Deconstructing Violence

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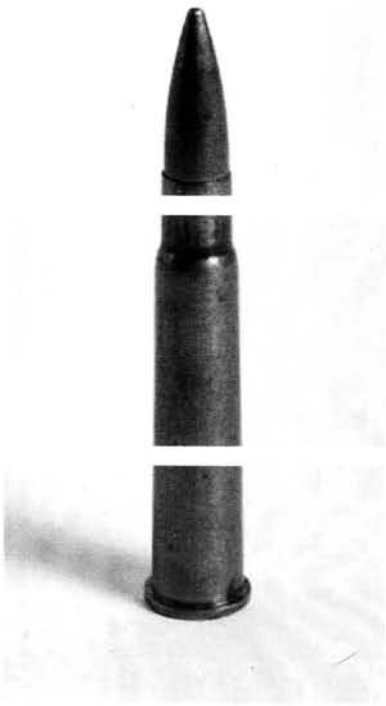
MAOA Variants and Behavior Genetics

"...a man pushed to acts where his own free will stood for nothing."
– Jacques Lantier, in Emile Zola's *La Bête Humaine*, 1889

Using the latest genetic technologies, scientists have identified certain gene variants in human populations that they say predispose individuals to antisocial behavior. Most prominent among these genes is monoamine oxidase A (MAOA). Here, we explore the available research on MAOA, scientists' claims about their findings, and the media's reaction to these studies. A review of the research reveals that a MAOA-antisocial behavioral link is far from conclusive and has garnered media attention disproportionate to its significance. These MAOA studies illustrate how relatively minor and unconfirmed genetic findings, when poorly communicated, can be misinterpreted, amplified by the media, and inappropriately incorporated into discussions of legal and social policy.



The notion that criminal behavior is heritable is a long-standing societal belief predating any knowledge of genetics. One can see the seeds of this popularized thinking as far back as 1889 in *La Bête Humaine* [*The Human Beast*], Emile Zola's story of a family that passed on abnormal behavioral defects from generation to generation.[1] Zola derived much of his thought on the subject of human antisocial behavior from Cesare Lombroso's influential proposals that human physiognomy could predict criminal behavior. With the rediscovery of Mendel's laws of inheritance in 1900 and the subsequent dramatic successes of genetics came claims that criminal behavior, and indeed much of human behavior, could be explained by genes. This view so permeated popular thinking that laws passed in most of the United States promoted sterilization of those considered hereditarily criminal as well as individuals with low I.Q.s. Eugenics propaganda and legislation reached its zenith in the 1920's; however, the later horrors of Nazi Germany's eugenics programs caused many to turn to more environmental explanations of human social behavior.

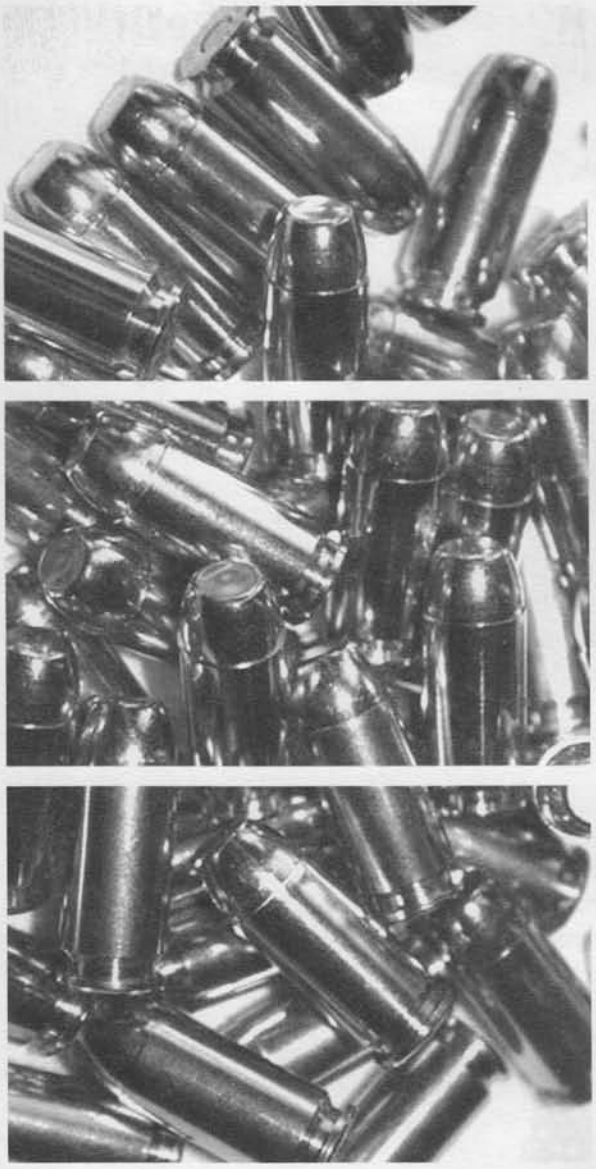
Significant public focus on a genetic basis of criminality was not revived until the 1960's when Patricia Jacobs and her coworkers published an article in *Nature* entitled "Aggressive Behavior, Mental Subnormality and the XYY Male," presenting the results of chromosomal screening of inmates in an institution for the criminally insane.[2] Finding a frequency of XYY males in this institution of about 3% and assuming that this frequency was much higher than in the general population (later found to be about 0.1%), the authors suggested a correlation between possession of an extra Y chromosome and aggressive behavior. Despite the very preliminary nature of the results and numerous criticisms of the conclusions by other scientists, the study received extensive public attention.[3] This quickly-dubbed "criminal chromosome" study provoked widespread discussion about the genetic basis of criminal behavior, leading to the use of the "XYY defense" in murder trials and screening for XYY males by prison officials in some states. Since its publication, the original study has been criticized for its small sample number and for problems of ascertainment bias.* By examining patients in a special kind of institution as opposed to the population at large, the authors could not draw any valid conclusions of a significant correlation. In fact, in 1975, the first of many studies to follow was published, showing that when researchers screened the general population for males with the extra Y chromosome, they did not observe the claimed associated aggressiveness. The only replicable features of XYY males in these studies were greater than average height and certain deficiencies in language and motor skills.

In one of the few studies to report an increased likelihood of incarceration of XYY males, researchers in the

* Ascertainment bias is the systematic bias introduced when non-random criteria are used to select individuals and/or implicated genes in which genetic variation is assayed.

United Kingdom reported in 1999 that criminal behavior was mediated by the lower intelligence of XYY subjects rather than a direct consequence of the extra Y chromosome. Of course, the correlation of lower intelligence and criminal behavior could also reflect an interaction of poorer school performance, etc. with class, social attitudes and social values. In addition, the crimes committed by the subjects of this study were crimes against property, not the physically aggressive crimes suggested in the title of the original paper. In fact, Patricia Jacobs, in her 1982 speech upon receiving an award from the American Society of Human Genetics, publicly regretted the title of her 1965 paper that had provoked the media interest. Despite the subsequent moderating studies and Jacobs' apology, the myth of the criminal chromosome died a slow death. As late as 1993, the movie *Alien 3* featured a remote planet populated by "extra Y chromos" who had been exiled from Earth and who were, according to their leader, "thieves, murderers, rapists and child molesters...all scum."

Nevertheless, crime is becoming genetic again, but with a new twist. In contrast to earlier research on antisocial behavior, some of the current studies, including those on MAOA, have begun to examine the interactions between people's genes and their environment, specifically, the particulars of their upbringing. Does this more expansive view, which attempts to account for the environment as a factor influencing human behavior, truly represent a change from the past in this area of behavioral genetic research?



DO ALTERED MAOA GENES CONTRIBUTE TO ANTISOCIAL BEHAVIOR?

Monoamine oxidases (MAOs) are a class of enzymes that catalyze the deamination of certain amine-containing molecules. One member of this class, MAOA, plays a significant role in the metabolism of the neurotransmitters serotonin, dopamine, and norepinephrine.* Because they function in breaking down key neurotransmitters in the brain, the role of MAOs in modulating behavior has been extensively studied. Research on the role of MAO in violent behavior began in the mid 1970's with work in rats.[4] By the early 1980's, researchers were studying human thrombocyte (blood platelet) MAO levels and looking for correlations between low MAO activity and social introversion, aggressive behavior, antisocial behavior, and criminal behavior.

In 1993, scientists reported on a Dutch family in which many of the males+ exhibited a specific MAOA mutation.[5,6] These males exhibited mild mental retardation and occasional aggressive or violent behavior. Although this correlation was intriguing, the applicability of this finding to the general population was questionable. The mutation was exceedingly rare – it completely eliminated MAOA enzyme activity and, in the years since, has not been reported to have been found in any other men tested. Given that the complete loss of MAOA activity resulted in mild mental retardation, some suggested that the violent behavior might be attributable to the individual's frustration experienced upon being unable to effectively communicate.[7] Indeed, loss of MAOA activity causes severe behavioral abnormalities in mice including increased aggressiveness, tremulousness and fearfulness, as well as defects in brain development.[8] Thus, the variety of structural abnormalities caused by the complete loss of MAOA activity may be responsible for the observed behavioral abnormalities and may not be the direct effect of monoamine metabolism in neuronal function per se.[9]

In 2002, the journal *Science* published research that suggested a broader connection between MAOA levels in human populations and antisocial behavior (referred to hereafter as "Caspi").[10] In contrast to the Dutch report on the rare MAOA mutation that eliminated the enzyme activity completely, this study was based on the existence of common human variants (polymorphisms) of the MAOA gene that affect the levels of the enzyme

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* Neurotransmitters are chemicals that are used to relay signals between cells of the nervous system.

+ The MAOA gene is on the human X chromosome; thus, males have only one copy of the gene (males have only one X chromosome), while females have two copies (females have two X chromosomes). Therefore, most of the MAOA studies conducted in humans, and described here, examine the behavior of males because they are able to inherit only one copy of the gene, while studies in females are complicated by the fact that they can inherit two different copies of the gene.

made in cells. Surprisingly, several studies had shown that the two major human versions of the MAOA gene – a high activity and a low activity polymorphism – represent approximately 65% and 35%, respectively, of males tested in diverse populations. It should be noted that the effect of the polymorphism on levels of activity was measured not in brain cells, but in fibroblast cells in the skin, which raises concerns about the conclusions of this and subsequent studies. Do the polymorphisms have the same effect on MAOA levels in brain cells? In addition, while the average difference in levels of MAOA between the two classes was approximately 7-fold in one study, the differences were quite variable (2- to 10-fold in another study) and the levels between the two classes overlapped.

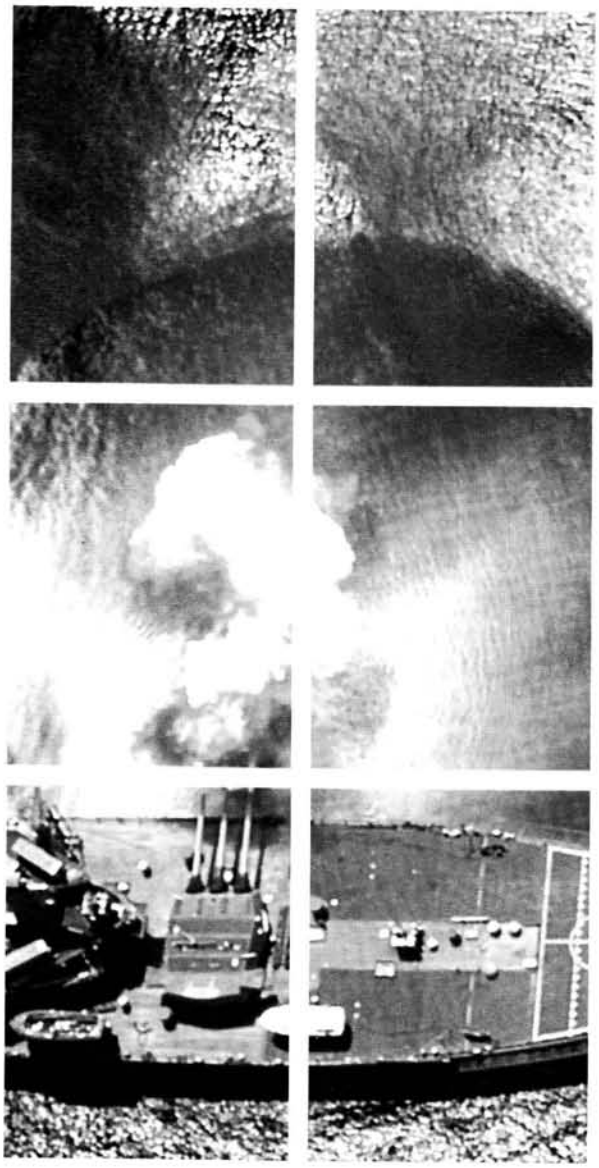
Using knowledge of the MAOA polymorphisms, Caspi and coworkers assessed the correlation of MAOA activity with antisocial behavior of 442 males followed in a longitudinal study conducted in Dunedin, New Zealand. The researchers used various criteria to assess the behavior of those who exhibited the low and high activity versions of the MAOA gene. They found those individuals with the low activity MAOA allele and who had been subjected to severe maltreatment as children were more likely to exhibit antisocial behavior than those with the high activity version. In contrast, those children with the low activity version but who had not been subject to abuse did not show any greater degree of antisocial behavior than those who had the higher activity MAOA gene. Moreover, those children with the higher activity MAOA gene who experienced child abuse were also more likely to exhibit antisocial behavior, albeit not to the same extent as the low activity group. The authors concluded that the high activity form of the gene might be "protective," preventing a continuing cycle of violence for maltreated males; the potential for antisocial behavior in males with the lower expressed version of MAOA only appeared when the subjects had experienced significant child abuse. The authors concluded that the correlation between MAOA polymorphism, maltreatment as a child, and subsequent antisocial behavior represented what is known as a gene-environment interaction (G x E).

Importantly, the Caspi research team noted that replication of their findings was needed before any solid conclusions could be drawn. Subsequent replication attempts have yielded mixed results; some studies have reached similar conclusions[11-13], another reported a "non-significant trend" toward the Caspi conclusions[14], and yet others completely failed to replicate the findings, including the gene-environment interaction.[15-17] Moreover, in addition to those studies that failed to replicate the Caspi findings, at least two studies found a contradictory inverse relationship – that is, an association between the high activity MAOA genotype and aggression in males.[18-19] Furthermore, even the 2004 Foley et al. study, which is frequently referenced as a replication of Caspi, can be interpreted differently upon closer reading; the authors acknowledge that on controlling for early adversity and the interaction of adversity and MAOA genotype, low activity MAOA was associated with lower (not higher) risk of conduct disorder.[11,20]

Studies of MAOA function in non-human primates have also been inconsistent with the original Caspi report. For example, Rhesus macaque monkeys with the low MAOA activity allele, raised in the absence of parental input, were even less aggressive than mother-raised macaques with either the low or high MAOA activity alleles.[21] Curiously, despite this contradictory observation, studies have cited the Newman et al study as supporting the findings of the Caspi et al. (2002) study.[12,21]

Recently, several authors of the original Caspi study published a meta-analysis[22], which included new data, the original Caspi data[10] as well as three other reports.[11,12,14] However, the criteria used to choose studies for this meta-analysis resulted in inclusion of only the original Caspi study, two studies that replicated the findings, and only one study that had a partial failure to replicate. Thus, given the

included data, the meta-analysis replication of the Caspi data is relatively unsurprising. In addition, one should consider that published results are frequently biased toward positive findings, skewing such meta-analyses. Indeed, at a Ciba Foundation Symposium in 1995, one scientist reported finding the inverse correlation between the low activity allele and delinquent behavior, but did not seek publication because "it was contrary to what [he] had predicted." Han Brunner, the lead author on the original MAOA Dutch study, responded: "It's extremely important that these sorts of negative findings are published to avoid the meta-analysis later coming up with the wrong answer."[23] Nevertheless, the contradictory study was never published.



Even if the Caspi results are conclusively confirmed, such findings are not de facto translatable to all populations. For example, while the earlier studies had focused on "white" males[10, 11], a recent study was the first to directly compare "white" and "non-white" populations.[13] For "non-white" individuals, the authors found no significant interaction between MAOA allele, maltreatment as a child, and subsequent violent and antisocial behavior. Interestingly, the authors chose to reanalyze the data using only "blacks." Again, they found no significant interaction. In addition to the mixed results from attempted replications of the Caspi et al. study, the failure to identify significant association in "non-white" individuals further challenges the generalizability of any results obtained in select "white" populations.

This same study also found that for "white" subjects, a statistically significant interaction was only found between MAOA genotype and maltreatment with juvenile violence, and was not significant for subsequent adult behavior.[13] This result suggests that knowing the genotype and childhood environment has no predictive power for the propensity for violence of the adult, arguing that life history modulates adult behavior irrespective of MAOA genotype. Indeed, as the recent meta-analysis study notes, "both scientists and the public are becoming increasingly aware that like many developmental processes, the nature of gene effects on behavior, too, is often contingent upon experience."[22]

Despite the many discrepancies and failures to replicate, two results were clearly consistent among all of the studies: first, childhood maltreatment is the strongest predictor of violent or antisocial behavior, and second, variation in the MAOA gene is not predictive of antisocial behaviors later in life. From the research completed to date, there are no well-established relationships between MAOA genotype and violent or antisocial behaviors. Furthermore, maltreated children who are at the highest genetic risk predicted by MAOA genotype appear to comprise a relatively small fraction of maltreated children overall. Even if the gene-environment interaction suggested by Caspi and others is eventually demonstrated to be correct, the relationship is merely probabilistic. That is, one would not be able to predict which children with the low-expressing version of the MAOA gene will exhibit antisocial behaviors.

We know from psychological and sociological research, which long predates behavioral genetic studies on the subject of antisocial behavior, that in general, individuals exposed to childhood maltreatment are more likely to have behavioral problems as adults (i.e. continue "the cycle of violence"). As even some of the behavioral geneticists studying the effects of MAOA polymorphisms note, "eradicating child maltreatment is clearly the preferred way to combat risk for psychiatric problems...social support can protect even genetically vulnerable children from the negative sequelae of maltreatment."[22]

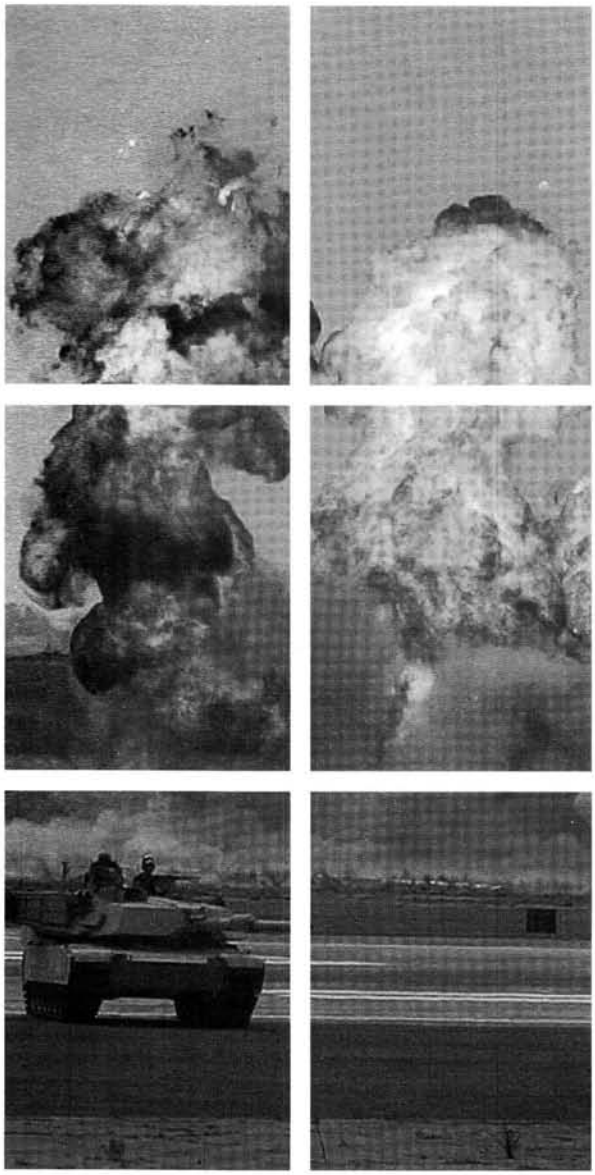
Even with this data, the authors of the original Caspi report and many of the replication studies allude to the possibility that understanding MAOA gene variation may permit the development of "improved pharmacological treatments" for children with the low-expressing version of MAOA.[10] Some may point to studies in MAOA-deficient mice that show that administration of serotonin synthesis inhibitors can counteract the development of behavioral abnormalities resulting from the loss of MAOA activity.[8] However, given that these findings are limited to MAOA-deficient animals, and loss of MAOA activity in humans is extremely rare[5], it is unclear how such pharmacological interventions would be useful. Taken to a logical conclusion, some may suggest that pharmacotherapy should be used to treat susceptible children living in abusive homes so that they can withstand the effects of the abuse.

Numerous questions about the utility of these reports are raised by the uncertainty of the science conducted so far. These uncertainties are confounded by the lack of pre-

dictive power of the proposed correlations, the stunning variability in MAOA levels among males with the same "low-expressing" polymorphism, the absence of understanding of mechanism for the effects reported, the contradictory studies that attempted to replicate the findings, and the much deeper knowledge of the overall role of child abuse in the development of antisocial behavior. At the very least, we are far from any certainty about the significance of this research field and similarly far from conclusions relating to any intervention, let alone rational pharmacological treatment.

A number of authors of the behavior genetic studies have suggested that knowledge of the MAOA allele variant could permit the development of programs to reduce antisocial behavior by, for example, "leading to more focused interventions"[13] or "screen[ing]" for children who are highly likely to develop severe conduct problems."[24] Such proposals raise serious ethical and social questions. These proposals disregard the fact that the findings of replication studies are mixed and that children with the "protective" high MAOA activity polymorphism may also exhibit violent behavior as a result of maltreatment. Thus, these polymorphisms are unlikely to have any real predictive power calling into question the utility of targeting only for "intervention" or "screening" those maltreated children with the "low MAOA activity" polymorphism for experiential (or still non-existent pharmacological) therapies. Such a strategy seems to be an unfair and dangerous approach allowing

avoidance of the real problem – child abuse. Maltreatment has consistently been demonstrated to predispose children to violent behavior[25], and experiential therapeutic interventions have been shown to reverse the effects of maltreatment on behavior.[26] Thus, it is irresponsible to focus treatment on a subset of maltreated children based on a possible genetic susceptibility to experience-induced violent behavior. Directing societal efforts to reduce child abuse and to provide experiential therapies for both victims and their families is the most effective, most ethical, and most socially rational method for counteracting the negative effects caused by maltreatment.



MAOA AND SOCIETY

Since the late 1980's, the monoamine oxidase (MAO) enzymes have taken a star role in several psychological dramas played out in the press. In addition to numerous articles on MAO's role in depression and depression-related disorders, The New York Times alone has profiled studies reporting MAO level hypotheses in risk-taking behavior, thrill-seeking behavior, alcoholism, drug addiction, cigarette addiction, and violent behavior.[27-32]

The news reports of the early 1990's on the MAOA-deficient Dutch family were no exception. The authors of the scientific studies, in their conclusions, hinted at a possible generalization of their finding in this one family to the larger problem of aggression in society. This hint was not missed. A *Science* reporter suggested "it might be possible to identify people who are prone to violent acts by screening for MAOA gene mutations..."[33] The provocative conclusions in the papers and the *Science* news article aroused tremendous media interest. A *Newsweek* article entitled "The genetics of bad behavior," included a photograph of a violent confrontation between Palestinians and Israelis, implying a genetic basis for world strife.[34] A television news report used films of U.S. street gang violence as a backdrop for a report on the MAOA study.[35] An issue of *U.S. News and World Report* that carried a story reporting on the Dutch study featured an infant dressed in prison clothing to indicate the supposed deterministic relationship between heredity and criminal behavior.[36]

Not surprisingly, these media reports on genetics and criminality have had their impact on the legal system. A *New York Times* article recalled that, "researchers were besieged by calls from lawyers, who wanted their clients tested for the genetic defect to use as a possible defense."[37] Indeed, in at least two court cases defendants have attempted to claim genetics as a mitigating defense for violent crimes. In *Turpin v Mobley*, the court rejected the defendant's claim in the sentencing phase that his behavioral/personality problems had a genetic basis. In this 1998 case, the defense attorney initially contacted Dr. Xandra Breakefield, one of the authors of the Dutch study; however, the lawyer subsequently decided not to have the defendant tested for the MAOA gene defect because he doubted that his client actually had the same genetic trait.[37] In November 2006, a defendant charged with murder unsuccessfully attempted to use the MAOA gene as a defense.[38] Despite the ultimate finding in this case, the language of the court is disturbing for its unquestioning acceptance of a genetic basis for violence: "Genetic testing did not show that the appellee had the MAOA gene, *which is the gene related to violent behavior*, but revealed that the appellee had a genetic vulnerability to becoming depressed and dysfunctional, especially in stressful, crisis-type situations" [emphasis added]. The Dutch study illustrates the way in which a relatively minor genetic finding can rapidly be transmitted to the public, be misinterpreted, and then be incorporated into discussions of legal and social policy.

The reporting by the media on the 2002 Caspi article, in many ways demonstrates a more careful approach to the subject than the reporting of the 1993 Dutch study. The later study, by its very nature an attempt to show that gene and environment interact to generate behavioral problems, had a less genetically deterministic flavor. Generally speaking, the articles qualified the study findings at some point and emphasized the importance of environmental factors, with at least two of the articles stating that the discovery is not of a "gene for violence."[36,40] The *Chicago Sun-Times* also stressed the environmental element and expressly referred to the need to replicate the study. Such attention to key details of the science is encouraging.

Other media outlets recognized the social and ethical confusions of the study. The *New Scientist* categorically rejected the Caspi study's suggestion of an eventual drug intervention, stating that these people are "victims of child

abuse, not bad genes."[41] ABC Health Minutes concluded soberly that the solution is not to move toward genetic testing but to prevent child abuse.[42] The *Guardian* (UK) predicted a day when the military, firefighters and police would screen recruits, and otherwise echoed a possible pharmacologic intervention for higher risk people, yet at the same time cautioned against the medicalization of social problems.[39] The *Hindu* suggested that the Caspi findings highlighted uncertainty about how much social practices might influence individual behavior and "why racial and caste discrimination and child abuse are not just uncivilized, but even dangerous," and ended with a declaration that "genetics is telling us how to behave."[43]

Nevertheless, the Caspi article's suggestion of pharmacological approaches to the problem based on genetic knowledge focused attention on the genetic side of the interaction between genes and environment and some in the media used unqualified declarative headlines such as "Study Finds Genetic Link to Violence," "Study Links Past Abuse, Gene to Violent Acts"[44], and even a mathematical equation "Gene + Abuse = Trouble."[36] *Popular Mechanics*, which provides "informative articles on automotive technology," carried the story under the headline "Criminals Share a Common Genetic Flaw," and employed various analogies using car keys to illustrate the relationship of the MAOA gene to the outcome of violent behavior.[41]

The tendency to explain human behavior as largely genetic in nature, even in the absence of supporting evidence, is often willingly propagated by scientists themselves. The *San Francisco Chronicle* quoted a Stanford psychiatrist, "people have to get used to the idea that there are probably genetic influences on many kinds of behavior."[44] The *Boston Globe* quoted one scientist not associated with the study, as saying that "If the results can be replicated, their public policy applications could be 'explosive.'"[45] With such hyperbole, it is little wonder that judges would convene, as they have, to begin to prepare themselves for the onslaught of legal issues that could arise from a genetic link to violence, however premature that assertion.

The era of strict genetic determinism of human behavior and aptitudes is rapidly disappearing. While the Human Genome Project advocates initially argued that genes for human behaviors would be rapidly identified, the opposite has been the case. This has led many researchers to emphasize the complexity of such behaviors and to suggest that multiple genes and environmental factors, difficult or impossible to separate, are involved in these human traits.[46] Researchers are increasingly considering these multiple factors and presenting more nuanced explanations when proposing genetic influences on behavior. Nevertheless, many scientists still lack sufficient information outside of their narrow fields of study and are not cautious or scholarly in presenting results, prematurely drawing conclusions about complicated and preliminary results that are difficult to replicate, and suggesting treatments that raise serious ethical and social issues. These sorts of presentations may direct the media to present more dramatic reports on the studies that often feature old-style deterministic headlines. In the case of antisocial or criminal behavior, the claims of genetic correlates are introduced relatively rapidly into the legal system in an unwarranted fashion. Over a century of history of scientific attempts to find these correlations illustrates the sometimes-severe impact of this field on society. The social consequences of this area of research both in terms of the legal system and how society, in general, deals with antisocial behavior call for a much more cautious and informed presentation both by scientists and the media. ■■■

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