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Dangerous DNA: The truth about the 'warrior gene'

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*The story of one gene epitomises popular misconceptions about how our DNA shapes us. But it can also teach some crucial lessons, says **Ed Yong***

Our tale begins two decades ago, when a group of Dutch women set out to find an explanation for the antisocial behaviour of the males in their family. As well as having learning difficulties, these men and boys were prone to outbursts of aggression and were racking up a list of serious offences, including arson, attempted rape and murder. Suspecting that the behaviour might be hereditary, the women approached geneticist Hans Brunner at the University Hospital in Nijmegen, the Netherlands. Eventually, in 1993, he tracked down the culprit: a defunct variant of a gene called *monoamine oxidase A*, or *MAOA*, located on the X chromosome.

Understandably, the announcement created a sensation. It was the first time a gene had been linked to human aggression - and *MAOA* seemed to be responsible for a history of violence stretching back five generations.

In the following years, evidence poured in to bolster the connection between *MAOA* and aggression. Then, in 2004, journalist Ann Gibbons sealed the link by giving *MAOA* the headline-friendly label "warrior gene". The moniker stuck, raising the profile of *MAOA*, and at the same time fuelling misconceptions about how our behaviour is affected by our genetic make-up.

Unravelling the interaction between genes and behaviour is one of the toughest tasks in biology. The publication of the entire human genetic code, a decade ago, dispelled any illusions that having the genetic blueprint of our species would give a clear insight into our nature. Instead, what became clear is that we have far fewer genes than anyone imagined, and that understanding how these shape us as individuals is going to be a colossal task.

Genes simply make proteins. So for behavioural geneticists the question is how, when and why they influence our behaviour. Clearly, this involves a complex tango between genes and environments. Working out the exact steps is devilishly difficult, and conveying the findings to those without a scientific background is just as problematic. The mis-selling of *MAOA* is a salutary lesson in what can go wrong. It provides four key messages for anyone trying to get to grips with the interplay between genes and behaviour.


Lesson 1

A catchy name is bound to be misleading

The so-called "warrior gene" is actually just a molecular garbage collector. It encodes a protein that breaks down some of the brain's signalling molecules when they have outlived their usefulness - including serotonin, noradrenalin and dopamine. If it slacks on the job, the build-up of these neurotransmitters leads to abnormal moods and behaviour. The gene comes in several varieties, distinguished primarily by their levels of activity. Because it is found on the X chromosome, females may have two different forms, while males have just one. Brunner's aggressive Dutchmen had a rare and completely inactive variant. The so-called low-activity variant, or *MAOA-L*, has a slightly shorter than usual promoter - the region that controls the gene's activity - and so produces less protein. Another common variant, *MAOA-H*, is more active.

Like the defunct version of *MAOA* found in the Dutch family, *MAOA-L*, is associated with violence and aggression. Last year, Kevin Beaver from Florida State University, Tallahassee, found that boys who carry *MAOA-L* were more likely to join gangs, and those who did were four times more likely to use

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weapons in a fight (*Comprehensive Psychiatry*, vol 51, p 130). Headlines proclaimed that "gang culture may be due to warrior gene" and that "boys who carry the gene are likely to be dangerous, violent and carry weapons". But this kind of deterministic thinking is wrong-headed. *MAOA-L* is actually very common: a third of white people have this version and most of them have nothing to do with gangs.

Besides, aggression is not the only behaviour associated with *MAOA* and, moreover, problem behaviours are not just associated with underactive versions of the gene. *MAOA-H* has been linked with risky financial choices, such as playing the lottery and not buying insurance. Low-activity variants, meanwhile, are implicated in numerous other traits including depression, anxiety, attention-deficit hyperactivity disorder, anorexia, schizophrenia, neuroticism, pathological gambling, smoking and alcoholism. While some of the findings are questionable, *MAOA*'s reach is likely to be extensive because the brain signalling molecules it helps break down have a wide range of roles. In a recent Taiwanese study, for example, high *MAOA* activity was implicated in gout, a painful condition caused by uric acid building up in the blood. The link is not obvious, but *MAOA* affects levels of dopamine, and dopamine affects the kidneys' ability to remove uric acid (*Human Genetics*, vol 127, p 223).

Although *MAOA* can certainly influence our behaviour, it is no puppet-master - if anything, it is just one of many different strings. Other genes, including *MAOB* and *COMT*, affect the creation, use and breakdown of the same neurotransmitters. It is highly likely that these genes work together to affect our behaviour, and genetic studies are starting to reveal the dalliances between these dancing partners. For example, one found that women with *MAOA-L* are more likely to become depressed while pregnant, but only if they also carry a low-activity version of *COMT*.

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At the very least, the "warrior gene" tag typecasts *MAOA* in a role that is just part of its repertoire. Behaviours are not simple traits like eye colour, and to label them as such will inevitably mislead.

Lesson 2

Nature and nurture are inextricably linked

MAOA is not a gene "for" aggression. Instead, certain carriers may be more aggressive in certain situations. Brunner reported that the men in his Dutch family mostly became aggressive when afraid, frustrated or angry. A recent study suggests this may be because people with low-activity variants of *MAOA* are more likely to overreact to threats or challenges that other people would shrug off.

Rose McDermott of Brown University in Providence, Rhode Island, asked volunteers to play a game against an anonymous partner who would split a pot of money between them. If they were unsatisfied with their share they could, at a small cost, punish their partner by administering an unpleasantly spicy sauce. Although *MAOA-L* carriers were always slightly more likely to dole out the sauce, the big difference in aggression between them and others came as a reaction to their partner taking the lion's share of the loot (*Proceedings of the National Academy of Sciences*, DOI: [10.1073/pnas.0808376106](https://doi.org/10.1073/pnas.0808376106)).

The discovery that *MAOA-L*-related aggression appears mostly as a reaction to certain circumstances is perhaps not surprising, since this is how most aggression manifests itself. However, the clearest sign yet that the gene is no ruthless determinant of behaviour came in 2002 when Avshalom Caspi and Terrie Moffitt of Duke University in Durham, North Carolina, published their findings about a sample of 442 men from New Zealand who they had followed from birth. A third of these men carried the

MAOA-L variant. Now, aged 26, this group was indeed more likely than the others to have developed antisocial disorders and violent behaviour - but only if they had been poorly treated or abused as children. Moffitt and Caspi concluded that the so-called "warrior gene" affects a child's sensitivity to stress and trauma at an early age. Childhood trauma "activates" bad behaviour, but in a caring environment its effect is quashed.

Since then, similar interactions between nature and nurture have become part and parcel of the *MAOA* story. Carriers of *MAOA-L* are more likely to show delinquent behaviour if they were physically disciplined as children. They are also more likely to be hyperactive in late childhood if their first three years were stressful, and to develop conduct disorders if their mothers smoked cigarettes while pregnant with them. The list goes on. Likewise, Beaver found that *MAOA-H* carriers were more likely to commit fraud, but only if they hung around with delinquent peers.

So environments can set the degree to which genes make their presence felt, creating the stage upon which genes express themselves. The stage-set can also affect the action of genes. Smoking, for example, can reduce the activity of *MAOA* regardless of which variant someone has. And there is evidence from mice that a fatty diet can do the same. Genes may be able to influence our behaviour, but our behaviour also influences our genes.

Lesson 3

Beware of reinforcing stereotypes

With *MAOA* being billed as an architect of aggression, it was perhaps just a matter of time before it became linked with a racial stereotype. That happened in 2006 at the 11th International Congress of Human Genetics in Brisbane, Australia. There, Rod Lea from the ESR Kenepuru Science Centre, Porirua, New Zealand, presented unpublished results showing that 56 per cent of the 46 Maori men he had tested carried *MAOA-L* - almost twice as many as in white populations. Based on an even smaller sample of 17 men, he also concluded that *MAOA* was recently shaped by natural selection, and that *MAOA-L* may have given Maoris an advantage in the war-filled period when they were colonising Polynesia.

The media could not resist a story connecting the so-called "warrior gene" with a group of people historically perceived as fearsome warriors and who were often now involved in domestic violence. Headlines such as "Warrior gene blamed for Maori violence" soon followed and, as you might expect, Maori communities and geneticists were outraged.

Lea tried to set the record straight in interviews in the days following the media furore, and later in an article in the *New Zealand Medical Journal*. He pointed out that his sample sizes were small. And he said that the goal of his research was actually to improve the health of New Zealanders by looking for genes that would affect their risk of alcohol or tobacco addiction. Other scientists made it clear that the association between *MAOA-L* and aggression is based solely on studies with white men and that Lea had never researched this link in Maoris. But it was too late, the media circus had moved on.

It is a classic example of how easily genetic findings can be taken out of context, and how carefully they must be framed. As far as we know, many populations have a similar level of *MAOA-L* carriers to Lea's sample of Maoris, but it would be ludicrous to say that all Africans, Pacific Islanders, Japanese and Chinese men are twice as likely to be violent as Caucasians. In fact, one of the few studies on *MAOA-L* and aggression to look beyond white males found no link in non-white Americans (*Biological Psychiatry*, vol 60, p 684). The Maori case study highlights the risk of extrapolating results from behavioural genetics, particularly from one ethnic group to another.

Lesson 4

Genes do not dictate behaviour

MAOA has been used by the media to condemn a race, so it is ironic that it has also been used by a judge to offer leniency to an individual. In November 2009, Abdelmalek Bayout, an Algerian immigrant living in Italy, was jailed for murdering a man called Walter Perez who had mocked him for wearing

kohl eye make-up.

Bayout's history of mental illness got him a soft nine-year sentence. The defence team managed to reduce this by a further year on appeal, arguing that carrying the *MAOA-L* gene predisposed Bayout to violence. This is the first time that behavioural genetics has affected a sentence in a European court. But it is not the first instance of lawyers trying the "my genes made me do it" defence, which is now the subject of heated debate.

"In principle, I'm not opposed to the use of behavioural genetics in criminal cases for affecting sentences," says [Nita Farahany](#) from Vanderbilt University, Nashville, Tennessee. A report by the British Nuffield Bioethics Council agrees, noting that judges could reasonably use genetic information in the same way as any other information about a defendant's background. It won't necessarily work in the defendant's favour, however. "It really does cut both ways," says Beaver. You could argue that if violence is wrapped up in someone's DNA, the law should come down even harder on them because they are likely to reoffend. Farahany agrees. "Defence attorneys are starting to realise that it may not be in their client's best interests to raise this evidence because prosecutors have seized on this double nature to denigrate the defendant's character," she says.

If violence is wrapped up in someone's DNA, the law might come down even harder on them

Farahany also believes that behavioural genetics should never be used to determine whether a defendant is guilty or innocent. It is about "explanations, not justifications", adds Beaver. Most experts agree that finding someone has a variant of a gene associated with crime no more removes their guilt than discovering they were abused as a child. Far better, says Moffitt, to consider a defendant's family history, which combines information from all their genes, together with environmental and social factors.

The story of *MAOA* so far is not pretty, but if we learn these four lessons, we might yet harness the potential that behavioural genetics offers. Once we move beyond genetic determinism, the nature/nurture dichotomy and simplistic generalisations, the discovery of genes related to mental or behavioural disorders can only improve our knowledge of ourselves. It will also help us make better decisions. For example, adoption agencies might want to place children with the *MAOA-L* gene with particularly stable families. If a purported link between *MAOA* and alcoholism holds up, people with certain variants might choose to become teetotal to avoid the risk of addiction. And perhaps we could tailor effective criminal rehabilitation programmes to the variant of *MAOA* and other crime-related genes a prisoner has.

For Moffitt, the best defence against the future misuse of genetic information is a "more realistic, nuanced understanding of the causes of behaviour, in which some genes' effects depend on lifestyle choices that are often under human control". This view is echoed by Beaver. "We could look at how to manipulate the environment to affect genetic predispositions," he says. "It's a way to harness this type of information and use it in a very progressive and humane fashion."

Ed Yong works for Cancer Research UK in London and blogs at [Not Exactly Rocket Science](#)

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