

Questioning a Putative Solution to Violence:

Genetics & Public Policy

Sanjay Basu

What broke in a man when he could bring himself to kill another?
—Alan Paton (1948)

Violent crime rates in the United States have risen by as much as 300 percent in just forty years, even as the proportion of young males—the sector of the population most likely to become violent criminals—has decreased dramatically.^{1,2} Putative solutions to the problem abound, but among the most controversial is Jack Westman’s case for “parental licensure”: A proposal to require individuals to be licensed for parenthood.³ Those persons whom supporter David Lykken refers to as “immature, overburdened, unsocialized, or otherwise incompetent” parents would be denied a license, and therefore denied the opportunity to have children—which, Lykken claims, would cause “the source of most crime and violence to dry up.”⁴ He calls Westman’s proposal a form of “eumemics,” an alternative to eugenics based on the selection of memes (Richard Dawkin’s term for units of environmental influence) rather than genes.⁵ Would such a licensing process work, and if so, would we want to institute it? A thorough review of the genetics and environmental correlates to violence suggests otherwise; biological and psychological data question the proposed effectiveness of parental licensure, and ethical problems with the plan can be used to argue against its implementation.



Genes and Violence

The proposal to institute parental licensure rests on the premise that “parents matter”—that, in other words, whatever factors contribute to violent behavior can be mitigated or otherwise altered by parental influences. Is this indeed the case?

Years ago, biologists might have readily agreed. But modern geneticists are unlikely to accept Watson's challenge to "give me a dozen healthy infants and my own specialized world to bring them up in, and I'll guarantee to take any one at random and train him to become any type of specialist I might select."⁶ We now know that life outcome is not so extremely mutable, and genetic evidence seems to demonstrate that the propensity to commit violent crime may have a genetic contribution.

Key evidence for this conclusion has come from studies of monozygotic and dizygotic twins. A 1986 investigation by Rushton and his colleagues included 573 adult twin pairs (raised together) from the University of London Institute of Psychiatry Volunteer Twin Register. The twin pairs received questionnaires assessing both their altruistic and aggressive tendencies, and the resulting scores produced a correlation of 0.40 between monozygotic (MZ) pairs and 0.04 for same-sex dizygotic (DZ) pairs. From these correlation values, a heritability estimate of 72 percent was obtained, suggesting that 72 percent of the total variability in the aggressiveness measurement could be attributed to genetic differences between individuals. Maximum-likelihood model-fitting of the data in this study revealed that approximately 50 percent of the variance on the aggressiveness test was associated with genetic effects, virtually zero percent was associated with the twins' common environment, and the remaining 50 percent was associated with each twins' specific environment or with testing error.

These dramatic results have since been replicated. A study of 500 healthy MZ and DZ twin pairs (some raised together, others apart) reported a heritability of 41 percent for scores on a different self-administered aggressiveness questionnaire; model-fitting procedures in this case were consistent with a genetic but not a shared environmental influence on the trait.⁷

The results from these two studies were further supported by a third research group, who administered the Multidimensional Personality Questionnaire (MPQ) to 217 MZ and 114 DZ reared-together adult twin pairs and 44 MZ and 27 DZ reared-apart adult twin pairs.⁸ Their study fitted a four-parameter biometric model (including genetic, additive versus nonadditive, shared family environment, and unshared environment components) to data obtained with eleven primary MPQ scales and three higher-order scales. The heritability of aggression estimated by this model was 0.44, with the intraclass correlation of MZ twins raised apart equaling 0.46, as opposed to just 0.06 for DZ twins raised apart (whereas MZ

twins together had a correlation of 0.43 and DZs raised together had a correlation of 0.14). Given these numbers, the variance due to the common environment could be considered negligible; between 1 and percent of the variance could be attributed to the environment shared by a pair of twins but different between twin pairs (using the formula $c^2 = r_{MZ} - h^2$, where c^2 is the common environment effect, r_{MZ} is the MZ twin correlation coefficient, and h^2 is the heritability).

The results of these three studies are robust; a recent review of over 100 such studies of aggressiveness suggests that, averaging between the studies, genetic differences account for about half of the total variation in risk for aggressiveness, even when using different scales.⁹ But heritability estimates between studies are far-ranging, indicating either that differences in research methods were significant between studies, or that moderators of genetic risk factors are likely to exist.

It is notable in reviewing this evidence that violent crime was not the specific phenotype being correlated to genetic factors in these studies. Rather, "aggression" was the trait being correlated, on the basis that violent crime is itself not a distinct phenotype. It is assumed here that violent crime is likely to be the result of a propensity to be aggressive. As the psychologist Michael Lyons has stated: "Criminality per se is not a biological characteristic of the individual—it is a social construct."³⁰

But the problem of clearly defining the phenotype of "violence" versus other phenotypes of "aggression" or even "antisociality" cannot be easily resolved. Given our current understanding, violence is likely to be the manifestation of a general highly aggressive phenotype, given that it is difficult to imagine individuals who are "nonviolent" but still "highly aggressive." Violence may also be part of a general "antisociality," although violence is not necessarily a prerequisite for the diagnosis of syndromes such as antisocial personality disorder.¹⁰ No matter how we specify conditions for "antisociality" and "aggressiveness," the problem of clearly defining a violence phenotype is not simply solved with current data from twins or through semantic debates. Rather, investigations into the underlying mechanisms of the trait may be more revealing; generally speaking, if the mechanism underlying one behaviorally defined trait is identical to the mechanism underlying another, then definitions of each trait should be redefined so that the two traits can be treated as a single, more general phenotype.

Mighty Mean Mice

A clear mechanism for the emergence of violence and a more concrete understanding of its

phenotype could help us evaluate the parental licensing strategy. To understand the putative genetic mechanisms underlying violence, and thereby specify both its phenotype and its causation more precisely, biologists have turned to animal studies. Several years ago, pharmacological evidence indicated that levels of serotonin were inversely related to behavior in mice; it is no surprise, then, that serotonin was among the first chemicals relating to aggressive behavior to be studied in detail.¹¹ 5-HT_{1B} receptor knockout mice were among the most intensely examined model systems. Although these mice did not display gross abnormalities and appeared normal during development, breeding, eating, exploration, and “anxiety” testing, they attacked intruders faster and with more intensity than did wild-type mice.¹² After this observation was replicated, scientists began to study the serotonin pathway differences between violent and nonviolent humans.¹³

One study of a Dutch family with X-linked mild mental retardation received considerable attention because it correlated a detrimental point mutation in monoamine oxidase A (MAOA), one of two enzymes that metabolizes 5-HT, to the degree of impulsive aggressive behavior exhibited by individual family members.¹⁴ Only those individuals in the family who displayed violent behavior (including murder attempt, rape, and arson) had the polymorphism. But because only fourteen individuals were studied and many of these individuals lived together, it would be incorrect to conclude that deficiency in MAOA necessarily led to the exhibition of violence from these individuals. Even if the MAOA polymorphism (rather than other genetic and environmental factors) was responsible for the differences between the violent individuals and their normal relatives, all of the violent persons in this study exhibited verbal abusiveness and several other antisocial tendencies. This suggests that violence alone could not be treated independently of a larger personality change manifest in these individuals. The MAOA polymorphism in this study is, in any case, so rare that it cannot account for most violent behavior in the United States. Another polymorphism in the promoter of the gene, however, has been more recently correlated to aggressive behavior in a community sample of 110 men from Pittsburgh, who were subjected to a standardized interview and a questionnaire measuring impulsivity, hostility and lifetime aggression history.¹⁵ The polymorphism in this study was associated with general traits of impulsive control and antagonism. Given such data, it would be presumptuous to declare that MAOA is the “mean gene” specifical-

ly responsible for violent behavior.¹⁶ However, MAOA may increase the propensity to behave aggressively. In this regard, MAOA deficiencies do not support a simple causal relationship between metabolic abnormality and behavioral problems. Aggressive or otherwise abnormal behavior has not been noted in depressed patients receiving MAO-inhibiting drugs, and MAOA deficiency actually increases functional 5-HT levels in the brain (contradicting previous hypotheses that low serotonin levels contribute to aggressive phenotypes).^{17,18,19}

Low serotonin levels were correlated to aggression in studies of other genes involved in serotonin metabolism. For example, mice heterozygous for a mutation in the gene encoding alpha-calmodulin-dependent protein kinase II (CaMKII) exhibit both increased defensive aggression and decrease fear responses.²⁰ Extracellular and whole-cell patch-clamp recordings from brainstem slices of these mice indicated that serotonin release in the dorsal raphe was reduced. Since CaMKII is required for the activation of tryptophan hydroxylase, the rate-limiting enzyme in 5-HT synthesis, a deficiency in the CaMKII mouse would likely lead to decreased 5-HT levels. Using a standardized interview and a questionnaire on aggression and anger-related traits of personality for 251 men and women from the Pittsburgh area, Manuck (1999) found an association between increased “antagonistic behavior” (tendency to experience unprovoked anger) and a polymorphism in the tryptophan hydroxylase gene.²¹

Because so much research has focused on this serotonin pathway, however, the possibility that other molecular pathways might contribute to aggression or violence has been largely obscured. One study in mice does suggest that aggression can be exhibited through a pathway not directly related to serotonin. Mice with a targeted disruption of neuronal nitric oxide synthase (nNOS), the enzyme that catalyzes the production of nitric oxide neurotransmitter, exhibited grossly normal appearance, locomotor activity, breeding, long-term potentiation, and long-term depression. But they also displayed a large increase in aggressive behavior and excessive, inappropriate sexual behavior.²² A human equivalent of this phenomenon has yet to be investigated. If polymorphisms do exist in genes related to nitric oxide metabolism, then association tests investigating a correlation between these and aggressive behavior in humans should be performed.

Nevertheless, the mouse data on nNOS, along with the diverse data accumulated from serotonin studies, indicates that a biological mechanism for aggressive personalities is unclear at the present

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time. Both animal models and genetic association studies have, however, indicated that violence in humans is probably not the effect of a single gene; rather, individual genes will probably be associated with discrete factors that increase or decrease the risk for aggressiveness. In addition, the survey studies correlating behaviors to polymorphisms in genes involved in serotonin metabolism suggest that violence or even aggression is very unlikely to be a phenotype independent of other personality traits. Aggression is probably the result of several different genes working together to elevate the risk of exhibiting violent tendencies and several other related traits.

Emergenesis and the Unshared Environment

What do these studies imply for our evaluation of the parental licensing hypothesis? Do they indicate that we should throw up our hands and presume that all violence will be fated upon us by genes that generate mean people?

Far from it. The fact that twin and adoption studies reveal about 50 percent heritability for aggressive behavior indicates that roughly half of the total variability in this trait can still be attributed to nongenetic differences between individuals. That may seem like a strong argument for the parental licensing strategy, but we have yet to establish whether the nongenetic influences on individuals are from parents or from other sources such as peer groups. Discriminating between parental influence and other nongenetic influences will be crucial to our evaluation of the parental licensing model. But it has been suggested that even if parents don't affect their children through environmental means, using measures like parental licensure could prevent the genes of violent or incompetent parents from generating violent or incompetent offspring (note, however, that this was neither the premise under which Westman (1994) originally proposed his parental licensure theory nor the premise under which Lykken (2000) endorsed it).²³ Therefore, to refute the parental licensing strategy on scientific grounds, one would need to present evidence that violence does not run in families despite the evidence that it is heritable and that the environmental influences of parents on their offspring is either negligible or superceded by other environmental influences.

At first glance, this task seems impossible. But significant data does exist to strongly support both of these arguments against parental licensing. First of all, the animal models and human association studies reviewed earlier in this paper suggest that a single "mean gene" is unlikely to exist, and so a

single allele for violence is probably not passing from parent to offspring. In addition, several heritable traits do not run in families, and a tendency toward aggression or violence is likely to be one of these traits. Traits that are influenced by a configuration of polymorphic genes (rather than a simple additive sum of such genes) are known as "emergenetic" traits.²⁴ Rather than being the product of several genes, each contributing a small bit to the resulting trait, emergenic traits are the product of an "assembly line" of genes, where each gene is essential to producing the phenotype. The absence or change of any one gene will not simply lessen the strength of the phenotype, but will instead produce a large quantitative or qualitative change in the trait. Lykken and his colleagues (1992) provide an analogy to the eye: A person who fails to receive a gene crucial for eye formation will not simply have a smaller eye, but will have a terribly malformed one.

Emergenic traits that follow this rule might include traits like facial beauty. While genes obviously affect facial beauty, the configuration of genes is also important. "As a rule," Lykken writes, "the attractiveness of a given nose or chin or pair of ears depends to an important extent on the context of the other features." A small, feminine nose and dimpled cheeks will not go so well with bushy eyebrows and a square jaw. But not all emergenic traits are to be evaluated as subjectively as facial beauty. Frequency of the alpha rhythm on an electroencephalogram, the exhibition of serious psychiatric syndromes, extraversion, and electrodermal response and habituation have all been cataloged as emergenic traits.^{25,26,27,28}

One can deduce that if a trait is emergenic, MZ twins should share a strong correlation for the trait because they share their entire assembly line of alleles. Also, since DZ twins are unlikely to share all the genes in the configuration, they should be much less than half as similar as MZ pairs (unless their shared environment contributes to their similarity). Twin studies of aggression indicate that DZ twins are also much less than half as similar on aggression scales as MZ twins, so aggression is probably emergenic and is minimally affected by the shared rearing environment. The trait's emergenic property indicates that aggression will be only weakly correlated between parents and offspring, so the trait will not simply "run in families."

During one study of twins, a Self-Rating Inventory was given to 198 pairs of middle-aged twins from the Minnesota Twin Registry. These twins gauged the seriousness of their aggression when they got mad (rating themselves a "five" if they got physically violent when angry) and were

retested on the same scale three years later. The cross-time, within-twin correlations were 0.89 for MZ twins and 0.03 for DZ twins.⁴

Similarly, the Rushton, Fulker, Neale *et al.* (1986) study reviewed earlier in this paper produced a correlation of 0.40 between MZ pairs and 0.04 for same-sex DZ pairs from a London twin population tested on a scale of aggressiveness; this data again supports the emergenic model for aggressive behavior.²⁹

If the DZ twin similarity had been half or more of the MZ similarity value, then one might hypothesize that aggression measured by these scales was either not emergenic or was strongly affected by the shared rearing environment. Neither of these cases appears to be true, and the diminished importance of shared environment indicates that parental influences on offspring with regard to this trait are minimal. If the parents treat their twins differently in such a way that the differential treatment has an affect on personality, and the treatment is not predictable from the child's innate characteristics (because parental treatment correlated to a child's genetic makeup will be included as "genetic" during behavioral analysis), then it is possible that parents do still have an affect. However, it is reasonable to say that these conditions are collectively implausible, validating our initial conclusion that

parents probably have little or no effect on offspring in this case (Pinker S, e-mail communication, May 2, 2001).

The fact that shared environmental influences probably fail to contribute to aggression is supported by all three of the twin studies reviewed in the first section of this paper. The researchers who conducted these studies used maximum-likelihood model-fitting of their data to conclude that zero percent of the variance between aggressiveness scores was associated with twins' common environment.

It has been argued, however, that characteristics of the shared or family environment can still promote antisocial behavior during childhood and beyond. Lyons, True, Eisen *et al.* (1995) suggested this after administering the Diagnostic Interview Schedule Version III-revised (which tests for symptoms of antisocial personality disorder) by telephone to 3,226 pairs of male twins from the Vietnam Era Twin Registry.³⁰ The results indicated that on a variety of symptoms, the unique environment (plus measurement error) explained the largest proportion of variance in both juvenile and adult antisocial traits. But shared environment also correlated to some juvenile antisocial traits, even though it did not significantly correlate to adult traits.

In addition, Lykken (2000) has defended his

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idea that parents significantly contribute to a child's manifestation of antisocial behavior, particularly violent crime, on the basis of results from his study of 465 30-year-old Caucasian male men born in Minnesota.⁴ Of this group, 118 were labeled "less socialized" for having served jail terms or for admitting that they had engaged in illegal activities during or after adolescence and for having delinquent friends. These men and their "more socialized" peers were asked to rate how "good" their parents were (based on degree of "affability," whether the parents were "strict but warm," "good company," and "not themselves focused on drinking or drugs"). The men also rated their "citizenship," their "religious commitment," whether they had a "happy home" or an antagonistic one, whether their parents were religious, and whether their friends were delinquent or got into fights. The findings indicated that a significant negative correlation (of effect size -0.50) was found between having "good parents" and being "unsocialized." Lykken has made much of this result, although the correlation is not indicative of causation.

A review of Lykken's data, however, shows that he has failed to correlate his "unsocialized" phenotype to that of aggressiveness or violence.

Simply serving jail time or engaging in illegal activities does not imply that an individual is violent, and so these results cannot be easily extrapolated to our consideration of factors contributing to violence. But an even greater problem lies with Lykken's population: It is constructed to avoid measuring the effect of peer environments on antisociality. Lykken gives the label "unsocialized" to individuals who not only serve jail time or engage in illegal activities, but also have friends who can be described as delinquent. Since his population of "unsocialized" individuals is comprised entirely of persons with delinquent friends, it is not surprising that Lykken found a large correlation (effect size +0.98) between being "unsocialized" and having "friends who get into fights," and an even greater correlation (effect size +1.14) between being "unsocialized" and having "delinquent high school friends." But it cannot be determined whether these results indicate that antisocial behavior in general is truly correlated to having delinquent peers or whether these correlations are merely a result of including only criminals with delinquent peers in the sample. By requiring that an individual have delinquent friends to be included in his "unsocialized" population, Lykken has avoided gauging whether peer behaviors correlate more strongly to antisocial behavior than do "good parents." A better study would have labeled individuals "unsocialized" if they had independently conducted violent or criminal behavior, regardless of their friends' behavior. Such a study would separately determine the correlation between being "unsocialized" and having delinquent or violent peers and compare that correlation to the one between being "unsocialized" and having "good parents."

Similar comparisons have been made: J. R. Harris has, for several years, indicated that influences experienced by children outside their homes are likely to affect their psychological traits more than parenting influences and other experiences shared by siblings in a family.³¹ While parents may provide basic nurturing, Harris has suggested, they have little effect on how children "behave the way they do in the world outside the home."³² Chief among the evidence in support of Harris are twin studies showing that DZ twins and siblings are often less than half as similar as MZ twins for most psychological traits. Had parenting influences and other factors shared between siblings in a family significantly affected these traits, then DZ twins and biological siblings should be more than half as similar as MZ twins. But this is not the case with most psychological variables—and, as previously shown, it is not the case with aggression.

Lykken argues that even if peer environments are the primary environmental influences on children's behavior, including aggressive behavior, parental licensure should still reduce violence because parents have a say in their child's choice of peers.⁴ Although Lykken presents no evidence for this conclusion (relying entirely on anecdotes), it is logical that his assertion is at least partly true. But such an extreme measure as parental licensure seems to be inappropriate public policy, particularly when other measures that can mitigate violence and misbehavior among peer groups—such as after-school and gang-reduction programs—are more likely to directly reduce the problem of bad peer influences without exerting control over an individual's reproductive opportunities. Sociological evidence suggests that programs providing after-school supervised work and play opportunities, along with marketable job skills and gainful employment opportunities to gang members, dramatically reduce overall community violent crime levels as well as the rates of violent crime committed by individual gang members or those living in gang-ridden neighborhoods.^{33,34}

Although gangs are certainly not the only forum through which violence is exhibited, successful gang-reduction programs demonstrate that initiatives affecting only peer environments, without directly affecting the dynamic of parent-child interactions in the home, can significantly mitigate violence. Unlike these programs, parental licensure would not directly address the peer environment or other neighborhood factors that contribute to aggression and violence.

Ethical Problems with Parental Licensing

Although this review of the genetic data on aggressive behavior allows for an undercutting of the parental licensing strategy on scientific grounds, ethical reasons to avoid parental licensing must also be discussed. Principle among these is the question of who would be licensed. Lykken (2000) suggests that both parents be "over 21, married, self-supporting" and that neither parent can have "been convicted of a violent crime."⁴ He would make exceptions, to be regulated by a "family court" for "wealthy professional women, such as the film actress Jodie Foster...or gay and lesbian couples" who wish to raise children. Westman (1996) offers slightly different criteria, suggesting that "18 would be a reasonable

age" for parenting if an 18-year-old makes a "pledge" to care for and nurture the child and if the applicant possesses "basic knowledge of child-rearing."³⁵ He admits, however, that "at this time large-scale testing for signs of parental incompetence is premature. Efforts to predict the parenting potential of pregnant women by testing have yielded inconsistent results."

But even if Westman's or Lykken's list of criteria become usable, there is no reason to believe that all Americans share the ideals the two men have listed. Parental licensing would inherently impose one group's ideals onto a population of persons with varied and protected rights to hold their own values. Under Lykken's criteria, and using current demographic statistics, persons of low socioeconomic status would account for most of those who would fail to obtain licenses to reproduce.³⁶ One wonders whether these groups would have a voice in the matter or simply, as Lykken has suggested, "be required to submit to an implant of Norplant [a chemical contraceptive] as a way to keep them from having another baby."³⁷

Such comments conjure images from Orwell's 1984 or scenes from television shows about CIA conspiracies. One can reasonably suppose that few American citizens would want their government officials forcing them into infertility by requiring chemicals be surgically implanted into their bodies. Parental licensing, it can be concluded, is fundamentally incompatible with our country's basic claim to the preservation of liberty.

Conclusion

It is recommended that a parental licensing strategy to reduce violent crime not be instituted. Studies of the genetics of violent crime suggest that no single gene is responsible for violence and that aggression itself is probably an emergent trait. As a result, parental licensing cannot be justified on the grounds that the trait simply runs in families. Studies also show that peer environments and other environmental influences aside from parenting are likely to be the primary environmental contributors to aggressive or violent behavior. On the basis that parental licensing proposals suffer from several ethical problems and might only indirectly address peer environments or other nonshared environmental contributions to aggressive behavior, it is suggested that alternative strategies, such as gang reduction measures and after-school programs, be instituted instead of parental licensing. ■

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