Though the emphasis of cognitive science has been on elements of higher cognition such as rationality and decision making, even to the point where the affective realm was factored out 'to the maximum extent possible' on the belief that 'if one were to take into account these individualizing and phenomenalistic elements, cognitive science might become impossible' (Gardner, 1985b, p. 41), it seems likely that the mechanisms allowing us to account for the future are as dependent on the strategic emotions as on rational planning or other aspects of conscious information processing.

Are the Higher Cognitive Emotions Modular?

According to Griffiths there are two ways of introducing irruptive motivational states like the higher cognitive emotions into psychology. The first, in which 'the conscious affect (feeling) associated with emotion acts as an internal source of reinforcement for behaviour' which 'is entirely consistent with the affect program theory' (Griffiths, 1997, p. 121) is represented by Frank's commitment model. The second way is that of proposing additional psychological mechanisms, the development of which may or may not depend on the existence of affect pro-

grams. Griffiths' own view of these emotions is that they are 'heterogeneously constructed'

> The developmental system which constructs the psychological phenotype includes traditional biological factors such as genes and traditional cultural elements such as stories and norms of behaviour. It contains many other resources, from child-rearing practices to landscapes. All of these may differ across cultures and induce variants of human psychology… Variation is of interest to evolution whenever it is reliably self regulating (Griffiths, 1997, pp. 131-132).

This view contrasts with that of evolutionary psychologists such as Cosmides and Tooby who view a species-typical suite of adaptations as the necessary outcome of the interaction of genes and any normal environment. Although some earlier sociobiologists tended to view individual traits as highly variable, they have also tended to discount the role of environment and have attributed significant differences, such as individual and racial differences in intellectual ability as measured by IQ tests, to the influence of polymorphic genes (Jensen, 1998; Rushton, 1997). But, although sociobiologists have emphasised differences in particular traits, in common with evolutionary psychologists they do tend to view human traits as a whole as being universally distributed. Therefore both of these groups of researchers can be said to subscribe to the doctrine of the monomorphic mind. Within this worldview it seems almost inevitable that any statistically atypical variance in traits will be ascribed either to pathology or culture, with the consequence that the 'nature versus nurture' dichotomy would be perpetuated rather than resolved.

Griffiths rightly points out that, from the developmental systems perspective, the environment can be the source of novelty, and that interactions between genes and environment will be nonadditive. Therefore, although the higher cognitive emotions are hypothesised to have evolved as a solution to the commitment problem, they may be highly variable across different environments. It is for this reason that Griffiths views the higher cognitive emotions as not being 'isolated modules, or special adaptations of higher-level cognition [but] manifestations of the central purpose of higher cognitive activity – the understanding and manipu-

lation of social relations' (Griffiths, 1997, p. 243). However, Griffiths appears to have an impoverished notion of modularity drawn from work in the cognitive sciences, and also to rely on the notion of a central repository of plasticity, rationality, and agency.

In the dedication to his seminal work *The Modularity of Mind* (1983), which, as we have seen, is largely responsible for the renewed interest in modularity amongst cognitive scientists and evolutionary psychologists, Jerry Fodor writes:

> One day – it must have been five years or so ago – my friend, colleague, and sometime co-author Merrill Garrett made what seems to be to be the deepest remark that I have yet heard about the psychological mechanisms that mediate the perception of speech. "What you have to remember about parsing," Merrill said, "is that basically it's a reflex." This work is, in effect, a sustained meditation on Merrill's insight, and is gratefully dedicated to him (Fodor, 1983, dedication)

Based principally on ideas arising from his arbitrary distinction between mechanisms subserving perception and cognition (1985, p. 3), and a complete neglect of emotion, Fodor subsequently describes the functioning of modules as i). domain specific; ii). mandatory; iii). inaccessible to central processes; iv). fast; v). informationally encapsulated; vi). producing 'shallow' (preliminary) outputs; vii). associated with fixed neural architecture; viii). prone to characteristic and specific breakdown patterns, and ix). having an ontogeny of characteristic pace and sequencing. Although he has a reputation as an opponent of evolutionary psychology (Fodor, 1998a; 1998b), Fodor claims that 'no facts now available contradict the claim that the neural mechanisms subserving input analysis develop according to specific, endogenously determined patterns under the impact of environmental releasers' (1983, p. 100). This view of the development of modules seems entirely in keeping with deterministic models of human mental endowment eschewed by some as 'Darwinian fundamentalism' (Gould, 1997a) or 'neurogenetic determinism' (Rose, 1997). Howard Gardner has described Fodor as driven to 'archnativism because of the difficulties of understanding how knowledge can be acquired' (1985a, p. 13).

In some respects Fodor's characterisation of modules as discrete, autonomous, and inevitable products of development represents a retreat from the more sophisticated models of the nineteenth century. The neurologist John Hughlings Jackson (1882; 1884) proposed a model of the nervous system as a functional hierarchy in which 'diseases or damage that affected the highest levels would produce dissolution, the reverse of evolution: the animals would still have a repertory of behaviours, but those behaviours would be simpler, more typical of an animal that had not yet evolved the missing brain structure' (Kolb & Whishaw, 1996, p. 15). Hughlings Jackson believed that functions were dependent on distributed components in which disconnection syndromes were likely, and could result from damage to areas not thought to be involved in the function in question:

> Thus if, for example, the nondominant (the nonlanguage) hemisphere is not involved in language but in spatial organization, then damage to that hemisphere would be revealed not just in spatial disabilities but also in language impoverishment because spatial concepts cannot be employed. Hughlings Jackson was particularly modern – so much so, in fact, that his ideas are receiving more serious consideration today than they did in his own time (Kolb & Whishaw, 1996, p. 15)

Hughlings Jackson considered that the 'lowest centres were the simplest and most rigidly organized, while the middle and highest regions were less tightly organized and more complex. The middle centres were said to 're-represent' the lowest, while the highest centres 're-re-represent' the lowest centres' (Grigsby & Schneiders, 1991, p. 25). Modularity needs to be accompanied by the concepts of an evolutionary hierarchy (though not a control hierarchy) of integrated mechanisms, and of functions based on distributed components, if we are to determine the varieties of modules and the nature of their interaction. Unfortunately, the consequences of the idea (derived from phrenology) that modularity and the anatomical localization of function are inseparable have already been detrimental as 'cognitive neuropsychology disappeared from science for more than half a century because of the diagram-maker's premature… attempts to express their functionally modular theories as also anatomically modular' (Coltheart & Langdon, 1998, p. 140), and confusion about these issues still

abounds today. There is no reason in principle why the affect programs, the higher cognitive emotions, and the socially constructed emotions should not be subserved by a dedicated neural architecture, but we need to be aware that these 'modules' are constructed through a very different contribution of causal co-determinants and have different properties. Our first group of developmentally more rigid affect programs arose to satisfy basic survival needs: these are our best candidates for functions that we share in common with many other species.

The second group of higher cognitive emotions can be considered adaptations of higher cognition, but these display considerably more plasticity than the affect programs, and may show considerable variability because of the influence of environmental (including cultural) factors in ontogeny. The socially constructed emotions may be subserved by a modular architecture, but this architecture is dependent on the plasticity of our psychological mechanisms. One recent study, for example, reported a multi-component reading system in which the components 'are differentially weighted depending on culture-specific demands of orthography' (Paulesu, et al., 2000). Clearly, searching for the instructions for this differential weighting of components of the reading module within the genome, or for selection pressures in an appropriate environment of evolutionary adaptedness, would be without justification, and yet it does seem appropriate to view the system as a functional module, or dedicated neural system. However, we should be aware that even this type of module cannot arise unless there is a highly developed and constrained suite of adaptations upon which this 'cultural adaptation' can be built. It is not simply fashioned from some central repository of plasticity supplied to meet any contingency.

Making Sense of Hierarchies

As I claimed earlier, one of the most insidious consequences of the genetic blueprint idea is the expectation that phenotypic characteristics should be innate, that is, *hereditarily determined*, or arising independently of environment or experience (Lehrman, 1953, p. 341). In his commentary on Stoljar and Gold's (1998) discussion of the biological neuroscience thesis (1998) Ian Ravenscroft

writes ‘future science would vindicate the thesis only if it were discovered that selective forces played no part in shaping mental modules, that the child’s social environment has no significant impact on its cognitive development, and that any number of other wide claims made by contemporary psychology are false’ (1998, p. 137). Griffiths has made a good attempt to integrate developmental systems theory with the concept of ‘modularity’ in his taxonomy of the emotions. However, Griffiths’ model accommodates the idea that there is also some sort of central processing unit or ‘Cartesian Theatre’ which acts as a store of general-purpose plasticity in addition to modular adaptations. This is a retreat from the concept of mind as a collection of mindless organs and, accordingly, I would like to suggest an entirely modular perspective.

I have argued that we should be careful not to conflate ‘modules’ with ‘Fodorian modules’, or genetically-determined modules called forth by environmental releasers, as this is likely to generate the expectation that psychological processes are likely to be subserved by indefeasible and culturally invariant entities. Although many theorists in evolutionary psychology agree that our concern should be with the evolution of the mechanisms subserving behaviour, rather than with behaviour itself, the concession made to phenotypic variability is often manifested in the notion that the environment might select from an innate repertoire of characteristics. Hence Michael Gazzaniga writes:

> Selection theory provides a link by which knowledge of how genes and environment interact can be bootstrapped to issues of cognition... If this hypothesis is accurate, it is quite possible that we humans are living in a delirious frame of mind about what influences what and what we can do about it. The deceptively simple notion of applying biological constructs to psychological processes challenges our whole philosophy of life – including the importance we place on personal achievement, intelligence, and acquired beliefs. Even though at the psychological level much of what happens to a person appears to be the result of instruction, at the molecular level we consistently see signs that selection is operating… It is my aim to show that the selection process governs not merely low-level neural circuit events like synaptic relationships (or how neurons talk to each other), but also the complex circuits responsible for higher functions, such as language and problem solving, and that, indeed, these were built into the brain as the result of millions of years of evolution (Gazzaniga, 1994, pp. 4-5).

This supposedly revolutionary resolution of the 'nature versus nurture' or what Gazzaniga describes as the 'selection versus instruction' dichotomy does little more than combine an impoverished view of modularity drawn from cognitive science with an impoverished view of evolution by natural selection. The idea that the environment selects from a massive repertoire of possibilities residing in the genome pays some regard to phenotypic variability, but is ultimately compatible with the view of human nature as relatively uniform, hardwired, and immutable. In essence this model postulates a genome containing a number of immutable types. Hence, any phenotypic variability is ascribed either to genetic variation or cultural inscription on our systems subserving 'general plasticity'. Recent years have witnessed a resurgence of models in which phenomena as diverse as intelligence and morality are viewed as variable, but genetically determined, traits unequally distributed amongst a familiar hierarchy of classes, races and sexes (Herrnstein & Murray, 1996; Jensen, 1998; Murray, 1998; Rushton, 1997). All of these models are based on the notion of a general-purpose architecture resting on top of a number of basic instinctual drives shared with other animals. Ironically, the only difference between these biological determinist models and models favoured in many branches of the human sciences is that in the latter the general-purpose architecture is viewed as such a powerful source of plasticity that, in effect, it erases our evolutionary heritage, and allows theorising to proceed without reference to the ideas of evolutionary biology.

In a response to some of my thoughts on a module for interpreting and predicting social behaviour (the theory of mind module) and its possible significance for an understanding of autism and schizophrenia, the evolutionary biologist George Williams wrote:

> I have [a problem] with the way evolutionary psychologists postulate a module whenever they find it convenient, with little thought as to how many modules there can be and how they might relate to each other. Maybe I am waiting for someone to propose some kind of module hierarchy, analogous perhaps to Tinbergen's instinct hierarchy proposed a few decades ago… At the top would be a prioritizing module that would decide which others to activate and when… There is an enormous number of different kinds of

> behaviour that require nerves for sensory-motor coordination and the thinking that organizes it, but very few are performed at the same time. I imagine that natural selection for the economic use of resources leads to the same nerves (and same brain regions) playing roles in javelin throwing and eye closing and letter typing, but not all at the same time. This does not mean that modules are not real, it merely means that they do not correspond to parts that can be identified in dissecting the brain (personal communication, 1998).

Williams displays the 'primacy of mind' syndrome discussed in chapter two. The evolutionary biologist Stephen Jay Gould, an opponent of 'panselectionism' and 'panadaptationism' was the main example whose work was addressed, but here we see another biologist, one who takes a mainstream genic selectionist viewpoint, resistant to the idea that everything about the mind can be described in mechanical terms. At the top of Williams hierarchy we find the 'master control module', a module that *knows* how other modules should be activated.

As we have seen, the work of Hughlings Jackson in the nineteenth century disconnected the ideas of modularity and brain localization and placed an emphasis on the notion of an evolutionary hierarchy of functions. I believe Hughlings Jackson's ideas should inform current models. In his book *Darwin's Dangerous Idea* (1995) Daniel Dennett proposes 'an outrageously oversimplified structure… for synoptic insight' called the Tower of Generate-and-Test each new floor of which 'empowers the organisms at that level to find better and better moves, and find them more efficiently' (1995, p. 373). This simplified hierarchy of organisms provides an illustration of how natural selection becomes increasingly dependent on developmental processes as more information is stored in the environment, rather than in the genome (see table 3).

Darwinian creatures	organisms with hardwired phenotypes
Skinnerian creatures	organisms with conditionable plasticity
Popperian creatures	organisms capable of previewing candidate acts
Gregorian creatures	organisms capable of being informed by the designed portions of the outer environment

Table 3: The creatures inhabiting each level of Dennett's Tower of Generate-and-Test (Dennett, 1995, p. 373).

Darwinian creatures have different hardwired phenotypes and selection of one favoured phenotype results in the multiplication of the favoured genotype, and as Bolton explains, 'early in phylogenesis it is the physical properties of organisms that are exploited for the purpose of information processing' (1998, p. 563). *Skinnerian creatures* are capable of generating reinforceable behaviours provided that the first response is not fatal. *Popperian creatures* can generate and test hypotheses and thus are capable of preselecting from alternative behaviours such that they 'make better-than-chance first moves' (Dennett, 1988, p. 375). Finally, *Gregorian creatures* can arrive at smart moves by employing designed portions of the outer environment, as Dennett explains:

> …tool use is a two-way sign of intelligence; not only does it *require* intelligence to recognize and maintain a tool (let alone fabricate one), but tool use *confers* intelligence on those who are lucky enough to be given the tool. The better designed the tool (the more information embedded in its fabrication), the more Potential Intelligence it confers on its user. And among the pre-eminent tools, Gregory reminds us, are what he calls ''mind tools': words. Words and other mind tools give a Gregorian creature an inner environment that permits it to construct ever more subtle move-generators and move-testers (Dennett, 1995, pp. 377-378).

Following Dennett, I would like to suggest that it may also be outrageously simple but illuminating to propose at least four types of module, each appropriate to a level in the hierarchy of generate-and-test, each partly dependent on modules in the level below it, and each capable of transmitting information to other modules. Many basic survival needs may be subserved by developmentally rigid modules sharing the characteristics of Fodor's input systems as described in *The Modularity of Mind* (1983). Such *Darwinian modules* may appear to be 'hardwired' in that their construction is dependent on a developmental system for which the components are present with high reliability. In our taxonomy of emotions the 'tactical' affect programs would be mediated primarily by Darwinian modules. *Skinnerian modules* are built on top of the Darwinian modules, but are not entirely dependent on them; they facilitate simple learned behaviours

and may exhibit the type of learning preparedness thought to underlie phobic responses. Certain of these modules are readily activated by elicitors in the environment that were linked to significant dangers in the environment of evolutionary adaptedness (Mineka, Keir & Price, 1980; Nesse, 1987; Seligman, 1970; 1971), and their constitution helps to explain the existence and persistence of phobias caused by commonplace elements in experience such as spiders, snakes, open places and the dark (Nesse & Williams, 1995). *Popperian modules*, are built on top of the Darwinian and Skinnerian modules and evolved under selection pressures of the complex social environment in which the optimum strategy is influenced by the strategies of the other actors and which must therefore remain highly plastic inn order to facilitate appropriate configuration to local circumstances. Although, like all evolved structures, they have an indispensable genetic component, much of the information affecting the structure and function of these modules is derived from the social environment. Popperian modules, and (to some extent) their Skinnerian and Darwinian subcomponents, would be the principal mechanisms underpinning the higher cognitive emotions, and other solutions to the problems of living a complex social environment, in which the optimal strategy is in part determined by what strategy other players are pursuing. Finally, *Gregorian[23] modules* (such as the reading module discussed earlier) subserve culturally variable aspects of our psychology and arise from the constrained plasticity afforded by the underlying modular components from which they derive a high proportion of their functionality. Gregorian modules may be universal or culture-specific, and as they track the complexities of the social environment they are amongst our principal *conscious* future detectors. This is certainly a simple model, but one with a sufficient correspondence to an idealised phylogeny to free us from the oversimplified picture of modules as the relatively uniform and autonomous components of a genetically determined cognitive architecture.

The general model I am proposing is of a completely modular mind, in which each system retains adaptive plasticity, but in which there is no general-purpose plasticity or *tabula rasa*. Popperian and Gregorian modules in particular are

[23] These are named after British psychologist Richard Gregory.

highly plastic, but they are functional only because they are integrated with and dependent on other modules in the hierarchy. Accordingly, this model allows us to leave behind the quasi-theological notion of a central Cartesian Theatre and the pseudo-scientific ideas of genetic determinism.

Asymmetric Connections Between Modules

Because some modules serve basic survival needs, and are phylogenetically ancient, homologous structures may exist in many species, and a commitment to the phylogenetic and comparative perspectives should be fundamental to evolutionary psychology. Although evolutionary psychologists place an emphasis on the Pleistocene period as the most relevant environment in understanding specifically human adaptations we should remember some functions are so fundamental that they have been preserved for many millions of years. According to the hierarchical model presented above it is also likely that modules arising later in phylogeny are constructed on top of, and by modifications to, existing modules. The ancient serotonergic systems, for example, modulate motivational drive and sensitivity to risks and rewards in the environment, and may be implicated in human psychological phenomena as diverse as anxiety, anorexia and bulimia nervosa, stress, obsessive-compulsive disorder, sleep disorders, substance abuse, and depression (Allman, 1999, pp. 26-27).

Because of their fundamental importance in subserving basic survival needs it is also likely that interconnections between Darwinian modules and other modules in the hierarchy is grossly asymmetrical. The connections between the amygdala (an ancient structure partly responsible for mediating fear conditioning) and the cortex are known to be far stronger than the connections from the cortex to the amygdala (Amaral, et al., 1992). Joseph LeDoux has speculated that it is this asymmetry in the connections between the cortex and the amygdala that explains ‘why it is so easy for emotional information to invade our conscious thoughts, but so hard for us to gain conscious control over our emotions’ (1998, p. 265). Such asymmetries may also partly explain why certain conditions, such as phobias, are particularly resistant to psychotherapy. Investigations by Sperry, Gazzaniga and, LeDoux employing split-brain surgery and ani-

mal models of fear conditioning have revealed ‘a fundamental psychological dichotomy – between thinking and feeling, between cognition and emotion’ (LeDoux, 1998, p. 15). However, as I have argued throughout, we should not imagine that cognitions and emotions are subserved by separate and discrete systems. They are functional precisely because they are so thoroughly interconnected.

The study of the neural substrate of psychological functions reveals that there are multiple systems responsible for psychological phenomena such as *emotion* or *memory* that we often regard as unitary. Furthermore, many of these systems are highly conserved throughout evolutionary history and can function below the level of conscious awareness. In a remarkable paper demonstrating that ‘preferences need no inferences’, Robert Zajonc (1980) showed that preferences can be formed even without conscious registration of stimuli, contrary to the postcognitive theories of affect that were pre-eminent at that time (Schachter & Singer, 1962). Zajonc’s paper has generally been interpreted as evidence for the primacy and independence of affect over cognition (see Zajonc, 1984), but it is more realistic to view these integrated systems as neither cognitive nor emotional but simply as operating below our level of conscious awareness. Zajonc appears to be confusing cognition and consciousness. Wilson had also shown previously that simple exposure to stimuli was sufficient to generate ‘positive feelings toward a previously encountered object [which] are not dependent on consciously knowing or perceiving that the object is familiar’ (Wilson, 1979, p. 811). The existence of a cognitive unconscious, unavailable to introspection (Nisbett & Wilson, 1977), and having its origins early in evolutionary history, helps to explain why implicit and explicit processes are subject to ontogenetic differences, different patterns of dissociation and pathology, and to different patterns of functioning across the life course (Reber, 1992a; 1992b). As LeDoux claims ‘knowing ‘where’ a function is located is the first step to understanding ‘how’ it works’ (1998, p. 73). LeDoux’s research on the emotional brain, concentrating on the fear system, indicate that emotional learning can be mediated by two different systems in the brain. Implicit or unconscious learning can be subserved by circuits in the thalamus and the lateral and central nuclei of the amygdala, or by the thalamo-cortical system, which is capable of making finer,

but slower, distinctions among stimuli. The older thalamo-amygdala pathways are retained because

> The information received from the thalamus is unfiltered and biased toward evoking responses. The cortex's job is to prevent the inappropriate response rather than to produce the appropriate one… [the] fear reaction system… involve[s] parallel transmission to the amygdala from the sensory thalamus and sensory cortex. The subcortical pathways provide a crude image of the external world, whereas more detailed and accurate representations come from the cortex. While the pathway from the thalamus only involves one link, several links are required to activate the amygdala by way of the cortex. Since each link adds time the thalamic pathway is faster. Interestingly, the thalamo-amygdala and cortico-amygdala pathways converge in the lateral nucleus of the amygdala (LeDoux, 1998, p. 165).

Additionally, contextual conditioning, or incidental learning involves an integration of individual stimuli 'into a context that no longer contains the individual elements' (LeDoux, 1998, p. 168). Fear conditioning dependent on context seems to be mediated by another brain structure, the hippocampus, the development of which is known to be controlled by a highly conserved homeobox gene known as *Lhx5* (Zhao, et al., 1999). Emotional disorders may result from an uncoupling of these separate systems, with a dissociation of thalamo-cortical and thalamo-amygdala systems resulting in fear conditioning not representative of events as consciously perceived, or a dissociation of the hippocampal systems resulting in the expression of emotions inappropriate to context (LeDoux, 1998, p. 169). LeDoux makes an important distinction between *emotional memories*, which are dependent on fear conditioning and can be inaccessible to consciousness, and *memories of an emotion*, which are explicit declarative memories (1998, p. 184). The latter initially depend on the temporal lobe memory system, but eventually 'the hippocampus relinquishes its control over the memory to the neocortex', where 'memory appears to remain as long as it is a memory, which may be a lifetime' (LeDoux, 1998, p. 193), a conclusion strengthened by a recent study published by Bontempi and colleagues (1999) showing that interaction between the hippocampal formation and the neocortex mediates the establishment of long-lived cortical representations. Consequently, the creation of new declarative memories can be impaired after bilateral hippo-

campal damage, whilst long-term memories can remain intact (Teng & Squire, 1999). A further consequence of the influence of the temporal lobe memory system is the phenomenon of *state-dependent learning* in which the recall of information is dependent on one's emotional state. This phenomenon may explain why those suffering from depression find it easier to recall sad events, sometimes with discomforting clarity. Rossi (1987) has hypothesised that the existence of state-dependent memory, learning and behaviour mechanisms, operating through autonomic, endocrine, immune and neuropeptide systems, helps us to understand mind-body interactions, and to explain various forms of healing promoted by hypnosis, placebo and relaxation responses. We should always remember that in evolutionary terms systems providing details of the emotional salience of cognitions are as much 'informational' as cognitions themselves, whether such information is accessible to consciousness or not. Given the limitations of our working memory and the construction of our modular minds, there is little reason to believe that much of the information processed by our brains can be conscious.

Perhaps the finding that, in terms of volume, the centromedial complex of the amygdala is the only brain structure to correlate with life-span in both strepsirhine[24] and haplorhine primates (Allman, McLaughlin & Hakeem, 1993) helps to put our contemporary obsession with higher cognition and selection pressures in the Pleistocene into perspective. The centromedial complex is involved in behavioural, autonomic and endocrine responses to danger such as the freezing and startle reflexes and increases in blood pressure and stress hormones (LeDoux, 1998, p. 161). We should expect that selection for mechanisms responsible for helping us to avoid any chance of reproducing has been paramount in evolution, to the extent that any extensive ability of higher cognition to inhibit basic survival responses would be strongly selected against. The brain can allocate fitness values to events via proximate emotional mechanisms, and memories with strong fitness consequences can be subject to different physiological processes than less important memories (Dukas, 1999, p. 44).

[24] *Strepsirhine primates* include lorises and lemurs, *haplorhine primates* include tarsiers, monkeys, apes, and humans.

Hierarchies, Heterarchies, Redundancy and the Evolution of Modularity

Although hierarchies are an important feature of our psychological architecture I have also indicated that it can be maladaptive for mechanisms at higher levels in the hierarchy to have too much control over those in the levels below. Patrick Bateson and Paul Martin compare the organization of the structures mediating behaviour to that of modern companies in which 'the organizational structure tends to be a matrix of project teams rather than a traditional top-down hierarchy' (1999, p. 98). These arrangements are known as 'heterarchies'. Although there is sufficient interaction between components to ensure that the organism functions as a coherent whole distributed systems are also favoured because of their greater efficiency and reliability; this is another reason why we should not expect to find Williams' master control module at the top of our hierarchy. In 1971, building on an idea of the palaeontologist William King Gregory, the neuroscientists John Morgan Allman and Jon Kaas 'suggested that evolution of cortical areas proceeded by replication of pre-existing areas' (Allman, 1999, p. 40). Allman also provides a possible answer to why older cortical areas have been maintained in evolution:

> One reason for the retention of older mechanisms occurred to me during a visit to an electrical power-generation plant belonging to a public utility. The plant had been in operation for many decades and I noticed that there were numerous systems for controlling the generators… When I asked why the older control systems were still in use, I was told that the demand for the continuous generation of power was too great to allow the plant to be shut down for the complete renovation that would be required to shift to the most up-to-date computer-based control system, and thus there had been a progressive overlay of control technologies… integrated into one functional system for the generation of electrical power. I realized that the brain has evolved in the same manner as the control systems in the power plant. The brain, like the power plant, can never be shut down and fundamentally reconfigured, even between generations, All the old control systems must remain in place, and new ones with additional capacities are added on and integrated in such a way as to enhance survival (Allman, 1999, p. 41).

Rilling and Insel's (1999b) comparative MRI study of the primate neocortex confirms the finding that the human brain is slightly over three times larger than

would be expected for a primate of the same body size. However, the data indicate a striking discrepancy between human and pongid brains in the extensive gyrification in the prefrontal cortex of the former, an important finding given the role of this region in complex problem-solving (Koechlin, et al., 1999), and social intelligence (Rowe, et al., 2001; Shallice, 2001; Stuss, Gallup & Alexander, 2001). As Rilling and Insel conclude this departure from allometric trends 'suggests selection for increased gyrification in the prefrontal cortex throughout hominid evolution' (1999b, p. 191). The other area noted for significantly more gyrification than expected is the seventh coronal slice, a region incorporating Wernicke's area, long implicated in the production and comprehension of language. Rilling and Insel also note that the increase in human neocortical gray matter is not proportional with the increase in the volume of the rest of the brain and that, although the increase in white matter outpaces that in grey, this increase falls well short of that necessary to retain the same level of interconnectivity between neurons. Ringo (1991) has also reported similar findings, together with the conclusion that larger brains must show more specialisation. This decline in interconnectivity indicates a greater reliance on the local processing of information and is compatible with the idea that many of our psychological mechanisms are modular.

Another scan of 11 primate species concentrating on the corpus callosum and anterior commissure demonstrates that the increase in primate brain size has resulted in increasingly independent hemispheres (Rilling & Insel, 1999a). Through their work on the insular cortex of bottlenose dolphins Manger and colleagues (1998) have found that although brain sizes vary dramatically across animal species, the range of module size is restricted, though the number of cortical areas across species is highly variable (Kaas, 1993; Kaas & Reiner, 1999). A large range of evidence on mosaic brain evolution compatible with the idea of modularity has recently become available – see particularly Barton and Harvey (2000) and de Winter and Oxnard (2001). Barton and Harvey conclude that 'mammalian brain evolution involved size changes concentrated in specific structures and functional systems' (2000, p. 1055). De Winter and Oxnard note that 'the relative proportions of different systems of functionally integrated brain

structures vary independently between different mammalian orders' and conclude that their 'findings provide more detailed evidence of mosaic evolution in brain organization, and rule out an overriding influence of uniform developmental constraints on mammalian brain evolution' (2001, p. 713). These findings confirm that brain evolution is characterised by the independent evolution of brain structures with anatomical and functional links. One of the most distinctive features of the neocortex is its modular organization (Jones, 2000; Mountcastle, 1997; Rockland, 1998). Although it is clear that these neural modules are not the same as functional cognitive modules it seems sensible to conclude that structure is a guide to function. Just as we do not assume that the cell is accidentally partitioned into organelles, we should not assume that the brain is divided into neural modules and distinct cytoarchitectonic regions merely so that it can perform as a mass of undifferentiated connectoplasm.

The existence of a neuronal type found only in the brains of pongids and hominids is also likely to be of importance. Using samples of the anterior cingulate cortex (Brodmann's area 24) of 28 primate species Nimchinsky and colleagues (1999) found a spindle-shaped cell in layer Vb specific to humans and great apes. The anterior cingulate is known to be involved in response selection (Awh & Gehring, 1999; Turken & Swick, 1999), and performance monitoring (Carter, et al., 1998), but also appears to have a number of discrete, functional regions subserving important aspects of cognition, emotion, and notably vocalization (Bush, Luu & Posner, 2000). Nimchinsky and colleagues note that

> …the emergence of this unique neuronal type in a neocortical area involved in vocalization in primates coincides with the evolution as a definable anatomic structure of the planum temporale, a region that is important for language comprehension. In view of the language comprehension abilities of great apes, it is therefore possible that several cortical structures involved in the production of specific vocalizations and in communicative skills sustained simultaneous, considerable, adaptive modifications during brain evolution in hominoids' (1999, p. 5272).

In considering neuroevolutionary matters we should always keep the issue of sexual dimorphism in mind. There are two types of human brain, male and female, and it is reasonable to expect that these have been subject to different

selection pressures. For example, women have a higher proportion of grey matter to cranial volume, whereas men have a higher proportion of white matter and cerebrospinal fluid to cranial volume. Women also have a relatively larger corpus callosum than men. Gur et al. (1999) found that of the top ten performers in a spatial task, nine were men, and seven of these men had greater white matter volumes than any of the women in the study. Our large brains probably do not simply provide an excess of plastic neurons capable of subserving any function, but may be a solution to the problem of retaining adequate functioning over a prolonged life span (Humphrey, 1999), something that could be of particular importance to caregivers. Allman and colleagues have found that there is a significant correlation between brain weight and maximum life-span in haplorhine primates (Allman, McLaughlin & Hakeem, 1993), and that the maximum human life-span is close to what would be expected for a primate of our relative brain size (Allman, 1999, p. 172). Allman et al. have also discovered in a variety of species that caregivers live longer, whether male or female, and 'that there is no difference in survival between the sexes in species in which both parents participate about equally in infant care' (1998, p. 6866). The fact that human females are the primary caregivers, and that human grandmothers are able to enhance their fitness post-menopausally by assisting the reproductive success of their daughters may also help to explain the structural and functional differences between the brains of men and women (Hawkes, et al., 1998; O'Connell, Hawkes & Blurton Jones, 1999). It would be remarkable if sexual dimorphism in brain structure were to have no relevance for our understanding of pathology, including psychopathology, and the issue of sex differences should be central to any classification of psychiatric disorders.

All adaptations have costs and benefits, and it is certain that psychological mechanisms are not cost-free because the rate of DNA damage in mammalian cells is extremely high, amounting to tens of thousands of DNA damages per day. This implies an enormous metabolic cost in maintenance and repair (Dukas, 1999). Also, as many of the processes within the brain are mediated by the same neurochemicals, functional systems must have the capacity to ensure that the correct information is elicited as required. One benefit derived from the piecemeal addition of overlapping systems is explained by Dukas in his analysis

of the costs of memory: redundancy helps to reduce the amount of error and noise in the system, and therefore 'probably plays a key role in ensuring a high level of accuracy' (1999, p. 41). The cost of redundancy is in terms of increased brain mass and energetic expenditure on maintenance, repair and replication. As George Heninger explains,

> One of the main features of the nervous system is the mutually dependent, diffuse, and often redundant biologic processes that subserve functions. In contrast to the relative specificity of sensory and motor systems, the systems subserving sleep-wakefulness, arousal-motivation, emotional reactivity, memory, and higher order behavioural functions are more widely distributed anatomically. The systems demonstrate extremely complex nonlinear response characteristics so that there is not a simple one-to-one correspondence between measures of neuronal function and the behaviours studied. In addition, there is a great deal of plasticity so that remaining systems can compensate for deficits' (Heninger, 1999, pp. 93-4).

Finally, we should be aware that the distinctive cultural traits of human beings appear to have emerged (or, more likely, grown in significance) during a period in which brain sizes have decreased. It appears that since the Late Pleistocene (around 30,000 years ago) human brain size has *decreased* by approximately ten percent with this decrease being paralleled by a decrease in body size. Maciej Henneberg notes 'it may be concluded that the gross anatomy of the hominid brain is not related to its functional capabilities. The large human brain:body size ratio may be a result of the structural reduction of the size of the gastrointestinal tract and, consequently, its musculoskeletal supports. It is related to richer, meat-based diets and extra-oral food processing rather than the exceptional increase in the size of the cerebrum. The exceptional mental abilities of humans may be a result of functional rather than anatomical evolution' (Henneberg, 1998).

As it is often said there are no general purpose problems in nature, and hence there are no general purpose solutions. The preservation and incremental modification of entrenched mechanisms is likely to represent a compromise between distributed, heterarchical functioning, supportive of mechanisms moulded by recent selection pressures, and hierarchical functioning capable of preserving the

influence of basic survival mechanisms. An evolutionary approach requires that we consider the costs as well as the benefits of any mechanism (as the latter must exceed the former for a system to persist), including the costs and benefits of those facilitating learning. Any animal may learn fitness-reducing information, or be exploited by other animals providing false information, unless systems are appropriately constrained (Crawford, 1989, p. 12). It seems unlikely, therefore, that the steady increase in brain size witnessed in the evolution of hominids up to between 150,000 and 290,000 years ago (Brace, 1995, p. 215) simply represents an incremental increase in general 'computing power' capable of being directed to any task. Given that there has been no increase in brain size during the emergence of specifically human traits the emphasis it is given in various theories of cognition seems without strong justification. It is well known, for example, that Neanderthals had brains equal in size to, or larger than, those of modern humans (Stringer, 1992, p. 247). C. Loring Brace has suggested that brain size should remain constant following the development of an effective way of transmitting culture, which is the 'primary human adaptive mechanism' (1995, p. 217). Because information is reliably stored in the environment less storage space is needed in brains.

Contrary to the hypothesis presented by Steven Mithen in *The Prehistory of the Mind* (1996) that the modern mind had its origins in a breakdown of barriers between what had been separate modules, it's possible that the evolution of language provided the higher-level processing capable of eliminating the need for the extensive redundancy that had been required to maintain the accurate storage and expression of information. Myths, poetry, songs and mnemonics all have the capacity to preserve a number of levels of information within a simple format. Existing modules and other brain tissue could have been released to respond to selection pressures, and this could in turn have produced an increase in modularity. Given the overarching importance of culture at this stage in human evolution Griffiths' opinion of the character of modular mechanisms that 'insofar as the mechanism reflects details of the evolutionary past, it does so in the form of learning preparedness' (1997, p. 116) seems particularly compelling. In passing we should note that Mithen's model of modular breakdown requires the convergent evolution of all separate human populations between

60,000 and 30,000 years ago. It is extremely improbably that this could have occurred.

Given that critics of evolutionary psychology such as Gould are keen to emphasise the role of developmental constraints in evolution, it seems strange that they see no constraint on the emergence of a massive general-purpose brain. Our closest animal relatives have no such structure, though clearly they do have specialised systems subserving perception, emotion, and cognition which are homologous to our own. The most reasonable conclusion is that our common ancestor had specialized systems and that these systems have been moulded incrementally over the last few million years, though it's quite possible that in geological terms there may have been rapid change even within this relatively short time period. In addition, even if a general-purpose mechanism had evolved this would not erase evolutionary history, and consequently the functions of the new structure would be integrated with earlier modular systems, and hence comparative psychology, neuroscience, palaeoanthropology, and cognitive archaeology would be central to understanding human psychology. In reality though, any massive (macromutational or saltational), change is likely to be highly maladaptive, if not instantly fatal, and the easiest way for evolution to proceed is by the selection of minor changes in each specialised structure, though many such structures may be moulded simultaneously. The evidence from comparative neuroanatomy demonstrates that this is what has happened.

All of the foregoing theoretical and empirical considerations indicate that the sudden emergence of a highly plastic general-purpose neocortex responsible for multimodal functioning is a distinctly implausible evolutionary event, and that evolutionary psychology's commitment to modularity is well-founded.

Heritability and Innateness

As James Chisholm has pointed out theorists working entirely from a 'genetic blueprint' perspective often employ the *phenotypic gambit*, 'the simplifying assumption that the relationship of the genotype to the phenotype is not especially important for understanding adaptation' (1999, p. 30). In his book, *Death, Hope*

and Sex: Steps to an Evolutionary Ecology of Mind and Morality (1999), Chisholm provides an emphasis on how the development of alternative reproductive strategies is contingent on environmental risk and uncertainty. This has the important consequence of confirming that human nature is 'biologically, adaptively local, contingent, and emergent' (Chisholm, 1999, p. xi). Ultimately, however, Chisholm's concession to the importance of development simply incorporates the idea that the environment can select from an innate repertoire of behaviours or mechanisms reliably responsible for those behaviours. This confuses the independent relevance of development (as embodied in the developmental systems approach) and life history theory. Chisholm is certainly correct that we require a combined evolutionary/developmental perspective in order to make sense of phenotypic characteristics, particularly those of human beings. However, his formulation, which relies principally on a more subtle variant of the phenotypic gambit rather than a rejection of it, goes only part of the way to addressing the fears of those critical of hyperadaptationism and hyperselectionism that 'the essence of biology – evolutionary theory – is inherently, essentially, deterministic and insensitive to historical contingencies, especially those affecting inequalities associated with race, ethnicity, class, and gender' (Chisholm, 1999, p. 6). Dylan Evans, for instance, goes as far as to claim 'that all the history of human civilization and culture, from the birth of agriculture some 10,000 years ago until the present, is irrelevant to understanding the design of the human mind' (Evans, 1999, p. 46). This perspective obscures that much of relevance to establishing the nature of evolved psychological mechanisms can be gained from cross-cultural studies. Such studies can demonstrate how the plasticity inherent in many evolved modules becomes 'adaptively local' and dependent for optimal functioning and form on information that does not reside in the genome. These cultural variants of psychological mechanisms can be analysed as 'descendants of a common ancestor', that is descendents of the form that the mechanism would have taken in a uniform ancestral environment. This is one of the ways in which evolutionary psychology can embark on a non-reductive analysis of cultural differences and need not concentrate solely on universal, or species-typical, forms.

The failure to distinguish between traits, mechanisms subserving traits, the genetic elements taking part in the developmental recipe, and other resources available during development generates hubris as to what can actually be selected for. Chisholm recounts the experience of Russian investigators in trying to select for reduced aggression in silver fox pups. After 25 years of selection for ease of handling the Russian team found that the difference between wild pups and the tame strain was that the latter had an extended period of primary socialisation resulting in a delay in the appearance of social fear. Thus the *innate* aggression of silver foxes failed to manifest itself not through selection against aggression genes but by selection for a longer period of primary socialisation. However, the silver foxes also changed in a variety of other ways: they wagged their tails, barked, and the females had abnormal ovulation patterns. These phenotypic characteristics responded to selection at the same time because they are genetically correlated, and therefore unavailable to selection individually. This is known as a pleiotropic effect (Belyaev, 1979; Majerus, Amos & Hurst, 1996, p. 75). The specific involvement of genes in this process remains ambiguous, but a developmental outcome (with mixed costs and benefits) of significance for handlers keen to retain their fingers can be selected for (Chisholm, 1999, p. 32). It is probable that in many instances desired outcomes can be achieved through changes in any aspect of the developmental recipe, but the expectation of a correspondence between a phenomenon *aggression* and a blueprint *genes for aggression* is likely to misdirect empirical investigation, particularly when dealing with species having a prolonged period of development in a complex psycho-social setting. As Lehrman puts it, the notion of 'innateness' applied to human psychology and sociology leads to 'a rigid, preformationist, categorical conception of development and organization' (1953, p. 359). The influence of this confusion about heritability and innateness can even be seen in the work of theorists committed to evolutionary psychology's emphasis on evolved psychological mechanisms who have suggested that some aspects of behaviour might best be understood in terms of *rape modules*, *homicide modules* (Buss, 1999; Buss & Duntley, 1998), or *gender modularity systems* (Cosmides & Tooby, 1999, p. 458). Analysis of this kind should be avoided, and is reminiscent of the phrenological emphasis on discrete areas subserving traits

such as integrity or depravity. Reification of this kind violates the principle that the mechanisms we seek should not embody the characteristics they subserve.

In her book *The Biology of Violence* (1999) Debra Niehoff describes attempts by a team of researchers under Robert Cairns at the University of North Carolina to create distinct breeds of mice varying to a maximum extent in their innate aggressiveness. Mice who 'froze' on being exposed to an intruder were bred to sisters of similarly timid mice, whilst aggressive males who readily attacked an intruder were bred to sisters of similarly aggressive males from other litters. This appeared to produce true breeding lines by the fourth generation, but though 'high aggressive mice were provoked by meeting an unfamiliar mouse for the first time, and low aggressive mice were immobilized… repeated exposure to the stranger normalized behaviour in both lines' (Niehoff, 1999, p. 251) until by the fourth encounter both strains of mice were equally aggressive. Furthermore,

> Even short periods of experience proved enough to override genetic background. When Cairns placed a high-aggressive male mouse, a low aggressive male, and a female together in the same cage, the high-aggressive animal, to no one's surprise, invariably attacked first. But two hours later, more than 40 percent of low aggressive mice had learned to fight back – and they did it effectively enough to take charge of the relationship. Their testosterone levels rose in the characteristic fashion of dominant males, their cortisol levels dipped, and their genes no longer mattered (Niehoff, 1999, p. 251).

Another example of how the neuro-behavioural system is open to experiential input is provided by research on the handling of young rat pups. Early-handled rats, on exposure to stressful stimuli in adulthood, show a rapid release of adrenocorticotrophic hormone from the pituitary in preparation for a response to the challenge, whereas non-handled animals show a slower and more sustained response less appropriate for dealing with stressful challenges. Early-handled rats also display slower neural degeneration and a more robust capacity to learn new tasks in old age as compared to non-handled rats, suggesting that factors operative early in development can have long-lasting effects. The characteristics of early-handled pups are also evident in pups whose mothers provide maternal care in the form of licking and grooming (Bateson & Martin,

1999, pp. 50-51). Apparently, disruption of any part of the developmental system affecting the modulation of the stress response in rats can have long-term consequences.

Perhaps one of the most important non-genetic (though not necessarily non-biological) variables having an impact on the developmental system is that of birth order. Frank Sulloway has collected data showing that 'sibling strategies typically entail emergent properties. Birth order, gender, and temperament all interact to produce personality characteristics that could not be anticipated based on a simple aggregate of these influences' (Sulloway, 1998, p. xvi.). Sulloway concentrates strongly on evidence for the effect of birth order on unconventional thought as expressed in scientific creativity and revolutionary thinking. Though Sulloway probably overstates his case (Rowe, 1997; Ruse, 1997), there are a number of studies suggesting that birth order and family size should be taken into consideration when considering phenomena as diverse as sexual orientation (Blanchard & Bogaert, 1998; Blanchard, et al., 1998); susceptibility to schizophrenia (Stompe, et al., 1999); general susceptibility to psychopathology (Richter, et al., 1997); hypochondria (Skinner, 1997); paedophilia (Bogaert, et al., 1997), and intellectual attainment (Zajonc & Mullally, 1997).

The multiplicity of developmental system variables and the non-additive nature of their interaction implies that we should have reservations about heritability figures based on attempts to quantify the genetic and environmental contribution to traits. Of course, no one doubts that both genes and environment matter but 'how much each of them matters defies an easy answer… [and] no simple formula can solve that conundrum' (Bateson & Martin, 1999, p. 66). Heritability is a population statistic representing the ratio between genetically caused variation and total variation (genetic and environmental) in a given population within a given environment. High heritability figures for any given trait are often deemed to represent immutability in that trait (Wahlsten, 1997), or, even more unrealistically, the genetic determination of that trait (Block, 1995). However, 'if the genetically caused variation is small compared to the environmentally caused variation, then the heritability is low, even when the characteristic is genetically determined' (Block, 1995, p. 450). The heritability of head number, for

example, is low in humans because there is no genetic variation. Where variation in an environmental characteristic is in part due to a heritable characteristic then that characteristic can also be highly heritable even if it is not genetically determined. As Susan Oyama explains,

> Heritability, as the proportion of phenotypic variance attributable to genetic variation under controlled conditions, is not a characteristic of traits but of relationships in a population observed in a particular setting. These relationships are expressed in numbers, which depend on the precise levels of genetic and environmental variables examined and the selection and operationalization of the dependent variable(s). Heritability, that is, is an attribute not of variants but of their statistical descriptions (variance). These descriptions are as dependent on the research design as they are on the traits themselves (Oyama, 1985, p. 37).

Block goes as far as to describe heritability as 'an uninteresting and misleading statistic' (1995, p. 459) because any indirect genetic effects, including gene-environment correlations outside the boundaries of what can be measured using prevailing atheoretical models, are included in the genetic component:

> If there is a genetic difference in the causal chains that lead to different characteristics, the difference counts as genetically caused even if the environmental differences are just as important. If we adopted the opposite convention, the convention that any environmental difference in two causal chains shows that the difference counts as environmentally caused, then we could not use the current methodology for measuring heritability, because we have no general method of detecting indirect genetic effects using current techniques. Heritabilities using the two different conventions would be radically different if there are substantial indirect genetic effects (Block, 1995, p. 468).

The gulf currently existing between theorists in psychology and psychobiology is illustrated sharply by a comparison of the opinion of a group of distinguished behaviour geneticists, 'quantitative genetic methods can detect genetic influence for complex traits… the size of the genetic effect is quantified by heritability' (Plomin, et al., 1997, p. 87) with that of Ned Block: 'heritability as it is construed by the field is a second-class concept that does not belong in anything that can be counted as science' (1995, p. 474). The developmental systems perspective suggests that we should err on the side of caution when consider-

ing heritability estimates. The measure only really comes into its own when breeding lines and their environments can be experimentally manipulated.

Natural Selection and Genetic Diversity

Although natural selection is generally regarded as a mechanism for producing uniformity, there are circumstances in which it can also support genetic diversity in a population, and it is imperative that just as we do not equate a genetic influence with immutability we should also not equate it with uniformity.

E. B. Ford (1940) suggested that where an heterozygote form is favoured over both homozygotes genetic variability would be maintained, a phenomenon known as *heterozygote advantage*. The classic example is that of sickle-cell anaemia. Homozygotes for the sickle allele produce abnormal haemoglobin and often die from anaemia before reaching maturity. Heterozygotes suffer from mild anaemia, but their abnormal haemoglobin molecules provide resistance against malaria. In regions with a high incidence of malaria heterozygotes are the most fit form because homozygotes for the sickle allele die from anaemia, whereas homozygotes for the normal allele are more susceptible to malaria (Livingstone, 1967; 1971; Raper, 1960). Instances of *temporally staggered heterozygote advantage* can occur when selection pressures vary over the life history. One allele coding for the enzyme mannose phosphate isomerase in red deer causes death in the first year of life, but the allele remains in the gene pool because heterozygotes reproduce earlier and are more fecund (Majerus, Amos & Hurst, 1996, p. 63; Pemberton, et al., 1991). An heterozygote advantage can also be conferred by temporal variation in the environment, for example, if different alleles are favoured at different times of the year. A *spatial heterozygote advantage* can occur where ‘particular alleles confer increased fitness in particular patches’ (Majerus, Amos & Hurst, 1996, p. 64). Heterozygotes moving between patches may have an advantage over homozygotes. *Frequency-dependent selection* occurs when fitnesses correlate with the frequency of the phenotype itself or with population density.

As homozygosity in human populations varies from 0.63 to 0.79 (Cavalli-Sforza, Menozzi & Piazza, 1994, p. 141), it is possible that heterozygote advantage and frequency-dependent selection are important in maintaining human psychological polymorphisms. Because of this high proportion of heterozygosity evolutionary psychology should not make a commitment to the idea that human minds are monomorphic, nor restrict itself to the study of 'species-typical' adaptations (Griffiths, 1997; Hull, 1986; Murphy & Stich, 2000; Wilson, 1994). However, a simplistic view of the action of natural selection can lead to what Gould has called the 'fatal flaw in human sociobiology', which is to follow the research strategy: 'break up the behavioural repertoire into items, posit and advantage for each item in terms of individual reproductive success, assume a genetic basis for the behaviour (not necessarily direct) and then infer that natural selection built the item for its implied advantages in the great calculus of reproductive struggle'[25] (1991, p. 50). When one moves away from simple one locus, two allele models, such as those on which the concept of heterozygote advantage depends, to less mathematically tractable models employing 'two loci with epistasis, fecundity selection, linkage disequilibrium and frequency dependence, will often (albeit not necessarily) result in adaptive landscapes characterized by maladaptive evolution in which selection drives the population 'down-hill'' (Pigliucci & Kaplan, 2000, p. 67). Amongst the alternatives to adaptationism enumerated by Pigliucci and Kaplan in a paper celebrating twenty years since Gould and Lewontin's (1979) famous critique of adaptationism are genetic drift (such as the *founder effect*, which seems to account for the prevalence of blood group B in aboriginal American populations), indirect selection (through association with another trait), selection without adaptation (as in a resource-limited species in which a mutation doubles fecundity), and adaptation without selection (in which behavioural plasticity is selected for but the behaviour in question is itself emergent) (2000, p. 67). Although any competent researcher should be careful to assess the impact of these and other factors it is equally important to remember that there is no general argument against the hypothesis that human

[25] For example, in *Introducing Evolutionary Psychology* Dylan Evans writes 'We can imagine genes as little beads threaded along a long string inside each cell. Each bead is an instruction (or group of instructions) that says something like: brown hair, blue eyes, short temper, etc' (Evans, 1999, p. 16).

beings have psychological adaptations, and of course there is compelling evidence consistent with it.

Life History Theory and Developmental Psychology

According to a recent contribution to *Archives of General Psychiatry* two of the important questions facing psychiatry in the 21st century are: 'How does life experience alter gene expression in vulnerable individuals?' and 'How does the aging progress affect disorder expression and treatment'. (Frank & Kupfer, 2000). Both of these questions could be subsumed under a more general enquiry as to how the functions of modules and other adaptations are modulated by life history invariants, that is, under the question as to how functioning changes to meet the perennial challenges to survival, development and reproduction encountered during a normal life span. The evolutionary study of life cycles and life history traits in an ecological context is known as *life history theory* (Chisholm, 1999, p. 35). *Lifespan psychology* aims to integrate data covering the entire course of development from conception and infancy to adolescence, adulthood and old age, by focussing on the study of the 'acquisition, maintenance, transformation, and attrition in psychological structures and functions… involved' and the '(a) interindividual commonalities (regularities)… (b) interindividual differences… and (c) intraindividual plasticity' observed (Baltes, Staudinger & Lindenberger, 1999). There are two main approaches: person-centred (holistic) and function-centred. Together these are sometimes described as *lifespan developmental psychology*. The concept of modularity, that is, of evolved psychological mechanisms, allows us to combine lifespan psychology and life history theory into one combined perspective.

Chisholm describes the *uncertain futures problem* as the problem of 'how to produce an adaptive match between organism and the environment when the organism takes time to 'build' but the 'instructions' for building it are received all at once and the organism's environment is changing the whole time' (1999, p. 19). A developmental module capable of setting the parameters of other modules in response to instructions from the environment could be one solution to the problem of how to create a more functional match between organism and

environment. Although Chisholm presents a reconciliation of evolution and development, claimed to be within the developmental systems tradition, in which 'adult, fully reproductive phenotypes are co-constructed by 'instructions' from their environments as well as their genes' (1999, p.19), the model is, in fact, comparable to Gazzaniga's (1994) selectionist model in which natural selection is responsible for a number of innate options available for expression during development according to the presence of (reliably) variable environmental elicitors. Indeed, just a few pages later he describes his model as accounting for 'developmental mechanisms (themselves produced by natural selection) that produced the individual differences that may be adaptive in particular social and physical environments' (Chisholm, 1999, p. 34). The word *phenotype* generally includes all aspects of an organism other than the genotype, and *phenotypic plasticity* refers to the ability of the genotype to produce more than one alternative form in response to environmental conditions. Both Chisholm and Gazzaniga argue not for phenotypic variability and novelty as envisaged by the developmental systems perspective, but for what is generally known as *polyphenism*, 'the existence of environmentally cued alternative phenotypes in the population' (West-Eberhard, 1989, p. 251).

Chisholm emphasises that optimality is local and contingent, and that the phenotype 'is not resident in or isomorphic with the genome but emergent – developmentally (i.e., historically) dependent on the dialectic between organisms and the environment from conception to death' (Chisholm, 1999, p. 33). Life is a series of trade-offs between survival, development and reproduction, and life history theory postulates that major stages in life history, such as puberty, menopause, and old age, represent shifts in the balance between these competing demands. Hence, as mentioned earlier, the female menopause is hypothesised to mark the point at which a woman's fitness is enhanced more by care of her grandchildren than by care of her own children (Clutton-Brock & Scott, 1991). In modern societies senescence occurs because of a piecemeal breakdown in the body's capacity to repair damage, but in a natural environment few would have lived to the age where selection pressures could not operate on genes whose effects exerted themselves only after individuals had cared for children and grandchildren. Consequently, though some aspects of old age may be the result

of the evolution of life history strategies, other aspects are simply the result of increased longevity promoted by our contemporary environment. However, although we can expect deficits as a result of aging Paul Baltes and colleagues remind us that an analysis based on the idea that 'deficits breed growth' may provide a useful perspective:

> This "deficits-breed-growth" mechanism may not only account for cultural-biological evolution, it may also affect ontogenesis. Thus it is possible that when people reach states of increased vulnerability in old age, social forces and individuals invest more and more heavily in efforts that are explicitly oriented toward regulating and compensating for age-associated biological deficits, thereby generating a broad range of novel behaviors, new bodies of knowledge and values, new environmental features, and, as a result, a higher level of adaptive capacity. Emerging research on psychological compensation is a powerful illustration of the idea that deficits can be catalysts for positive changes in adaptive capacity (Baltes, Staudinger & Lindenberger, 1999, p. 477).

As human beings have a prolonged period of development in the care of parents who can (consciously or unconsciously) communicate information about the social environment, and who can, to some considerable extent, determine many of the conditions of that social environment, Chisholm contends that natural selection should have favoured mechanisms for making decisions about the allocation of resources to survival, development and reproduction based on conditions during the attachment process.

Just after the Second World War John Bowlby received a commission from the World Health Organization to investigate the problems of children who had been orphaned or separated from their parents (Bateson & Martin, 1999, p. 168), and in 1951 Bowlby published findings indicating that such children were more likely to become socially disruptive adolescents and that deprivation of maternal care could have consequences throughout life. Subsequently, Bowlby took an evolutionary, ethological, view of *attachment behaviour* as an adaptation encouraging infants to maintain maximally close contact to the caregiver(s) during times of distress or uncertainty. The nature of the interpersonal interactions experienced during attachment behaviour would have a long-term impact on the capacity of

the infant to form strong emotional bonds through consistent patterns of thinking, feeling, and behaving, or *attachment style* (Bowlby, 1969).

Though early displays of family coercion have been found to be predictive of problem behaviour at age four, these are not as predictive as the absence of early positive interactions, such as 'affectively positive, educative exchanges between mother and child' (Pettit & Bates, 1989). Children with good attachment relations with their parents tend to have fewer tantrums, and 'use their parent as a secure base from which to explore the world' (Bateson & Martin, 1999, p. 24). Ultimately, an individual's *attachment style* (secure or insecure) could have consequences for that individual's reproductive fitness through it's affect on 'three major adaptive challenges: [to] survive to reproductive age, mate, and provide adequate care for offspring so that they, too, will survive to reproduce' (Zeifman & Hazan, 1997, pp. 237-238).

Bowlby thought attachment behaviour was originally selected for as a response to the threat of predation, but Chisholm regards it as a mechanism for 'learning about… one's past and one's present in order to predict one's future – and thereby to "evaluate" one's alternatives and "choose" (i.e., not necessarily consciously) one's optimal developmental pathway'. In fact a great deal of human development can be seen as about 'the ontogeny of reproductively relevant future detectors and value detectors' (1999, p. 119). Following Plotkin (1994), Chisholm views the emotions as value detectors, or innate 'information about the sources of security and danger in our ancestors environments… emotions are not simply irrational messages from our evolutionary past. They mark events' (1999, p. 87). The combined purpose of our cognitive-emotional mental architecture is to allow us to represent both facts and values, but where Chisholm refers to *emotion* he seems to have in mind what I have followed Ekman in calling *affect programs* or basic emotions. Chisholm argues that the subjective experience of fear, for example, 'may be understood as the representation in the phenotype (the embodiment) of environmental risk and uncertainty' (1999, p. 115). An internal representation of environmental risk and uncertainty derived via the attachment process provides the information by which resources can be

allocated between survival and reproduction in order to achieve local optimum functioning:

> …the ultimate reason that inconsistent, insensitive, unresponsive, or rejecting parenting is today associated with insecure attachment is that when our infant ancestors in the EEA experienced inconsistent, unresponsive, or rejecting parenting (through their failure to experience "felt security" in sufficiently many iterations of the attachment cycle) they also sensed emotionally that their larger environments were high in risk and uncertainty – and that they thus had low reproductive value… All else been equal, the optimal reproductive strategy under such conditions is likely to be to maximize current reproduction by producing many offspring while investing relatively little in each (Chisholm, 1999, p. 115-116).

Chisholm views the attachment process as one of fine tuning of behavioural phenotypes in which trade-offs and constraints define local optimality (1999, p. 50). This assumption of local optimality, rather than global optimality, implies that

> (1) perfection cannot exist, (2) the concept of a fixed or "essential" human nature is not useful, (3) the concept of "normal" is ambiguous, and (4) to understand human nature we would do well to adopt a processual approach, focusing on the evolutionary and developmental contingencies (selections, decision, choices, choices) that produce that phenotype (Chisholm, 1999, pp. 50-51)

The actual mechanism for *socioassessment* capable of generating a locally optimum allocation of resources between survival and reproduction is comprised of 'internal working models, theory of mind and Machiavellian intelligence' which become 'different facets of an evolved developmental psychological algorithm for detecting the social future' (1999, p. 120-121). Chisholm contends that TOM (theory of mind) is 'our species particular form of Machiavellian intelligence' (1999, p. 121) and that

> …both TOM and MI have their origins in internal working models of attachment relations… [because] parents' ability and willingness to invest were important correlates or determinants of their children's reproductive value, then perhaps the best way for children to avoid stepping of a [fitness] cliff or to set the stage for fu-

> ture good fitness moves would be to read their parents' minds (Chisholm, 1999, pp. 122-123).

Amongst the phenomena that this model seeks to explain are anomalies in the putative developmental rule 'if conditions are good, become sexually mature early; but if conditions are poor, delay maturity' (Bateson & Martin, 1999, p. 119). Although both sexes are maturing earlier, and the average age of menarche has declined by eleven days per year over the past hundred years as social conditions have improved (Bateson & Martin, 1999, p. 118), there are findings that run counter to this trend. Chisholm discusses a number of studies in which stress related to father absence predicted an earlier age at menarche. Since the publication of Chisholm's book, Bruce Ellis and colleagues have produced another significant study of 173 subjects showing that girls with close, supportive relationships with their parents tend to develop later, whilst those with cold or distant relationships develop earlier. In particular, the quality of the fathers' involvement was found to be the most important feature of the family environment to relate to the onset of puberty (Ellis, et al., 1999, p. 398). In an earlier study Herman-Giddens and colleagues (1988) found that one in fifteen girls who had experienced sexual abuse developed secondary sexual characteristics before eight years of age. Overall, Chisholm concludes that girls developing in conditions of chronic risk and uncertainty 'are likely to experience HPA system hyperactivation, which is implicated in both early menarche and young age at first intercourse' (1999, p. 186), and that 'at least in the US, women who begin childbearing in their teens also tend to have their children in quick succession' (1999, p. 187). Although this model confounds our expectations about such things as early menarche and early (often single) mothering, perhaps the actual human developmental program is: if material conditions are good become sexually mature earlier, but if parental investment, particularly from the father, is poor become sexually mature as soon as possible. In other words, where the social world is genuinely risky our mechanisms for socioassessment are configured by hormonal mechanisms to promote an allocation of resources to reproduction rather than development. Chisholm calls this *Young Female Syndrome* 'an evolved facultative adaptation' (1999, p. 189). The recent finding that 'frontal and parietal gray matter peaks approximately one year earlier in females, corresponding with the earlier age of

females, corresponding with the earlier age of onset of puberty, suggests a possible influence of gonadal hormones' (Giedd, et al., 1999) and may be relevant to the onset of the requirement to employ Machiavellian intelligence and strategies of mate choice as an independent reproductive agent.

In the presence of risk, the optimal strategy for men, as well as women, may also be to maximise current reproduction. The *Young Male Syndrome* (or *Absent Father Syndrome*) could also represent 'an evolved universal capacity that enables males to develop what may be (or have been) the optimal reproductive strategy under risky and uncertain conditions' (Chisholm, 1999, p. 173). Young Male Syndrome has been described as a 'taste for risk' that is 'socially facilitated by the presence of peers in pursuit of the game goals' resulting in homicide, dare-devilry and gambling (Wilson & Daly, 1985, p. 59). Though Chisholm concedes that 'more information is needed on the relationship between attachment history and adult sexual and parenting behaviour, he concludes that 'insecure attachment does seem to predispose both men and women toward an 'uncommitted' style of romantic/sexual behaviour. This, in turn, would seem to be consistent with the hypothetical adaptive function of a strategy for maximizing current reproduction' (1999, p. 202). Of course, an adaptation capable of producing a disposition toward such behaviour need not produce a conscious strategy, nor need it produce adaptive behaviours in the current environment. In our modern environment Chisholm regards the short time preference typical of the two syndromes as 'diagnostic of chronic poverty and inequality' (1999, p. 138).

Life history theory's approach to the Young Male Syndrome also cautions against over-hasty attempts at the biochemical and neuroanatomical individuation of disorders or behaviours, as Paul Gilbert notes,

> …in regard to theories of cause it does not really matter if… male aggression or depression is associated with low 5-HT [serotonin] or not, for such biochemical parameters may simply be the mediators of strategies for depression and high violence (low cooperation) in social contexts where violence pays more than cooperation. Different environments recruit different strategies and therefore different physiologies (Gilbert, 1998, p. 368).

Of particular relevance is the recent finding of heterochronous development in key brain areas[26] indicating that: 'adolescent brain may not be fully developed, and that the highest-level areas to do with social judgement and self-control may not be completely mature until we hit our twenties' (McCrone, 2000, p. 22), though lower-level areas of the cortex dealing with motor and sensory processing appear to mature earlier.

In a longitudinal MRI study of brain development in childhood and adolescence Giedd and colleagues found that increases in cortical grey matter were regionally specific with 'developmental curves for the frontal and parietal lobe peaking at about age 12 and for the temporal lobe at about age 16, whereas cortical gray matter continued to increase in the occipital lobe through age 20' (Giedd, et al., 1999, p. 821). In a comparison of the brains of a group of adolescents ranging from 12 to 16 years of age with a group of adults aged 23 to 30 Sowell and colleagues (1999) found small maturational changes in the parietal, temporal and occipital lobes but large group differences in the frontal lobes and the subcortical regions known to subserve emotional regulation and planning. Sowell and colleagues write:

> In regions of frontal cortex, we observed reduction in gray matter between adolescence and adulthood, probably reflecting increased myelination in peripheral regions of the cortex that may improve cognitive processing in adulthood. This was predicted by post-mortem, electrophysiological, positron-emission tomography and neuropsychological studies of normal cognitive and neurological development. Neuropsychological studies show that the frontal lobes are essential for such functions as response inhibition, emotional regulation, planning and organization. Many of these aptitudes continue to develop between adolescence and young adulthood. On the other hand, the parietal association cortices are involved in spatial relations and sensory functions, and the lateral temporal lobes are involved in auditory and language processing, aspects of cognitive development that are largely mature by adolescence. Thus, observed regional patterns of static versus plastic maturational changes between adolescence and adulthood are consistent with cognitive development (Sowell, et al., 1999, p. 860).

[26] Nonhuman primate studies generally reveal synchronous cortical development, that is, with similar timing in diverse cortical regions (Giedd, et al., 1999, p. 862).

The full import of these findings for an understanding of human rationality may become clearer within the context of Antonio Damasio's theory about the nature of the functional relationship between the frontal lobes and subcortical regions which is considered in the following section.

The Neurobiology of Human Machiavellian Intelligence

In his book *Descartes' Error* (1996a) Antonio Damasio reports new studies of frontal lobe functioning (in addition to re-examining earlier studies) and contends that rather being purely executive centres where reasoning takes place, circuits in the frontal lobes are in fact part of an adaptive system responsible for the integration of reason and emotion, and that it is this integration that allows rational decision-making to take place. Damasio has found that patients with ventromedial frontal lobe damage are not deficient on any neuropsychological test of reasoning ability, or in any aspect of reasoning about problems in social situations. However, such patients frequently become totally disorganised and unable to make successful decisions relating to work, relationships, finances, and so on. A particularly instructive case is that of Damasio's patient Elliot who, after demonstrating normal cognitive functioning and a 'superior intellect' (Damasio, 1996a, p. 41), took part in a series of controlled laboratory tasks concerned with social convention and moral value revealing that

> Elliot had a normal ability to generate response options to social situations and to consider spontaneously the consequences of particular response options. He also had a capacity to conceptualize means to achieve social objectives, to predict the likely outcome of social situations, and to perform moral reasoning at an advanced developmental level (Damasio, 1996a, pp. 48-49).

However, as Elliot himself admitted 'and after all this, I still wouldn't know what to do!' (Damasio, 1996a, p. 49). In fact, following his operation for a frontal lobe tumour Elliot had lost his job through inability to prioritise tasks, had become bankrupt after a business partnership with a disreputable character, had left his wife and children for another woman to whom a brief marriage also ended in divorce, and had drifted without income. Damasio remarks that

> The tragedy of this otherwise healthy and intelligent man was that he was neither stupid nor ignorant, and yet he acted often as if he were. The machinery for his decision making was so flawed that he could no longer be an effective social being. In spite of being confronted with the disastrous results of his decisions, he did not learn from his mistakes. He seemed beyond redemption like the repeat offender who professes sincere repentance as he leaves jail but commits another offence shortly thereafter (Damasio, 1996a, p. 38).

Damasio proposes that what Elliot and others like him lack, is not the capacity to reason, but the capacity to create and respond to *somatic markers:*

> When the bad outcome connected with a given response option comes into the mind, however fleetingly, you experience an unpleasant gut feeling. Because the feeling is about the body, I gave the phenomena the technical term *somatic* state ("soma" is Greek for body); and because it "marks" an image, I called it a marker… I use somatic in the most general sense (that which pertains to the body) and I include both visceral and nonvisceral sensation when I refer to somatic markers (Damasio, 1996a, p. 173).

Somatic markers function as automated alarm signals that protect us against future losses, and then allow us to choose from fewer alternatives. Though they allow a cost/benefit analysis to be conducted in due course, somatic markers allow the number of options under consideration to be reduced because 'emotions and feelings have been connected, by learning, to predicted future outcomes of certain scenarios. When a negative somatic marker is juxtaposed to a particular future outcome the combination functions as an alarm bell. When a positive somatic marker is juxtaposed instead, it becomes a beacon of incentive' (Damasio, 1996a, p. 173).

Despite deficiencies in moral reasoning, patients such as Elliot do not develop extremely amoral behaviour comparable to that of psychopaths, but Damasio hypothesises that psychopaths may demonstrate the features of future blindness, extreme violence, the incapacity to distinguish between the moral and the conventional, and display biological correlates such as reduced galvanic skin response, and hypofrontality as a result of a congenital defect in the somatic marker systems. Further studies with Elliot and other patients with ventromedial

frontal lobe damage have confirmed the combination of decision-making defect and flat emotion and feeling. Anderson and colleagues (1999) recently reported the cases of two adults who experienced prefrontal damage before sixteen months of age who had normal cognitive abilities but showed impaired social behaviour and defective social and moral reasoning comparable to that displayed by psychopaths. Damage to either the amygdala or the ventromedial prefrontal cortex results in impaired decision making, but those with amygdala damage are also unable to acquire conditioned skin conductance responses (a marker of somatic state) in response to reward or punishment. Bechara and colleagues (1999) report that all of their patients (ten with ventromedial prefrontal cortex damage and five with amygdala damage) were unable to develop anticipatory skin conductance responses when considering risky choice. In terms of an evolutionary hierarchy of survival mechanisms this is what one would expect. Damage to any of the components of a future detector, in this case a Popperian module subserving the creation of somatic markers, results in a deficient to ability to predict outcomes, especially social outcomes, but the specific pattern of deficits depends on the place that any sub-module occupies in the hierarchy of evolved mechanisms. Damage to more ancient components, such as Darwinian and Skinnerian modules, results in impaired biological functioning, and in impaired social functioning when those components serve as sub-components of Popperian or Gregorian modules.

Damasio's (1996b) evolutionary perspective reduces the emphasis placed on cognition, and on the brain more generally, so that the body 'is the driving force behind the creation, design, and maintenance of the brain' (Damasio, 1998). Chisholm notes that

> …to be fully Machiavellian – [is] to act strategically with regard to one's (body's) interests... In sum, human MI would seem to consist of (1) TOM (to explain and predict behaviour); (2) the prefrontal cortical capacity to inhibit behaviour (in order to set the stage for a valuable future); and (3) good connections between the prefrontal cortex and the amygdala (because what makes something valuable or not is subjective value experience, which involves the amygdala and the rest of the social brain). As Damasio argues… the emotional brain represents ("marks") the "body's interests" about which the evolutionarily recent prefrontal cortex was se-

> lected to make good decisions (i.e., to be rational)… (Chisholm, 1999, p. 130).

One serious deficiency of Chisholm's model integrating life history theory and developmental biology is that it predicts 'large differences in outcomes based on rather small changes to the assumptions and the parameters in a model' (Mace, 2000, p. 38), though this shortcoming is mitigated by the fact that many of his assumptions and predictions are open to empirical test. However, though Chisholm acknowledges that the production of many phenotypes from the same genotype can be an adaptive process, he forgets that any novel element within the developmental system can be the source of changes in the phenotype. Additionally, modules subserving more recently evolved functions are likely to be malleable in response to aspects of the environment for which no contingency exists in the genome.

Modules and Malleability

Bateson and Martin identify a number of processes capable of inducing psychological plasticity including 'social isolation, fasting, lowering blood glucose with insulin, physical discomfort, chronic fatigue and the use of disturbing lighting and sound effects' (1999, p. 189). Extreme fear and arousal also make individuals susceptible to radical changes in their beliefs and desires, though the biological link between stress and plasticity is unclear (Bateson & Martin, 1999, p. 191). Developmental modules responsible for setting the parameters of other modules, such as those involved in socioassessment, may be based on the same neuroendocrine mechanisms responsible for reconfiguring modules in response to important life events. Whereas the gonadal steroid hormones appear to be highly conserved regulators of sexual behaviour in a wide range of vertebrate taxa, the neuropeptides oxytocin (OT) and vasopressin (AVP) have a role in mediating species-specific sexual and social behaviour (Young, 1999). Both oxytocin and vasopressin act as signals in the central pathways involved in information processing (Ermisch, Landgraf & Mobius, 1986). Oxytocin is associated with changing connectivity within the brain and appears to facilitate reorganisation of the brain at important moments in the life cycle (Bateson & Martin,

1999, p. 194). Nancy Ostrowski has produced a model in which oxytocin plays a part in integrating and restructuring areas of the nervous system involved in 'steroid-sensitive reproductive behaviors; learning; and reinforcement' (Ostrowski, 1998). In particular oxytocin is involved in pair bonding in many species, and in social interaction in nonhuman primates (Winslow & Insel, 1991); its molecular structure may provide insight into the evolution of monogamy (Insel, et al., 1996). As an attenuator of memory oxytocin has been called the 'amnesic' neuropeptide, its effect being the opposite to that of vasopressin. Vasopressin participates in suppression of the immune system during stress (Shibasaki, et al., 1998); memory (Alescio-Lautier, Devigne & Soumireu-Mourat, 1987; Dietrich & Allen, 1997; Labudova, et al., 1998), in brain development (Boer, 1985), and in species-typical affiliative behaviour (Young, et al., 1999). In relation to psychopathology oxytocin may be involved in obsessive-compulsive behaviours (Insel, 1992; Insel & Winslow, 1992) and autism (Insel, 1997; Insel, O'Brien & Leckman, 1999). Individuals with bulimia nervosa have increased plasma and CSF levels of vasopressin (Demitrack, et al., 1992), but normal levels of oxytocin (Demitrack, et al., 1990).

In response to stress the group of neuromodulators called the catecholamines (dopamine, adrenaline and noradrenaline) act to prepare the heart and muscles for exertion, but also act on the brain to stimulate the amygdala and inhibit the prefrontal cortex. This gives priority to the phylogenetically older structures responsible for generating associations between stimuli and the emotions over structures mediating planned behaviour. Additionally catecholamine-induced activation of the amygdala stimulates the formation of declarative memories mediated by the hippocampus (Arnsten, 1998). Consequently short-term stress can enhance both conscious and unconscious memories of a stressful situation. The catecholamines thus have reciprocal effects on modules occupying different levels in the phylogenetic hierarchy: enhancing the operation of Darwinian and Skinnerian modules, but inhibiting the function of Popperian and Gregorian Modules. Prolonged stress can result in damage to the hippocampus and high levels of catecholamines in the prefrontal cortex cause cognitive dysfunction (Arnsten, 1998). An analysis of the different responses of cognitive-emotional modules according to their role and position in the hierarchy may help us to un-

derstand why prefrontal cortex deficits feature prominently in disorders related to stress, particularly the affective disorders, schizophrenia and post-traumatic stress disorder.

It seems likely that the initial parameters of modules can be set by neuroendocrine mechanisms in a way that may have long-lasting effects on functioning, and that similar neuroendocrine mechanisms can reconfigure modules according to expectable life events and novel features of the environment. Modules occupying different levels in the hierarchy of survival functions are likely to differ in their malleability, and in their response to the same neuromodulators.

Conclusion

In this chapter I have used developmental systems theory, a modular perspective on the evolution of psychological mechanisms, and life history theory to present a view of the mind as composed of mindless agents. These agents are integrated in hierarchies and heterarchies in order to balance the competing demands of functions dictated by basic survival needs and functions dictated by the need for social and mating success. Agents (or modules) display constrained ecological, developmental and cultural plasticity compatible with the requirement that psychological functioning should be configured to local conditions. The connections between these modules may be highly asymmetric, and most of their processes may be inaccessible to consciousness. In the following chapter I will develop these ideas within the context of evolutionary psychopathology.

Chapter 6

Evolutionary Developmental Psychopathology

> Since all aspects of the phenotype are products of ontogenesis, they are in some sense acquired. Means (developmental interactants) are inherited, results ("natures") are acquired by construction. A reproductively successful organism passes on the pertinent environment in many ways. This, to a large extent, is what it *means* to be reproductively successful, and it involves much more than having the "right" genes.
>
> (Oyama, 1985, p. 125)

> When the wrong question is being asked, it usually turns out to be because the right question is too difficult. Scientists ask questions they can answer. That is, it is often the case that the operations of a science are not a consequence of the problematic of that science, but that the problematic is induced by the available means.
>
> (Lewontin, 2000, p. vii)

Introduction

Before proceeding to an assessment of a number of mental disorders from the viewpoint of evolutionary developmental psychopathology as I have characterized it, I will summarize some of the main ideas evaluated so far. In chapter two I considered the 'separation of contradictory things' or the allocation of casual co-determinants to exclusive (and usually antagonistic) frameworks of explanation (or worldviews), and suggested that three damaging dichotomies could be avoided through an analysis based on the approach to evolution by natural selection known as developmental systems theory. In chapter three I assessed the current scheme of classification in psychiatry and highlighted its main deficiencies through an overview of the historical development of theories based on the neurochemical individuation of traits and disorders; the influence of tradition; of socio-political advocacy; and the incompatible needs of research scientists and clinicians. The causal homeostatic theory of natural kinds, which seeks to identify projectable categories at different (but mutually compatible) levels of analysis, was recommended as the foundation of good classification. In chapter four I outlined some of the main developments in contemporary biological

thought, including the concepts of inclusive fitness (kin selection), reciprocal altruism, gene selectionism or 'selfish gene' theory, sexual selection, parental investment, parent-offspring conflict, evolved psychological modules (domain-specific adaptations or agents), and mismatch theory.

In chapter five I looked at the role of the strategic (or 'higher' cognitive) emotions as a solution to the commitment problem; and advocated the view of emotions and cognitions as complimentary components of our evolved decision-making systems. I also looked at some of the evidence for the existence of multiple systems that have been fashioned by natural selection for their contribution to problem solving in our ancestral environment, and at empirical and theoretical reasons for accepting the modular view of the mind. I argued that modules participate in hierarchies and heterarchies in which there is no master control module, Cartesian Theatre, or central repository of general plasticity, and that together modular systems constitute the 'society of mind'. I also outlined some of the evidence suggesting that because modular systems subserve different functions, some related to basic survival and others related to social functioning, and because they occupy different levels in the hierarchy, they may have different responses to the same neurochemicals. I also advocated the following: the connections between modules may be highly asymmetric; modular systems may retain some plasticity allowing them to adapt to changing ecological, developmental, and social circumstances, though the parameters of some systems may be constrained early in development, especially during the attachment process. Evolutionary theory, life history theory, and developmental psychology/lifespan psychology can provide one coherent perspective on the ontogeny of modules. Systems capable of inducing plasticity function across the lifespan. As more recent systems are constructed on top of and out of more phylogenetically ancient modules homologous systems in other species (such as the fear and memory systems studied by LeDoux) can inform our theories of psychological functioning at any level, including that of 'higher' cognition. Studies of interindividual commonalities, interindividual differences, and intraindividual plasticity can all contribute to our theories, and therefore cross-cultural studies and studies of pathology, including psychopathology, are always relevant to the construction of hypotheses about our psychological mechanisms. It

is likely that much of the information processed by our modular systems is inaccessible to consciousness. Changes (or differences) in function should be taken to imply changes (or differences) in form as this will induce us to consider the possibility that modular systems are polymorphic, sexually dimorphic, and subject to change across the lifespan. Many of the components of modular systems may participate in more than one functional system, and systems may demonstrate considerable redundancy. Psychological functioning is mediated by modular systems and not by neurochemicals, and therefore traits and disorders cannot be neurochemically individuated. Our modular systems are not localized to a particular area in the manner envisaged by phrenology, but the distributed components participating in any function may map fairly reliably (in terms of statistical generalization) across individuals who display the same traits. Evolutionary psychology and evolutionary developmental psychopathology are not concerned primarily with behaviour but with the evolution, function, and dysfunction of the mechanisms that subserve psychological processes and behaviour. An important assumption is that because of mismatch current psychological functioning and behaviour may differ from that in the ancestral environment. Accordingly, cross-cultural studies should help to illustrate the degree of adaptive plasticity inherent in modular systems.

How to Proceed with the Investigation and Classification of Disorders

If we accept that our current schemes of classification are not only an unreliable guide to the nature of psychopathology, but an impediment to investigation, how are we to extract anything of value from the vast literature in psychiatry, psychology and related fields? First of all, we should adopt the theory driven approach of evolutionary developmental psychopathology, which will help us to interpret and synthesize existing findings, if the assumptions outlined so far are broadly correct, and above all we should remember that adaptations were forged to function in past environments and not necessarily in our current environment. Secondly, we should include in our investigations brain-damaged patients (who are often excluded from current research) as an analysis of pathology will help us to map psychological functions on to brain systems (Frith, 1992, p. 8). Thirdly, we should investigate not only behavioural abnormalities but in-

formation-processing abnormalities, in a scheme that acknowledges both cognition and affect as components of information processing. Fourthly, we should concentrate our investigations on specific signs and symptoms, rather than syndromes, as symptoms such as delusions and hallucinations, for example, are observed in patients who currently fall into a number of categories, including schizophrenia and affective psychosis (Frith, 1992, p. 9). Fifthly, we should expect that complex psychological processes should be broken down into simpler tasks that can be performed by the mindless agents in our 'society of mind'. Finally, we should be particularly attentive to any data showing sexual dimorphism and changes in psychological functioning and neural architecture across the lifespan, and to comparisons between adults, adolescents, and children. The remainder of this chapter will examine the applicability of this framework to existing findings in psychopathology. Although the evolutionary approach should inform the whole of psychopathology I will concentrate on those findings that illustrate most vividly the ideas discussed so far. I will also suggest a number of original hypotheses that enable us to integrate results from a range of research.

The Theory of Mind Module and Psychopathology

Perhaps our most distinctive attribute is the capacity to manage highly complex social interactions. As Sanjida O'Connell explains 'we do not interact with other people by looking at how they behave, rather, we think about what they are thinking and respond to them on that basis' (1997, p. 2). How do we establish reciprocal relationships, avoid (or initiate) confrontations, find mates, and establish our social roles? How does our capacity to engage in these activities develop and change over the lifespan? Does the development of social cognition relate to the changing balance between the need to allocate resources to survival, development, and reproduction? Using Tinbergen's framework we should ask: what are the mechanisms of social intelligence? How do they develop? How do they function? How did they evolve? When we are equipped with a knowledge of the mechanisms of social intelligence we will be better placed to investigate the nature and causes of its dysfunction as this relates to psychopathology.

Working within the modular framework Simon Baron-Cohen (1995) has elaborated a model of the evolution and development of ‘mindreading’. Baron-Cohen argues that we automatically and often unconsciously interpret human behaviour in terms of beliefs, desires and intentions through the operation of the adaptive cognitive mechanisms comprising the theory of mind module, and that children with autism (Kanner, 1943) suffer from ‘mindblindness’ as a result of an impairment of this module. The theory of mind module, or mindreading mechanism, is also referred to as a component of ‘Machiavellian intelligence’ (Byrne & Whiten, 1997; 1988) or social cognition (Adolphs, 1999). Baron-Cohen identifies four different mechanisms comprising the human mindreading system, the Intentionality Detector (ID), the Eye Direction Detector (EDD), the Shared Attention Mechanism (SAM) and the Theory of Mind Module (ToMM). These components roughly reflect four properties of the natural environment: volition, perception, shared attention and epistemic states.

The Intentionality Detector

The first component of the mindreading system is ID, the Intentionality Detector. This is ‘a perceptual device that interprets motion stimuli in terms of goal and desire’ (Baron-Cohen, 1995, p. 32) and which preferentially attends to stimuli exhibiting self-propulsion and direction. This most basic component of mindreading can take input from any modality (vision, touch, audition etc.) and from stimuli with hugely differing morphology and structure. It is, therefore, as easy for us to attribute intentionality to an insect, or a cow, as it is to a human being, and for us to mistakenly attribute intentionality, albeit briefly, to such things as pieces of paper blowing in the wind, or to collections of pixels making up computer sprites. The amodal property of ID is apparent in our capacity to attribute intentionality to tactile, auditory and other stimuli. Even young infants are sensitive to changes in an adult’s goal, for example, they respond to the distinction between a give and a tease (Reddy, 1991). In a classic study adults were found to explain the movement of geometrical shapes in a short film in terms of goals (Heider & Simmel, 1944), and this result has been repeated with children (Dasser, Ulbaek & Premack, 1989).

The Eye Direction Detector

This perceptual device has three basic functions: 'it detects the presence of eyes or eye-like stimuli, it computes whether eyes are directed toward it or toward something else, and it infers from its own case that if another organism's eyes are directed at something then that organism sees that thing' (Baron-Cohen, 1995, pp. 38-39). Both ID and EDD form dyadic representations, in the case of ID representations involving goal and desire ('Her *goal* is to go over there', 'It *wants* to get the cheese'), in the case of EDD those representations involving visual perception ('It *sees* me', 'Mummy *sees* the door'). These representations are termed dyadic because they describe intentional, or mentalistic, relations between two objects, either Agent and Object, or Agent and Self, and the mechanisms underlying them form the basis of an autistic universe, one in which agents and objects and the relations between them can be observed, but in which these observations do not form the basis of shared attention, which is the domain of a third component of the mindreading system. Both ID and EDD provide input for this third mechanism, the Shared Attention Mechanism.

The Shared Attention Mechanism

The function of the Shared Attention Mechanism is to form triadic representations, which is the representation of a triadic relation. Triadic representations specify the relations among an Agent, the Self and an Object (or another Agent) and can be expressed in the following form:

[Agent/Self-Relation-(Self/Agent-Relation-Proposition)]

For example,

[Mummy-sees-(I-see-the bus)]

These examples are taken from Baron-Cohen who notes that 'this attempt at formalism is useful because it brings out that a triadic representation contains an embedded dyadic representation' (Baron-Cohen, 1995, p. 45). SAM is like a

comparator in that it can fuse ‘dyadic representations about another’s current perceptual state and dyadic representations about the self’s current perceptual state into a triadic representation’ (Baron-Cohen, 1995, p. 46). SAM has a privileged relationship with EDD in that triadic representations are generally formed through the perception of eye direction, but SAM also makes the input from ID available to EDD so that eye direction can be read in terms of an agent’s goals or desires.

The Theory-of-Mind Mechanism or Module (ToMM) was first proposed by Alan Leslie (1994) as a system for inferring the full range of mental states from behaviour and has been adopted by Baron-Cohen, who notes that

> …the other three mechanisms have got us to the point of being able to read behaviour in terms of *volitional mental states* (desire and goal) and to read eye direction in terms of *perceptual mental states* (e.g., see). They have also got us to the point of being able to verify that different people can be experiencing these particular mental states about the same object or event (shared attention). But a theory of mind, of course, includes much more. (Baron-Cohen, 1995, p. 51).

In particular we need two additional things: the capacity to represent the complete range of epistemic mental states, and ‘a way of tying together all of the mental-state concepts (the perceptual, the volitional and the epistemic) into a coherent understanding of how mental states and actions are related’ (Baron-Cohen, 1995, p. 51). One of these requirements, that of representing epistemic mental states, is achieved through ToMM’s capacity to form M-Representations. These are representations of propositional attitudes that take the form:

[Agent-Attitude-‘Proposition’]

For example,

[Ian-believes-‘it is raining’]

ToMM may begin to emerge between 18 and 24 months as this period generally marks the onset of pretend play and

> …infants become able to construe the behaviour of other Agents as relating to fictional states of affairs, specifically, as issuing from the attitude of pretending the truth of a proposition that describes a fictional state of affairs. For example, a mother's actual behaviour of talking to a banana can be understood by constructing the M-representation, **mother pretends** (of) **the banana** (that it is true that) '**it is a telephone**'. This links her behaviour, via an attitude, to a fiction (Leslie, 1994, p. 141, emphasis in the original).

It is important to note that through M-representations ToMM can confer a key property of epistemic states, that of referential opacity (or non-substitutability) thus suspending the normal truth relations of propositions. Leslie explains:

> …the reference of terms in such embedded propositions becomes opaque (Quine, 1961). For example, "the prime minister of Britain" and "Mrs. Thatcher" refer at this time of writing to the same person. Therefore, anything asserted about the prime minister of Britain, if true, must be true of Mrs. Thatcher as well (and, likewise, false for one, false for the other). If it is true that the prime minister of Britain lives at No. 10 Downing Street, then it must be true that Mrs. Thatcher lives at No. 10 Downing Street. But put this proposition in the context of a mental state term and this no longer holds. Thus, "Sarah-Jane believes that the prime minister of Britain lives at No. 10 Downing Street" in no way entails the truth (or falsehood) of "Sarah-Jane believes Mrs. Thatcher lives at No. 10 Downing Street". In a mental state context one can no longer "look through" terms to see what they refer to in deciding such issues. The mental state term suspends normal reference relations. Quine (1961) called this *referential opacity* (Leslie, 1987, p. 416).

Hence the statement 'Snow White *thought* the woman selling apples was a kind person' can be true, while 'Snow White *thought* her wicked stepmother was a kind person' may be false (Baron-Cohen, 1995, p. 53).

Tying the Four Mindreading Mechanisms Together

Baron-Cohen suggests that ToMM receives inputs from ID and EDD via SAM because SAM's triadic representations have a relation slot that can take attitude terms and thereby be converted into M-representations.

Triadic representation: [Agent/Self-Relation-(Self/Agent-Relation-Proposition)]

M-representation: [Agent-Attitude-“Proposition”]

Therefore ToMM cannot develop without a functioning shared attention mechanism. The ontogeny of these mechanisms can be summarised as follows (Baron-Cohen, 1995):

Phase	Mechanism	Age	Representations
I ‘Primary Intersubjectivity’	ID Basic functions of EDD	Birth to 9 months	Dyadic
II ‘Secondary Intersubjectivity’	SAM	9 to 18 months	Triadic
III	ToMM	18 to 48 months	M-representations

In 1985 Simon Baron-Cohen, Uta Frith and Alan Leslie proposed that the three principal features of autism – abnormalities in social development, in the development of communication, and in pretend play – could arise through a failure in the development of mindreading. Since then a range of experimental results has confirmed that though ID and EDD appear to be functioning normally in autism, the shared attention mechanism does not.

> In most children with autism, SAM does not appear to be working through any modality - vision, touch, or audition. By and large, they bring an object over to someone, or point an object out, or lead someone to an object and place the person’s hand on it, only when they want the person to operate that object or get it for them. This is not shared attention in any sense; these behaviours are primarily instrumental, and do not indicate a desire to share interest with another person for its own sake (Baron-Cohen, 1995, p. 69).

This deficiency in SAM precludes the development of ToMM and therefore autistic children should be deficient in the understanding of false belief.

Is there evidence for the hypothesis that ID and EDD remain intact in autism whilst SAM is dysfunctional and that this results in deficiencies in the perception and understanding of epistemic states? Autistic children do use the word ‘want’ in their spontaneous speech (Tager-Flusberg, 1989; 1993) and in describing picture stories involving agents (Baron-Cohen, Leslie & Frith, 1986). They can

distinguish animacy, and understand that desires can cause emotions (Baron-Cohen, 1991b; Tan & Harris, 1991). They can detect when someone in a photograph is 'looking at them' (Baron-Cohen, et al., 1995) and interpret eye direction in terms of someone's 'seeing' something. Autistic children also use the word 'see' spontaneously (Tager-Flusberg, 1993) and can work out what someone else is looking at (Baron-Cohen, 1989b; Baron-Cohen, 1991a; Hobson, 1984; Tan & Harris, 1991). The evidence does suggest that ID and EDD remain intact. However, all of the evidence collected to date does show 'a massive impairment in the functioning of SAM in most children with autism' (Baron-Cohen, 1995, p. 66).

> Children with autism often do not show any of the main forms of join-attention behaviour. Thus, they do not show gaze monitoring (Leekam, et al., 1993; Loveland & Landry, 1986; Mundy, et al., 1986), nor do they show the related behaviours of attempting to direct the visual attention of others by using the pointing gesture in its "protodeclarative" form (Baron-Cohen, 1989b; Curcio, 1978; Mundy, et al., 1986). This is not because they cannot point at all – they do use the pointing gesture for some other, non-joint attentional functions, such as to request objects that are out of reach (Baron-Cohen, 1989b) and to identify different items in an array, for themselves (Goodhart & Baron-Cohen, 1993). And not only is the protodeclarative pointing gesture missing in young children with autism, but so are other declarative gestures, such as the showing gesture (which young normal toddlers use simply to show someone else something of interest (Baron-Cohen, 1995, p. 66)

Given that SAM is deficient in autism, is there evidence of a consequent incapacity in ToMM resulting in the failure to appreciate the epistemic mental state of belief?

The primatologists Premack and Woodruff (1978) first introduced the idea of 'theory of mind' as the ability to explain and predict the behaviour of intelligent agents in a paper considering the existence of mentalizing abilities in chimpanzees. The philosopher Daniel Dennett (1978) suggested that in the case of humans this ability might best be evaluated by investigating a child's capacity to understand that someone might hold a false belief. This idea was developed by Wimmer and Perner (1983) who came up with a false belief test and found that

normal children could pass it by the age of 3 or 4. The test was adapted for use with autistic children by Simon Baron-Cohen, Alan Leslie and Uta Frith (1985).

> The test involves seeing that Sally puts a marble in one place, and that later, while Sally is away, Anne puts the marble somewhere else. The child needs to appreciate that, since Sally was absent when her marble was moved from its original location, she won't know it was moved, and therefore must still believe that it is in its original location (Baron-Cohen, 1995, p. 70)

In other words, the child must understand that, whilst the proposition 'the marble is in its original location' is false, the M-representation [Sally *thinks* 'the marble is in its original location'] is true. Most autistic children fail this test, a result that has been replicated many times (Baron-Cohen, 1989a; 2000; Baron-Cohen, Leslie & Frith, 1985; Leekam & Perner, 1991; Leslie & Thaiss, 1992; Reed & Peterson, 1990) . Autistic children also fail a theory of mind task called the 'Smarties Test'. After having been shown that a Smarties tube actually contains pencils most autistic children predict that a new observer will also think that the tube contains pencils (Perner, et al., 1989). As Baron-Cohen concludes 'the robustness of this finding suggests that in autism there is a genuine inability to understand other people's different beliefs' (1995, p. 71). Sanjida O'Connell explains,

> It is only after the age of five that children can refer to the brain as an organ for thinking and talk about its mental functions, such as dreaming, remembering and imagining. Autistic children have no idea that the brain is used for thinking. To them it is an organ like any other. When asked what the brain does, they say things such as. "It makes you move". Uta Frith once conducted an experiment on reading with some autistic children. When one child did particularly well, she asked quite by accident, "Oh, how did you know that?" He replied, "By telepathy." (O'Connell, 1997, pp. 98-99)

Some evidence has shown that the theory of mind deficit is not a core cognitive deficit in autism, because some high functioning individuals pass second-order false belief tests. However, it is unlikely that these studies reveal a fully intact theory of mind in these cases. Some have considered second-order tests to be high-level tests of theory of mind, but whilst they do test for abilities beyond that for which first-order tests probe (those that can be passed by normal children at

four years of age, and in which the subject has to infer the beliefs of another person), these tests still only probe for the typical skills of 6-year-old.

The ‘levels’ or ‘orders’ referred to in theory of mind tests are levels of intentionality and normally we cope happily with three levels of intentionality (O'Connell, 1997, p. 7) and find anything above five levels extremely difficult. Sanjida O’Connell has a delightful example of levels of intentionality

> In the film, *The Lion in Winter*, Peter O’Toole plays Henry II and Katherine Hepburn his estranged wife, Eleanor of Aquitaine. The two of them are plotting against each other as to which of their three sons should inherit the throne. Henry says of Eleanor, “She knows I want John on the throne and I know she wants Richard. We’re very frank about it.” Which leaves the third son Jeff, who is equally frank. In a brilliant exposition of levels of intentionality, Jeff says, “I know. You know I know. I know you know I know. We know Henry knows and Henry knows we know it. We’re a very knowledgeable family.” After Jeff has left the scene, Eleanor pithily sums him up, “He’ll sell us all you know. But only if he thinks we think he won’t” (O'Connell, 1997, p. 117).

The Theory of Mind Mechanism and Schizophrenia

Though it is simple to discover similarities between conditions at an unhelpful level of generality, the key approach of cognitive neuropsychology is to identify fundamental deficits and explain these in terms of ‘a similar underlying dysfunction in the processing of information and, underlying this, a similar neurophysiological dysfunction’ (Frith & Frith, 1991, p. 66). The term ‘autism’ was originally coined in 1911 by Eugen Bleuler to characterise the social impairment that seemed characteristic of schizophrenia, and new work by Chris Frith and others has sought to establish similarities between the two disorders (Frith & Frith, 1991). Schizophrenia was long considered to be a neurodegenerative disease, but the failure to find the gliosis consistent with this hypothesis suggests that this is not the case (see particularly Heckers, 1997). The brains of some schizophrenics demonstrate gliosis (a sort of neural scar tissue) but most do not (Roberts & Bruton, 1990), and even brains with enlarged ventricles may show no sign of gliosis (Bruton, et al., 1990). Frith concludes that:

> On the basis of these results it is currently believed that the brain abnormality associated with schizophrenia occurs very early (e.g., before birth) and reflects a neurodevelopmental disorder (Murray & Lewis, 1987), that is "a disorder in which early, fixed pathology becomes manifest clinically during the normal course of the maturation of the brain" (Breslin & Weinberger, 1990). This idea fits in well with the assumption of a genetic basis, but does not exclude other biological causes that affect early development (Frith, 1992, p. 24).

Given that some types of autism and schizophrenia appear to be neurodevelopmental disorders can deeper parallels be drawn between them?

The negative symptoms in schizophrenia are those which are abnormal by their absence. These include poverty of speech, flattening of affect, retardation and social withdrawal. The positive signs are those things that are abnormal because of their presence in the clinical picture. These include hallucinations delusions, and incoherence of speech. Frith has suggested that some schizophrenics lack awareness of their own mental states and those of others, resulting disordered goals and intentions (1994, p. 151). Schizophrenia differs from autism in that it can be a (relatively) transitory disorder which generally affects people after puberty, but there are aspects of schizophrenia congruent with the notion of mindblindness. How would the world look during a sudden loss in the capacity to interpret behaviour in mentalistic terms?

> People would seem wooden, actors without real emotions (derealisation). In extreme cases, we might even think that our loved one had been replaced by a robot, as the creature did not have real mental states (Capgras syndrome). Likewise, if we could no longer "read" our own mental states then we would feel ourselves to be unreal (depersonalisation). If we found it so difficult to read other people's intentions we might conclude that this was a deliberate ploy; that people were deliberately disguising their intentions in order to gain some secret end. This could be the basis of a paranoid belief in a general conspiracy. This would apply particularly to people we knew well. As in these cases we would have gained some facility in reading their intentions….I propose then, that certain delusions can be explained as the consequence of losing the ability to "read" the intentions and beliefs of others. This can be seen as the most minor of a sequence of failures in "theory of mind" mechanisms (Frith, 1994, pp. 152-153)

Frith proposes that the positive symptoms of schizophrenia are caused by a disruption of the capacity to form M-representations, that the proposition (e.g., 'it is raining') becomes detached from the attitude (e.g., 'Ian believes') and that the content is perceived as a representation of the real world. The following table appears in Frith (1994, p. 154)

Normal proposition	**Detached content**	**Abnormal experience**
I know that 'my car is faulty'	My car is faulty	Thought insertion
I intend to 'make a cup of tea'	Make a cup of tea	Delusion of control
Eve thinks 'Chris drinks too much'	Chris drinks too much	Third-person hallucination

The clinical picture is likely to be very varied, owing to the (often relatively) transitory nature of symptoms and to a wide variation in the degree of deficit experienced between individuals and by one individual over time. The developmental stages identified in the study of autism (1) awareness of our goals (2) awareness of our own intentions and other mental states; and (3) awareness of other people's mental states can be identified with different classes of schizophrenic signs and symptoms (Frith, 1994, p. 156):

Loss of awareness of	**Positive features**	**Negative features**
Own goals	Grandiose ability	Depersonalisation lack of will
Own intentions	Delusions of control thought insertion	Poverty of thought loss of affect
Others' intentions	Delusions of persecution third person hallucinations	Derealisation social withdrawal

Frith's tripartite model, which postulates that the signs and symptoms of schizophrenia relate to dissociations reflecting the ontogeny of the mindreading mechanisms that are deficient in autism has received some empirical support. Corcoran and colleagues compared 55 patients with a diagnosis of schizophrenia with two groups of control subjects, first a group of 30 normal controls, and second a group of 14 psychiatric control patients during the performance of a newly-devised task examining the capacity to infer intentions behind indirect

speech. The responses of the two control groups were very similar and these were combined to create a single control group.

> Problems performing the… task were seen in patients with negative features and in those with paranoid delusions and related positive features. There was also limited support for the argument that patients with incoherent speech are poor at inferring the intentions behind indirect speech (Corcoran, Mercer & Frith, 1995, p. 10)

Patients suffering from passivity experiences and those in remission had no difficulty with the task.

> According to the model, these patients [with passivity experiences] have a representational disability involving the monitoring of their own intentions to act (Frith & Done, 1989). It is intriguing that these patients were perfectly capable of inferring the intentions of others from indirect speech in the present study. This suggests that these two skills are dissociable (Corcoran, Mercer & Frith, 1995, p. 10).

In a second study Frith and Corcoran studied mentalizing ability in 46 symptomatic schizophrenic patients as compared with 44 non-symptomatic controls. The subjects ‘heard six stories and simultaneously were shown simple cartoon pictures depicting the action sequencing occurring in the stories. All of the stories involved false belief or deception, so that it was necessary to infer the mental states of the characters in order to understand their behaviour (Frith & Corcoran, 1996). Those patients with paranoid delusions were impaired on the theory of mind tasks, but others manifesting negative features or incoherence had difficulties associated with memory and not mental state questions. Those with delusions of control and those in remission did not differ from normal controls. ‘These results are consistent with the hypothesis that certain of the positive symptoms of schizophrenia reflect an impairment in the ability to infer the mental states of others’ (Frith & Corcoran, 1996, p. 521). In commenting on both of these studies Frith notes that

> My colleague Rhiannon Corcoran has carried out a series of studies in which schizophrenic patients performed various “Theory of mind” tasks, some of which were derived from the autism literature

> (Corcoran, Mercer & Frith, 1995; Frith & Corcoran, 1996). The results of these studies suggest that patients with negative features perform worse on “Theory of mind” tasks than would be expected on the basis of their current IQs. There is also some evidence, though less strong, that patients with delusions about the intentions of other people (e.g., delusions of persecution and delusions of reference) perform “Theory of mind” tasks badly. Patients currently in remission have no problems with the tasks suggesting that this is a state, rather than a trait variable (Frith, 1996, p. 1512).

However Walston, Blennerhassett, and Charlton (2000), located four male schizophrenics between the ages of 32 and 43 whose symptoms appeared to be pure cases of persecutory delusions encapsulated to a specific group of persecutors with hostile *intenti*ons. These men were free from other detectable pathology in their reasoning processes, affect, and social interactions and showed no deficit on theory of mind tests. The authors of this study also noted that the content of these delusions ‘is consistent with the nature of hostile threats to men in the ancestral human environment’. In a previous study Walston, David, and Charlton (1998) reported sex differences in the content of persecutory delusions consistent with the idea ‘that men would tend to identify physically violent gangs of strangers as their persecutors, while women would tend to identify their persecutors as being familiar females whose persecution took the form of social exclusion and verbal aggression’ (1998, p. 257). Of the female cases studied 73 percent identified familiar people as their persecutors, while 85 percent of the men identified strangers.

Frith and Corcoran have clearly demonstrated theory of mind deficits in many of those diagnosed as schizophrenic, and in some patients with negative features these deficits are similar to those demonstrated by patients diagnosed as autistic. These patients ‘had a tendency to fail to recognise hidden intentions and false beliefs and tended not to use mental-state language in their explanations’ (Corcoran, 2000, p. 396). However, many of these patients also had general cognitive deficits in areas such as memory and language pragmatics, which may be responsible for the deficit in theory of mind. On the other hand, many patients with positive symptoms of formal thought disorder ‘tended to give bizarre misinterpretations… which did not appear to lack mental-state terminol-

ogy' and performed poorly on theory of mind tasks whilst manifesting symptoms, but not on recovery. Those with positive symptoms characterised as paranoid delusions also had theory of mind problems 'but the difficulty was not as grave [as those with negative symptoms or autism]. When these patients failed, the tendency was to fail to recognise hidden intentions or false beliefs and not to use mental-state language' (Corcoran, 2000, p. 397). Overall, patients with positive symptoms appeared to be 'cognitively intact' and to have specific deficits not related to memory impairments or other cognitive deficits, and for those patients in remission ToM skills returned to normal. Corcoran concludes: 'what is stressed in the schizophrenia literature is that the core deficit may lie in the use of previously acquired information and/or within the reasoning domain. In autism it is generally, though not universally, held that the theory of mind deficit is highly selective and independent of other cognitive skills' (Corcoran, 2000, p. 405). Although the deficits uncovered do not support Frith's explanation of schizophrenia it is clear that many of those categorised as 'schizophrenic' do have problems with theory of mind, and that some patients are more comparable to those with autism than others.

Theory of Mind Deficits in Other Disorders

As a consequence of the work in autism and schizophrenia by Baron-Cohen, Frith and others theory of mind tests have been administered to those diagnosed with a range of other disorders. In a study of theory of mind and psychoses Doody and colleagues (1998) compared the performance of people categorised into five groups: non-psychiatric controls, affective disorder, schizophrenia with normal pre-morbid IQ, schizophrenia with pre-morbid IQ in the mildly learning disabled range, and mild learning disability with no history of psychiatric illness. They found that impaired theory of mind on second order tests is specific to schizophrenia compared to mild learning disability and affective disorder control groups, but that subjects with schizophrenia and pre-morbid mild learning disability show greater impairment than subjects with schizophrenia and a pre-morbid IQ within the normal range. As some patients diagnosed as suffering from affective disorder with a psychotic component often display a range of symptoms comparable to those observed in schizophrenia it is significant that

this group was not impaired on second-order theory of mind tests. Blair and colleagues (1996) reported no deficits in theory of mind in their study of twenty-five adult psychopaths, all of whom displayed skill in using appropriate mental-state terminology. Other studies have reported no deficits in those with Gilles de la Tourette Syndrome (Baron-Cohen & Robertson, 1995), and Conduct Disorder (Buitelaar, et al., 1999; Happé & Frith, 1996) and Dysthymia (Buitelaar, et al., 1999). Mentalising difficulties have been reported in patients diagnosed as suffering from Borderline Personality Disorder (Fonagy, et al., 1996; Fonagy, Redfern & Charman, 1997; Fonagy, et al., 1995; Fonagy & Target, 1996; 1998), but these problems appear to be associated with various types of mistreatment during childhood, which could have provided an incentive to minimize empathy and the use and appreciation of mentalistic concepts (Corcoran, 2000, pp. 408-9).

Two studies have reported second-order theory of mind deficits in subjects diagnosed with Attention Deficit Hyperactivity Disorder (Buitelaar, et al., 1996; 1999), though the earlier report was of a single case-study, and only ten subjects have been studied in total. However, in clinical groups matched person-to-person on age and verbal IQ nine children with ADHD and twenty with pervasive developmental disorder-not otherwise specified (PDD-NOS) performed as poorly as the autistic children, on a set of first- and second-order ToM tasks and for the matching and context recognition of emotional expressions.

The Neurobiology of the Theory of Mind Module

The question of whether ‘theory of mind’ is a separate function independent of executive processes has been a subject of some debate, and there has also been some dispute over the neural substrate of the theory of mind module. Fortunately, several important new studies have helped to clarify the situation. First of all, Happé, Malhi, and Checkley (2001) have reported the first case of acquired theory of mind deficit following a surgical procedure. The patient P.B. underwent a stereotactic anterior capsulotomy in which ‘lesions target neuronal connections between the mid-line thalamic nuclei and the orbito-frontal cortex, as they pass in the anterior one third of the internal capsule, between the head of the caudate nucleus and the putamen’ (2001, p. 85). Subsequent to this pro-

cedure the patient was impaired on tests requiring mental state attributions, and although he also showed impairments in executive functioning these did not appear to cause problems in dealing with tests where mental-state attributions were not required. Rowe and colleagues found distinct theory of mind deficits in thirty one patients with unilateral frontal lobe damage and found that these deficits were 'independent of non-mental state inferencing' and that 'within the context of this experimental design, the ToM deficit and the executive functioning deficits in patients with frontal lobe lesions are not causally related' (Rowe, et al., 2001, p. 614). However, they also concluded that the theory of mind module is instantiated in the frontal lobes, which is unlikely to be correct for reasons I will examine shortly. Fifteen of the patients (six males and nine females) had right frontal lobe lesions, and sixteen (eight males and eight females) had left frontal lesions, involving the dorsolateral, orbital and medial areas. This suggests that the frontal lobe components of the ToM module are distributed, or that different task demands co-opt additional areas. The latter is suggested by the study of Stuss, Gallup, and Alexander (2001) which detected impairment on a deception task with bilateral inferior medial damage. Stuss and colleagues conclude:

> That bilateral, particularly right, orbital/medial, lesions might impair patients' capacity to incorporate the experience of another's deceptions into their own plans is consistent with existing knowledge about damage to this region. Lesions in this area result in a failure to activate relevant somatic markers so that past emotional experience can be used to guide response options (Bechara, et al., 1997)… Our results identify the brain regions necessary for some components of a theory of mind… The frontal lobes are essential, with the right frontal lobe perhaps particularly critical, maybe because of its central role in the neural network for social cognition, including inferences about the feelings of others and empathy for those feelings. The ventral medial frontal regions are also important perhaps because connections with the amygdala and other limbic structures give them a key role in the neural network for the behavioural modulation based upon emotions and drives (Stuss, Gallup & Alexander, 2001, p. 284)

The conclusion of Stuss and colleagues is supported by Valerie Stone (2000) who has found that the components of the theory of mind module are distributed in a number of brain areas including the orbitofrontal cortex, medial frontal cor-

tex, dorsolateral frontal cortex, and the amygdala. Fine, Lumsden, and Blair (2001) have reported a case of an individual with specific damage to the lateral part of the basal nuclei of the left amygdala. This patient displayed impairment in theory of mind tasks, but his performance on all tests of comprehension, memory and executive functioning was normal to good. In a post-mortem study of six brains of individuals diagnosed with autism five showed increased neuron-packing density in basal and medial lateral nuclei of the amygdala, but in contrast five of the six showed no abnormality in the lateral nuclei (Bauman & Kemper, 1994). It is undoubtedly significant that the most substantial projection to the hippocampus originates in the basal nucleus (Pikkarainen, et al., 1999), and that this nucleus appears to be involved in memory consolidation, particularly during emotional arousal (Roozendaal, et al., 1999).

Baron-Cohen and colleagues (1999) have reported left amygdala activation during a task requiring the inference of mental state from a picture of the eyes (the 'Eyes Test') in an fMRI study. Individuals with Asperger's syndrome (now often designated as high functioning autism) who are impaired in theory of mind showed reduced activation of this region. Those with Asperger's syndrome often display high levels of intellectual ability and can pass second-order theory of mind tasks, but show impairment in more subtle adult-level tasks such as recognising gender from the eye region of the face, and recognising basic emotions from the whole face (Baron-Cohen, et al., 1997). Because of the centrality of the amygdala as a component of social intelligence Baron-Cohen and colleagues (2000) have presented an 'amygdala theory of autism' based on their fMRI study showing that subjects in the autism group activated frontal cortex to a lesser extent than a control group and demonstrated no activation of the amygdala at all. Baron-Cohen and colleagues suggest that the amygdala is essential for the identification of mental state from complex visual information. The autism group showed greater activation of on the 'temporal lobe structures specialized for verbally labelling complex visual stimuli and processing faces and eyes. This may arise as a compensation for an amygdala abnormality' (Baron-Cohen, et al., 2000, p. 360). In a study of nine adult patients Critchley and colleagues (2000) also found that high functioning individuals with autistic disorder

(used here to cover autism and Asperger's syndrome) do not activate the left amygdala region when implicitly processing emotional facial expressions.

Although dysfunction of the amygdala may represent a core neural deficit in autism, it is important to note that dysfunction in the other components thought to be involved in the theory of mind module have also been reported. Happé and colleagues (1996), for example, reported that normal controls accessed the left medial prefrontal cortex during a theory of mind task, but no task related activity was observed in a PET scan of five subjects with Asperger's syndrome, although they displayed normal activity in immediately adjacent areas. In a study of 23 autistic children Ohnishi and colleagues (2000) matched symptom profiles with regional cerebral blood flow and found altered perfusion in the medial prefrontal cortex and anterior cingulate gyrus to be related to deficits in theory of mind, and altered perfusion of the right medial temporal lobe to be related to an obsessive desire for sameness. In comparison with the control group decreases in regional cerebral blood flow were identified in the bilateral insula, superior temporal gyri and left prefrontal cortices. Thomas and colleagues (2001) have found predominantly left amygdala and substantia innominata activity during the presentation of fearful faces, but whereas adults showed increased left amygdala activity for fearful faces relative to neutral faces children showed greater amygdala activity with neutral faces than with fearful faces. For the children there was also a gender difference: boys but not girls showed less activity with repeated exposure to the fearful faces. However, as this was the first study to examine developmental differences in the amygdala response to facial expressions using functional magnetic resonance imaging the results are tentative, but I shall seek to demonstrate that age- and sex-related differences in functioning are likely to be central to our understanding of the evolution, development, and breakdown of the theory of mind module.

Dawn Bowers presented preliminary findings to the International Neuropsychological Society in February, 2001 demonstrating that although men and women are equally expressive, men display most of their joy, disgust or other sentiments in the lower left quadrant of their face. Women, on the other hand, were found to show their emotions across their entire countenance. Bowers be-

lieves that these data support the conclusion that the brains of men are more compartmentalised than those of women and that the emotional priming systems for men may be located in the right hemisphere but are more dispersed for women. Significantly, Van Strien and Van Beek (2000) have detected a positive emotional bias of the left hemisphere in women. On the other hand language functions seem to be concentrated in the left hemisphere of male brains, whereas in women they are more equally distributed across the brain (Shaywitz, et al., 1995). It is also notable that Harasty and colleagues (1997) have found the volume of the superior temporal cortex, expressed as a proportion of total cerebral volume, to be significantly larger in females, with the Wernicke and Broca language-associated regions proportionally larger than those of males. Broca's area in females was 20.4 percent larger than in males.

Emery and Perrett (2000) have studied the neurophysiology of social cognition in the macaque and have found that there are a variety of anatomical subregions and distinct cell populations in the anterior section of the superior temporal sulcus (STS) in the temporal lobe. These include cell populations involved in 'the visual appearance of the face and body while they are static or in motion'; 'particular face and body movements'; and 'face and body movement as goal-directed action' (Emery & Perrett, 2000, p. 285). There is also another cell type involved in coding 'movement which is not a predictable consequence of the monkey's own actions'. Tomasello, Call, and Hare (1998) have reported that five primate species, rhesus, stumptail, pigtail macaques, sooty mangebeys, and chimpanzees all utilise the direction of attention of conspecifics to orient their own attention. Tomasello, Hare, and Agnetta have found that chimpanzees follow the gaze directions of other animate beings, including humans, 'geometrically to specific locations' and do not simply turn in the general direction and try to find something interesting (1999, p. 769). Chimpanzees can also identify the emotional significance of the facial expressions of conspecifics (Parr, in press). Importantly, for this analysis of the neurobiology of ToM, the cell populations in the temporal cortex have been found to provide the visual specification of body and face signals to the amygdala through the basolateral nuclear complex:

> …the temporal cortex cells… can provide a window into the minds of others. They can, in principle, support an understanding of what other individuals are attending to, what they feel emotionally, what aspects of the environment cause these feelings, how others are interacting, and the goals of these interactions. Of course, and observer may not explicitly realise the feelings and plans of others; nonetheless the visual specification supplied by the temporal cortex allows the observer to capitalise on the minds and behaviour of others and to react in the most appropriate way. Provided the visual system can specify what others are doing, one need not understand intentions or be able to mind read in order to come up with appropriate behavioural reactions (Emery & Perrett, 2000, p. 297)

In their analysis of the distributed human neural system for face perception Haxby, Hoffman, and Gabbini (2000) identify a core system consisting of the inferior occipital gyri for early perception of facial features; the superior temporal sulcus for the changeable aspects of faces and the perception of eye gaze, expression and lip movement; and the lateral fusiform gyrus for the invariant aspects of faces and the perception of unique identity.

According to a magnetic resonance imaging study of 121 healthy children (all aged between 4 and 18) amygdala volume during development increases significantly more in males than in females, but hippocampal volume increases more in females. ‘These sexually dimorphic patterns of brain development may be related to the observed sex differences in age of onset, prevalence, and symptomatology seen in nearly all neuropsychiatric disorders of childhood.’ (Giedd, et al., 1997, p. 1185, also see Giedd, et al., 1996). Males also show greater age-related losses in the frontal and temporal lobes in the left hemisphere, whereas women show equal rates of decline in both hemispheres, with perhaps a small bias to the right (Murphy, et al., 1996).

Using a population-based sample of twins aged 5-17 Scourfield and colleagues (1999) have found a considerable genetic influence on the development of social cognition, and that males have poorer social cognition than females. Skuse and colleagues (1997) have discovered an X-linked imprinted locus affecting social cognition of which only the paternal copy is expressed. Males are substantially more vulnerable to a variety of developmental disorders, including au-

tism and language impairment. Skuse and colleagues conclude ‘our findings are consistent with the hypothesis that the locus described, which we propose to be silent in males… acts synergistically with susceptibility loci elsewhere on the genome to increase the male-to-female ratio of such disorders’ (Skuse, et al., 1997, p. 707). A separate study involving Skuse has also identified eight girls with Xp deletions, three of whom showed symptoms similar to autism (Thomas, et al., 1999). Fombonne (1999) reviewed twenty-three epidemiological surveys of autism published in the English language between 1966 and 1998 covering four million subjects. In these studies 1533 cases of autism were reported. The median prevalence rate across the surveys was 5.2 per 10,000 and the average male to female ratio was 3.8:1. The overall estimate for cases of all forms of pervasive developmental disorders was 18.7 per 10,000.

A Closer Look at Lateralized Responses in the Amygdala

As so many separate lines of enquiry covering humans, nonhuman primates, and other animals have identified an important role for the amygdala in social cognition I shall examine some of the more recent studies in more detail.

On the basis of a PET study of ten healthy subjects performing a recognition memory task with food and non-food items Morris and Dolan (2001) have concluded that the left amygdala and regions of the right orbitofrontal cortex subserve the integration of perceptual (food), motivational (hunger), and cognitive (memory) processes in the human brain. The fact that the degree of activity in the left amygdala during memory encoding is predictive of subsequent memory of emotionally intense scenes also suggests ‘that amygdala activation reflects moment-to-moment subjective emotional experience and that this activation enhances memory in relation to the emotional intensity of an experience’ (Canli, et al., 2000, p. 1). Damage to the left amygdala impairs memory for emotional stimuli, but leaves memory for neutral stimuli intact (Adolphs, Tranel & Denburg, 2001). During a study of subjects exposed to combat sounds activation of the left amygdala was detected only in those suffering from PTSD (Liberzon, et al., 1999). The left amygdala also appears to form part of the brain’s ‘deviance detection system’ as it has been found to be activated during the presentation of a

series of nouns only when an item in the series has discrepant emotional import (Strange, et al., 2000). In a visual encoding task involving the presentation of photographs of single faces and paired faces the left amygdala and hippocampus were observed using fMRI to be active only during paired face encoding, which suggests that these structures are involved in associative learning (Killgore, et al., 2000). A separate study showed that the left amygdala was activated in a face processing task only during exposure to unfamiliar faces (Dubois, et al., 1999).

The amygdala is involved in reward and punishment feedback in animals, and in humans in the comparable situation of winning and losing. In an fMRI study of participants engaged in a fictitious competitive tournament during which the frequency of positive and negative trials was parametrically varied by the experimenters independently from the subjects' actual performance and without their knowledge the parametric increase of winning was associated with left amygdala activation whereas the parametric increase of losing was associated with right amygdala activation (Zalla, et al., 2000). This suggests that the amygdala responds differentially to changes in the magnitude of positive or negative reinforcement. There is also differential activation dependent on the subject’s level of awareness of the stimuli with the left amygdala being activated during conscious processing and the right amygdala during unconscious processing (Morris, Öhman & Dolan, 1998). In rats greater serotonin concentration in the right versus the left amygdala is correlated with anxiety (Andersen & Teicher, 1999). Blood flow to the left amygdala has been found to increase during exposure to aversive odorants, and the degree of activity was significantly correlated with subjective assessment of perceived aversiveness (Zald & Pardo, 1997). It is undoubtedly significant that the left amygdala has been found to be smaller in depressed patients (von Gunten, et al., 2000), and patients with temporal lobe epilepsy and dysthymia (chronically depressed mood) have enlarged left and right amygdala volumes, with those of females being significantly larger than those of males (Tebartz van Elst, et al., 1999). Activity in the left amygdala increases during gaze monitoring, and in the right amygdala during eye contact (Kawashima, et al., 1999).

In summary, it seems reasonable to conclude that nuclei of the left amygdala are significantly involved in the cognitive-emotional assessment of reward and risk in the natural and social environments and with the long-term storage of memories based on these assessments. Any pathology affecting these nuclei is likely to be devastating to the functioning of a variety of key tactical and strategic modules.

How Does the Brain Read Minds?

The most reasonable hypothesis based on the studies discussed is that the distributed neural components of the theory of mind mechanism include the superior temporal sulcus, the amygdala, the medial prefrontal cortex and (possibly) the orbitofrontal cortex. This is compatible with the proposed neurobiological basis of social intelligence first articulated by Leslie Brothers (1990) and developed by Simon Baron-Cohen (1995). As Frith and Frith conclude,

> The physiological basis of one aspect of social cognition, theory of mind, is just beginning to be understood. Brain-imaging studies suggest that a network of areas linking medial prefrontal and temporal cortex forms the neural substrate of mentalizing, that is, representing one's own and other people's mental states. The medial prefrontal areas are prominent also in tasks that involve self-monitoring, whereas the temporal regions are prominent also in tasks that involve the representation of goals of actions (Frith & Frith, 2001, p. 151).

There are age- and sex-related differences in the development, maturation, and breakdown of these components, and in the degree to which these components are accessed during theory of mind tasks. Most of these important factors are not controlled for in studies of psychopathology. This fundamental flaw makes it extremely difficult to extract valuable data from most existing studies.

The Neurobiology of Schizophrenia and Related Disorders

In this section I shall outline briefly some of the most important studies that have highlighted pathology in the regions that have been identified tentatively as the location of sub-components of the theory of mind module.

In a study of a series of brains collected over 40 years ago from well-documented schizophrenic cases (the Vogt collection), it was found that the amygdala and the hippocampus were substantially and significantly decreased in volume in comparison with a control series. These studies were performed on single, primarily left, hemispheres (Reynolds, 1992). Reynolds reported the first finding of increased dopamine levels in the left amygdala in a postmortem study published in *Nature* almost twenty years ago (1983), and concluded on the basis of subsequent studies that 'the dopaminergic innervation of the amygdala provides a means of understanding the action of antipsychotic drugs in a disease with primarily temporal lobe pathology' (Reynolds, 1992, p.571). Falkai and Bogerts (1986) have found significant losses of nerve cells in the hippocampus, and abnormal orientations of pyramidal cells, and dendritic irregularities disrupting the normal synaptic pattern have been found in the hippocampus by Scheibel and Kovelman (1981), who suggest that these abnormalities represent a congenital, developmental disorder specific to schizophrenia. Damage to the hippocampus may result in schizophrenia-like symptoms. Torrey and Peterson (1974) have pointed out that tumours, infarctions, infections and traumas affecting the medial temporal lobe are often associated with symptoms similar to or indistinguishable from schizophrenia (Lantos, 1988). It is also notable that the hippocampus is particularly vulnerable to the hypoxia that can result from the kind of obstetric complications that have often been implicated in the aetiology of schizophrenia (Murray, et al., 1988). The hippocampus is involved in long-term declarative memory encoding (Alkire, et al., 1998), and appears to operate in concert with the amygdala when encoding information with emotional content. Male schizophrenics with hallucinatory symptoms display impaired recruitment of the hippocampus during conscious recollection (Heckers, et al., 1998). Patients with temporal lobe epilepsy can develop 'a schizophrenic-like state with prominent positive symptoms' (Strange, 1992, p. 253). Maier and colleagues (2000) have reported that patients with schizophrenia and patients with temporal lobe epilepsy and psychosis (but not those without psychosis) display volume reductions in the left hippocampus and amygdala. Bryant and colleagues (1999) found volume reductions in the superior temporal gyrus and the amygdala/hippocampal complex in male patients diagnosed with schizophrenia, but not in female patients. Reductions in left amygdala and hippocampus have

been reported in patients with schizophrenia and affective psychosis at first hospitalization, though those in the latter category showed no reduction in the left posterior superior temporal gyrus (Hirayasu, et al., 1998).

Pearlson and colleagues (1997) have reported finding that the left amygdala was smaller and that the right anterior superior temporal gyrus was larger in patients with bipolar disorder. A lesion specific to the left amygdala was found in a postmortem study of a case of chronic psychosis (Fudge, et al., 1997). Reduced volume in the left amygdala has also been reported in healthy children of schizophrenics (Keshavan, et al., 1997). Significantly, those taking MDMA (Ecstasy) in a PET study experienced psychological changes such as heightened mood, increased extroversion, slight derealisation and mild perceptual alterations, and difficulty in concentrating, and these changes were accompanied by increased regional blood flow in the ventromedial prefrontal cortex, left amygdala, cingulate cortex, insula and thalamus (Gamma, et al., 2000). In a study showing heterogeneity of functioning consistent with the modular analysis Evangeli and Broks (2000) found that in a test of social cognition some schizophrenics showed deficits associated with amygdala damage while others did not.

Although patients diagnosed with schizophrenia do not exhibit the same pathology (or pathology specific to schizophrenia) the principal brain changes found are ‘fairly specific neuronal reductions in certain temporal lobe regions such as the hippocampus, amygdala, and parahippocampal gyrus, and there is some evidence for altered frontal lobe (prefrontal cortex) function’ (Strange, 1992, p. 253). For an assessment of all 193 MRI studies conducted between 1988 and August 2000 see Shenton (2001). Birchwood, Hallett, and Preston have concluded that the data on schizophrenia suggest that there are at least three forms of the disorder. One form has a lower genetic risk and is characterised by predominantly negative symptoms at onset, poor pre-morbid functioning, a poor prognosis and poor response to neuroleptic medication, and prominent intellectual impairment, and is more often associated with males. A second form is characterised primarily by positive symptoms, is associated more often with females, and has a later onset, a better prognosis, a strong affective component,

and good pre-morbid adjustment. The third form shows a mixture of symptoms, and affects men and women equally. Those in this third category have a higher genetic risk, mainly positive symptoms at the outset, and are often in the younger age range. These patients respond well to medication, but have a very mixed pre-morbid history and prognosis. These categories are offered as rough guides in the search for more specific formulations that can be related, potentially, to environmental factors capable of raising the liability to schizophrenia throughout development (Birchwood, Hallett & Preston, 1989, pp. 325-327).

An interaction between the prefrontal cortex, amygdala, and nucleus accumbens seems to subserve the regulation of goal-directed behavior by affective and cognitive processes. In rats stimulation of the basolateral amygdala sufficient to cause mild behavioural activation causes dopamine release in the prefrontal cortex. The prefrontal cortex influences the behavioral impact of amygdala activation via the active suppression of dopamine release in the nucleus accumbens, and absence of this influence appears to result in an aberrant pattern of behavioral expression in response to amygdala activation, including the tendency to repeat responses to an experience in later situations where it is not appropriate. (Jackson & Moghaddam, 2001). It has also been found that depletion of dopamine in the medial prefrontal cortex potentiates the stress-evoked dopamine release in the nucleus accumbens shell. This system could be involved in the symptoms of schizophrenia and other disorders that are influenced by stress (King, Zigmond & Finlay, 1997). It has been known for many years that relapse rates are highest for those individuals living in stressful environments in which there is a high degree of expressed emotion (Leff, 1976). The medial prefrontal cortex also attenuates sensory-driven affective responses through the recruitment of inhibitory neurons in the basolateral nucleus of the amygdala that suppress sensory cortical inputs. In stressful situations, during which dopamine levels in the basolateral nucleus of the amygdala increase, regulation by the medial prefrontal cortex could be reduced resulting in a disinhibition of sensory-driven affective responses.

The dopamine agonist apomorphine attenuates inputs from the medial prefrontal cortex, whilst augmenting inputs from temporal area three of the sensory cor-

tex. (Rosenkranz & Grace, 2001). Presumably dopamine antagonists have the opposite effect and therefore decrease sensory-driven affective responses. This may explain their efficacy in reducing the positive symptoms of schizophrenia. Ironically, the dopamine D1 receptors that are more common in the prefrontal cortex are down-regulated as a result of treatment with many common antipsychotics. In a study using nonhuman primates Lidow, Elsworth, and Goldman-Rakic (1997) administered eight different drugs at therapeutic doses for six months and found that all of them down-regulated the levels of both D1 and D5 mRNAs in the prefrontal cortex by 30 percent to 60 percent compared with a control group. I conclude that, while some patients with primarily temporal lobe pathology and positive symptoms may be helped by the dopamine antagonists usually administered in the treatment of psychotic disorders, those with neurodevelopmental impairment of the prefrontal cortex and primarily negative symptoms may suffer additional damage. The course and outcome of schizophrenia are both better in the developing ('Third World') than in the developed world, despite the much greater availability of resources and health care in the latter, and though some have speculated that the explanation of this strange phenomenon rests in variations in the distribution of genetic and environmental risk factors (Jablensky, 2000), it could be that many in the developing world simply escape the damage inflicted by inappropriate administration of substances capable of altering the distribution of neurochemicals and receptors in the brain.

Mice lacking the *tailless* gene protein product show a reduction in the size of the limbic structures including the amygdala, both males and females are more aggressive than usual, and females show no maternal instincts (Monaghan, et al., 1997). This indicates that the morphology of these important structures can be influenced by genetic factors and that disruption of these genes can have behavioural consequences. In *Drosophila* the *dissatisfaction* gene, which encodes a nuclear receptor closely related to the vertebrate *tailless* proteins is involved in sex-specific neural development (Finley, et al., 1998). In mice lacking the *Lxh5* gene the hippocampus fails to form with its normally layered structure because of a disruption in the pattern of cell migration during development (Zhao, et al., 1999). Because this gene is a highly conserved homeobox gene it is likely that its homologue is involved in hippocampal development, and therefore

memory and functions related to social cognition, in humans. All of these genes would seem to be good candidates for the basis of research into the aetiology of the symptoms of psychosis, though to the best of my knowledge they are not being investigated at the moment. Indeed, as one should have come to expect, the search for genes involved in schizophrenia appears to be largely atheoretical (see, for example, Kendler, 1999), though there are a few notable exceptions (Crow, 2000). David Skuse, whose work on sex chromosomes and social cognition was mentioned earlier, has appealed to researchers to abandon the 'one gene one disease' model in favour of a 'focus on the search for the genetic processes underlying specific cognitive functions that, in turn, underpin child psychiatric disorders, especially those that are neurodevelopmental in origin' (Skuse, 1997, p. 354). However, in the following section, through a consideration of a number of studies that have exogenous variables as their focus, I aim to demonstrate that contributing factors to developmental systems other than genes may be highly significant in the aetiology of many forms of mental illness.

Adverse Conditions and the Functioning of Psychological Mechanisms

Torrey and colleagues (1997) have reviewed over 250 studies covering 29 Northern and five Southern Hemisphere countries and have found a consistent winter-spring excess of births for both schizophrenia and bipolar disorder of 5-8 percent. These authors also report seasonal birth excess in schizoaffective disorder (December-March), major depression (March-May), and autism (March), and a seasonal birth effect for anorexia (January-June with the peak March-June) has just been reported (Eagles, et al., 2001). Amongst the factors held likely to be responsible are 'seasonal effects of genes, subtle pregnancy and birth complications, light and internal chemistry, toxins, nutrition, temperature/weather, and infectious agents or a combination of these are all viable possibilities' (Torrey, et al., 1997, p. 1). The hypothesis that infectious agents are responsible for some cases of schizophrenia received strong support earlier this year when Karlsson and colleagues (2001) reported that they had found nucleotide sequences related to those of the human endogenous retroviral (HERV)-W family of endogenous retroviruses and to other retroviruses in the murine leukemia virus genus in the cerebrospinal fluid of 29 percent of 35 patients diag-

nosed with recent-onset schizophrenia and in one of twenty patients diagnosed with chronic schizophrenia, but in none of 22 individuals with neurological conditions or 30 individuals with no neurological or psychiatric conditions that they examined. The authors note that ‘there are several mechanisms by which retroviral sequences might be transcribed within the nervous system… For example, the long terminal repeat regions of many retroviral RNAs contain binding sites for a number of different transcription factors and enhancers … [which] can activate… human genes located downstream from the site of retroviral integration’ (Karlsson, et al., 2001, pp. 4637-8)

Bunney and Bunney (1999, p. 225) have suggested that a physical trauma or virus experienced by women in the second trimester of pregnancy, during which neurons migrate from the ventricular walls to the cortical plate, could result in disordered connectivity in the prefrontal cortex. This could be associated with the hypofrontality (reduced activity in the prefrontal cortex) observed in some schizophrenics, which seems to be related to negative symptoms (Berman & Weinberger, 1999, p. 255). In some studies hypofrontality has been seen to be associated only in patients with negative symptoms (Andreasen, et al., 1992; Byne, et al., 1999, p. 239). Decreased blood flow during prefrontal tasks is strongly correlated with reduction in the dopamine metabolite HVA in cerebrospinal fluid (Weinberger, Berman & Illowsky, 1988), and blood flow in the prefrontal cortex increases after administration to schizophrenic patients of the dopamine agonists apomorphine and amphetamine (Byne, et al., 1999, p. 238; Davis, et al., 1991). One interesting finding in terms of the emphasis I have placed on development is that even psychological trauma in the second trimester of pregnancy could be implicated in a susceptibility for schizophrenia and other disorders. Meijer (1985) found that offspring of mothers who were exposed to the threat and the occurrence of the six day Arab-Israeli war during pregnancy displayed developmental delays and behavioural deviance (discussed in van Os & Selten, 1998). Huttunen and Niskanen (1978) used the Finnish population register for people born between 1925 and 1957 to identify 167 people whose fathers had died before their children's births and a control group of 168 people whose fathers died during the first year of their children's lives. The incidence of alcoholism and personality disorders was relatively high in

both groups, but the number of diagnosed schizophrenics and the number committing crimes were significantly higher in the index than in the control group. The investigators concluded that maternal stress may increase the risk of the child for psychiatric disorders, especially during months three to five and in the final month of gestation. Van Os and Selten (1998) found that in the cohort of offspring born to women who were pregnant during the May 1940 invasion of The Netherlands by German forces had a higher incidence of schizophrenia than unexposed controls. In the second trimester men, but not women, were particularly vulnerable. With regard to possible mechanisms van Os and Selten note

> The fetus is protected to a degree from the growth retarding and neurotoxic effects of glucocorticoids by placental enzymes. It is possible, however, that the capacity of these enzymes is exceeded in the case of greatly elevated maternal cortisol levels. A further possibility is that high levels of cortisol are produced by the fetus itself, in response to fetal hypoxia induced by high levels of maternal catecholamines and uterine vasoconstriction. Indirect mechanisms can also influence later risk. For example, there is increasing interest in the possible association between maternal exposure to stressful life events and preterm delivery, which may increase the risk of schizophrenia in the child. Similarly pregnant women who experience stressful life events may develop depressive symptoms, which have been in turn associated with greater risk of complications of birth and pregnancy (van Os & Selten, 1998, p. 326).

The period from the second trimester of pregnancy to the second year of infancy is crucial to brain development and is also therefore a period during which the brain can be affected by poor nutrition. During the Second World War a German blockade resulted in what has been called the Dutch Hunger Winter of 1944-1945. The birth cohort conceived at the height of this famine showed a twofold increase in the risk for schizophrenia (Bunney & Bunney, 1999, p. 232; Susser, et al., 1996).

It has been suggested that in bad conditions a pregnant woman can modify the development of her unborn child such that it will be prepared for survival in an environment in which resources are likely to be short (Bateson & Martin, 1999, p. 110) resulting in a *thrifty phenotype* (Hales & Barker, 1992). Individuals with a

thrifty phenotype will have ‘a smaller body size, a lowered metabolic rate and a reduced level of behavioural activity… adaptations to an environment that is chronically short of food’ (Bateson & Martin, 1999, pp. 110-111). Those with a thrifty phenotype who actually develop in an affluent environment may be more prone to disorders such as diabetes, whereas those who have received a positive maternal forecast will be adapted to good conditions and therefore better able to cope with rich diets. This idea, which is also known as the *Barker hypothesis* (Barker, 1992), is now widely (if not universally) accepted and is a source of grave concern for societies undergoing a transition from sparse to better nutrition (Robinson, 2001). However, just as the mother may be able to provide a forecast of environmental conditions perhaps she can also send a forecast of social conditions via the mechanisms discussed above. In most hunter-gatherer societies the death of the mother’s mate, and the consequent probability of low paternal investment, could well be as significant as, if not more significant than, environmental conditions of food shortage. The probability of a poor outcome could also affect the willingness of the mother to invest in the offspring after the birth of the child, and a poor socioassessment could be communicated during the attachment process, as hypothesized by Chisholm. Those with the resulting ‘thrifty cognitive phenotype’ could be at a higher risk of sustaining developmental damage responsible for various symptoms of mental illness. Certainly, it seems extraordinary to imagine that the processes involved in the production of the thrifty phenotype would be sensitive purely to maternal nutrition and would result only in physical, rather than psychological, changes.

Gaudino, Jenkins, and Rochat (1999) used linked 1989-1990 birth and death certificates of singleton infants in Georgia to calculate the relative risks for 38,943 infants with no father’s name listed on the birth certificate compared to 178,100 with father's names listed. Compared to the rate for married mothers listing the name of the father, the relative risk of death was 2.5 for unmarried mothers not listing fathers, 1.4 for unmarried mothers listing fathers, and 2.3 for married women not listing fathers. The risk remained significant after taking into consideration other factors such as maternal race, age, adequacy of prenatal care and medical risks; congenital malformations, birth weight, gestational age, and small-for-gestational age. Gaudino and colleagues concluded that paternal

involvement is protective against low birth weight and infant mortality. Hultman and colleagues (1999) examined the cohort of all children on the Swedish birth register between 1973 and 1979 who were subsequently listed as having been admitted to hospital aged 15-21 with a diagnosis of schizophrenia, affective psychosis, or reactive psychosis. Schizophrenia was found to be positively associated with multiparity, maternal bleeding during pregnancy, and birth in late winter. Boys who were of low birth weight for their gestational age, number four or more in birth order, and whose mothers had been bleeding during late pregnancy were at greater risk. In females none of these variables was related to schizophrenia. Affective psychosis was found to be associated with uterine atony[27], and late winter birth. Reactive psychosis (often diagnosed as schizophrenia outside Scandinavia) was associated with multiparity. A study of all patients diagnosed with autism in North Dakota matched with their birth certificates identified the five pre- and perinatal risk factors associated with autism as decreased birth weight, low maternal education, later start of prenatal care, having a previous termination of pregnancy, and increasing father's age (Burd, et al., 1999).

Ramrakha and colleagues (2000) found that young people diagnosed with substance dependence, schizophrenia spectrum, antisocial disorders, and depression were more likely to engage in risky sexual intercourse, contract sexually transmitted diseases, and have sexual intercourse at an early age (before 16 years). The likelihood of risky behaviour was increased by psychiatric comorbidity. These associations were not moderated by sex, and adjustment for socioeconomic background made no difference to the results. These findings are in keeping with Chisholm’s idea that those receiving a negative socioassessment during the attachment process will be more likely to engage in risky behaviour.

Vivette Glover and Tom O’Connor of Imperial College, London are also about to publish data showing that the mother’s anxiety during the last few weeks of pregnancy can affect the unborn baby’s developing brain. The women's stress levels were assessed at 18 and 32 weeks of pregnancy, and their children were

[27] relaxation of the uterus after the birth of the baby.

assessed for behavioural and emotional problems just before they turned four. The study only included women who were anxious before the birth of the child, but not after, in order to rule out the possibility that the mother's anxiety was transmitted to the child after birth. Women with the highest stress levels were 50 percent more likely to have hyperactive children; boys were particularly affected and were twice as likely as normal to be hyperactive. Myhrman and colleagues (1996) collected data prospectively on the Northern Finland 1966 Birth Cohort of 11,017 individuals. In the sixth or seventh month of pregnancy mothers were asked whether the pregnancy was wanted, mistimed but wanted, or unwanted. Those born from unwanted pregnancies were two and a half times more likely to develop schizophrenia than those who are either wanted or wanted but mistimed, and the result remained significant even after adjustment for confounding sociodemographic, pregnancy and perinatal variables. The authors suggested that stress during pregnancy may affect fetal brain development, and that continuing stress after childbirth, leading to an abnormal family atmosphere during childhood, may affect emotional and cognitive development, giving rise to schizophrenia. They also speculate that being wanted and reared in a propitious family atmosphere may be a protective factor for schizophrenia in those who may be vulnerable for other reasons. Using data on the same Finnish cohort Jones and colleagues (1998) identified 76 cases of DSM-III-R schizophrenia that arose by age 28; 67.1 percent of these were men. Low birth weight and the combination of low birth weight and short gestation were more common among the schizophrenic subjects.

The hypothesis presented here that the thrifty phenotype could be a result of a negative maternal forecast of not only nutritional but familial/social early life circumstances implies that there should be a higher than expected correlation between schizophrenia and diabetes. The comorbidity of schizophrenia and diabetes is higher than for the general population (Dixon, et al., 2000; Holden & Pakula, 1999; Odawara, et al., 1997); and the evidence suggests that 'a higher prevalence of diabetes in schizophrenic patients may be a universal phenomenon' (Mukherjee, et al., 1996, p. 68).

The Barker Hypothesis and the Trivers-Willard Hypothesis

The evolutionary theorist Ronald A. Fisher (1890-1962) assumed that biotic and abiotic environmental effects would act equally on male and female phenotypes, and this assumption was widely held until challenged in an influential paper published by Trivers and Willard in *Science* (1973). Trivers and Willard argued that in a population of mammals females would vary considerably in their condition. Those is a good condition would be more likely to produce large healthy young, and those in poor condition would be more likely to produce small, weak offspring. In most mammalian species males compete for access to females; therefore a larger size carries a greater advantage for males rather than females, and, of course, males also have a greater variance in reproductive success, with many failing to reproduce at all. Following this line of reasoning Trivers and Willard suggested that females in good condition should favour sons, and females in poor condition should favor daughters. The sex ratio may be adjusted pre-natally or post-natally. Although there are many potential confounding factors the hypothesis has received empirical support from a remarkable range of studies. In guppies fed a high protein diet and in wild populations of American opossums whose food supply was experimentally manipulated, females in a poor condition favoured daughters over sons (Badcock, 2000, p. 182). When fed a sub-standard diet female wood rats bring about death by starvation in their sons by preferentially feeding their daughters (Trivers, 1985). In the population of red deer on the Isle of Rhum in Scotland subordinate females have been found to prefer daughters and dominant females sons (Cartwright, 2000, p. 121). In subsequent studies with this deer population birthweight was found to be a significant determinant of total lifetime reproductive success in males, with heavier-born males being more successful than lighter ones. In contrast, birthweight did not affect female reproductive success (Kruuk, et al., 1999). Pregnant female house mice maintained on a consistent low-food diet were found to give birth to a lower proportion of males than control females and females deprived of food every other day one week before mating and those deprived every third day during gestation produced a lower proportion of males than did controls (Meikle & Thornton, 1995). In a study of golden hamsters

physiologically-stressed females were found to skew offspring sex ratios to favour daughters (Huck, et al., 1988).

In the case of humans Gaulin and Robbins (1991) used longer interbirth interval and duration of breastfeeding as indicators of parental investment in a study of 906 mothers. In poor conditions there was greater investment in daughters for both of these variables, and more investment in sons in good conditions. Of the fourteen variables studied five showed 'marked and significant sex-by-condition interactions of the type and in the direction predicted by Trivers and Willard; none showed significant effects in the opposite direction' (Gaulin & Robbins, 1991, p. 61). Lee Cronk has found evidence of female-biased parental investment under poor conditions in the 'Mukogodo of Kenya; the Cheyenne of North America; the Kanjar of south Asia; the Mundugumor of New Guinea; persons living in contemporary North America; as well as persons living in historical Germany, Portugal, and the US' (Cronk, 1991, p. 387). Chacon-Puignau and Jaffe (1996) found a Trivers-Willard effect related to the marital status of the mother through demographic information collected from registration data in Venezuela. Their results indicated 'that the investment in females associated with environmental adversity is greater than the investment in males associated with good environmental conditions' (Chacon-Puignau & Jaffe, 1996, p. 257). Koziel and Ulijaszek (2001) also found support for a weak Trivers-Willard effect among a large contemporary Polish sample using first birth interval and extent of breastfeeding as measures of parental investment. They found evidence of greater investment in female offspring at the lower extremes of income, and greater investment in males at higher levels of income, in particular a greater proportion of first-born boys were breastfed longer than girls, while the opposite trend was found among families with fathers with lowest levels of education. Using reliable demographic data Mealey and Mackay (1990) studied 1314 Mormon women who married before 1851 when polygyny was legal. There was a significant bias towards male children in the wives of men of the highest rank (Badcock, 2000, p. 184).

I believe that the Barker (or thrifty phenotype) hypothesis should be understood within the context of parent-offspring conflict, parental investment theory, and

the Trivers-Willard hypothesis. We should not distinguish between the possible effects of biotic, abiotic, or socio-psychological factors on the developmental system. From this perspective psychological factors such as father absence or an unwanted pregnancy are as capable of producing a poor maternal forecast, and contributing towards the development of the thrifty phenotype, as poor nutrition. These are all physical effects with material consequences. In the context of the maternal-fetal conflict identified by David Haig a poor maternal forecast represents a change in the balance of the 'stable tug of war'. The consequences could range from spontaneous abortion to the triggering of the thrifty phenotype, depending on the severity of the reduction in maternal investment. It is difficult to accept that a system facilitating such a process could ever be adaptive, but conditions for our ancestors were probably often much harsher than they are for many today, and in some circumstances it may have been adaptive to abandon or withdraw support even from newborns. Amongst the foraging people known as the Aché a child with no father is four times more likely to die before the age of two, and mothers sometimes kill fatherless infants because of their poor prospects. Some foraging peoples even bury orphans alive with the deceased parent (Hill & Hurtado, 1996; Hrdy, 1999, pp. 236-7). Fatherless young children are also in greater danger of being killed (deliberately or through neglect) by new partners. Tribal raiders intent on capturing fertile women have also been known to target young children intentionally. Elena Valero, a Brazilian captured by the Yanomamö describes this vividly:

> …the men began to kill the children; little ones, bigger ones, they killed many of them. They tried to run away but [the Karawetari raiders] caught them, and threw them to the ground, and stuck them with bows, which went through their bodies and rooted them to the ground. Taking the smallest by the feet, they beat them against the trees and the rocks (Hrdy, 1999, p. 242).

Amongst the Ayoreo people of Bolivia and Paraguay expectant mothers move to the forest with a band of close kinswomen when labour begins. During labour a woman sits on or hangs from a tree branch, and when the baby is born it falls into a hole prepared by the kinswomen. Unwanted children are pushed into the hole with a stick and buried, without ever being touched by human hands. Ayoreo women have been known to bury several children before settling into a

permanent marriage and raising children successfully. The ‘principal reason for such a drastic decision, according to the mothers themselves, is lack of parental support. Other reasons that mothers offer are deformities, the birth of twins, or the arrival of a new baby so soon after an older sibling as to overburden the mother and imperil the older child’s survival’ (Daly & Wilson, 1988, p. 39). To take an example closer to home: between 1902 and 1927 approximately 48 percent of the women incarcerated in Broadmoor special hospital in England had committed infanticide (Hrdy, 1999, p. 289). It is, perhaps, easy to view infanticide as deplorable or pathological, and somewhat less easy to comprehend the mechanisms that allowed our ancestors to make (consciously or unconsciously) hard decisions about the appropriate allocation of resources in harsh environments.

The Trivers-Willard hypothesis should lead us to expect that a poor maternal forecast will be particularly detrimental to males, and may predispose them to a range of medical and psychiatric conditions, and may also predispose them to develop risky life-history-strategies, though at any stage in development other factors may either compound or ameliorate the effects of early influences. Ironically, risky life-history strategies themselves may expose vulnerable individuals to a variety of factors likely to increase the probability of incurring further psychological and physiological damage. These ideas help to explain the problem of the ‘fragile male’ (Kraemer, 2000). Although at conception there are more male than female embryos exposure to severe life events before and during the periconceptional period, including smog, earthquakes, and flood, might be associated with a decline in the sex ratio. Hansen, Møller, and Olsen (1999) used the Danish population based medical birth registry to identify all Danish women who gave birth between 1st January 1980 and 31st December 1992. They subsequently identified all women exposed to severe life events in the year of birth and the previous year but included only the women exposed before the second trimester. This resulted in an exposed cohort of 3072 singleton pregnancies and a control group of 20,337 singleton pregnancies was randomly selected. The effect of psychological stress related to severe life events on the sex ratio was clearly demonstrated with the proportion of boys found to be 49.0 percent in the exposed group and 51.2 percent in the control group. As Kraemer puts it

> From this point on it is downhill all the way. The male fetus is at greater risk of death or damage from almost all the obstetric catastrophes that can happen before birth. Perinatal brain damage, cerebral palsy, congenital deformities of the genitalia and limbs, premature birth, and stillbirth are commoner in boys, and by the time a boy is born he is on average developmentally some weeks behind his sister: “A newborn girl is the physiological equivalent of a 4 to 6 week old boy.” The male brain is heavier, with a larger hypothalamus, probably from the influence of a surge of testosterone in the third trimester of pregnancy, which also promotes greater muscle bulk. Similar differences have been observed in chimpanzees… By the time a boy is born the pattern seems set. Developmental disorders such as specific reading delay, hyperactivity, autism and related disorders, clumsiness, stammering, and Tourette's syndrome occur three to four times more often in boys than in girls, although girls, when they have such a disorder, may be more severely affected. Conduct and oppositional disorders are at least twice as common in boys. Genetic factors are known to play a part, varying from low heritability in conduct disorder to high in autism, but why are they all commoner in boys? (Kraemer, 2000, p. 1609).

Kraemer indicates that an evolutionary perspective should be helpful here, and even remarks that ‘a hominid male of, say, half a million years ago may have needed all the opportunities for risk taking he could get, just to procreate. Charles Darwin noted this’ (Kraemer, 2000, p. 1611). As Kramer notes the biological fragility of the male from conception onwards is little known or understood. There is a clearly a great deal to be gained from an evolutionary perspective. It may be that the insights provided by evolutionary developmental psychopathology as discussed above are so completely counterintuitive that they cannot be attained through any other approach. The mechanisms producing the thrifty phenotype in offspring were almost certainly adaptive in our ancestral environment, but in many current environments the physiological, psychological, and behavioural effects of these adaptations may be extremely damaging. For a pregnant woman in contemporary developed societies the loss of a mate may have consequences only vaguely comparable to those experienced by a woman in hunter-gatherer society, but the adaptations capable of triggering the thrifty phenotype may still be operative. Many of the ‘pathologies’ we seek to explain by reference to endogenous mechanisms may well be better explained by reference to mismatch theory.

Conclusion: The Evolution and Ontogeny of the Theory of Mind Module

In terms of the evolutionary framework that I have advocated it appears that the theory of mind module is composed of tactical (short term response and survival) and strategic (long term response and survival) systems. The tactical systems are based on phylogenetically older components such as the amygdala and hippocampus and facilitate rapid sensory-driven cognitive-emotional responses. Within the modular scheme these are Darwinian and Skinnerian modules. The more recent prefrontal systems subserve long-term planning through the integration of cognition and the strategic (higher cognitive) emotions, and these are Popperian and Gregorian modules capable of functioning correctly only if their tactical subcomponents are also intact. In order to ensure rapid responses to sensory stimuli in dangerous and stressful situations the prefrontal mechanisms can be inhibited by the same neurochemicals that potentiate the tactical systems. In normal circumstances the modules work together in concert to facilitate the mentalistic interpretation of behaviour, the learning of cognitive-emotional responses, and the storage of these responses in long-term memory through the mediation of the hippocampus and the prefrontal cortex.

The nature of the interaction between the components subserving theory of mind may be adjusted during development, and the social cognition associated with the thrifty phenotype may have a substantially different configuration, and may subserve behaviours associated with the Young Male/Female Syndromes. The theory of mind mechanisms are sexually dimorphic and there are changes in their structure and functioning across the lifespan, but equal numbers of men and women suffer from the major psychoses, and as males are more susceptible to developmental damage, the number must equalise because of some other factor or factors affecting only females. It may be that female mechanisms are more susceptible to damage caused by stress, or that woman are particularly susceptible to some exogenous factor such as a pathogen. One important risk factor that is not currently appraised by modern medicine will be discussed in the following section on premenstrual syndrome. Ultimately, the explanation of the fact that females have different, more effective, and more robust mechanisms of social intelligence may be explained in terms of parental investment

theory. Women should be more discriminating about the choice of a long term mate because of the gross asymmetry in the investment that males and females make in offspring, and because of the benefits that can be derived from the choice of a mate more likely to participate in parental care. In addition to sex the other important factors in assessing the nature of the impairment are the location of the damaged component, the stage of development at which the impairment was caused, and the ecological and social circumstances under which development took place.

A number of studies using diverse methods from neuroimaging, neuroanatomy, and neuropathology have confirmed that damage to the amygdala, particularly the left amygdala, can result in substantial theory of mind deficits. If this damage occurs very early in development then various aspects of social cognition will be severely impaired across the lifespan because the subject will be unable to engage in cognitive-emotional learning or respond appropriately to sensory stimuli. If the hippocampus is damaged learning may take place, but the results may not be stored in long-term memory where they can guide (consciously or unconsciously) cognitive-emotional strategic planning. Damage to the prefrontal cortex will result in faulty switching between tactical and strategic responses to sensory stimuli, and long-term dysfunction may cause down-regulation of receptors in the amygdala. This may explain why positive symptoms as identified in sub-types of schizophrenia are superseded by negative symptoms over time. Overall, the fact that theory of mind deficits are detected as a consequence of multifarious neuropathology and in conditions as diverse as schizophrenia, autism, Asperger's syndrome, and Attention Deficit Hyperactivity Disorder suggests that these conditions are not discrete entities.

The picture of mental illness emerging here is compatible with the model proposed by Murray and Fearon (1999) of an interaction between multiple genes and environmental factors, with the latter being divided in to predisposing and precipitating factors. I would add that many of the genes involved in these developmental processes need not be 'disease genes' and that many of the predisposing and precipitating factors may arise because of a mismatch between the (environmental and/or social) conditions anticipated by the adaptations sub-

serving the prenatal maternal forecast and the actual conditions of development. Adaptations designed to contribute to the production of a phenotype modified pre- or post-natally to meet the psychological (i.e., information processing) and physiological demands of a risky and impoverished hunter-gatherer environment may function less than optimally in many current environments, with resulting impairment to separate but co-dependent physiological and cognitive-emotional systems. If this is correct then an assessment of psycho-social conditions of early development should contribute to our understanding of a range of medical and psychiatric disorders, and indeed our understanding of the aetiological factors operative in these conditions may be completely transformed. It seems likely that we will discover unusual links between a variety of developmental system variables and a range of physical and mental conditions which under current medical and psychiatric hypotheses should have no connection.

Early in the nineties molecular genetic techniques were used successfully to identify a new type of mutation called the trinucleotide repeat amplification. This phenomenon is now known to be the cause of conditions such as myotonic dystrophy, fragile X syndrome, Kennedy's disease, Huntington's disease, spinocerebellar ataxia type 1, and dentatorubral-pallidoluysian atrophy (Petronis & Kennedy, 1995). There were hopes that this mutation would provide insights into bipolar disorder and schizophrenia as both seemed more amenable to interpretation in this framework than in terms of polygenes because the amplification of trinucleotide repeats over time seemed to offer an explanation of the greater severity and earlier age at onset in subsequent generations, a phenomenon known as anticipation (Kendler, 1999, p. 204), observed in both of these conditions. However, subsequent studies attempting to establish the existence of trinucleotide repeats failed to find any differences either between affected and unaffected individuals or across generations (Petronis, et al., 1996). This and other failures of molecular genetics lead one prominent researcher to complain that

> Ten years of intensive molecular genetic searches for DNA mutations that would cause or predispose to major psychosis, unfortu-

> nately, have not been very productive. Experimental data of genetic linkage and association studies accumulated over this decade are either controversial or negative. Research strategies that worked relatively well in other complex diseases, such as breast cancer and Alzheimer's disease, turned out to be significantly less efficient in major psychosis (Petronis, 2000, p. 8).

It seems clear that there are probably many exogenous as well as endogenous factors capable of causing damage to one or more of the distributed components of the theory of mind mechanism and we should expect the search for endogenous causal factors responsible for causing hypothetical diseases such as schizophrenia to remain as unsuccessful as they have been to date. Until we have projectable categories in psychiatry valid explanations of mental disorders will elude us, as will a coherent assessment of the causes and transmission of disease within a population.

Premenstrual Mood Disorder and Female Mating Strategies

In 'Appendix B' of DSM-IV (American Psychiatric Association, 1994, p. 703) there are a number of proposals for new categories of mental illness including 'dissociative trance disorder', 'caffeine withdrawal', and 'premenstrual dysphoric disorder'. Premenstrual mood disorder (PMDD, also called premenstrual dysphoria) is not officially a psychiatric disorder but the term has been used by psychiatrists for many years (DeJong, Rubinow & Roy-Byrne, 1985), and the condition has generally been regarded as another aspect of depression or neuroticism (Van der Ploeg, 1987), though women themselves have been found to rate the symptoms of premenstrual stress as 'normal experiences reflecting ordinary behaviour' (Sveinsdottir, Lundman & Norberg, 1999, p. 916). Premenstrual syndrome occurs during the luteal phase of the menstrual cycle, with a symptom-free period during the follicular phase. In this section I would like to propose an explanation of the symptoms of premenstrual syndrome in terms of adaptations for female reproductive strategies. Approximately three quarters of women experience some premenstrual changes (American Psychiatric Association, 1994, p. 716; Steiner & Pearlstein, 2000), and symptoms decline with age. Amongst a sample of girls in the 13-18 age group 88 percent reported moderate to severe symptoms and 56 percent reported extreme symptoms including food

cravings, breast swelling, abdominal discomfort, mood swings, stressed feeling, and dissatisfaction with appearance (Cleckner-Smith, Doughty & Grossman, 1998). The younger teenagers (13-15) reported less severe symptoms that those in the older (15-18) group. It is notable in terms of the analysis to follow that peaks in women's sexual desire occur most frequently during fertile phases (Regan, 1996).

Recent years have witnessed an upsurge of interest in the function of smell, including the possible existence of pheromones and their potential role in mate choice in humans. Savic and colleagues (1997) have demonstrated that women who smelled an androgen-like compound activated the preoptic and ventromedial nuclei of the hypothalamus, whereas men who smelled an oestrogen-like compound activated the paraventricular and dorsomedial nuclei of the hypothalamus. ‘This sex-dissociated hypothalamic activation suggests a potential physiological substrate for a sex-differentiated behavioral response in humans’ (Savic, 1997, p. 661). Karl Grammer (1993) asked 289 women to rate the smell of the male hormone androstenone. The subjects rated this component of male body odour unattractive, but the rating changed to a neutral emotional response at the conceptive optimum around ovulation. Grammer speculated that this ‘cyclic-dependent emotional rating of androstenone might facilitate active female choice of sex partners and may be a proximate cue for female mate-choice’ (Grammer, 1993, p. 201). In a study of body odour during which subjects wore a T-shirt for three consecutive nights under controlled conditions Rikowski and Grammer (1999) found positive relations between body odour and attractiveness, and negative ones between smell and body asymmetry for males, only if the female odour raters were in the most fertile phase of their menstrual cycle. Asymmetry was assessed by the measurement of seven bilateral traits and a separate group of judges rated photographs of subjects for attractiveness. Gangestad and Thornhill (1998) used the same ‘T-shirt method’ to establish that in a group of 41 female subjects those near the peak fertility of their cycle tended to prefer the scent of shirts worn by symmetrical men, and individual women’s preference for symmetry correlated with their probability of conception. The subjects at the low fertility phase of their cycle and women who were taking

the contraceptive pill showed no significant preference for either symmetrical or asymmetrical men.

The resistance to parasites conferred by heterozygosity is thought to be one of the reasons for the evolution of sexual reproduction. Claus Wedekind has hypothesized that odours could act as signals directly revealing the existence of resistance genes. Signals of this type would promote the survival of the man's offspring by allowing choosy females to optimize costs and benefits of each resistance in the progeny (Wedekind, 1994a; 1994b). But can females detect resistance genes through odours? The major histocompatibility complex (also called the HLA - human leukocyte antigen – in humans) is a cluster of over 20 linked genes on chromosome 6. These genes are highly polymorphic, with some of them having over 50 alleles. They have a major function in the immune response against pathogens and parasites. The MHC is also responsible for producing the tissue type that allows the immune system to identify tissue as self, and is used as a method of kin recognition, at least in mice (Majerus, Amos & Hurst, 1996, p. 109). Certain MHC combinations, usually heterozygous ones, are superior under selection by pathogens. This implies that females should attempt to identify mates with MHC genes differing to their own in order to increase the chance of producing offspring with the desirable heterozygosity and enhanced parasite resistance. Wedekind and Furi (1997) asked 121 men and women to score the odours of six T-shirts, worn by two women and four men and found that their scorings of pleasantness 'correlated negatively with the degree of MHC similarity between smeller and T-shirt-wearer in men and women who were not using the contraceptive pill [but not in those who were]… This suggests that in our study populations the MHC influences body odour preferences mainly, if not exclusively, by the degree of similarity or dissimilarity' (Wedekind & Furi, 1997, p. 1471). These findings suggest that women can detect the MHC differences (rather than specific combinations) that would increase the heterozygosity of offspring. Remarkably, in a study of 137 male and female students who had been typed for their MHC Milinski and Wedekind (2001) found that individual preferences for perfume ingredients correlated with a person's MHC genotype. This finding supports the hypothesis that perfumes are chosen 'for self' in order to amplify body odours that reveal a person's im-

munogenetics. Platek, Burch, and Gallup (2001) have recently discovered sex differences in olfactory self-recognition. In their study 59.4 percent of females, but only 5.6 percent of males could recognise their own odour, and females rated their own secretions as significantly lower on a pleasant-positive factor than males rated their own odours. These authors remark:

> It has been argued that females were selected for a better sense of smell, although it might be more appropriate in this case to say that they have been selected for a better pheromonal detection/chemical communication system. If this were the case, then maybe a female's perception of her own proximate chemosecretions could act as (1) a priming mechanism to better detect more subtle and minute changes in the surrounding environment and (2) to better integrate incoming chemical information with her proximate chemosignal state (as well as other sensory systems) in an attempt to make the best possible assessment of any particular context. Because differences in preference for the odour of another individual of the opposite sex has been shown to be linked to the donor's makeup at their HLA loci and the degree to which they show fluctuating asymmetry, this ability to integrate and assess incoming volatiles might be associated with mate choice preferences and/or menstrual cycle phase (Platek, Burch & Gallup, 2001, p. 639).

In mice, Rülicke and colleagues (1998) have found that female eggs could select specific sperm. During an epidemic of mouse hepatitis virus the proportion of MHC-heterozygous embryos increased, which suggests ‘that parents are able to promote specific combinations of MHC-haplotypes during fertilization according to the presence or absence of a viral infection’ (1998, p. 711). Through an analysis of 189 human societies Bobbi Low has found that there is a strong relationship between the number of parasites a population is exposed to (pathogen stress) and the degree of polygyny, i.e., the custom of having more than one wife (Low, 1990). In these regions of high pathogen stress both women and men rate the importance of the physical attractiveness of a prospective mate more highly than in other regions of the world (Gangestad & Buss, 1993).

Men with more symmetrical body measures have more sexual partners; have more sexual partners outside their primary relationship (Scheib, Gangestad &

Thornhill, 1999), and women prefer the scent of symmetrical men during their fertile phase (Thornhill & Gangestad, 1999). Aggregate measures of FA, i.e., *fluctuating asymmetry*, the asymmetry resulting from errors in the development of normally symmetrical bilateral traits under stressful conditions, correlate very significantly with the number of sexual partners, though the effect may be mediated through a preference for indicators other than symmetry (Gangestad, Bennett & Thornhill, 2001). Women generally appear to prefer slightly feminized to average male faces (Perrett, et al., 1998), even though testosterone-dependent secondary sexual characteristics may be a signal a robust immune system (Ditchkoff, et al., 2001; Kirkpatrick & Ryan, 1991), and should be favoured by females according to the 'good genes' model of sexual selection. Using computer graphics to manipulate the feminized and masculinized features of human faces Perrett and colleagues (1998) found that for a group of female Japanese and Scottish subjects increasing the masculinized features of faces altered the perception of personality characteristics, increasing the ratings of perceived dominance, masculinity, and age, but reduced the ratings of perceived warmth, emotionality, co-cooperativeness, honesty, and quality as a parent. The authors conclude that 'the results indicate that judgements of male attractiveness reflect multiple motives. Females may adopt different strategies, giving preference to characteristics that are associated with dominance and an effective immune system, or to characteristics that are related to paternal investment' (Perrett, et al., 1998, p. 886). This balance between selection pressures favouring highly masculine features such as large body mass, upper body strength, and other features promoting success in male-male competition and selection pressures favouring feminized features helps to reduce sexual dimorphism in appearance in humans, but clearly promotes sexual dimorphism in the psychological mechanisms subserving mate choice.

In a second study using images manipulated by computer software Penton-Voak and colleagues (1999) decided to test the hypothesis that females would be more sensitive to markers of immunological competence during the phase of the menstrual cycle when conception is most likely. Thirty-nine Japanese subjects who reported regular menstrual cycles and no use of oral contraceptive were asked to select the face they found most attractive from five Caucasian,

and separately from five Japanese male faces. Subjects preferred faces that were less feminized in the high-conception-risk phase, and no effect for stimulus origins (Japanese or Caucasian) was found. There were trends indicating that women with a partner preferred more masculine faces, and these underwent a great cyclic change in preference than those without a partner. In a second experiment British subjects were allowed to manipulate images and asked to choose the most attractive face for a 'long-term relationship' or a 'short-term relationship'. Subjects preferred a less feminine face during the high-conception-risk phase, but those taking oral contraceptives showed no cyclic changes in face preference. The authors reviewed the previous evidence suggesting that dominance and parental qualities were judged to lie at the opposite ends of a continuum related to facial masculinity, and that suggesting the benefit of selecting good gene for parasite resistance might incur the cost of low paternal investment. However, low paternity uncertainty in humans caused by the lack of visual similarity between father and offspring, and concealed ovulation, together with cyclic changes in face preference, suggest that female reproductive strategies could be mixed under some ecological and social circumstances. A female could secure the advantage of extra-pair copulation with a man with more masculinized features and good immunocompetence whilst choosing a long-term partner more likely to cooperate in paternal care.

Reliable estimates of the rate of cuckoldry in human populations are hard to obtain, and the figures reported in various studies have ranged from 1 percent to over 25 percent (Geary, 1998, p. 135). Fortunately, however, there is a another, if somewhat unlikely, source of information about female promiscuity: the size of male testes. A Swedish physiologist Gustaf Retzius (1842-1919) first noticed that in different primate species the size of the testes relative to body size varies dramatically, and in the 1970s this phenomenon was also noticed by Roger Short, who speculated that the different species varied in their need to produce sperm. On encountering Geoffrey Parker's work on *sperm competition* Short realized that this was one reason why males needed large sperm supplies (Birkhead, 2000, pp. 76-7). Parker had established that when two males copulated with the same female in one reproductive cycle the ejaculates of the two males could compete to fertilize the females eggs. It is also known that in many

species females can discriminate between the sperm of different males, a phenomenon known as *sexual selection by cryptic female choice* (Eberhard, 1996). In chimpanzees females copulate 500-1000 times with many males per each pregnancy, but female gorillas copulate around 30 times with a much smaller number of males per pregnancy. Not surprisingly, male chimpanzees have very much larger testes (relative to body size) than male gorillas, and relative testis size has been confirmed as a reliable predictor of the intensity of sperm competition across a wide range of animal species. The modest relative human testis size, which is closer to that of gorillas than chimpanzees, suggests that we have evolved to cope with modest levels of sperm competition, and that human females have probably been moderately promiscuous (Birkhead, 2000, pp. 79-83).

Scores on measures of psychoticism, anxiety, and extraversion have been found to increase in women tested during the premenstrual stage (Mohan & Chopra, 1986), and in the postmenstrual stage significant *decreases* in extraversion and Lie scale scores have been measured (Layton, 1988). Young women have been found to score higher on measures of increased impulsivity during the premenstrual phase of the menstrual cycle than during the other phases (Howard, Gifford & Lumsden, 1988), and co-variations between menstrual symptoms and state anxiety, depression and Neuroticism on the Eysenck Personality may be influenced strongly by genetic factors (Silberg, Martin & Heath, 1987). Thiessen has proposed that female reproductive strategies are more variable than those of males because ‘females track the quality of the environment and link their sexuality to reproductive opportunities, while successful male reproduction depends less on quality environments and more on the availability of females’ (Thiessen, 1994, p. 167), but it is also probable that females do track the quality of males within the context of particular environments, and that their assessment of males varies according to the proximity of their reproductive optimum.

Serotonin, Motivation, and Premenstrual Syndrome

In studies of primates female gorillas were found to initiate mating during the periovulatory period, but mated at other times only under intimidation (Nadler, 1980). Dee Higley and Steve Soumi have hypothesized that animals with low serotonin levels are more sensitive to hazards and opportunities in the environment, whereas those with high serotonin levels are socially dominant and more stable (Allman, 1999, p. 26). Rhesus monkeys with low serotonin levels display high levels of aggressive behaviour, take more risks, and have shorter lifespans (Higley, et al., 1996). These findings help to explain the association between low cholesterol and an increased risk of violent death from accidents and suicide. Jay Kaplan and colleagues have found that monkeys fed on a low-cholesterol diet are more aggressive and have reduced levels of serotonin (Allman, 1999, p. 27; Kaplan, Potvin Klein & Manuck, 1997). This reduction in serotonin levels leads to an increase in food-seeking behaviour and general risk taking. In terms of the modular analysis advocated here the serotonergic systems designed to subserve risk-taking in the pursuit of nutrients, i.e., a basic survival need, also serve as the sub-components of mechanisms designed to mediate risk-taking in pursuit of an enhanced position in the status hierarchy and the pursuit of mates, i.e., social and reproductive survival needs. This ensures that the relationship between the neurotransmitter serotonin and the various mechanisms on which it acts is highly convoluted. In passing I would like to emphasise once again that no simple relationship between serotonin and mood exists, and the treatment of depression with substances designed to promote a general increase in serotonin is bound to result in very mixed outcomes.

Men with low serotonin turnover have been found to exhibit daytime hyperactivity and disrupted sleeping patterns (Mehlman, et al., 2000). The serotonergic systems have reciprocal relationships with the gonadal hormones and selective serotonin reuptake inhibitors (SSRIs) increase the amount of serotonin in the brain, but they also reduce the libido (Vega Matuszcyk, Larsson & Eriksson, 1998), and a review of the effects of antidepressants indicates that most interfere with sexual functioning (Ferguson, 2001). The newer SSRI ‘wonder drugs’ appear to be particularly potent in causing sexual dysfunction. In a study of 610

women and 412 men who had previously shown no previous sexual impairment, and who were questioned about libido, orgasm, ejaculation, erectile function, and general sexual satisfaction, the overall incidence of sexual dysfunction was found to be 59.1 percent; men had a higher frequency of sexual dysfunction (62.4 percent) than women (56.9 percent), although women had higher severity (Montejo, et al., 2001). A number of placebo-controlled trials have indicated that these drugs are effective in treating the symptoms of premenstrual dysphoric disorder, and 'several preliminary studies indicate that intermittent (premenstrual only) treatment with selective SRIs is equally effective in these women and, thus, may offer an attractive treatment option for a disorder that is itself intermittent' (Steiner & Pearlstein, 2000, p. 17). A study of the blood serotonin levels of women with premenstrual syndrome has shown that they have significantly lower levels than matched controls, which suggests that 'the physiologic basis of premenstrual syndrome involves an alteration in serotonin metabolism' (Rapkin, et al., 1987, p. 533).

A study by Rasgon and colleagues (2001) attempted to find evidence for differences in neurochemical brain changes across the menstrual cycle in premenopausal women with and without PMDD, with the expectation that the latter would show signs of abnormal functioning. They found the ratio of N-acetylaspartate to creatine (NAA/Cr) in the region of the medial prefrontal cortex and the cingulate gyrus declined significantly from the follicular to the luteal phase in both groups of subjects, and a significant increase in the ratio of choline to creatine (Ch/Cr) was observed in occipito-parietal white matter. These phenomena appeared to reflect ovarian steroid-related changes in neurotransmission. These findings support cycle-associated changes in brain excitability, with lower frontal brain activation premenstrually. The changes also resemble those described in affective disorders (Rasgon, et al., 2001, p. 54). Unfortunately Rasgon and colleagues offer no explanation for the change in occipito-parietal Ch/Cr ratio, although it is curious to note that decreased levels of NAA and elevated levels of choline in this region appear to be related to poorer intellectual functioning. In one study these metabolites accounted for a large proportion (around 45 percent) of the variance in performance on intelligence tests (Jung, et al., 1999). We should remember, however, that even though the results were

interpreted in terms of pathology, the study by Rasgon and colleagues showed no differences between the control group and the PMDD group. Indeed, David Rubinow and Peter Schmidt of the US National Institute of Mental Health have concluded that there are no luteal phase-specific biological abnormalities in MRMD (i.e., Menstrual Cycle-Related Mood Disorders) and 'there does not appear to be a disturbance of reproductive endocrine function that underlies MRMD' (1999, p. 911). Apparently the only evidence that PMDD is a disorder is the commitment by some researchers that it should be one.

The overall function of the serotonergic system appears to be to modulate the strength of neural connections 'so as to produce stable neural circuits as the organism engages in a wide variety of different behaviours… reducing the strength of serotonergic modulation increases motivational drive and sensitivity to both risk and reward, which can in some circumstances confer adaptive benefits' (Allman, 1999, 26). The serotonergic systems are thus implicated in diverse conditions that are typified by changes in motivation including anxiety, depression, and sleep disorders, and these systems may serve different functions in the left and right hemispheres (Regard & Landis, 1997). I hypothesise that the serotonergic, hormonal, neurochemical, motivational, emotional, and cognitive changes observed in premenstrual syndrome are part of an adaptive system designed to reduce satisfaction temporarily with the prevailing conditions and to promote extra-pair mating with males of complimentary MHC configurations and desirable traits, though there will, of course, be many other factors capable of influencing actual behaviour of any given individual. The mechanisms by which these changes are effected may produce unpleasant experiences for the majority, and perhaps maladaptive changes for an unfortunate minority, but all that is required for a system to be favoured by natural selection is that it should promote an outcome likely to enhance survival and reproduction, not that it should promote stability or contentment.

The production of offspring with a range of MHC configurations and other traits through mixed mating strategies is likely to have been extremely beneficial in many past environments of evolutionary adaptation, and the female 'extra-pair copulation' mechanisms may still be adaptive in the current environment. How-

ever, the patterns of reproduction in contemporary Western society are very different to those in traditional societies, and are therefore probably very different to those in our recent ancestral hunter-gatherer environment. Malcolm Potts and Roger Short explain,

> On average US women with college degrees postpone child bearing until they are over 26 years old, perhaps a decade and half after they went through puberty. Only 7 percent of US women will breastfeed their babies for twelve months or longer. By contrast, women in modern hunter-gatherer societies have their first birth in the later teens or early twenties (only a few years after they first menstruate) and they will have four to eight children, each of which may be breastfed for three to four years, two or three of which are associated with the suppression of ovulation. Our Stone Age ancestors (or a contemporary woman in the Highlands of Papua New Guinea) may have had an average of fifty menstrual cycles in a lifetime, while a modern woman has about 450 – nine times as many. Breast cancer is 120 times as common in a Western woman today as in a hunter-gatherer. It seems that incessant ovulation and the accompanying hormonal turmoil is abnormal and highly dangerous (Potts & Short, 1999, p. 268).

Working with slightly different figures Robert Sapolsky estimates that a hunter-gatherer woman may have only about 24 periods across her lifespan; a modern Western woman about 500 (1998, p. 115). At this stage we can only speculate as to the cost of the cognitive, motivational, and behavioural changes that occur during the 450-500 menstrual cycles experienced by women in the developed world, but it is likely to be considerable. It is known that from early adolescence through to adulthood women are twice as likely to suffer from depression as men (Nolen-Hoeksema, 2001). The constant hormonal and neurochemical changes associated with this massive increase in the number of menstrual cycles may well account for some of the stress experiences and stress reactivity that appear to interact to create women's greater vulnerability to depression and other mental disorders. The steroid hormones known as glucocorticoids mediate the stress response and these are known to be capable of causing both depression (Sapolsky, 1998, p. 248) and frank psychosis in some cases (Jeffcoate, 1993, p. 82). These hormones have been found to act as a potent suppressor of neurons that possess both dopamine D5 and D2 receptors and thereby alter dopamine-mediated neurophysiology in critical regions of the brain

implicated in psychosis (Lee, et al., 2000). This suggests that further work on the relationship between stress depression, anxiety disorders, and schizophrenia could be fruitful, although one leading neuroendocrinologist noted recently that 'it seems likely that the future handling of stress induced mental illness is likely to be as cross disciplinary as the research into its causes. Sadly, for the biomedical scientists of this country, with a track record unsurpassed, all this excitement comes at a time when resources for multidisciplinary research work are almost impossible to obtain' (Herbert, 1997, p. 535).

In many contemporary environments our adaptations are also flooded with artificial stimuli and hence their functioning may be maladaptive for this reason. Any complex functional system may be damaged in many different ways as a result of both endogenous and exogenous processes, but systems may also be bombarded with faulty or inappropriate information, resulting in what could be called *cybernetic dysfunction* in Crawford's (1998) terminology. As David Buss explains,

> The media images we are bombarded with daily… have a potentially pernicious consequence. In one study, after groups of men looked at photographs of either highly attractive women or women of average attractiveness, they were asked to evaluate their commitment to their current romantic partner. Disturbingly, the men who had viewed pictures of attractive women thereafter judged their actual partner less attractive than did men who had viewed analogous pictures of women who were average in attractiveness. Perhaps more important, the men who had viewed attractive women thereafter rated themselves as less committed, less satisfied, less serious, and less close to their actual partners. Parallel results were obtained in another study in which men viewed physically attractive nude centrefolds – they rated themselves as less attracted to their partners. The reasons for these distressing changes are found in the unrealistic nature of the images (Buss, 1994, p. 65).

The type of supernormal stimuli encountered in the mass media and in many novel situations may be responsible for symptoms as diverse as depression caused by an unrealistic assessment of one's position in the social hierarchy, hypervigilance caused by exposure to unusual life-threatening dangers as experienced in modern warfare, or relationship dissatisfaction originating in a

faulty appraisal of the availability (or unavailability) of prospective mates. Consequently, to ask whether anxiety, depression, posttraumatic stress disorder, and hypo- or hypersexuality are disorders has no meaning outside of an assessment of the functioning of particular mechanisms in particular environments, and an investigation of the possible functions of a system within an evolutionary framework may yield extremely counterintuitive results, as I hope my analysis of premenstrual syndrome has shown.

On a more general note we should expect that the administration of non-specific substances such as the Selective Serotonin Uptake Inhibitors capable of altering the function of many systems may have a less than desirable effect in many circumstances, and that variable outcomes ranging from good improvement to catastrophic impairment will continue to be reported in the psychiatric literature.

Delusional Misidentification: Modular Disconnection Disorders?

In this section I will examine some of the complex disorders that are hypothesised to result from the disconnection of modules.

Brain damage can result in a number of specific delusional beliefs including *anosognosia*, which is an unawareness of impairment, leading to denial of disability, and *duplication* or *substitution*, in which things and/or people are claimed to be duplicates or copies of the real object (Stone & Young, 1997). Examples of disorders that combine these features are *somatoparaphrenia*, thinking that your arm is someone else's; *Cotard delusion* (Cotard, 1882), thinking that you are dead; *Frégoli delusion* (Courbon & Fail, 1927), thinking that disguised people are following you; *reduplicative paramnesia* (Luzzatti & Verga, 1996; Pick, 1903), thinking you are somewhere other than where everyone around you claims to be; *Capgras delusion* (Capgras & Reboul-Lachaux, 1923), thinking that someone close to you has been replaced by a duplicate; and *intermetamorphosis*, thinking you have been turned body and soul into someone else (Courbon & Tusques, 1932). In contrast to the wide-ranging delusions often seen in schizophrenia these delusions are 'monothematic and often circum-

scribed' (Stone & Young, 1997, p. 329). Delusions of this type have all been found to follow damage to the right hemisphere of the brain.

Patients with Capgras delusion typically believe that someone close to them has been replaced by a duplicate. The condition is believed to be rare, having an incidence rate of about 0.12 percent (Dohn & Crews, 1986). The delusion has been found to co-occur with other disorders such as obsessive-compulsive disorder (Sverd, 1995) and schizophrenia (Silva & Leong, 1992). Delusional misidentification may in fact be a fairly common feature in schizophrenia (Walter-Ryan, 1986). V. S. Ramachandran has discussed the case of a man, 'Arthur', who suffered a car accident and thereafter became convinced that his parents had been replaced by well-intentioned impostors (Ramachandran & Blakeslee, 1999, pp. 159-173). The patient could think of no reason why someone should pretend to be his parents but speculated that the impostors were employees of his real father. Significantly, the patient did not treat either of his parents as impostors when he spoke to them on the telephone. Ramachandran also describes the case of a man who believed his pet poodle to have been replaced by an impostor, and there is a report of a case in which a woman believed her cat to have been replaced by a duplicate that was ill-intentioned towards her (Reid, Young & Hellawell, 1993). Although some cases of Capgras have a relatively benign outcome, others do occasionally have very serious consequences. One man who was convinced that his stepfather had been replaced by a robot decapitated the man in order to search his skull for tell-tale microchips (Ramachandran & Blakeslee, 1999, p. 166; Silva, et al., 1989).

Ramachandran decided to test the hypothesis that Arthur would have normal face recognition, but an impaired emotional response, by using a measurement of galvanic skin response (GSR). Arthur shown a series of pictures of his parents interleaved with those of strangers, and measurements were also taken from six individuals who served as controls. Those in the control group showed large differences in the GSR in response to pictures of their parents, but Arthur showed a uniformly flat response to all of the pictures. Further tests showed that Arthur had no deficit in his ability to recognise and compare faces, and that he had a full range of human emotions that were appropriately expressed. As Ar-

thur had no deficit in either his capacity to experience emotion or his ability to recognise faces most plausible explanation was that he was impaired in his ability to link the two. Patients with Capgras delusion differ from those with frontal lobe damage and those with damaged amygdalas, who show uniformly low GSRs and no emotional response, because they do have normal emotional experiences and therefore have a baseline for comparison. As Ramachandran explains:

> This idea teaches us an important principle about brain function, namely, that all our perceptions – indeed, maybe all aspects of our minds – are governed by comparisons and not by absolute values. This appears to be true whether you are talking about something as obvious as judging the brightness of print in a newspaper or something as subtle as detecting a blip in your internal emotional landscape… You can discover important general principles about how the brain works and begin to address deep philosophical questions by doing relatively simple experiments on the right patients. We started with a bizarre condition, proposed an outlandish theory, tested it in the lab and – in meeting objections to it – learned more about how the healthy brain actually works (Ramachandran & Blakeslee, 1999, p. 167).

Ramachandran's observation that Arthur did not suspect his parents of being impostors when speaking to them by telephone implied that a separate dissociation between voice recognition and emotion could also take place (Hirstein & Ramachandran, 1997). As there are separate pathways from the auditory regions of the temporal lobe to the amygdala this possibility had long been acknowledged. The existence of this auditory form of Capgras delusion has recently been confirmed by Lewis and colleagues (2001) whose patient H. L displayed normal autonomic responses for faces but reduced autonomic responses for famous voices. The disorder of *prosopagnosia*, in which individuals fail to recognize familiar faces, but exhibit normal GSR responses indicative of covert recognition (Ellis, et al., 2000), suggests that this condition is the mirror-image of Capgras delusion (Ellis & Young, 1990). Prosopagnosia appears to be the result of damage to the occipito-temporal regions (Damasio, Damasio & Van Hoesen, 1982), whereas Capgras seems to be the result of parieto-termporal lesions (Stone & Young, 1997, p. 337).

Patients suffering from Cotard delusion believe that they are dead; will sometimes ask to be buried, and often claim to smell rotten flesh. They may also speak in sepulchral tones or be completely mute; may not respond to threatening gestures or noxious stimuli (Weinstein, 1996, p. 20-21), and may be akinetic and refuse to eat (Silva, et al., 2000). There seems to be no difference between men and women in terms of clinical profile, and the risk of developing the condition increases with age (Berrios & Luque, 1995b), though one case in a prepubescent child has been reported (Allen, et al., 2000). Though Cotard delusion can be considered a distinct syndrome it is best viewed as a symptom that can occur in a number of mental disorders where nihilistic delusions are present (Young & Leafhead, 1996, p. 150). Cotard himself seems to have believed condition to be a subtype of depression (Berrios & Luque, 1995a). Many suffering from depression, for example, often speak of themselves as feeling like the 'living dead', and patients with schizophrenic symptoms sometimes claim that they have ceased to be human. Young and Leafhead write: 'Feelings of lack of emotional responsiveness, unreality of events, detachment from the world, strangeness and unfamiliarity were prominent features in our clinical cases, and they frequently crop up in reports of the delusion of being dead or preoccupation with death… we think that their significance is often underestimated' (1996, p. 164). Ramachandran has suggested that Cotard delusion is an exaggerated form of Capgras delusion. Instead of a disconnection between face perception and emotion Cotard delusion may be caused by a complete disconnection of sensory areas and the limbic system resulting in a complete lack of emotional contact with the world. If this hypothesis is correct then people with Cotard delusion should show a complete lack of GSR response to all external stimuli. Unfortunately, the necessary experiments have not yet been carried out, though clinical case studies yield much information that is consistent with Ramachandran's hypothesis.

Stone and Young (1997) propose that patients with the Cotard and Capgras delusions are unable to correct their mistaken perceptions because they also have a biased attributional style as well as a fundamental cognitive deficit. This biased style affects the way in which unusual perceptual experience is misinterpreted. Persecutory delusions and suspiciousness are noted in cases of Cap-

gras delusion because ‘forming an account in terms of impostors [arises] because of a more general tendency to attribute negative events to external causes’ (1997, p. 345) whereas those with Cotard are believed to be predisposed toward attributions to internal causes, resulting in depressive symptoms. However, it seems unnecessary to appeal to the skewed or faulty functioning of other systems to account for the symptoms of these disorders. It is more parsimonious, and in keeping with the idea of the mind as completely modular, simply to account for these symptoms in terms of cybernetic dysfunction. The inability of other systems to compensate for malfunction in a core module leads inevitably to malfunction in ‘downstream’ modules, though of course the particular content and explanation of any delusion will reflect the patient’s prior knowledge and experiences.

In the discussion of theory of mind left-hemisphere pathologies were implicated in a number of dysfunctions. It is interesting to note that various forms of delusional misidentification are generally associated with the right hemisphere. Although cognitive neuropsychiatry can help clarify the relationship between specific pathologies and specific cognitive deficits highly localized damage is extremely rare. The heterogeneity of many mental disorders, especially schizophrenia, is probably explained by the fact that these syndromes encompass signs and symptoms arising from the simultaneous disruption of many different systems.

Psychopathy: Pathology or Adaptation?

Philippe Pinel (1745-1826) used the term *insanity without delirium* to describe behaviour that was marked by complete remorselessness, but the modern concept of ‘psychopathy’ was put forward by Hervey Cleckley (1903-1984) in his classic work *The Mask of Sanity* (1941). According to Cleckley’s criteria a psychopath is an intelligent person characterised by poverty of emotions, who has no sense of shame, is superficially charming, is manipulative, who shows irresponsible behaviour, and is inadequately motivated. Interspersed in Cleckley’s vivid clinical descriptions are phrases such as ‘shrewdness and agility of mind,’

'talks entertainingly,' and 'exceptional charm' (Hare, 1993, p. 27). Cleckley also provides a striking interpretation of the meaning of the psychopath's behaviour:

> The [psychopath] is unfamiliar with the primary facts or data of what might be called personal values and is altogether incapable of understanding such matters. It is impossible for him to take even a slight interest in the tragedy or joy or the striving of humanity as presented in serious literature or art. He is also indifferent to all these matters in life itself. Beauty and ugliness, except in a very superficial sense, goodness, evil, love, horror, and humour have no actual meaning, no power to move him. He is, furthermore, lacking in the ability to see that others are moved. It is as though he were colour-blind, despite his sharp intelligence, to this aspect of human existence. It cannot be explained to him because there is nothing in his orbit of awareness that can bridge the gap with comparison. He can repeat the words and say glibly that he understands, and there is no way for him to realize that he does not understand (Cleckley, 1941, p. 90 quoted in Hare, 1993, pp. 27-28).

The terms 'psychopathy' and 'sociopathy' are used interchangeably with the latter often being used to avoid confusion with psychoticism and insanity, though the choice of term also often reflects the user's views on whether the determinants of the condition are psychological, biological, and genetic on the one hand or social forces and early experience on the other (Hare, 1993, p. 23). The DSM category of *antisocial personality disorder* (introduced in *DSM-III*, 1980) was supposed to have had covered psychopathy, but because clinicians were not thought sufficiently competent to assess personality traits the *DSM* definitions have concentrated on the antisocial and criminal behaviours associated with the condition. This has blurred the distinction between psychopaths and criminals, and of course most of the latter are not psychopaths. Antisocial Personality Disorder (category 301.7) is described in *DSM-IV* simply as 'a pervasive pattern of disregard for, and violation of, the rights of others that begins in childhood or early adolescence and continues into adulthood… This pattern has also been referred to as psychopathy, sociopathy, or dyssocial personality disorder' (American Psychiatric Association, 1994, p. 645). This confusion of terminology is especially damaging for research because whereas DSM-IV describes APD as 'associated with low socio-economic status' (1994, p. 647) psy-

chopathy ‘seems *less* likely to be associated with social disadvantage or adversity’ (Rutter, Giller & Hagell, 1998, p. 110).

Robert Hare has described his attempts to identify true psychopaths as a prison psychologist in the early 1960s. Most of the personality ‘measures’ or ‘instruments’ popular at that time, such as the Minnesota Multiphasic Personality Inventory (MMPI), were questionnaires based on self-reporting. When administered to psychopaths, who are expert at ‘impression management’ (Hare, 1993, p. 30) these instruments are less than reliable. One of the inmates in Hare’s research program even had a complete set of MMPI tests and interpretation manuals and, for a fee, would advise fellow inmates on the correct answers to show the steady improvement more likely to lead to parole. Another inmate ‘had an institutional file that contained three completely different MMPI profiles. Obtained about a year apart, the first suggested that the man was psychotic, the second that he was perfectly normal, and the third that he was mildly disturbed’ (Hare, 1993, p. 31). Each of these profiles had been treated as genuine, but each had in fact been produced to meet specific objectives: the inmate’s desire first to transfer to a psychiatric hospital, then to transfer back to the main prison after he found that conditions were not to his liking, and finally to secure a supply of Valium. Hare decided to construct his own *Psychopathy Checklist* in order to have a method of separating psychopaths from the rest of the prison population, and this method is now used throughout the world. The *Checklist* highlights the key emotional and interpersonal symptoms of psychopathy: psychopaths are said to be glib and superficial; egocentric and grandiose; to lack remorse or guilt; to lack empathy; to be deceitful and manipulative; and to have shallow emotions. In terms of social deviance the psychopath is also said to be impulsive; to have poor behavioural controls; to need excitement; to show lack of responsibility; to show early behaviour problems, and to demonstrate adult antisocial behaviour problems (Hare, 1993, pp. 34-82).

It is difficult to appreciate just how different the functioning of psychopaths is compared to that of the non-psychopath. After killing a waiter who had asked him to leave a restaurant Jack Abbott denied any remorse because he hadn’t done anything wrong, and after all ‘there was no pain, it was a clean wound’

and the victim was ‘not worth a dime’ (Hare, 1993, pp. 42-3). The psychopathic serial killer John Wayne Gacy murdered thirty-three young men and boys, but described himself as the victim because he had been robbed of his childhood. Kenneth Taylor battered his wife to death and then couldn’t understand why no one sympathised with his tragic loss. One woman allowed her boyfriend to sexually abuse her five-year-old daughter because she was too tired for sex, but then was outraged that social services should have the right to take the child into care. Diane Downs murdered her three children, wounding herself in the process in order to provide evidence for story of an attack by a stranger. Asked about her feelings regarding the incident Downs replied ‘I couldn’t tie my damned shoes for about two months… The scar is going to be there forever… I think my kids were lucky’ (Hare, 1993, p. 53, quoted from The Oprah Winfrey Show, September 26, 1988). Clinicians refer to the emotions of psychopaths as proto-emotions, that is, primitive responses to immediate needs. Hare remarks:

> Another psychopath in our research said that he did not really understand what others meant by “fear”. However, “When I rob a bank,” he said, “I notice that the teller shakes or becomes tongue tied. One barfed all over the money. She must have been pretty messed up inside, but I don’t know why. If someone pointed a gun at me I guess I’d be afraid, but I wouldn’t throw up.” When asked to describe how he *would* feel in such a situation, his reply contained no reference to bodily sensations. He said things such as, “I’d give you the money”; “I’d think of ways to get the drop on you”; “I’d try and get my ass out of there.” When asked how he would *feel*, not what he would think or do, he seemed perplexed. Asked if he ever felt his heart pound or his stomach churn, he replied, “Of course! I’m not a robot. I really get pumped up when I have sex or when I get into a fight” (Hare, 1993, pp. 53-4).

The prevalence of APD is estimated at three percent in males and one percent in females (American Psychiatric Association, 1994, p. 648), but the rate of psychopathy according to the Cleckley/Hare criteria is probably about one percent (Hare, 1993, p. 74). Half of all serial rapists may be psychopaths (Prentky & Knight, 1991). The recidivism rate of psychopaths is roughly double that of non-psychopathic offenders, and the violent recidivism rate is about triple that of other offenders (Hare, 1993, p. 96). Insight-oriented therapies actually appear to make psychopaths (but not non-psychopaths) more likely to recidivate (Quinsey

& Lalumière, 1995; Rice, et al., 1999), possibly because psychopaths use psychotherapy sessions to develop their skills in psychological manipulation, and because they see no need to change their admirable personalities (Hare, 1993, pp. 192-206). Because of a lack of research and the confusion over terminology it is not clear whether there are differences between males and females in the prevalence of psychopathy. However, Hare estimates that abut 20 percent of male and female prison inmates are psychopaths and that psychopaths are responsible for more than 50 percent of the serious crimes committed (1993, p. 87). Cloninger's 'two-threshold' model suggests a polygenic and sex-limited contribution to psychopathy according to which more men than women would pass the threshold for activation of predisposing genes. This model predicts that males should be more susceptible to environmental influences and females who do become psychopathic should have a greater genetic predisposition; this is confirmed by the finding that the offspring of female psychopaths are more vulnerable than those of male psychopaths (Cloninger, Reich & Guze, 1975; Mealey, 1995, pp. 526-7). As Mealey explains,

> The two-threshold model thus explains in a proximate sense what sociobiologists would predict from a more ultimate perspective. The fact that males are more susceptible than females to the environmental conditions of their early years fits well with sociobiological theory in that the greater variance in male reproductive capacity makes their "choice" of life strategy somewhat more risky and therefore more subject to selective pressures (Buss, 1988; Mealey & Segal, 1993; Symons, 1979). Sociobiological reasoning thus leads to the postulate that males should be more sensitive to environmental cues that (1) trigger environmentally contingent or developmentally canalised life history strategies or (2) are stimuli for which genetically based individual differences in response thresholds have evolved (Mealey, 1995, p. 527).

In the previous chapter and in the earlier section on theory of mind I discussed Chisholm's model of the development of alternative reproductive strategies being contingent on environmental risk and uncertainty. This model was built upon work by Draper and Harpending (1982) on the relationship between adolescent reproductive strategies and father absence. The optimality of any reproductive strategy is dependent on local environmental contingencies. In addition to the cue for reproductive strategies provided by father absence Chisholm suggests

that a socioassessment can be communicated via the attachment process, and that the nature of this socioassessment can have an impact on variance in reproductive strategies including age at menarche, age at first sexual activity, and number of mating partners. A poor socioassessment can contribute to the patterns of behaviour identified as the Young Male/Female Syndromes. A similar model has been proposed by Belsky, Steinberg, and Draper (1991) in which the developmental trajectory is part of a reproductive strategy 'hypothesized to be associated with earlier timing of puberty, earlier onset of sexual activity, unstable pair bonds, and limited parental investment' (Belsky, 1995, p. 545). Linda Mealey argues that males who are 'competitively disadvantaged with respect to the ability to obtain resources and mating opportunities… who are least likely to outcompete other males in a status hierarchy, or to acquire mates through female choice are the ones most likely to adopt a cheating strategy' (1995, p. 527). Harpending and Sobus (1987) predicted that human cheaters should have the following traits

> Human cheaters would not be detectable by instruments routinely available to his or her conspecifics… [and] should be very mobile during their lifetimes. The longer a cheater interacts with the same group of conspecifics the more likely they are to recognise the cheater's strategy and to refuse to engage in interactions with him or her. There will be costs of mobility, since the mobile cheater will have to learn a new social environment after a move, and he or she will need to be skilled at it. A third prediction is that human cheaters would be especially facile with words, language, and interpersonal empathy… Human male and female cheaters should exhibit very different patterns of cheating, reflecting the obligate mammalian dimorphism in reproductive strategy and potential. A male cheater should be especially skilful at persuading females to copulate and at deceiving females about his control of resources and about the likelihood of his provisioning future offspring. Females, on the other hand, should feign lack of interest in copulation in order to deceive males about their paternity confidence. They should also exaggerate need and helplessness in order to induce males to provide them with more resources and support then they might otherwise provide. Finally, female cheaters might abandon offspring as soon as they perceived that the chance of offspring survival exceeded some critical value (Harpending & Sobus, 1987, 65S-66S).

In Mealey's terminology *primary sociopaths* are biologically *contraprepared* to learn empathy and consequently demonstrate psychopathic behaviour at an early stage, whereas *secondary sociopaths* encounter a combination of risk factors such as a large number of siblings, low socio-economic status, urban residency, low intelligence and poor social skills. These variables contribute to the development of secondary sociopathy in a two stage process involving initially parental neglect, abuse, inconsistent discipline, and punishment as opposed to rewards. In the second stage children may be at a social disadvantage because of poor social skills and may therefore interact primarily with a peer group comprised other unskilled individuals, including primary sociopaths. Mealey hypothesises that 'antisocial behaviour may then escalate in response to, or as a prerequisite for, social rewards provided by the group' (1995, p. 534). According to Mealey primary sociopaths are 'designed for the successful operation of social deception and… are the product of evolutionary pressures which… lead some individuals to pursue a life strategy of manipulative and predatory social interactions' (Mealey, 1995, p. 524). Primary sociopathy is thus a frequency-dependent adaptation, but secondary sociopathy is a facultative cheating strategy.

The ethologists Eibl-Eibesfeldt (1970) and Lorenz (1966) proposed mechanisms that limit aggression in social animals, and an alternative model of psychopathy based on this research has been put forward by James Blair (1995). In animals such as dogs, who bare their throats when attacked by a stronger opponent, a display of such submission cues results in a termination of the attack. Blair has proposed a functionally analogous mechanism in humans: a violence inhibition mechanism (VIM) that would be activated by non-verbal communications of distress. This mechanism is said to be a prerequisite for the development of three aspects of morality: the moral emotions (such as sympathy, guilt, remorse and empathy), the inhibition of violent action and the moral/conventional distinction. Blair has suggested that psychopaths lack a functional VIM and could not be negatively reinforced by distress cues and further predicted '(1) that psychopaths will not make a distinction between moral and conventional rules; (2) that psychopaths will treat moral rules as if they were conventional; that is, under permission conditions, the psychopaths will say that moral as well as conven-

tional transgressions are OK to do; (3) that psychopaths will be less likely to make references to the pain or discomfort of victims than the non-psychopath controls' (Blair & Morton, 1995, p. 13). Using subjects identified by Hare's *Psychopathy Checklist* Blair found that

> …while the non-psychopaths made the moral/conventional distinction, the psychopaths did not; secondly, and in contrast with predictions, that psychopaths treated conventional transgressions like moral transgressions rather than treating moral transgressions like conventional transgressions; and thirdly, and in line with predictions, that psychopaths were much less likely to justify their items with reference to victim's welfare (Blair & Morton, 1995, p. 20).

As Blair and Morton note 'this study has not proven that psychopaths lack VIM, [but] it has provided evidence that is in line with the position' (1995, p. 25).

Mealey has proposed two different aetiologies for sociopathy, but in her framework those displaying chronic antisocial *behaviour* are placed in the same functional category. This implies that they have similar or identical psychological mechanisms. On the other hand, Blair concentrates on the mechanisms subserving psychopathic behaviour, but concludes that psychopaths have a dysfunctional psychological mechanism and are disordered in comparison to other members of society. With Blair I believe that psychopaths do have very different psychological mechanisms, but with Mealey I believe that these mechanisms may well be the result of a frequency-dependent adaptation. Most of those who meet the criteria for Antisocial Personality Disorder do not fall into this second category, and research that fails to distinguish to distinguish between these categories is likely to be extremely misleading. In one significant study it was found that the *Psychotherapy Checklist* could not distinguish between psychopathic and schizophrenic offenders in 50 consecutive male admissions to an English Special Hospital (Howard, 1990). This may indicate that some schizophrenics with a history of antisocial behaviour are suffering from what could be called *state-dependent psychopathy*. These individuals would probably not meet the criteria for either primary or secondary sociopathy as discussed by Mealey and others. In terms of appropriate scientific, psychological, social and

therapeutic approaches to psychopathy it is clearly essential to distinguish between the different aetiologies involved.

What is most outstanding about psychopaths is that they appear extremely at ease with themselves. They can be articulate, are often highly intelligent, and are regularly described as ‘charming’, and ‘convincing’. Psychopathy is not associated with low birth weight, obstetric complications, poor parenting, poverty, early psychological trauma or adverse experiences, and indeed Robert Hare remarks ‘I can find no convincing evidence that psychopathy is the direct result of early social or environmental factors’ (Hare, 1993, p. 170). No sound evidence of neuroanatomical correlates for psychopathic behavior has been found, though an interesting (and highly significant) negative correlation has been found in 18 psychopaths between the degree of psychopathy as assessed by the *Checklist* and the size of the posterior half of the hippocampi bilaterally (Laakso, et al., 2001). Lesions of the dorsal hippocampus have been found to impair acquisition of conditioned fear, a notable feature of psychopathy, but it is not clear whether this neuroanatomical feature is the cause of, or is caused by, psychopathy. A study of 69 male psychopaths identified by the revised edition of Hare’s *Psychopathy Checklist* found no support for the hypothesis that psychopaths are characterized by verbal or left hemisphere dysfunction (Smith, Arnett & Newman, 1992). One particularly striking feature of psychopathy is that extremely violent and antisocial behaviour appears at a very early age, often including casual and thoughtless lying, petty theft, a pattern of killing animals, early experimentation with sex, and stealing (Hare, 1993, p. 158). In a study of 653 serious offenders by Harris, Rice, and Quinsey childhood problem behaviors provided convergent evidence for the existence of psychopathy as a discrete class, but ‘adult criminal history variables were continuously distributed and were insufficient in themselves to detect the taxon’ (1994, p. 387). In a recent study psychopathic male offenders were found to score lower than nonpsychopathic offenders on obstetrical problems and fluctuating asymmetry, and in fact the offenders meeting the most stringent criteria for psychopathy had the lowest asymmetry scores amongst offenders (Lalumière, Harris & Rice, 2001). As the authors note this study provides no support for the idea that psy-

chopathy results from developmental stability of some kind, but does give partial support for life-history strategy models.

An evolutionary game-theoretic explanation for the low but stable prevalence of psychopathy has been modelled successfully (Colman & Wilson, 1997), and though this provides some tentative support for Mealey's suggestion that psychopathy is a frequency-dependent strategy, cross-cultural work using reliable measures will be needed to establish whether there is a stable proportion of sociopaths in traditional societies (Archer, 1995). Given the paucity of evidence in favour of developmental instability and brain damage in psychopaths the suggestion that psychopathy is an adaptation is worthy of further exploration. Particular attention should also be paid to the probability that child psychopaths are mislabelled as suffering from Attention Deficit Hyperactivity Disorder, Conduct Disorder (see American Psychiatric Association, 1994, p. 85), or Oppositional Defiant Disorder (see American Psychiatric Association, 1994, p. 91). According to Hare 'none of these diagnostic categories quite hits the mark with young psychopaths. Conduct disorder comes closest, but it fails to capture the emotional, cognitive, and interpersonal personality traits… that are so important in the diagnosis of psychopathy' (1993, p. 159).

A Taxonomy of Modular Disorders

Although a taxonomy based on the principles outlined in this chapter would take many years of research work to compile Dominic Murphy and Stephen Stich have suggested a high-level classification of mental disorders based on the core ideas of evolutionary psychology. They agree with the emphasis placed on modularity or the idea of 'interconnected processing systems' some of which may 'have proprietary access to a body of information that is useful in dealing with its domain' (Murphy & Stich, 2000, p. 63). In their view the mind is composed of domain-specific modules, general-purpose modules, proprietary and non-proprietary stores of information, and 'a variety of other sorts of mechanisms' (2000, p. 65). Their first category of mental disorders contains those hypothesised to result from pathology internal to the module. The second category includes disorders caused as a result of faulty information received from a bro-

ken upstream module. If a number of downstream systems receive inputs from such a module there may be a variety of different clusters of symptoms. This phenomenon could constitute one explanation of the high rate of co-morbidity found in psychiatric medicine, an assessment of which was provided by a survey of common psychiatric syndromes published by the *British Journal of Psychiatry* in 1998. Patrick Sullivan and Kenneth Kendler found that: 'The *DSM-III-R* and closely related *DSM IV* nosology did not capture the natural tendency of these disorders to co-occur. Fundamental assumptions of the dominant diagnostic schemata may be incorrect' (1998, p. 312). Of the 1898 female twins they studied 62.3 percent had two or more disorders. Sullivan and Kendler's opinion of the validity of *DSM* nosology is comparable to that expressed in chapter three.

> Psychiatric classification has been heavily influenced by the views of certain advocates or by expert consensus. Given the profound influence of the dominant psychiatric classification schema on clinical practice and research, it is remarkable that empirical study has played a relatively minor role in the overarching nosological questions concerning these syndromes. Moreover, most nosologies have been based on the analysis of clinical samples despite substantial evidence that such samples are biased subsets of the general population (Sullivan & Kendler, 1998, p. 316).

Murphy and Stich's third category covers those disorders caused by the mismatch between current and ancestral environments. The final category consists of 'disorders that may not be', that is, disorders that are probably adaptations. As Murphy and Stich conclude 'one of the virtues of the evolutionary approach to psychopathology is that, in some cases at least, it provides a principled way of drawing the distinction between mental disorders and patterns of antisocial behaviour produced by people whose evolved mind are beset by no problems at all' (2000, p. 92).

Conclusion

Toward the end of the nineteenth century W. Lloyd Andriezen, Pathologist and Assistant Medical Officer of the West Riding Asylum published a long and detailed paper in the journal *Brain* 'On Some of the Newer Aspects of the Pathol-

ogy of Insanity' (1894). Andriezen applauded the growth of the scientific method in psychology in the 'spirit of Darwin', which he believed had helped to bring the discipline closer to neurology:

> The gradual recognition of the inadequacy in the methods of the older metaphysico-psychological writers, and the increased interest in the study of the brain and nervous system themselves by various physiological and pathological methods, and the further feeling that an attempt should be made to correlate these with the actual activities of life, growth, and conduct of the individual – all these are slowly working towards the desired result… But little progress could be said to have been made in the study of insanity and its treatment, till physicians came to look at it in precisely the same way as they did ordinary disease; to study mind as a brain function which is found in nearly all animals in varying degrees; which in man arises from small beginnings like any other function, then gradually develops and attains the acme of its complexity and activity in adult life, and finally fails and disappears with the decay of old age – in a word, as distinctly correlated with the anatomico-physiological development, growth, and decay of the brain and the nervous system (Andriezen, 1894, pp. 548-9).

Towards the end of his paper Andriezen extolled the virtues of biological studies in the development and life histories of nervous systems throughout the animal kingdom combined with sociological studies of hereditary as influencing mental as well as physical traits. Indeed, Andriezen argues that 'the intrinsic vice of organisation is within, and requires but little stress of circumstances to reveal itself' (1894, p. 686). Amongst the stressors discussed are chemical poisons such as alcohol and psychological/behavioural phenomena such as sexual excess and worry. Although Andriezen's model was compelling (though clearly enmeshed in the nature/nurture dichotomy), far-sighted, and perceptive the implausibility of Darwin's model of heredity and the rise of alternative schools of thought in psychology, together with the perennial inclination toward the 'separation of contradictory things' derived from the Western philosophical and theological traditions contributed to the submergence of Darwinian ideas in psychology and psychiatry.

Unfortunately, although Darwinian ideas are now thriving in psychiatry and psychology the overall framework adopted by many falls into the scheme of dichotomous approaches criticised in chapter two. As Oyama puts it,

> The search goes on for the chimerical genetic essences underlying individual or species characteristics. We doggedly stalk through the phylogenetic underbrush and the thickets of heritability coefficients, pursuing the hidden reality that will unify and categorize the variety of the living world. Whether the spectral essence is sought in the form of programmed development toward genetic templates, universal repertoires of unlearned behavior or inherited core temperaments, the form of the questions we put to nature becomes numbingly familiar, in spite of increasingly impressive jargon and obligatory disclaimers. (Oyama, 1985, pp. 108-109)

I have endeavoured to demonstrate that the perspective of evolutionary developmental psychopathology can help us to understand the development, malleability, and impairment of psychological mechanisms across the lifespan. In particular, I have argued extensively that the application of theories of parental investment and parent-offspring conflict can yield insights into many aspects of psychopathology and normal psychological functioning. I have identified mechanisms that could account for some of the sex differences in mental disorders, and have suggested a novel interpretation of the connection between the conditions prevailing during pregnancy and the physiological, psychological, and behavioural predispositions and characteristics of offspring. In contrast to contemporary psychiatry's emphasis on the search for 'inborn errors' I have indicated that a search for pathogenic features of the current environment, including the social environment, should also be a worthwhile endeavour. Furthermore, I have attempted to establish that a nosological schema based on the general principles of evolutionary developmental psychopathy should allow us to delineate the projectable categories that will provide insights into the aetiology and pathophysiology of conditions that cannot be revealed by psychiatric research within the atheoretical framework advocated by traditional biological psychiatry. Indeed, I believe that an appreciation that minds consist of developmentally plastic, polymorphic, and sexually dimorphic psychological mechanisms, which are subserved by distributed neural components that participate in

more than one faculty, provides an eminently coherent basis for research into human nature and the nature of psychopathology.

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