How much can genetics tell us about the causes of crime and violence?

Joe Schwartz debunks the assumptions behind studies into the genetic causes of criminal behaviour.

In most ways, science has replaced religion as a way to understand the world. The efficacy of science is due to the fact that it locates cause and effect in the material world, an approach that has yielded invaluable understandings of the world that we inhabit. Yet when we approach the question of the genetics of human behavioural difference we encounter what we call genetic fundamentalism – a belief in a mythic, not a real genetics. This is how it works.

Reports of genetic differences in human behaviours, including crime and violence, fall into three distinct classes. Less than one per cent are based on the isolation of a protein or other biomolecule that can correlate with the observed behaviour. An example of this tiny minority of studies might be the possibility that a deficient thyroid receptor protein could make a child vulnerable to hyperactivity.

A second class of studies, currently the most popular, is based on theoretical evolutionary arguments. The argument is simple. One simply assumes that the behaviour in question is adaptive. This is the basis, for example, of the infamous Thornhill and Palmer book justifying rape (Thornhill *et al*, 2000). Rape exists. Therefore it must be adaptive. Such stories are modern 'just so' stories in the tradition of Rudyard Kipling (Gould and Lewontin, 1979).

The third class of studies is based on giving tests or questionnaires to twins. The twin literature on the genetics of differences in IQ performance is based entirely on this procedure (Plomin, 1999 and 1991).

All three approaches have been abused by zealots of genetic explanation. But that by itself does not necessarily mean we have nothing to learn from their efforts. Let us see for ourselves.

Class 1: Assays of biochemicals

The actual laboratory work correlating the existence of specific biochemicals to a human behavioural condition is the only work that connects to a real instead of an imagined genetics but such studies must be carefully controlled which often they are not.

A famous cautionary example is given by the Nobel Prize-winner Julius Axelrod (Healy, 1996). In a project trying to establish whether there were any biochemicals specifically associated with the condition diagnosed as schizophrenia, Axelrod's group came up with a striking result. A chromatographic analysis of the urine samples of a group of schizophrenics always showed two pink spots compared to controls. Many less capable investigators would have rushed into print with this result claiming that a metabolite unique to schizophrenics had been found, possibly of genetic origin. The newspapers would then have swung into action with predictable stories about a possible cure for schizophrenia being found. But Axelrod was too good a biologist to believe that a complex condition like schizophrenia could be reduced to two pink spots. As he recalled: "It was too good to be true". Instead he made a very close examination of the diets of his two groups. He found that the control group was a group of Mennonites who didn't drink coffee. What the chromatographic analysis had picked up was the presence of a harmless metabolite of coffee in the urine of the schizophrenic sample which was absent in the urine of the Mennonites.

Class 2: Conclusions based on evolutionary arguments about adaptation

This class of studies can be discarded completely. They are nothing more than stories after the fact.

The complexities of human motivation do not figure in these adaptationist accounts of human action. Consider the case of Jones and his umbrella (Fodor, 2005). Jones is observed to carry an umbrella. Is such behaviour adaptive? We can only begin to speculate on the adaptive nature of Jones' carrying his umbrella if we know why, in fact, Jones is carrying his umbrella. If Jones thinks it will rain we could say this is adaptive because Jones' ancestors survived better by keeping dry. On the other hand perhaps Jones is simply returning Smith's umbrella. In that case we need to find another reason for why returning things would have made Jones' ancestors have greater reproductive success. Or on the third hand perhaps Jones just likes to carry an umbrella because he thinks it is quite sophisticated. Further Jones may not even know why he likes to carry his umbrella.

The evolutionary psychologists' answer to these complexities is that it doesn't matter what Jones thinks or feels. All we have to do, whether Jones likes it or not or whether Jones knows it or not, is to show that Jones' carrying an umbrella maximises the chance that his genes will survive better than another's. And now we are in the realm of pure speculation, in other words a 'just so' story.

Evolutionary psychology has nothing to teach us about any form of human behaviour let alone about the complex, many-faceted origins of human crime and violence. All these arguments do is replace God's will by natural selection.

Class 3: Twin studies

In principle twin studies could provide clues about the possible genetic components to human differences in behaviour. But in practice this field is dominated by academic ideologues who aim to prove that the behaviours in question are genetic rather than trying to find out whether there are genetic factors or not.

The basic methodology is simple enough. A sample of identical twins (MZ twins) is given a set of questions to answer. It can be questions about anything from IQ questions to radicalism to questions about anti-social behaviour. The results of the test to pairs of MZ twins are compared to the results of the same test given to pairs of non-identical twins (DZ twins). If the pairs of identical twins are more similar to each other in test scores than the pairs of non-identical twins are to each other, this extra similarity **is assumed** to be due to the extra genetic similarity

of identical twins.

It cannot be stressed too strongly that the extra similarity **is assumed** to be due to extra genetic similarity. In statistical language such an error is called confusion between hypothesis testing and parameter estimation. In the IQ controversy of a generation ago a reconsideration of the celebrated case of separated identical twins showed that an environmental model with no genetic component at all fit the data equally well (Schwartz and Schwartz, 1974).

For twin studies to have value they need not only to test alternative hypotheses but also to offer the usual experimental controls. For example in MZ-DZ comparisons there needs to a comparison, for example, of DZ twins of the same sex to DZ twins of the opposite sex. Since DZ twins and siblings each have 50 per cent of their genes in common any differences in similarity would have to be due to environmental influence. And such differences in similarity of test scores have been shown routinely to be as large as the reported differences between MZ and DZ twins (Schwartz and Schwartz, 1974). But these controls do not appear to have been offered in any twin study published in the major scientific journals over the past 40 years in spite of extensive criticism in the scientific journals (some selected references are: Fehr, 1969; Moran, 1973; Cohen, 1973; Tizard, 1974; Adams *et al,* 1976).

In general, experimental controls are not considered necessary by the investigators. This is the hallmark of fundamentalist belief. Observed differences seem so self-evidently genetic that any tests of the hypothesis seem redundant. So embedded is this belief system that summer schools, similar to bible study, exist to teach others how to estimate genetic parameters (MRC 2005).

The genetics of crime and violence

The genetics of crime and violence offers its own special challenge to investigators. Usually without stating the case explicitly the crime and violence in question is class based. In white collar crime the genes of the perpetrators are rarely of interest. Street crime and violence are of course of great concern especially in communities where they are most common. But it is only street crime that is given a genetic analysis as in the recent twin study of Viding *et al* of a supposed genetic risk of psychopathy in seven-year-olds (Viding *et al,* 2005). Duly picked up by the media without question, the *Times* then headlined the usual implication: "If psychopaths are born, not made, social policy can't do much to help" (Ahuja, 2005).

Such studies are not worth the paper they are printed on. The investigators have simply assumed, as usual, that a greater similarity of answers to their questions by MZ twins compared to DZ twins is due to the greater genetic similarity of MZ twins. No account is taken of the fact that identicals who look exactly alike are frequently mistaken for each other, and can bond so closely to each other that they develop their own language. No controls are offered in this study nor are environmental alternative models tested. Instead the observed similarities are simply assumed to be genetic.

These fundamentalist presentations of genetic influences on human behaviour belong in philosophy classes alongside discussion of Intelligent Design (Dawkins and Coyne, 2005). They simply do not have the status of controlled scientific studies and are able to pass the ordinary science journal reviews because genetic causation is so deeply embedded in our culture that the reviewers themselves are already convinced of the plausibility of the genetic hypothesis and do not question it further.

However there is the possibility that the class of studies involving actual assays of biomolecules and correlating their absence or presence in the case of violent offenders may be useful. These investigators tend to distance themselves from ideologically motivated 'gene hunters.' They insist that the causal factor in street violence is a personal history of abuse. Childhood maltreatment is a universal risk factor for anti-social behaviour. However not all maltreated children end up as perpetrators of violent crime. As such, these investigators have been motivated to look into whether there could be a genetic factor that tips the balance (Caspi *et al,* 2002; Foley *et al,* 2004).

Studies along these lines, if validated, (Haberstick *et al,* 2005) could in principle lead to medication that would reduce the risk that abused boys (only) could have for violent behaviour. But again a certain discipline is needed to recognise the limitations of this approach.

As is well known from epidemiology, an infective causal agent may be present but there is no disease. The tuberculosis bacterium is the classic example. Many people are exposed to the bacteria but only a few contract tuberculosis. Nevertheless the tuberculosis bacterium is the causal agent of the disease. Similarly it is maltreatment that is the causal agent of street crime and violent behaviour. Low activity of the MAOA enzyme in the absence of maltreatment does not produce violent behaviour. As one group of investigators cautions their colleagues: "We suggest that in psychiatric genetics, ignoring nurture handicaps the field's capacity to make new discoveries about nature" (Moffitt *et al,* 2005). The old game of 'A gene for....' is now increasingly understood to be a projection of gene hunters' desires onto their data rather than a properly balanced analysis of the activity in question (Kendler, 2005).

But we must also more importantly note that studies that may isolate a biochemical vulnerability to the effects of maltreatment do not teach us anything about the causes of street violence. For an understanding of the causes of street violence we must look, as we have always had to look, not at the human genome, but at the maltreated human being in his or her social-developmental environment, the long-acknowledged infective agent leading to street crime.

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