

The Commentators
What your genes reveal about you
Richard Dawkins

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Polly Toynbee is my hero, a journalistic knight in shining armour. To tone down the metaphor, she can usually be relied upon to hit any nail squarely on the head. But nobody is perfect, and even the best-placed and best-intentioned thumb occasionally sports a bruise. Her column last Thursday, "The nature of our DNA will always lag behind nurture", must not pass without correction, and I offer it in a spirit of pained admiration.

Owing to some weird tribal or union practice of the newspaper world, authors don't write their own headings, so Polly Toynbee cannot be blamed for: "Gene testing is pointless. Our fates are more likely to be shaped by our postcodes". Nevertheless, it is an accurate reflection of what she wrote.

But even if the postcode remark were true, gene testing wouldn't be pointless. Insurance actuaries would still want to read our genes, just as they record our smoking habits though the link between smoking and disease is statistical, not absolute. Both genes and smoking contribute to your risk. Nature and nurture are not competitors such that one wins and the other loses.

She gives the game away when she misuses the word "predictor": "Even those genes that show a strong disposition to specific conditions such as heart disease are not predictors. If those who know they are in danger eschew a diet of fried Mars bars they will not die of heart disease."

But we can simultaneously say, without contradiction: Even those environmental factors that show a strong disposition to specific conditions such as heart disease are not predictors. If you are fortunate in your genes you can eat fried Mars bars all day and you won't die of heart disease.

Both diet and genes contribute to the probability of heart disease. So do other factors such as stress. In the real world, prediction means statistical prediction, prediction of probability. When a tipster recommends a particular horse, he weighs up the past form of all the horses, adding in rumours and stable lad gossip together with expectations about the "going" (which in turn depend upon statistical weather forecasts). The result is a probability. On average you'll win more by following a good tipster than by betting completely at random.

When a successful racehorse is sold for stud, his purchaser is betting (lots of) money on a statistical prediction about the horse's genes. If there never had been genes for racing ability in horses, there wouldn't be a separate breed of racehorse at all. By analogy, there wouldn't be separate breeds of trotting horses, carthorses, polo ponies, sheepdogs, gundogs, or fighting bulls.

Is Homo sapiens some sort of bizarre exception to the rule? That doesn't sound a very Toynbeeish suggestion. She says "there is no gene for intelligence . . ." but she qualifies this by adding "there are a large number of genes responsible . . ." Yes indeed, but there are a large number of genes responsible for running speed in horses, milk yield in cows, pugnacity in fighting cocks and pit bull terriers. Why should the presence of many genes make prediction any less feasible?

Without getting into the notorious problems of defining intelligence, we can prove to ourselves that, in a powerfully predictive sense, there have been genes for intelligence for millions of years of human history. All you must assume is that, by whatever definition you are prepared to accept, we are more intelligent than our ape ancestors. Certainly our brains are spectacularly bigger than our fossil ancestors'. Right then, how has that evolutionary change come about?

There is only one way: genes for intelligence (or whatever you want to call the qualities that separate us from our ancestors) have been favoured in the gene pool. No evolutionary change in X can take place unless there are genes for X varying in the population. It follows that, during the

millions of years in which we have been pulling ahead of our ape ancestors, some of us have been brighter than others, and it has been predictable from our genes. To deny that, you must deny Darwinism, something that a person of Polly Toynbee's education and intelligence will not do, however tempted by liberal good intentions.

Genes are important causal agents, combining with other genes and with environmental agents in the statistical determination of our abilities. The way in which they combine is best understood in terms of the statistical technique called "analysis of variance". Improvement in our understanding of the world is equivalent to an increase in our ability to predict outcomes as we take into account more causal agents. Equivalently, our uncertainty is progressively reduced, measured as increasing proportions of variance explained.

Variance is a measure of how variable a population is, and therefore how ignorant we are about any random member of it. The total variance is the sum of variance due to diet, due to education, due to genes, due to this, that or the other, plus finally a residue of unexplained variance. If all you know is that I am human, your best guess of my ability to run a mile is that I am average for the whole population. But your confidence is negligible. The population includes everything from aged cripples to babes in arms: the variance is large, and at this stage it all lies in the unexplained residue.

If I now tell you that I am male, in my twenties and in regular training, your confidence increases as portions of variance are shifted from unexplained residue to explained categories. If I now tell you that my father is called Roger Bannister, your estimate and your confidence change again. With each new piece of information, whether genetic or environmental, the unexplained variance decreases and the accuracy of the prediction increases.

There is a complication. Not all variance is "additive". Sometimes there are "interactions". In the statistical sense, this means something other than addition, and it is often treated as equivalent to multiplication. If a boy is very slightly cleverer than his brother for genetic reasons, the difference may be just enough to push him, but not his brother, through the eleven-plus and into grammar school. The eventual result of this may be that one brother becomes a professor, the other an unemployed labourer.

The difference in their genes is nowhere near enough to account for the final difference in worldly success. It has been multiplied by a threshold effect, the eleven-plus examination. The genes' contribution to the variance is no longer simply additive. There is a "genes x education" interaction. In the full analysis of variance, the total variance is partitioned into its additive components (genes, diet, education, etc) plus all the interactions (genes x education, genes x diet, education x diet, etc). Once again, there is a residue of unexplained variance, but we have reduced it by subtraction not only of the additive components but of the interactions too.

Interaction is a technical expression of what we see as "unfair" or "double jeopardy". Some people are disadvantaged by their genes and this will affect their lives and their health. That's bad enough. If insurance companies are allowed to use this information to penalise them again, they will end up penalised twice. Polly Toynbee is right - and typically so - to call for legal sanctions to make insurers spread the risk. Spreading risk is, after all, what insurance is all about. But we shan't help anybody, and might play into the hands of unscrupulous insurers, if we falsely underestimate the importance of genes.

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