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The evolution of intelligence

Natural genius?

The high intelligence of Ashkenazi Jews may be a result of their persecuted past

THE idea that some ethnic groups may, on average, be more intelligent than others is one of those hypotheses that dare not speak its name. But Gregory Cochran, a noted scientific iconoclast, is prepared to say it anyway. He is that rare bird, a scientist who works independently of any institution. He helped popularise the idea that some diseases not previously thought to have a bacterial cause were actually infections, which ruffled many scientific feathers when it was first suggested. And more controversially still, he has suggested that homosexuality is caused by an infection.

Even he, however, might tremble at the thought of what he is about to do. Together with Jason Hardy and Henry Harpending, of the University of Utah, he is publishing, in a forthcoming edition of the *Journal of Biosocial Science*, a paper which not only suggests that one group of humanity is more intelligent than the others, but explains the process that has brought this about. The group in question are Ashkenazi Jews. The process is natural selection.

History before science

Ashkenazim generally do well in IQ tests, scoring 12-15 points above the mean value of 100, and have contributed disproportionately to the intellectual and cultural life of the West, as the careers of Freud, Einstein and Mahler, pictured above, affirm. They also suffer more often than most people from a number of nasty genetic diseases, such as Tay-Sachs and breast cancer. These facts, however, have previously been thought unrelated. The former has been put down to social effects, such as a strong tradition of valuing education. The latter was seen as a consequence of genetic isolation. Even now, Ashkenazim tend to marry among themselves. In the past they did so almost exclusively.

Dr Cochran, however, suspects that the intelligence and the diseases are intimately linked. His argument is that the unusual history of the Ashkenazim has subjected them to unique evolutionary pressures that have resulted in this paradoxical state of affairs.

Ashkenazi history begins with the Jewish rebellion against Roman rule in the first century AD. When this was crushed, Jewish refugees fled in all directions. The descendants of those who fled to Europe became known as Ashkenazim.

In the Middle Ages, European Jews were subjected to legal discrimination, one effect of which was to drive them into money-related professions such as banking and tax farming which were often disdained by, or forbidden to, Christians. This, along with the low level of intermarriage with their gentile neighbours (which modern genetic analysis confirms was the case), is Dr Cochran's starting point.

He argues that the professions occupied by European Jews were all ones that put a premium on intelligence. Of course, it is hard to prove that this intelligence premium existed in the Middle Ages, but it is certainly true that it exists in the modern versions of those occupations. Several studies have shown that intelligence, as measured by IQ tests, is highly correlated with income in jobs such as banking.

What can, however, be shown from the historical records is that European Jews at the top of their professions in the Middle Ages raised more children to adulthood than those at the bottom. Of course, that was true of successful gentiles as well. But in the Middle Ages, success in Christian society tended to be violently aristocratic (warfare and land), rather than peacefully meritocratic (banking and trade).

Put these two things together—a correlation of intelligence and success, and a correlation of success and fecundity—and you have circumstances that favour the spread of genes that enhance intelligence. The questions are, do such genes exist, and what are they if they do? Dr Cochran thinks they do exist, and that they are exactly the genes that cause the inherited diseases which afflict Ashkenazi society.

That small, reproductively isolated groups of people are susceptible to genetic disease is well known. Constant mating with even distant relatives reduces genetic diversity, and some disease genes will thus, randomly, become more common. But the very randomness of this process means there should be no discernible pattern about which disease genes increase in frequency. In the case of Ashkenazim, Dr Cochran argues, this is not the case. Most of the dozen or so disease genes that are common in them belong to one of two types: they are involved either in the storage in nerve cells of special fats called sphingolipids, which form part of the insulating outer sheaths that allow nerve cells to transmit electrical signals, or in DNA repair. The former genes cause neurological diseases, such as Tay-Sachs, Gaucher's and Niemann-Pick. The latter cause cancer.

That does not look random. And what is even less random is that in several cases the genes for particular diseases come in

different varieties, each the result of an independent original mutation. This really does suggest the mutated genes are being preserved by natural selection. But it does not answer the question of how evolution can favour genetic diseases. However, in certain circumstances, evolution can.

West Africans, and people of West African descent, are susceptible to a disease called sickle-cell anaemia that is virtually unknown elsewhere. The anaemia develops in those whose red blood cells contain a particular type of haemoglobin, the protein that carries oxygen. But the disease occurs only in those who have two copies of the gene for the disease-causing haemoglobin (one copy from each parent). Those who have only one copy have no symptoms. They are, however, protected against malaria, one of the biggest killers in that part of the world. Thus, the theory goes, the pressure to keep the sickle-cell gene in the population because of its malaria-protective effects balances the pressure to drive it out because of its anaemia-causing effects. It therefore persists without becoming ubiquitous.

Dr Cochran argues that something similar happened to the Ashkenazim. Genes that promote intelligence in an individual when present as a single copy create disease when present as a double copy. His thesis is not as strong as the sickle-cell/malaria theory, because he has not proved that any of his disease genes do actually affect intelligence. But the area of operation of some of them suggests that they might.

The sphingolipid-storage diseases, Tay-Sachs, Gaucher's and Niemann-Pick, all involve extra growth and branching of the protuberances that connect nerve cells together. Too much of this (as caused in those with double copies) is clearly pathological. But it may be that those with single copies experience a more limited, but still enhanced, protuberance growth. That would yield better linkage between brain cells, and might thus lead to increased intelligence. Indeed, in the case of Gaucher's disease, the only one of the three in which people routinely live to adulthood, there is evidence that those with full symptoms are more intelligent than the average. An Israeli clinic devoted to treating people with Gaucher's has vastly more engineers, scientists, accountants and lawyers on its books than would be expected by chance.

Why a failure of the DNA-repair system should boost intelligence is unclear—and is, perhaps, the weakest part of the thesis, although evidence is emerging that one of the genes in question is involved in regulating the early growth of the brain. But the thesis also has a strong point: it makes a clear and testable prediction. This is that people with a single copy of the gene for Tay-Sachs, or that for Gaucher's, or that for Niemann-Pick should be more intelligent than average. Dr Cochran and his colleagues predict they will be so by about five IQ points. If that turns out to be the case, it will strengthen the idea that, albeit unwillingly, Ashkenazi Jews have been part of an accidental experiment in eugenics. It has brought them some advantages. But, like the deliberate eugenics experiments of the 20th century, it has also exacted a terrible price. ■

Trust

Paying through the nose

A person's level of trust can be changed with a chemical spray

SUSPICION and trust are two sides of the same coin. Over the course of evolution, humans and other animals have walked a line between the need for self-preservation and the benefits and delights of social co-operation. When a swarthy man beckons you into a dimly lit alley, you would do well to walk briskly away, but in reality you might be losing an opportunity to discover a delightful but out-of-the-way little restaurant.

A paper in this week's *Nature*, by Michael Kosfeld and Markus Heinrichs of the University of Zurich and their colleagues, explores the biological underpinnings of trust in such interactions. The researchers found that trust is surprisingly mechanistic: sniffing a spray containing a hormone called oxytocin increases a person's level of trust in others.

Oxytocin, a hormone produced by part of the brain called the hypothalamus, plays many roles. It stimulates contractions during childbirth and, once a child is born, helps to release milk when its mother feeds it. In some species, notably voles, it has been shown to regulate behaviours such as pair bonding, maternal care and the ease with which an animal will approach a stranger. Dr Kosfeld and Dr Heinrichs therefore had good reason to suspect that it plays a role in trust. They also knew from the work of others that hormones consisting of protein fragments known as peptides can cross into the brain if administered as a nasal spray. Oxytocin is one such peptide.

I'd trust him to the end of the Earth

To probe oxytocin's role in promoting trust between people, the researchers invented a game. This game involved an "investor" and an anonymous "trustee" in whom money, in the form of "monetary units" worth 40 Swiss centimes (32 cents) was invested. Investor and trustee never met, and were allowed to interact only once. In addition to being paid for their time, participants were able to cash their monetary units in at the end of the game, in order to get the proper economic juices flowing. Each investor received 12 units. He could choose to keep all of them, or to give four, eight or all 12 of them to the trustee—which would result in their value being tripled. The trustee then chose whether to reward or abuse the investor's trust by sharing a portion of the proceeds with him.

All the investors and all the trustees had something sprayed up their noses before the experiment started. In some cases, though, there was no oxytocin in this spray. Of the investors who were sprayed with oxytocin, 45% invested the maximum of 12 units, while only 21% of those who received the control spray did so. On average, the oxytocin-sprayed group transferred 17% more money to their trustees than the controls. Oxytocin, therefore, seems to promote trust.

The proof that it is trust that is being promoted, rather than a general bonhomie towards others, or a reduced aversion to risk, comes in two parts. The first is the response of the trustees. These people did not, as some might expect, simply take the money and run. The investors usually got something back, albeit less than half of the trebled amount. But the sum returned did not depend on whether there was oxytocin in the spray a trustee had sniffed—as it might have been expected to if oxytocin promoted generally sociable behaviour, rather than trust specifically.

The second piece of proof that oxytocin is "trust-specific" came when the investors were told that a computer rather than a human trustee would be on the other end of the transaction, and that the amount returned would be decided at random. In this set-up, the oxytocin-sprayed group and the control group invested equal amounts. The researchers thus concluded that oxytocin was not simply lowering a ▸▸