The Integration of Genetic Propensities into Social-Control Models of Delinquency and Violence among Male Youths

Guang Guo
University of North Carolina-Chapel Hill

Michael E. Roettger
University of North Carolina-Chapel Hill

Tianji Cai
University of North Carolina-Chapel Hill

This study, drawing on approximately 1,100 males from the National Longitudinal Study of Adolescent Health, demonstrates the importance of genetics, and genetic–environmental interactions, for understanding adolescent delinquency and violence. Our analyses show that three genetic polymorphisms—specifically, the 30-bp promoter-region variable number tandem repeat (VNTR) in MAOA, the 40-bp VNTR in DAT1, and the Taq1 polymorphism in DRD2—are significant predictors of serious and violent delinquency when added to a social-control model of delinquency. Importantly, findings also show that the genetic effects of DRD2 and MAOA are conditional and interact with family processes, school processes, and friendship networks. These results, which are among the first that link molecular genetic variants to delinquency, significantly expand our understanding of delinquent and violent behavior, and they highlight the need to simultaneously consider their social and genetic origins.

Why do some individuals become serious and violent delinquents while others do not, despite growing up in similar social contexts and participating in similar social processes? We maintain that part of the answer lies in genetic propensities. Social conditions may be sufficient to produce delinquency in some individuals, whereas for others both social conditions and genetic propensities may be needed to make a difference. The relationship between social conditions and genetic propensities may be additive or interactive. To illustrate, suppose that family disruption and genetic propensities each increase the probability of a delinquent act by .1. If the two are additive, the total increase would be .2 when both are present. If the two are interactive, the total could be .4, significantly larger than .2. In such a scenario, genetic propensities could amplify the effect of family disruption or family disruption could amplify the effect of genetic propensities.

Genetic propensities should be of relevance to sociological inquiry. Indeed, ignoring gene–environment interactions may result in the neglect of pivotal social processes. And, even if genetic propensities are noninteractive and uncorrelated with social controls—meaning that disregarding genetic propensities will

Direct correspondence to Guang Guo, CB#3210, Department of Sociology, University of North Carolina, Chapel Hill, NC 27599-3210 (guang_guo@unc.edu). This research uses data from Add Health, a program project designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris, and funded by a grant P01-HD31921 from the National Institute of Child Health and Human Development, with cooperative funding from 17 other agencies (www.cpc.unc.edu/addhealth/contract.html). Special acknowledgment is due to Andrew Smolen and John K. Hewitt of the Institute for Behavior Genetics, University of Colorado for DNA isolation and genotyping. We gratefully acknowledge support from NIH: P01-HD31921 to Add Health, R03 HD042490-02 to Guang Guo, R03 HD053385-01 to Guang Guo, and support from NSF, SES -0210389 to Guang Guo.

not bias the estimated effects of social controls—they can improve predictions.

We address the challenging question of genetic and genetic–environmental influences in this article by incorporating measures of genetic propensities into a classic sociological model of delinquency. The following section reviews two hitherto largely independent lines of research on delinquency: the social-control life-course approach and the recent developments in molecular genetics and related evolutionary perspectives. We then bring together these two lines of inquiry, describing and motivating the gene by social-control interaction analysis reported in this article.

BACKGROUND

The Social-Control Life-Course Theory of Delinquency

Contemporary social-control theories (Gottfredson and Hirschi 1990; Hirschi 1969; Sampson and Laub 1993) can be traced to the eighteenth-century writings of Beccaria ([1764] 2004) and Bentham ([1789] 1970). For both, all human beings intrinsically seek pleasure and avoid pain. "Nature has placed mankind under the governance of two sovereign masters: pain and pleasure. It is for them alone to point out what we ought to do, as well as to determine what we shall do" (Bentham [1789] 1970:11). Applying Beccaria's principle to criminal behavior, Bentham believed that the pursuit of human pleasure is likely to lead to criminal acts unless the pursuit is checked by painful consequences. Such pain does not have to be physical, nor does it have to originate from the criminal justice system. The pain can be physical, political, moral, religious, or social. Individuals contemplating a criminal act simply weigh the pleasure against the pain involved.

A major theoretical and empirical development over the past two or three decades has been the formulation and testing of a life-course theory of informal social control. The theory emphasizes informal vis-à-vis formal social control. The latter includes such state sanctions as surveillance, enforced conformity, and incarceration. Informal social-control theories call attention to social bonds between an individual and society, suggesting that an individual is more likely to commit a crime when the bonds are weak or broken (Durkheim 1897; Gottfredson and Hirschi 1990; Hirschi 1969; Kornhauser 1978; Sampson and Laub 1993).

The life-course perspective, while stressing the importance of informal social ties at all ages across the life course, differentiates the roles played by important institutions of informal social control on the basis of age or life span. Family, school, and peer groups are the dominant institutions of informal social control in childhood and adolescence. In adulthood, the dominant social-control institutions are marriage and employment. Structural social conditions, such as neighborhood poverty and family socioeconomic status, are considered an underlying but necessary part of the explanation. The family and school processes are embedded in these structural conditions. These processes mediate the structural context.

According to Sampson and Laub (1993), the key to familial informal social control is linking the child to family and, ultimately, society. This may be accomplished by discipline, supervision, or attachment. Any family process that undermines any of the three aspects would undermine familial social control.

Schools are another dominant institution of social control in adolescence (Gottfredson and Hirschi 1990). With their authority and resources, schools are better organized and better equipped than the family to provide social control. Also, schools often are more impartial than the family in recognizing delinquency.

Peers are considered a major source of influence to mediate the impacts of structural background factors. Ethnographic studies repeatedly report the extraordinary importance of friends for adolescents, suggesting adolescents' vulnerability to peer influence (Corsaro and Eder 1990; Cusick 1973). The differential association theory (Sutherland 1947) and the differential reinforcement theory (Akers 1985) both regard intimate involvement with delinquent friends as essential for adoption of delinquency.

In the study of adolescent delinquency, the association between friends' delinquency and a respondent's delinquency is one of the most replicated findings (Matsueda 1982; Matsueda and Anderson 1998; Matsueda and Heimer 1987). Haynie (2001) examined the structural characteristics of adolescent friendship networks, including density (how closely an adolescent is integrated into the peer network), centrality (an adolescent's position in the net-

work), and popularity (prestige of an adolescent). She shows that these network characteristics influence the level and direction of the association with friends' delinquency.

Individual Propensities for Delinquency and Crime

The concept of self-control figures prominently in research on delinquency. This is because social control can have different effects due to differences in self-control or individual propensities. Measuring individual propensities, however, has proved to be difficult. All the propensity measures developed to date are based on delinquent behavior, and often delinquent behavior in early life. These endogenous propensity measures gauge behavior rather than propensity. They consequently tend to confound and mask the true effects of social control.

Caspi and colleagues (1994) suggest that crime-prone individuals tend to have a higher negative emotionality and a weaker constraint. Moffitt (1993) proposes two distinct categories of individuals: *adolescence-limited* and *life-course persistent.* The delinquent careers of adolescence-limited offenders are short and tend to be over when adolescence ends, whereas life-course persisters start early and persist in crime over the life course. The small number of life-course persisters is responsible for a disproportionately large number of adult crimes. Moffitt (1993) argues that the differences between the two categories are rooted in childhood, with life-course persisters associated with difficult temperament, poor verbal IQ, and low self-control.

Nagin and Land (1993), relying on statistical techniques to identify propensities, developed a mixed regression method that identifies groups of offenders with similar patterns of delinquency over the life course. They separated four distinct offending trajectories in a sample of British males: adolescence-limiteds, high-level chronics, low-level chronics, and nonoffenders. The group identification depends ultimately on the observed delinquency of the individuals.

Gottfredson and Hirschi (1990) devote their book *A General Theory of Crime* to the concept of self-control rather than societal control, which is the central theme of Hirschi's (1969) previous book, *Causes of Delinquency.* Gottfredson and Hirschi suggest measuring crime-prone propensity by a number of individual characteristics and behaviors, namely, an urge to gratify desires immediately; a lack of diligence and persistence in a course of action; a lack of commitment to job, marriage, and children; a lack of skills and planning; and a tendency to drink excessively, use illegal drugs, or gamble. These characteristics and behaviors indeed tend to be correlated with delinquent and criminal behavior. The measures, however, can be criticized for being tautological because they do not define self-control or propensity for delinquency separately from delinquency (Akers 1991). Any correlation thus merely means that delinquency predicts delinquency.

Below, we propose genotype as a measure of individual propensities for delinquency. Although DNA sequences are determined at conception and do not change except through mutation, their effects are, with rare exceptions, not deterministic because their expressions are subject to environmental influences immediately after conception. By examining genetic variations across individuals and their links to delinquency, we can begin to develop what Akers (1991) called "independent" indicators of self-control, for a more accurate definition of self-control.

Genetic Propensities for Delinquency and Crime

The evolutionary perspective argues for a role of genes in aggressive behavior. Here, aggressive behavior is adaptive because it could lend advantages in reproduction, protection of the young, and food acquirement. As Wilson (1975) points out, however, not all levels of aggression are adaptive or equally adaptive. Depending on the specific contexts of food attainment, living arrangements, physiology, and courtship patterns in each species, some level of aggressiveness may be optimal, above which fitness is reduced.

Wilson discusses two possible constraints that evolution may impose on aggressiveness. First, consciously or unconsciously, aggressiveness may be directed against genetically-related relatives. Reduced survival among relatives would lead to reduced survival of the genes shared among the aggressor and the relatives—including the genes that underlie aggressiveness. This process would constrain

aggressive behavior at an optimal level. Second, energy invested in brute force aggression could be invested in nonaggressive courtship, food attainment, and caring for the young. The evolutionary theory outlines only the plausibility of genetic influences in very general terms. All the relevant details will have to be worked out empirically.

An intriguing line of research derives from field observation of violent behavior among chimpanzees in the African wild (Boesch and Boesch-Achermann 2000; de Waal 2005; Wilson and Wrangham 2003; Wrangham and Peterson 1996). Chimpanzee violence differs from the pattern observed in other nonhuman primates and is characterized by premeditated deadly attacks on neighboring lone individuals or parties. The norm is intergroup hostility, which includes "group battles" and "gang attacks" among males. In group battles, many males join in but grievous injuries are not commonly observed. Gang attacks involve many males attacking a single individual and frequently result in grievous injuries and deaths. Males often brutally attack females and females with young offspring. On many occasions, males deliberately target a female's infant, killing and eating it.

Assuming that chimpanzees mirror who we were millions of years ago, the descriptive data suggest that human violence is rooted in prehuman history. One may even consider the descriptive data evidence for an adaptive role of violence in human evolution. Similar to observational human data, though, field observation of chimpanzees is hardly capable of separating genetic from nongenetic origins of violence.

Human geneticists have had remarkable success in identifying individual genes with variations that lead to simple Mendelian traits and diseases such as phenylketonuria (PKU), sickle-cell anemia, Tay-Sachs disease, and cystic fibrosis (Botstein and Risch 2003; Risch 2000). Mendelian traits and diseases are characterized by a one-to-one or near one-to-one correspondence with the individual genes. Diseases with simple Mendelian patterns of inheritance are uncommon. Most human diseases, traits, and outcomes (e.g., alcohol dependence and high blood pressure) are complex. That is, they are the consequence of many genes that typically contribute a small to moderate effect and that often interact with other genes and the environment. Most delinquent and violent behaviors are considered complex. Understanding these behaviors requires understanding both their socioeconomic–cultural components and their genetic components.

A number of twin and sibling studies report a genetic contribution to delinquency (Christiansen 1977; Gottesman, Carey, and Hanson 1983; Malone et al. 2004; Rodgers, Buster, and Rowe 2001). Rowe and Osgood's (1984) study represents an early investigation into genetic sources of delinquency using identical and fraternal twins. Molecular genetic studies of human delinquent and criminal behavior, however, are rare. Most evidence comes from animal models and psychiatric studies. Monoamine oxidase A *(MAOA)* is a major enzyme that catalyzes the oxidative deamination of a number of biogenic amines in the brain, including dopamine.[1] Using knockout-mouse models,[2] Cases and colleagues (1995) and Shih and Thompson (1999) developed a line of mice with a targeted disruption of the *MAOA* gene. As a result, they observed an increase in the brain levels of dopamine, serotonin, and norepinephrine, as well as an increase in manifested aggression among the males. Brunner and colleagues (1993) reported mental retardation and impulsive aggression among eight males in an extended Dutch family with an uncommon sex-specific point mutation in the *MAOA* gene.

Zhu and colleagues (Zhu, Chen, and Shih 1994; Zhu et al. 1992) and Sabol, Hu, and Hamer (1998) identified a 30-bp promoter region variable number tandem repeat (VNTR)[3] in *MAOA* affecting the level of transcriptional

[1] Dopamine is a type of neurotransmitter or chemical messenger that affects the brain processes that control movement, emotional response, and the capacity to feel pleasure and pain.

[2] A knockout mouse is a mouse that has had one or more of its genes made nonfunctional through a gene knockout. By comparing the phenotypes of these knockout mice with those of wild-type mice that still have all their genes intact, researchers can deduce the function of the targeted gene.

[3] Variable number of tandem repeats (VNTR) is one type of polymorphism in human DNA. It is the number of repeated segments at a locus that varies among individuals. The VNTRs can be used as molecular genetic markers.

activity that may be associated with psychiatric disorders and behavioral traits. The PCR product usually comprises five possible fragment sizes of 2, 3, 3.5, 4, and 5 copies of the repeat sequence. The 3 and 4 repeats are much more common than the 2, 3.5, and 5 repeats in human populations. Caspi and colleagues (2002) report that maltreated male children in New Zealand with the 3R or 5R of the VNTR in *MAOA* are more likely to engage in violent behavior than are maltreated children with the 3.5R or 4R of the VNTR. Guo and colleagues (2008) report evidence from the National Longitudinal Study of Adolescent Health (Add Health) that male youth possessing the 2R of the VNTR in the *MAOA* gene are associated with a much higher level of delinquency and a much lower level of promoter activity.

Many studies on genetic variants and aggression focus on the role of dopamine and its receptors and transport sites (de Almeida et al. 2005; Miczek and Fish 2005; Miczek et al. 2002). The pharmacotherapeutic interventions most commonly applied for human aggression use dopaminergic antagonists (de Almeida et al. 2005). For example, the dopamine D2 receptor *(DRD2)* antagonist haloperidol has long been used to treat aggressive behavior in psychotic patients. Civelli and colleagues were the first to clone a *DRD2* gene (Bunzow et al. 1988) and the first to describe the 3′ Taq1 polymorphism in the gene. The Taq1 polymorphism is a T to C transition in the 3′ noncoding region of the gene. There have been mixed findings with regard to the functional significance of this polymorphism. The *DRD2**A1 allele was shown to be associated with decreased receptor density in one study (Pohjalainen et al. 1998) but not in others (e.g., Laruelle, Gelernter, and Innis 1998). Evidence from Add Health shows that the *DRD2**178/304 genotype is associated with higher levels of delinquency than the *DRD2**304/304 or *DRD2**178/178 genotype (Guo, Roettger, and Shih 2007).

The dopamine transporter gene *(DAT1)* codes for a dopamine transporter protein (DAT), which limits the level and duration of dopamine receptor activation (Bannon and Whitty 1995). A knockout mice study, which selectively disabled the *DAT1* gene, established the central importance of the dopamine transporter in controlling synaptic dopamine levels and its role as an obligatory target for the behavioral and biochemical action of amphetamine and cocaine (Giros et al. 1996). Vandenbergh and colleagues (1992) identified a polymorphic 40-bp repeat in the transcribed portion of the gene, which most commonly repeats 9 (*DAT1**9R) to 10 times (*DAT1**10R). One study found that human subjects homozygous[4] for the 10R allele exhibit significantly lower dopamine transporter binding than do carriers of the 9R allele (Jacobsen et al. 2000), although this finding was not replicated in another study (Heinz et al. 2000). A number of studies demonstrate an association between the 10R allele in the *DAT1* gene and attention deficit hyperactivity disorder (ADHD) (Cook et al. 1995; Cornish et al. 2005; Daly et al. 1999; Gill et al. 1997; Waldman et al. 1998). The *DAT1**9R allele is reported to be associated with both a lower score in novelty seeking and greater success in smoking cessation (Sabol et al. 1999). Guo and colleagues (2007) show that the trajectories of serious and violent delinquency among youths in Add Health for the *DAT1**10R/10R and *DAT1**10R/9R genotypes are about twice as high as that for the *DAT1**9R/9R genotype.

Gene–Environment Interactions

Gene–environment interaction refers to the assumption that an environment may influence individuals' sensitivity to the effects of a genotype and vice versa (Hunter 2005). A classic example is that of PKU, an autosomal recessive disease that potentially causes hopeless mental and physical degeneration. However, only individuals with recessive mutations in the phenylalanine hydroxylase gene who are exposed to phenylalanine in the diet are susceptible to PKU (Khoury, Adams, and Flanders 1988). The disease or the gene expression can be controlled effectively by restricting the dietary intake of phenylalanine, starting within the first month after birth.

Another important social-science example comes from the work of Caspi and colleagues (2002). They found that a functional polymorphism in *MAOA* modifies the effect of maltreatment. Only maltreated children with a

[4] An organism is homozygous if it has the same allele on both of its homologous chromosomes. Different versions of genes are called alleles.

genotype generating low levels of *MAOA* expression tend to develop a violent behavior problem. Maltreated children with a genotype that produces high levels of *MAOA* activity, in contrast, are less affected.

Some recent educational performance studies using twins and siblings similarly report evidence of gene–environment interactions. Guo and Stearns (2002) show that heritability for a cognitive measure is much lower among those growing up in disadvantaged social environments than among those living in "normal" environments, suggesting genetic potential's dependence on social environments. Turkheimer and colleagues (2003) analyzed scores on the Wechsler Intelligence Scale for a sample of 7-year-old twins from the National Collaborative Perinatal Project. The results demonstrate that the proportions of IQ variance attributable to genes and environment vary with socioeconomic status. These models suggest that in impoverished families, the shared environment accounts for 60 percent of the variance in IQ and the contribution of genes is close to zero. In affluent families, the result is almost exactly the opposite.

Environmental measures used in a gene–environment interaction study, of course, may not be purely environmental. They may be partially determined by genetic influences. In this regard, animal models often are in a position to create genuine environmental conditions by manipulation. Suomi and colleagues assigned rhesus monkeys to one of two groups at birth: mother-reared (MR) or nursery- and peer-reared (NPR). During the first six months, the MR infants were reared in a group that consisted of 8 to 12 adult females, including their mothers. The NPR infants were separated from their mothers at birth and reared in a neonatal nursery. From the 37th day on, each NPR monkey was placed with three other monkeys of similar ages. No adult was included in the group. Using these experimental monkeys, a number of studies demonstrate interactions between the 5-HTTLPR polymorphism in the serotonin transporter gene (5-HTT) and rearing type. Among nursery- and peer-reared monkeys, the 5-HTT*l/l genotype has lower cerebrospinal fluid concentrations than the 5-HTT*l/s genotype (an indicator of central nervous system function) (Bennett et al. 2002), higher adrenocorticotropic hormone (ACTH) levels during a separation/stress experiment (interpreted as exaggerated limbic-hypothalamic-pituitary-adrenal [LHPA] responses to stress) (Barr et al. 2004b), lower visual orientation scores assessed on days 7, 14, 21, and 30 of life (Bennett et al. 2002), and an increased level of alcohol consumption among females (Barr et al. 2004a).

The mechanisms of gene–environment interaction are understood only in a few isolated cases. A particularly interesting case is the interplay between the maternal behavior of mother rats and the glucorticoid receptor gene for offspring's responses to stress (Meaney, Szyf, and Seckl 2007). Mother rats are classified into low or high licking/grooming (LG) and arched-back nursing (ABN). The latter is characterized by a mother rat nursing her offspring with her back arched and legs splayed outward. The offspring of low LG-ABN mothers were found to grow up more fearful and abnormally sensitive to stress than were offspring of high LG-ABN mothers. Cross-fostering studies, in which pups born to low LG-ABN mothers and high LG-ABN mothers were switched at birth, exclude the possibility of a direct transmission of maternal care to offspring stress responses (Francis et al. 1999).

One mechanism for gene–environment interaction is methylation, a process in which DNA sequences are chemically modified by acquiring methyl groups to cytosine bases. DNA methylation plays an important part in the regulation of gene expression. Mounting evidence shows that the silencing of tumor suppressor genes by DNA methylation is a typical process in cancer development (Baylin et al. 2001). Methylation is a main component of epigenetics, which are chemical instructions for gene activity that do not alter DNA sequences (Tsankova et al. 2007). Epigenetics promises to be the key to revealing the mechanisms that regulate gene expression in response to environment.

Meaney and colleagues (Weaver et al. 2004) discovered that rats' maternal behavior alters the dynamics of methylation and demethylation of the promoter in offspring's glucorticoid receptor genes. In response to stress, this receptor protein helps bring about gene expression in the brain. Methylation is only observed in the gene promoter shortly after birth (not before birth) and among offspring of low LG-ABN mothers.

It is hypothesized that low LG-ABN nursing causes the methylation, which leads to lowered levels of gene expression and produces more stressful animals. These biochemical and behavioral changes are stable and tend to last for the remainder of an animal's life.

In the analyses that follow, we examine the potential interactions between genetic variants and social-control processes. A genetic variant can correspond to higher or lower risks of delinquency. When a genetic variant is linked to a higher risk of delinquency, its detrimental effect may not be constant across social-control groups. Our general hypothesis is that *the effect of a delinquency-increasing genetic variant will tend to be suppressed among individuals exposed to higher levels of social control (e.g., those who have a strong attachment to school and those who grow up with two biological parents), and that the same effect tends to be amplified by lower levels of social control.*

We must add the caveat, however, that it is likely such effects will differ by sex. Female delinquent and criminal involvement has consistently been much lower than that of males. In the United States, large gender differences have been documented by official data since the FBI began data collection during the 1930s and by self-reported survey data. Gottfredson and Hirschi (1990) suggest that the two genders may be subject to quite different self-control mechanisms. Geneticists have recently discussed the sex-specific genetic architecture that underlies complex human traits (Weiss et al. 2006). Our preliminary analysis suggests that the genetic variants act on males and females differently. For these reasons, we focus on males in this study.

Objectives

Our analyses have two specific objectives. First, we examine the main effects of genetic propensities on serious and violent delinquency by adding—both separately and jointly—three genetic polymorphisms to a social-control model of delinquency. The three genetic polymorphisms, mentioned earlier, are the 30-bp promoter-region VNTR in the *MAOA* gene, the 40-bp VNTR in the *DAT1* gene, and the Taq1 polymorphism in the *DRD2* gene.

Second, and no less important, we examine potential interactive effects on serious and violent delinquency between the three genetic polymorphisms and key attributes and processes pertaining to family, school, and friend network social controls.

DATA AND MEASURES

Data Source

Our data are drawn from the DNA subsample in the National Longitudinal Study of Adolescent Health (Add Health), which started as a nationally representative sample of about 20,000 adolescents in grades 7 to 12 in 1994 to 1995 (Wave I) in the United States (Harris et al. 2003). Add Health is longitudinal; initial interviews with respondents were followed by two additional in-home interviews in 1996 (Wave II) and 2001 to 2002 (Wave III). Our analysis uses the sibling sample of Add Health because DNA measures collected at Wave III in 2002 are available only for this subset of the respondents. The subset consists of about 2,500 monozygotic (MZ) twins, dizygotic (DZ) twins, full biological siblings, and singletons. This study is based on approximately 1,100 males whose DNA and social-control measures are available.

Measures

Serious and violent delinquency. We construct a serious delinquency scale and a violent delinquency scale using 12 questions asked of all the Add Health respondents in Waves I to III. The questions and scaling weights used to create the scales are reported in the Online Supplement on the *ASR* Web site: http://www2.asanet.org/journals/asr/2008/toc064.html. These two scales are variations of a type of scale widely used in contemporary research on delinquency and criminal behavior (Thornberry and Krohn 2000). Our scales are closely related to the scales used, for example, by Hagan and Foster (2003) and Haynie (2001, 2003) in analyses of Add Health data and by Hannon (2003) in an analysis of data from the 1979 National Longitudinal Study of Youth.

Following the delinquency literature (Hagan and Foster 2003; Hannon 2003; Haynie 2001, 2003), we divide the 12 questions/items into nonviolent and violent categories. Nonviolent delinquency includes stealing amounts larger or smaller than $50, breaking and entering, and selling drugs. Violent delinquency includes seri-

ous physical fighting that resulted in injuries needing medical treatment, use of weapons to get something from someone, involvement in physical fighting between groups, shooting or stabbing someone, deliberately damaging property, and pulling a knife or gun on someone. The serious delinquency scale is based on all 12 items, and the violence scale is based on a subset (8) of the 12 items.

Cronbach's alpha values for the serious delinquency scale are .81 for Wave I, .79 for Wave II, and .73 for Wave III. Our serious delinquency scale overlaps Hagan and Foster's (2003) delinquency scale to a substantial extent. The serious delinquency scale is designed to capture a wide range of serious delinquent behavior that could result in state sanction, such as arrest, conviction, and incarceration. Hagan and Foster's (2003) 15-item scale includes most of the 12 items used in our scale, as well as a number of items on acts typically viewed as common adolescent deviance, such as lying to parents or guardians about where they had been, minor vandalism, being loud in a public place, and driving a car without its owner's permission. As the name suggests, our violent delinquency scale focuses on an array of violent delinquent behavior that could potentially be classified as violent offenses by the criminal justice system. The Cronbach alpha values for the violent delinquency scale are .75 for Wave I, .74 for Wave II, and .66 for Wave III.

Measuring delinquency, violence, and crime is admittedly challenging. Official measures based on police reports and the prison and court system substantially underestimate delinquency and crime because they reflect not only the behavior of offenders but also the political processes in the justice system (Hood and Sparks 1970; Murphy, Shirly, and Witmer 1946; Robison 1936; Thornberry and Krohn 2000). For these reasons, many criminologists have turned to self-reports in recent decades (Hindelang 1981; Hindelang, Hirschi, and Weis 1979; Thornberry and Krohn 2000). Self-reports, currently a fundamental method of measuring criminality, are capable of yielding reliable and valid data (Hindelang 2001; Thornberry and Krohn 2000).

As with any survey of sensitive private information, reporting accuracy is a concern. To protect confidentiality, reduce nonresponses, and increase reporting accuracy, this section of the interview in Add Health was self-administered by audio computer-assisted self-interview (audio-CASI). Sensitive questions were read to respondents by means of audio headphones. The computer gave respondents instructions on how to complete their answers. Self-reported rates of illegal and embarrassing behavior are higher when computer-assisted techniques, particularly self-administered techniques, are used (Tourangeau and Smith 1996; Wright, Aquilino, and Supple 1998).

The percentage of the U.S. adult population that has ever been incarcerated in a state or federal prison increases sharply among 25- to 34-year-olds, compared to 18- to 24-year-olds (Bonczar 2003). This points to a likely heavier sample attrition among more chronic offenders because of incarceration at Wave III but not at Waves I and II. Add Health Wave III records the specific reasons why some Wave I and Wave II respondents were not interviewed at Wave III. Approximately 12 individuals from the sibling sample were not interviewed due to incarceration. Chantala, Kalsbeek, and Andraca (2004) estimated the extent of underreporting at Wave III relative to Wave I, using the respondents and the reports at Wave I and taking advantage of the observation that some of the respondents at Wave I were nonresponders at Wave III. Their estimates indicate that most of the delinquent and violently delinquent activities could be underrepresented by 1 to 2.5 percent in the Wave III data relative to the Wave I population and that selling drugs, carrying a weapon, and shooting or stabbing someone could be underrepresented by about 5 percent. To reduce the potential impact of disproportional sample attrition at Wave III, we remove observations of serious and violent delinquency measured at age 24 or older.

SOCIAL CONTROL: STRUCTURAL AND DEMOGRAPHIC VARIABLES. Table 1 provides the descriptions, means, and standard deviations of the variables used in our analysis. The declining delinquency scores from Wave I to Wave III reflect the underlying age patterns of delinquency. The PVT, a slightly abridged version of the Peabody Picture Vocabulary Test (Lubin, Larsen, and Matarazzo 1984; Rice and Brown 1967), is usually considered a verbal IQ test. About 4 percent of the participants are missing on the PVT. The original religiosity, measured

Table 1. Variable Description, Means, and Standard Deviations

Variable Name	Description	Mean	SD
Serious and Violent Delinquency			
Wave I	Serious Delinquency Scale, Wave I	2.43	4.32
Wave II	Serious Delinquency Scale, Wave II	1.65	3.45
Wave III	Serious Delinquency Scale, Wave III	1.18	2.35
Wave I	Violent Delinquency Scale, Wave I	1.68	3.13
Wave II	Violent Delinquency Scale, Wave II	1.05	2.36
Wave III	Violent Delinquency Scale, Wave III	.69	1.62
Structural/Demographic			
Age/Ethnicity			
Age	Respondent's age at time of interview at Wave I	17.6	2.89
White	Respondent's race reported as white at Wave I	.603	.48
Black	Respondent's race reported as black at Wave I	.167	.372
Hispanic	Respondent's race reported as Hispanic at Wave I	.149	.357
Asian	Respondent's race reported as Asian at Wave I	.081	.271
Cognitive Development			
PVT < 90	Verbal IQ less than 90 at Wave I	.247	.431
PVT 90 to 110	Verbal IQ between 90 and 110 at Wave I	.484	.499
PVT > 110	Verbal IQ greater than 110 at Wave I	.269	.444
PVT Missing	Missing on IQ score at Wave I	.044	.205
Religiosity			
Weekly or more, WI	Respondent attends church weekly or more at Wave I	.352	.478
Weekly or more, WII	Respondent attends church weekly or more at Wave II	.475	.499
Weekly or more, WIII	Respondent attends church weekly or more at Wave III	.173	.378
Family SES			
Household size	Number of individuals living in household at Wave I	5.02	1.48
Parent jobless	Parent unemployed at Wave I	.053	.224
Jobless missing	Parent missing response on employment at Wave I	.127	.330
< High school	Parent interviewed has less than high school education	.238	.426
High school	Parent interviewed has high school education only at Wave I	.272	.456
> High school	Parent interviewed has education beyond high school	.490	.499
Contextual Traits			
Proportion black	Proportion black in census tract at 1990 Census	.128	.245
Family Process			
Daily family meals	Eats meals with parent 6 days per week at Wave I	.479	.50
Social services	Taken out of home by social services by 6th grade	.013	.11
Two biological parents	Lives with both parents at Wave I	.640	.480
Parental attachment	Emotional attachment to resident parent, Wave I	4.48	.74
Dad jailed	Biological parent served time in jail, Wave III	.14	.35
School Process			
Repeated a grade	Repeated grade by Wave I	.257	.437
School attachment	Emotional attachment to school at Wave I	2.21	.83
Peer problems	Problems getting along with other students, Wave I	.076	.26
Truancy in last year	Has 5 or more unexcused absences from school, Wave I	.097	.30
Being expelled	Expelled from school by Wave I	.031	.174
Social Networks Wave I			
Friends delinquency	Friends' delinquent behavior at Wave I	5.96	3.75
Centrality	Respondent's centrality in friends social network	.81	.67
Density	Respondent's density in friends social network	.28	.14
Popularity	Respondent's popularity in friends social network	4.84	4.00
Genotype			
9R/9R	Proportion of 9R/9R genotype in *DAT1*	.053	.223
10R/9R	Proportion of 10R/9R genotype in *DAT1*	.348	.476
10R/10R	Proportion of 10R/10R genotype in *DAT1*	.599	.490
178/304	Proportion of A1/A2 genotype in *DRD2*	.372	.497
178/178	Proportion of A2/A2 genotype in *DRD2*	.549	.483
304/304	Proportion of A1/A21genotype in *DRD2*	.079	.271
2R	Proportion of 2R/Other genotype in *MAOA*	.008	.089

Notes: N = 1,111 persons; 3,071 person-observations; fewer when some family, school, and social network variables are considered.

by church attendance in all three waves, has four categories: never, less than monthly, less than weekly, and weekly or more. Our exploratory data analysis shows that the main distinction is between "weekly or more" and the other three categories. We create a dummy variable to reflect this result.

Household size measures household crowding and includes all individuals living in the household at Wave I. *Parent jobless* measures parental unemployment, which is coded 1 if one or two parents were unemployed at Wave I and 0 otherwise. *Education* refers to the education level of the adult interviewed at home at Wave I (categories are less than high school graduation, high school graduation, and at least some college education). We also consider a number of contextual characteristics and, in our final analysis, focus on the percentage of African Americans in the Census tract.

SOCIAL CONTROL: FAMILY PROCESS VARIABLES. *Two biological parents* is based on a family structure variable in Add Health that has categories of two biological parents, single parent, stepparent, and other families including children from adopted families and foster homes (Harris, Duncan, and Boisjoly 2002). We created a dummy variable for two biological parent families versus all others. *Daily family meals* is based on the Add Health Wave I question, "On how many of the past 7 days was at least one of your parents in the room with you while you ate your evening meal?" We coded the answer as a dummy variable with six or seven days as 1 and fewer than six as 0. Wave I parental attachment is an average of two variables constructed from "How close do you feel toward your resident mother or resident father?" and "How much do your parents care about you?" Both range from 1 to 5. *Dad jailed,* coded 0 or 1, is constructed from the Wave III question, "Has your biological father ever served time in jail or prison?" *Social services,* also from Wave III, is coded 1 if the respondent reported having been taken out of the home by social services before the sixth grade.

SOCIAL CONTROL: SCHOOL PROCESS VARIABLES. *Repeating a grade* is coded 1 if the respondent had repeated a grade by Wave I. About one-fourth of Add Health respondents had repeated a grade by Wave I. *School attachment* (Haynie 2001) is an average of the responses (each ranging from 1 to 5) to the three Wave I questions concerning whether a respondent last year felt close to people at school, felt like being part of school, and was happy to be at school. *Peer problems* is based on the Wave I self-report of daily problems getting along with peers at school. The variable is coded 1 if the answer is "almost everyday" or "every day" and 0 otherwise. *Truancy* is a measure of skipping school for a full day without an excuse last year. It is coded 1 if the number of unexcused absences is 5 or more.

SOCIAL CONTROL: FRIEND SOCIAL-NETWORK VARIABLES. These variables include centrality, density, popularity, and friend delinquency (Haynie 2001). Our centrality measure, developed by Bonacich (1987), attempts to gauge adolescents' positions within their friend networks. It is a measure of the number of links required to connect all other adolescents in a person's friendship network. The lower the number of links required, the more central the adolescent. The measure is weighted by the centrality of those a person nominates as friends. This measure of centrality takes into consideration not only a respondent's position but also the person's friends' social positions.

The most dense network possible is one in which every member has ties to every other member. Density is measured by the observed number of ties divided by the number of possible ties in an adolescent's friendship network, standardized by the maximum number of friends a respondent can nominate. The ties include both "send" and "receive" nominations. An average density value of .28 indicates that 72 percent of the potential pairwise ties in an adolescent's social network are not nominated.

Popularity is measured by the number of receive nominations, or the number of times the respondent is nominated by other students in school. Each adolescent was nominated as a friend an average of 4.84 times.

Friend delinquency is measured by the average number of self-reported minor delinquency items over the past 12 months per send-and-receive friend nomination. The minor delinquency items include *smoked cigarettes, drank alcohol, got drunk, skipped school without an excuse, did dangerous things on a dare,*

and *raced vehicles such as cars or motorcycles*. The measure is based on responses obtained directly from the friends themselves at the Add Health Wave I school interview. Almost all studies on peer influences use data based on a respondent's perceptions of a friend's behavior instead of the actual behavior of a friend. Perceptions of friends' behavior are considered unreliable because reporters tend to project their own behavior onto others (Bauman and Ennett 1996). This perception bias can be corrected only with data that allow the measures of friends to be taken directly.

Compared with the delinquency items obtained from the in-home surveys at Waves I to III, and used to construct our dependent variables, the friend delinquency items are fewer and more minor, but they are the only delinquency items available from the friends themselves. Friend delinquency has a mean value of 5.96, indicating that friends committed an average of six minor delinquent activities over the past 12 months.

GENETIC VARIANTS. At Wave III, in collaboration with the Institute for Behavioral Genetics in Boulder, Colorado, Add Health collected, extracted, and quantified DNA samples from the sibling subsample. This article reports findings from three genetic polymorphisms in three genes: a 40-bp VNTR polymorphism in the 3′ region of the *DAT1* gene, a polymorphic Taq1A restriction endonuclease site about 2,500 bp downstream from the coding region of the *DRD2* gene, and the 30-bp VNTR in the promoter region of the *MAOA* gene. Additional details about these genetic polymorphisms can be found in our Online Supplement and at the Add Health Web site.

ANALYTICAL STRATEGY AND RESULTS

We present results from both exploratory contingency table analysis and regression analysis. The contingency table analysis compares the mean scores of serious delinquency and violent delinquency across genotypes and age group. The regression analysis uses mixed regression models (Searle 1971; Searle, Casella, and McCulloch 1992), which are essentially random-effects models or multilevel models. Our sample consists of twins and siblings measured repeatedly over Add Health waves. These measures are not independent. The mixed models have long been established in the statistical literature for analysis of data that are not independent. The following equation describes the basic structure of our models:

$$Delinquency_{jit(s)} = \beta_{0ji(s)} + \mathbf{D}_{jit}\,\boldsymbol{\beta_1} + \mathbf{SC}_{ji}\,\boldsymbol{\beta_2} + \mathbf{G}_{ji}\,\boldsymbol{\beta_3} + e_{jit(s)}\text{ , (level 1 model)}$$

$$\beta_{0ji(s)} = \beta_{0j(s)} + \nu_{ji}\text{ , (level 2 model)}$$

$$\beta_{0j(s)} = \beta_0 + u_{0j(s)}\text{ , (level 3 model)}$$

where *delinquency* is either serious delinquency or violent delinquency measured at Wave *t,* for individual *i,* in sibling cluster *j,* and for type of sibling cluster *s* (MZ twins, DZ twins, or full siblings); **D** is a row vector of demographic covariates for Wave *t,* individual *i,* and sibling cluster *j*; **SC** is a row vector of social control covariates measuring social-structural conditions, family processes, school processes, and social networks; **G** is a row vector of covariates measuring genetic variants in *DAT1, DRD2,* and *MAOA;* and $u_{0j(s)}$, ν_{ji}, and $e_{jit(s)}$ are random effects at the level of sibling cluster, individual, and Add Health wave, respectively. Gene–environment interaction terms can be added to this equation readily.

The basic trajectory of serious and violent delinquency is described by age and age^2 and their parameters. The model allows the random effect at the sibling cluster level and the level of delinquency measures to vary by type of sibling cluster because the strength of the correlation in these types of sibling clusters varies considerably. Random coefficients for age and age^2 were tested and dropped because they were not significant. Conditional on the three random intercepts at the level of sibling clusters and one random intercept at the individual level, the siblings and repeated measures are independent.

Population stratification is a major concern in genetic association studies (Marchini et al. 2004).[5] We address the potential impact of pop-

[5] An association between a genetic variant and a human outcome can be false-positive (or false-negative) because individuals with the outcome and individuals without the outcome have different genetic backgrounds or different ancestral population origins.

ulation structure by adjusting for self-reported race/ethnicity in all regression analyses so that the comparisons across genotypes are made after adjusting for the effects of race/ethnicity. We also apply Allison and colleague's (1999) procedure for testing for possible population stratification (data not shown). Our findings do not seem to be affected by population stratification.

Findings From Exploratory Analysis

Table 2 compares the mean score of serious delinquency and violent delinquency across genotypes by age group among males. Both serious delinquency and violent delinquency peak at ages 16 to 18, consistent with the age patterns of delinquency typically found among adolescents and young adults. For the *DAT1* gene, the most prominent result is the sharply reduced serious and violent delinquency scores in each of the three age groups for individuals with the *DAT1**9R/9R genotype as compared with the *DAT1**10R/10R genotype and the *DAT1**10R/9R genotype. For example, individuals ages 16 to 18 with the 9R/9R genotype scored an average of 1.28 on the serious delinquency scale, which is much lower than 2.11 for the 10R/10R genotype and 2.23 for the 10R/9R genotype.

For the *DRD2* gene, the participants heterozygous for the 178 and 304 alleles consistently scored higher on both the serious delinquency and the violent delinquency scales across all three age groups than did the homozygotes of the *DRD2**304/304 and *DRD2**178/178 genotypes. Comings and MacMurray (2000) review this pattern of much higher or lower value for the heterozyotes relative to both types of homozygotes and describe it as heterosis. For the *MAOA* gene, the male participants with a 2 repeat on the X chromosome reported much higher levels of serious and violent delinquency. These initial exploratory associations concerning *DAT1, DRD2,* and *MAOA* do not seem to vary by age group, suggesting an absence of interaction between the genotype effects and life stages of adolescence and young adulthood. The next section presents significance test results for the genotype effects obtained from the mixed regression-models that take into account the sibling clustering.

Main Effects of Social Control and Genetic Propensities

The model of social control in Table 3, which does not consider genetic variants, reveals significant effects, and in the expected directions, particularly for repeating a grade in school, religiosity, parental unemployment, and daily family meals. Repeating a grade is associated with higher serious delinquency, although the result is only marginally significant ($p = .09$). Attending church weekly or more corresponds with a much lower serious delinquency score,

Table 2. Mean Score of Serious Delinquency and Violent Delinquency by Genotype and Age; Add Health Males, Waves I to III

	Genotype	Genotype Frequency at Wave I	Serious Delinquency			Violent Delinquency		
Age Range			12–15	16–18	19–23	12–15	16–18	19–23
Gene								
DAT1	10R/10R	654	2.11	2.11	1.17	1.37	1.40	.710
	10R/9R	378	2.28	2.23	1.23	1.63	1.51	.73
	9R/9R	**56**	**1.17**	**1.28**	**.62**	**.76**	**.92**	**.29**
	Other/Other	42	1.97	1.59	1.65	1.46	1.04	1.03
	Sample size	1130	872	1292	1095	872	1292	1095
DRD2	178/178	619	2.03	1.84	1.20	1.33	1.21	.68
	178/304	**425**	**2.38**	**2.56**	**1.20**	**1.68**	**1.75**	**.67**
	304/304	89	1.52	1.69	.95	1.09	1.13	.62
	Sample size	1113	868	1284	1098	868	1284	1094
MAOA	**2R**	**11**	**5.78**	**3.23**	**2.20**	**4.33**	**2.53**	**1.70**
	No 2R	1115	2.07	2.10	1.17	1.40	1.40	.70
	Sample size	1126	865	1281	1095	865	1281	1095

Table 3. Coefficients (standard errors) of Random-Effects Models of Serious Delinquency among Male Adolescents and Young Adults: Social Control and Genetic Propensities (Add Health Waves I to III)

Models	Social Control	*DAT1*	*DRD2*	*MAOA*	3 Combined
Intercept	–2.207(2.209)	–2.996(2.230)	–1.975(2.212)	–2.259(2.209)	–2.755(2.232)
Age/Ethnicity					
Age	.628(.242)**	.624(.241)**	.622(.242)**	.632(.242)**	.621(.241)**
Age^2	–.022(.007)***	–.022(.007)***	–.022(.007)***	–.022(.007)***	–.022(.007)***
White					
Black	–.013(.326)	–.023(.326)	–.020(.327)	–.057(.327)	.008(.326)
Hispanic	.527(.256)*	.551(.256)*	.599(.262)*	.511(.257)	.599(.261)*
Asian	.537(.312)	.540(.315)	.524(.315)	.534(.313)	.529(.317)
School Attachment					
Repeated grade	.339(.19)	.332(.189)	.331(.19)	.343(.19)	.329(.19)
PVT < 90	.037(.254)	.031(.256)	.035(.256)	.04(.255)	.036(.255)
PVT 90 to 110	.213(.192)	.216(.192)	.191(.193)	.207(.193)	.195(.192)
PVT > 110					
PVT missing	–.398(.415)	–.373(.416)	–.446(.417)	–.387(.416)	–.406(.415)
Religiosity					
Weekly or more	–.768(.14)***	–.767(.139)***	–.758(.14)***	–.766(.14)***	–.755(.143)***
Family SES					
Two bio. parents	–.178(.182)	–.171(.182)	–.152(.182)	–.173(.182)	–.143(.181)
Others					
Household size	.018(.056)	.023(.056)	.022(.056)	.023(.056)	.031(.056)
Parent jobless	.781(.379)*	.782(.379)*	.821(.379)*	.769(.378)*	.808(.378)*
Jobless missing	.164(.32)	.178(.32)	.174(.321)	.170(.322)	.194(.321)
< High school	–.280(.285)	–.299(.284)	–.263(.285)	–.291(.286)	–.293(.285)
High school					
> High school	.106(.2)	.126(.2)	.117(.2)	.113(.2)	.121(.199)
Daily fam. meals	–.459(.157)**	–.458(.156)**	–.448(.157)**	–.454(.157)**	–.454(.156)**
Contextual Characteristics					
Proportion black	.756(.472)	.741(.472)	.719(.472)	.708(.472)	.661(.471)
Genotype					
9R/9R					
10R/9R		.966(.361)**			.893(.362)*
10R/10R		.747(.356)*			.667(.357)
178/304					
178/178			–.276(.17)		–.259(.17)
304/304			–.843(.312)**		–.773(.311)*
2R				1.799(.870)*	1.726(.866)*
No 2R					
Random Effects					
σ^2_{u}, MZ	5.543(1.46)***	5.548(1.502)***	5.338(1.458)***	5.605(1.515)***	5.431(1.479)***
σ^2_{u}, DZ	4.292 (.955)***	4.316 (.988)***	4.440 (.955)***	4.225 (.979)***	4.334 (.981)***
σ^2_{u}, ful sib	1.755 (.4)***	1.810 (.399)***	1.774 (.398)***	1.740 (.398)***	1.813 (.393)***
σ^2_{v}, person	1.216 (.358)***	1.138 (.359)***	1.159 (.354)***	1.204 (.361)***	1.081 (.353)**
σ^2_{e}, MZ	11.171(1.018)***	11.212(1.018)***	11.200 (.99)***	11.174(1.01)***	11.238(1.019)***
σ^2_{e}, DZ	7.620 (.552)***	7.553 (.560)***	7.619 (.552)***	7.623 (.562)***	7.627 (.561)***
σ^2_{e}, ful sib	7.87 (.3)***	7.912 (.299)***	7.865 (.3)***	7.873 (.3)***	7.860 (.299)***
P against *social control*	—	.0247	.0136	.0404	.0035
–2 Log L	16,006.9	15,999.5	15,998.3	16,002.7	15,988.3
N of persons	1,111	1,111	1,111	1,111	1,111
N of measures	3,071	3,071	3,071	3,071	3,071

* $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$ (two-tailed tests).

and the result is highly significant. Parental unemployment is linked to a much higher serious delinquency score, and having meals daily with a parent has a large protective effect on serious delinquency. As expected, all four between-variances for MZ twin pairs, DZ twin pairs, full-sibling clusters, and repeated measures of the same individual are highly significant.

The models of *DAT1, DRD2,* and *MAOA* in Table 3 each add one genetic polymorphism in one of the three genes to the model of social control. The estimated effects of these genetic variants are highly consistent with those from the contingency table analysis in Table 2. The models of *DAT1* include only participants with the *DAT1**10R/10R, *DAT1**10R/9R, and *DAT1**9R/9R genotypes. The three genotypes amount to about 98 percent of the sample. The *DAT1* model shows that individuals with 10R/10R and 10R/9R genotypes scored, respectively, .96 and .75 points higher on the serious delinquency scale than did those with the 9R/9R genotype. The corresponding *p* values are .0079 and .034, respectively. A likelihood ratio test for the two categories of 10R/10R and 10R/9R against 9R/9R yields an χ^2 statistic of 7.4 with 2 df ($p = .025$).

In the model of *DRD2,* the homozygous 178/178 and the 304/304 genotypes scored lower than the heterozygotes (178/304) (.28 and .84 lower, with *p* values of .098 and .0054, respectively). A likelihood ratio test of the two categories of the homozygotes against the heterozyotes yields an χ^2 statistic of 8.6 with 2 df ($p = .014$). In the model of *MAOA,* the 2 repeat genotype is associated with a much higher serious delinquency score (1.80; $p = .040$) than are the other genotypes. When three genetic polymorphisms are added to the model of social control (last model in Table 3), the effects of these genetic variants and their *p* values are essentially the same as the models in which one polymorphism is added at a time, suggesting an absence of gene–gene correlations or that these three genetic variants are independently predicting delinquency. A likelihood ratio test against the social control model produces an χ^2 statistic of 18.6 with 5 df ($p = .0035$). The results for violent delinquency in Table 4 are similar to those for serious delinquency in Table 3. The estimated effects of genetic variants are very similar across the two sets of results.

Tables 3 and 4 both report the number of participants as well as the number of observations used in a model. Most participants contributed three observations to the analysis. A small number of participants contributed fewer than three. To facilitate comparison and likelihood ratio tests across different models to test genotype effects, we use exactly the same number of observations in the models on serious delinquency (3,071) and violent delinquency (3,071). Using samples with the maximum number of observations (3,243) yields almost identical results.

Gene–Environment Interactions

Table 5 presents models that investigate the gene–environment interaction between *MAOA* and *grade retention* and the interaction between *DRD2* and *having daily family meals.* All the interaction terms are statistically significant. The *p* values for the two *MAOA* interaction terms are .0005 (serious delinquency) and .0001 (violent delinquency), respectively. The *p* values for the two *DRD2* interaction terms are .023 and .0069, respectively. The likelihood ratio test of the model of serious delinquency *with* two interaction terms (two combined model) against the model (not shown here) *without* the two interaction terms produces an χ^2 of 15.6 with 2 df and a *p* value of .0003. The parallel model of violent delinquency produces an χ^2 of 23.6 with 2 df and a *p* value less than .0001.

These gene–environment interaction findings indicate that *certain genotype effects and the effects of social control are mutually dependent.* For example, in the *MAOA* model of serious delinquency, the effect of repeating a grade depends on whether one has a 2 repeat in *MAOA.* Without a 2 repeat, repeating a grade raises the serious delinquency score by only .30. With a 2 repeat allele, repeating a grade raises the score by the large value of 6.44. The aforementioned interaction term is interpreted as an effect of grade retention that depends on a genotype. An interaction term can also be interpreted as a genotype effect that hinges on the level of social control. For example, in the *DRD2* model of serious delinquency, for those who do not have regular meals with a parent, having the 178/304 genotype raises the delinquency score by .70 points. For those who do have daily meals with a parent, however, the negative effect of 178/304 is completely suppressed ($.70 - .72 \approx 0$).

The estimates in the last two models in Table 5 that consider two interaction terms jointly are very similar to those in the models that consid-

Table 4. Coefficients (standard errors) of Random-Effects Models of Violent Delinquency among Male Adolescents and Young Adults: Social Control and Genetic Propensities (Add Health Waves I to III)

Models	Social Control	*DAT1*	*DRD2*	*MAOA*	3 Combined
Intercept	–.498(1.565)	–.997(1.567)	–.279(1.562)	–.545(1.56)	–.779(1.581)
Age/Ethnicity					
Age	.308(.171)	.306(.171)	.304(.172)	.312(.172)	.305(.171)
Age^2	–.012(.005)**	–.012(.005)**	–.012(.005)**	–.012(.005)**	–.012(.005)**
White					
Black	–.026(.228)	–.001(.228)	–.011(.229)	–.063(.228)	–.024(.229)
Hispanic	.317(.18)	.335(.180)	.357(.183)*	.301(.18)	.355(.184)
Asian	.280(.218)	.293(.221)	.257(.22)	.276(.218)	.270(.222)
School Attachment					
Repeated grade	.344(.134)*	.341(.134)*	.335(.134)*	.347(.134)**	.336(.133)*
PVT < 90	.3214(.179)	.217(.179)	.208(.179)	.216(.179)	.215(.18)
PVT 90 to 110	.208(.135)	.214(.135)	.191(.135)	.205(.135)	.195(.135)
PVT > 110					
PVT missing	–.387(.293)	–.369(.294)	–.421(.294)	–.379(.293)	–.39(.293)
Religiosity					
Weekly or more	–.467(.102)***	–.46(.101)***	–.461(.10)***	–.466(.101)***	–.459(.099)***
Family SES					
Two biological parents	–.093(.128)	–.086(.128)	–.073(.128)	–.09(.128)	–.064(.128)
Others					
Household size	.017(.039)	.021(.039)	.02(.039)	.021(.04)	.026(.04)
Parent jobless	.342(.267)	.348(.266)	.367(.266)	.335(.267)	.362(.265)
Jobless missing	.108(.225)	.116(.226)	.095(.225)	.115(.226)	.130(.226)
< High school	–.244(.2)	–.257(.199)	–.237(.2)	–.252(.2)	–.257(.201)
High school					
> High school	–.042(.14)	–.043(.14)	–.046(.14)	–.045(.14)	–.044(.14)
Daily family meals	–.287(.11)**	–.308(.11)**	–.287(.11)**	–.284(.11)*	–.285(.11)**
Contextual Characteristics					
Proportion black	.624(.331)	.614(.331)	.591(.33)	.586(.332)	.547(.329)
Genotype					
9R/9R					
10R/9R		.634(.256)*			.578(.257)*
10R/10R		.442(.252)			.381(.252)
178/304					
178/178			–.271(.119)*		–.257(.119)*
304/304			–.617(.218)**		–.567(.218)**
2R				1.55(.621)*	1.499(.618)*
No 2R					
Random Effects Omitted					
P against *social control*		.0317	.0058	.0128	.0005
–2 Log L	13,889.1	13,882.0	13,878.8	13,882.9	13,866.8
N of persons	1,111	1,111	1,111	1,111	1,111
N of measures	3,071	3,071	3,071	3,071	3,071

* $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$ (two-tailed tests).

er one interaction term at a time. The parameter estimates in the joint model are slightly smaller, and the *p* values are slightly larger than those in the single-term models, suggesting the absence of major correlations among the two genetic polymorphisms.

Additional Gene–Environment Interactions with Family Processes, School Processes, and Friend Social Networks

We systematically test additional gene–environment interactions with indicators of family

Table 5. Coefficients (standard errors) of Random-Effects Models of Serious and Violent Delinquency among Male Adolescents and Young Adults: Interactions between Genetic Propensities and Social Controls (Add Health Waves I to III)

Models	*MAOA*		*DRD2*		Two Combined	
	Serious Delinquency	Violent Delinquency	Serious Delinquency	Violent Delinquency	Serious Delinquency	Violent Delinquency
Intercept	–2.366(2.207)	–.639(1.56)	–2.476(2.209)	–.738(1.562)	–2.622(2.206)	–.867(1.561)
Age	.639(.242)**	.318(.172)	.629(.242)**	.310(.172)	.639(.242)**	.319(.172)*
Age^2	–.022(.007)***	–.012(.005)**	–.022(.007)***	–.012(.005)**	–.022(.007)***	–.012(.005)**
White						
Black	–.068(.324)	–.076(.225)	.048(.325)	–.057(.226)	–.10(.323)	–.104(.225)
Hispanic	.548(.255)*	.331(.178)	.509(.256)*	.301(.18)	.531(.254)*	.318(.178)
Asian	.521(.311)	.267(.216)	.487(.316)	.235(.22)	.474(.311)	.224(.216)
School Attachment						
Repeated grade	.299(.190)	.309(.132)*	.306(.191)	.316(.134)*	.268(.189)	.284(.133)*
PVT < 90	.022(.253)	.2(.178)	.040(.256)	.215(.179)	.025(.253)	.200(.177)
PVT 90 to 110	.215(.191)	.211(.134)	.18(.193)	.181(.136)	.185(.191)	.185(.134)
PVT > 110						
PVT missing	–.358(.415)	–.356(.292)	–.411(.418)	–.401(.294)	–.370(.415)	–.370(.291)
Religiosity						
Weekly or more	–.761(.143)***	–.462(.101)***	–.779(.14)***	–.477(.099)***	–.772(.143)***	–.471(.099)***
Family SES						
Two biological parents	–.160(.181)	–.078(.126)	–.155(.183)	–.073(.128)	–.138(.181)	–.059(.127)
Others						
Household size	.030(.056)	.027(.039)	.027(.056)	.025(.04)	.038(.056)	.033(.039)
Parent jobless	.791(.376)*	.355(.263)	.826(.379)*	.381(.267)	.833(.376)*	.392(.264)
Jobless missing	.231(.322)	.163(.223)	.132(.322)	.083(.227)	.203(.320)	.138(.223)
< High school	–.358(.284)	–.303(.198)	–.280(.287)	–.244(.201)	–.357(.285)	–.302(.199)
High school						
> High school	.099(.198)	–.058(.138)	.107(.201)	–.051(.14)	.090(.199)	–.065(.138)
Daily family meals	–.464(.157)**	–.276(.11)**	–.198(.196)	–.067(.138)	–.191(.194)	–.065(.136)
Contextual Traits						
Proportion black	.708(.468)	.594(.327)	.708(.471)	.581(.332)	.663(.468)	.553(.326)

(continued on next page)

Table 5. *(continued)*

Models	*MAOA*		*DRD2*		Two Combined	
	Serious Delinquency	Violent Delinquency	Serious Delinquency	Violent Delinquency	Serious Delinquency	Violent Delinquency
Genotype						
178/304			.692(.219)**	.602(.155)***	.664(.219)**	.577(.153)***
178/178 or 304/304						
2R	–.213(1.036)	–.137(.739)			–.19(1.032)	–.118(.734)
No 2R						
GxE Interaction						
Repeated grade*2R	6.44(1.84)***	5.479(1.32)***			6.27(1.834)***	5.323 (1.312)***
Daily meals*178/304			–.719(.317)*	–.605(.222)**	–.693(.313)*	–.581(.219)**
Random Effects Omitted						
2 interact terms					$p = .0003$	$p < .0001$
–2 Log L	15,990.5	13,865.9	15,996.9	13,873.9	15,981.2	13,851.6
N of persons	1,111	1,111	1,111	1,111	1,111	1,111
N of measures	3,071	3,071	3,071	3,071	3,071	3,071

* $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$ (two-tailed tests).

processes, school processes, and friend social networks. In Table 6, we present interaction results between family processes and *DRD2**178/304, which is shown to be interacting with *family meals* in Table 5. The two models of *family meals* (Models 1 and 7) for serious and violent delinquency are the same as those presented in Table 5 except that the models in Table 6 use fewer observations because of missing values in the additional family process variables. The models in Table 6 show only the main and interaction effects directly relevant to the particular interaction. All the other coefficients are omitted. Models 2 to 5 and 8 to 11 replace *family meals* with another family indicator. Models 6 and 12 consider three family process indicators simultaneously: family meals, social services, and two biological parents.

From the perspective of gene–environment interactions, *social services* and *two biological parents* act similarly to *family meals.* In the case of *social services,* the joint presence of both the riskier allele (178/304) and *social services* substantially heightens serious (2.86) and violent (1.98) delinquency. In the case of *two biological parents,* growing up with two biological parents reduces the level of serious (–.736) and violent (–.458) delinquency for those possessing the riskier allele. *Parent attachment* and *dad jailed* do not seem to interact with the genetic variant.

Table 7 is parallel to Table 6 and presents interaction results for school process indicators. In addition to *repeating a grade, school attachment* shows significant interactions with *MAOA**2R. Consistent with expectations, both *school attachment* and *MAOA**2R are significantly associated with delinquency. However, for those *MAOA**2Rs, each level of school attachment reduces the serious delinquency scale by 3.32 and the violent delinquency scale by 2.23. *Peer problems, truancy,* and *being expelled* all have a strong positive main effect on delinquency, but they do not seem to interact with *MAOA**2R.

Table 6. Coefficients in the Random-Effects Models of Serious and Violent Delinquency among Male Adolescents and Young Adults: Interactions between Genetic Propensities and Family Processes (Add Health Waves I to III)

	Family Meals	Social Services	2 Bio-Parents	Parent Attachment	Dad Jailed	Family Meals	Social Services	2 Bio-Parents
	Serious Delinquency							
	Model 1	Model 2	Model 3	Model 4	Model 5		Model 6	
Family	–.209	1.10	–.134	–.834***	.748**	.033	1.27	–.229
*DRD2*178/304*	.629**	.311	.834**	–1.035	.277	1.13***		
GE interaction	–.583	2.86	–.736*	.295	.172	–.759*	2.62	–.558
	Violent Delinquency							
	Model 7	Model 8	Model 9	Model 10	Model 11		Model 12	
Family	–.076	.809	–.084	–.578***	.462*	.032	.905	–.099
*DRD2*178/304*	.548**	.270*	.598**	–.451	.234	.877***		
GE interaction	–.548*	1.98	–.458	.157	.204	–.48	1.82	–.51*
N of measures	2,798	2,798	2,798	2,798	2,798		2,798	
N of persons	1,023	1,023	1,023	1,023	1,023		1,023	

Note: The interaction terms are added to the same basic models as in Tables 3 to 5.
* $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$ (two-tailed tests).

The basic models in Table 8 are the same as those in Tables 3 and 4. To these basic models, we add indicators of friendship networks and gene–environment interactions. Haynie (2001) interprets only the two-way interaction effects between friend delinquency and centrality, density, and popularity. Only one interaction in our analysis, the one between friend delinquency and centrality, is consistent with Haynie's. One likely source for this discrepancy is analysis samples. Whereas Haynie's results are based on the entire Add Health sample of more than 13,000 individuals, we derive our results from 755 males for whom the network and DNA data are available.

The interaction effects between friend delinquency and *DRD2**178/304 indicate that for those possessing the riskier 178/304 genotype,

Table 7. Coefficients in the Random-Effects Models of Serious and Violent Delinquency among Male Adolescents and Young Adults: Interactions between Genetic Propensities and School Processes (Add Health Waves I to III)

	Repeating Grade	School Attachment	Peer Problems	Truancy	Being Expelled	Repeating Grade	School Attachment
	Serious Delinquency						
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	
School	.451**	–.59***	1.28***	1.62***	2.08***	.153	–.595***
*MAOA*2R*	–.24	14.1***	2.06*	1.98*	1.60*	6.10	
GE interaction	6.61***	–3.32**	–2.63	–2.87	–.31	4.83*	–1.56
	Violent Delinquency						
	Model 7	Model 8	Model 9	Model 10	Model 11	Model 12	
School	.26*	–.338***	.891***	1.21***	1.56***	.222	–.336***
*MAOA*2R*	–.15	–3.91*	1.70**	1.77*	1.36**	2.42	
GE interaction	5.56***	–2.23**	–1.70	–3.30	–.145	4.82**	–.637
N of measures	3,020	3,020	3,020	3,020	3,020	3,020	
N of persons	1,094	1,094	1,094	1,094	1,094	1,094	

Note: The interaction terms are added to the same basic models as in Tables 3 to 5.
* $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$ (two-tailed tests).

Table 8. Coefficients in the Random-Effects Models of Serious and Violent Delinquency among Male Adolescents and Young Adults: Interaction between Genetic Propensities and Friendship Networks (Add Health Waves I to III)

	Serious Delinquency	Violent Delinquency
Social Network		
Centrality	–.83**	–.69(.22)**
Density	1.79	1.73*
Popularity	.093	.091*
Friend delinquency	.2*	.19**
Centrality × Friend delinquency	.084	.064
Density × Friend delinquency	–.43*	–.39**
Population × Friend delinquency	–.01	–.01
Genotype		
178/304	–.65	–.43
178/178 or 304/304	—	—
GE interaction		
Friend delinquency × *178/304*	.13**	.10**
N of measures	2,050	2,050
N of persons	755	755

Note: The interaction terms are added to the same basic models as in Tables 3 to 5.
* $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$ (two-tailed tests).

one additional minor delinquency item per identified friend raises the level of serious delinquency by .13 and the level of violent delinquency by .10.

DISCUSSION AND CONCLUSIONS

The key innovation of this study is the incorporation of molecular genetic variants into a social-control life-course model of delinquency. We use three genetic polymorphisms in the *DAT1, DRD2,* and *MAOA* genes to measure genetic propensities for delinquency and criminality. All three genetic variants are significantly related to self-reported serious and violent delinquency in a model of social control—a social-control model that includes social-structural conditions and a number of indicators for family and school processes.

Importantly, our findings also highlight an interaction effect between the *MAOA* gene and *repeating a grade* and an interaction effect between the *DRD2* gene and *having daily family meals.* To test whether these interactions represent merely isolated effects or patterns of interactions between genetic variants and family and school processes, we examine the interactions with a host of other family, school, and friendship network characteristics.

The gene–environment analysis shows extensive interaction effects jointly produced by a genetic variant and a family, school, or friendship network characteristic. *Family meals, social services,* and *two biological parents* separately and jointly interact with *DRD2**178/304. *Repeating a grade* and *school attachment* separately interact with *MAOA**2R, and *friend delinquency* interacts with *DRD2**178/304. In every instance of these interactions, a stronger social-control influence of family, school, or social networks reduces the delinquency-increasing effect of a genetic variant, whereas a weaker social-control influence of family, school, and social networks amplifies the delinquency-increasing effect of a genetic variant. For example, in the model of serious delinquency for *two biological parents* in Table 6, for those with the *DRD2**178/304 genotype, *two biological parents* reduces the genotypic effect of .834 by .736.

Our study makes important contributions to the social-control model of delinquency. By incorporating fundamental individual differences at the molecular genetic level, we discovered effects of social control that would have been missed without the interaction analysis. For example, only in the gene–environment interaction model in Table 6 does *two biological parents* significantly predict delinquency. When *two biological parents* is included as a main effect, it is not related to delinquency (Tables 3, 4, and 5).

Our findings confirm that genetic effects are not deterministic. The expression of the genes may depend heavily on environment. In both the *MAOA* and *DRD2* models, the genotype effect changes dramatically when an interaction with environment is allowed. Conversely, the social-control effect also changes radically once an interaction with a genetic variant is introduced. The latter point can be illustrated by the *MAOA**2R grade retention interaction result for serious delinquency in Table 7. For those who do not have a 2R allele, *repeating a grade* raises serious delinquency by .451, but for those possessing a 2R allele, *repeating a grade* raises serious delinquency by a drastic 6.61.

The mechanisms that lie beneath statistical gene–environment interactions are, by and large, poorly understood. The interaction between *MAOA**2R and grade retention may be investigated from two ends: grade retention and *MAOA.* What is it about grade retention that makes an individual more susceptible to the effect of *MAOA**2R? Our theoretical frame points to social control. Retention may weaken social control. Compared with younger children, adolescents are more difficult to supervise and monitor. They typically have made extensive investments in social bonds in school (Stattin and Kerr 2000). Retained students' social bonds are likely disrupted by teachers' negative perceptions and low expectations, by peer ridicule and labeling, and by their own feelings of frustration, humiliation, shame, failure, and confusion (Pagani et al. 2001). Consequently, the weakened bonds to school may increase exposure to deviant peers. Nagin and colleagues (2003) report an effect of grade retention on physical aggression among school youths.

Little is known about how grade retention alters *MAOA* gene expression. Earlier, we reviewed the epigenetic mechanisms behind a gene–environment interaction effect on rats' responses to stress—an interaction between maternal care and the glucorticoid receptor gene. Studies of epigenetic mechanisms in *MAOA* in humans do not seem to have been performed.

Having meals daily with one or two parents is a powerful moderator for the effect of *DRD2.* Family meals define routine and consistency in a family (Wolin and Bennett 1984) and offer an opportunity for parents to communicate with their children (Gillman et al. 2000; Neumark-Sztainer et al. 2003; Neumark-Sztainer et al. 2000; Videon and Manning 2003). Family meals may be a formal or informal "check-in" time for the physical and emotional well-being of the teens. Several studies report a positive effect of family mealtimes (Neumark-Sztainer et al. 2004). Youth whose families eat meals together spend more time on homework and reading for pleasure (Tepper 1999). Regular family meals may serve as a proxy for parental involvement in a child's life and family connectedness in general (Neumark-Sztainer et al. 2004). Family connection is consistently associated with reduced risks for emotional distress, substance use, violence involvement, unhealthy weight control, and sexual behaviors (Borowsky, Ireland, and Resnick 2001; Eisenberg et al. 2004; Kingon and O'Sullivan 2001; Resnick et al. 1997).

Not only is epigenetics of crucial importance for understanding gene–environment interactions, but it may also prove to be important for interventions. Epigenetic changes are potentially reversible. Meaney and colleagues (Cervoni et al. 2002; Cervoni and Szyf 2001) reversed the epigenetic alterations on the glucorticoid receptor exon 17 promoter in grown-up offspring of low LG-ABN rats by treating them with TSA, a histone deacetylases inhibitor. Changes in responses to stress were also observed among those that experienced epigenetic reversal. Once treated by TSA, the stress response of low LG-ABN offspring came to resemble that of high LG-ABN offspring.

Our study is one of the first to measure propensities for delinquency and criminality nearly exogenously. Our propensity measures depend on the random recombination[6] of the two parents' genetic makeup and thus are fixed at conception. They are therefore different from variables such as one's own education and exogenous in the sense that no factors during an individual's lifetime could have an impact on the measures. However, these measures are subject

[6] Recombination is the formation of new allele (an alternative form of a gene located at a particular chromosome site) combinations in a gamete, which is a haploid reproductive cell. Two gametes, most often an egg and a sperm, join in fertilization to form a zygote.

to influences of parental behaviors and parental preferences before conception, including assortative mating. Thus, although genetic measures are unmistakably biological at one level, they inevitably have social and behavioral influences embedded in them. It must be recognized that some indicators of family, school, and social networks may not be purely environmental. These influences could be partially genetic. In such cases, the estimated gene–environment interactions become partially gene–gene interactions, but the genetic influences in these "environmental" factors are unidentified.

The indicators of family, school, and social networks may be endogenous or partially endogenous. For example, the association between friend delinquency and an individual's delinquency may result partially from selection rather than causality. When both selection and causation are present, the thorny task is to separate the two. This concern leads to two observations. The first observation is not new: we should caution against interpreting the associations with the indicators of social processes as causal. The second observation illustrates again the importance of "natural experiments," which aim at uncovering "causal" effects through research design. Molecular genetic measures may help to uncover a relationship with social processes, but they do not guarantee that the relationship is causal.

The present study explores only a single polymorphism in each of a very few number of genes. Other functional variants within each of the three genes could also be related to human aggression and violence. A human genome comprises thousands of genes and millions of genetic variations. Other functional variants in other genes should be examined as well. Finally, the findings must be replicated in large population-based studies before any firm conclusions are drawn.

Studies involving genetic propensities for delinquency have ethical, legal, and social ramifications (Rothstein 2005). For example, evidence of genetic propensities for criminal behavior may be used to challenge a basic assumption of the U.S. legal system: individuals have free will and consequently are held legally responsible for their behavior. If an individual's free will is weakened by innate propensities for criminality, should the person not be held fully responsible? In such a case, punishments like the death penalty might seem cruel and unusual. The same genetic evidence may also work against a defendant. In some states, sexual predator laws indefinitely jail offenders who have been convicted of multiple sex crimes against children, for fear the perpetrators may harm children again if released. Conceivably, genetic evidence could be used to suggest that individuals predisposed to commit sex crimes should not be released. Responsible use of genetic evidence in these and other ethical, legal, and social issues remains an unresolved challenge.

The public policy implications of these findings are just beginning to be discussed. If the higher-risk genotype *DRD2**178/304 could be neutralized by parental involvement, public policy may want to target individuals with 178/304 and specifically encourage parental involvement. Should that not be feasible, surrogate programs that involve social institutions beyond the nuclear family might be implemented. The case is similar to that for PKU, in which only individuals with mutations in the phenylalanine hydroxylase gene who are exposed to phenylalanine in the diet are susceptible to PKU. The two cases do have important differences. Genetic variants associated with delinquency are likely to carry more social stigma and consequences, such as incarceration and genetic profiling, than would the PKU gene. Public policy programs based on these delinquency-related genes are likely to be much more delicate. Their feasibility and implications must be considered carefully.

Although the particular focus of this study is on delinquency and criminality, the use of molecular genetic variants to measure genetic propensities for human behaviors and other characteristics may have much broader implications. Explicitly or implicitly, contemporary social sciences generally assume that all individuals are the same at birth and attribute individual differences solely to environmental forces. Downplaying individual differences in certain historical periods may not be inappropriate. In times and places of war, famine, sharp social inequality, and epidemic diseases, innate individual differences may be far less significant than in modern, peaceful, industrialized democracies.

Emerging molecular genetic evidence suggests that individuals may differ in innate

propensities for a wide variety of traits and behaviors. As genetic evidence increasingly points to intrinsic individual differences, the social sciences may need to incorporate this development. In studies investigating causes of delinquency, the incorporation of genetic evidence may help to estimate social-control effects (which may be correlated with genetic effects) more precisely, improve model prediction, and reveal interactions with social-control processes. The development of contemporary molecular genetics has created challenges and opportunities for the social sciences. Meeting the challenges and opportunities will advance our understanding of how individual traits and behaviors are affected by social processes.

Guang Guo *is professor of sociology and faculty fellow at Carolina Population Center and Carolina Center for Genomic Sciences at the University of North Carolina-Chapel Hill. His research focuses on the integration of genetics and sociology in the studies of social and health behavior in humans, and statistical and research methods. In addition to publications in sociological literature, he has published in genetics journals including* Behavior Genetics, Human Genetics, European Journal of Human Genetics, *and* American Journal of Medical Genetics.

Michael E. Roettger *is currently a PhD candidate at the University of North Carolina-Chapel Hill. His dissertation examines issues of social inequality and the U.S. criminal justice system.*

Tianji Cai *is currently a PhD candidate at the University of North Carolina-Chapel Hill. He is interested in sociological research methods and the interplay between biology and society.*

REFERENCES

Akers, Ronald L. 1985. *Deviant Behavior: A Social Learning Approach*. 3rd ed. Belmont, CA: Wadsworth.

———. 1991. "Self-Control as a General Theory of Crime." *Journal of Quantitative Criminology* 7:201–11.

Allison, D. B., M. Heo, N. Kaplan, and E. R. Martin. 1999. "Sibling-Based Tests of Linkage and Association for Quantitative Traits." *American Journal of Human Genetics* 64:1754–64.

Bannon, Michael J. and Christopher J. Whitty. 1995. "Neurokinin Receptor Gene-Expression in Substantia-Nigra: Localization, Regulation, and Potential Physiological Significance." *Canadian Journal of Physiology and Pharmacology* 73:866–70.

Barr, Christina S., Timothy K. Newman, Stephen Lindell, Courtney Shannon, Maribeth Champoux, Klaus Peter Lesch, Stephen J. Suomi, David Goldman, and J. Dee Higley. 2004a. "Interaction between Serotonin Transporter Gene Variation and Rearing Condition in Alcohol Preference and Consumption in Female Primates." *Archives of General Psychiatry* 61:1146–52.

Barr, Christina S., Timothy K. Newman, Courtney Shannon, Clarissa Parker, Rachel L. Dvoskin, Michelle L. Becker, Melanie Schwandt, Maribeth Champoux, Klaus Peter Lesch, David Goldman, Stephen J. Suomi, and J. Dee Higley. 2004b. "Rearing Condition and rh5-httlpr Interact to Influence Limbic-Hypothalamic-Pituitary-Adrenal Axis Response to Stress in Infant Macaques." *Biological Psychiatry* 55:733–38.

Bauman, Karl E. and Susan T. Ennett. 1996. "On the Importance of Peer Influence for Adolescent Drug Use: Commonly Neglected Considerations." *Addiction* 91:185–98.

Baylin, Stephen B., Manel Esteller, Michael R. Rountree, Kurtis E. Bachman, Kornel Schuebel, and James G. Herman. 2001. "Aberrant Patterns of DNA Methylation, Chromatin Formation and Gene Expression in Cancer." *Human Molecular Genetics* 10:687–92.

Beccaria, Casera. [1764] 2004. "Of Crimes and Punishments." Kila, MT: Kessinger Publishing.

Bennett, A. J., K. P. Lesch, A. Heils, J. C. Long, J. G. Lorenz, S. E. Shoaf, M. Champoux, S. J. Suomi, M. V. Linnoila, and J. D. Higley. 2002. "Early Experience and Serotonin Transporter Gene Variation Interact to Influence Primate CNS Function." *Molecular Psychiatry* 7:118–22.

Bentham, Jeremy. [1789] 1970. *An Introduction to the Principles of Morals and Legislation.* London, UK: The Athlone Press.

Boesch, Christophe and Hedwige Boesch-Achermann. 2000. *The Chimpanzees of the Taï Forest: Behavioral Ecology and Evolution.* Oxford, UK: Oxford University Press.

Bonacich, Philip. 1987. "Power and Centrality: A Family of Measures." *American Journal of Sociology* 92:1170–82.

Bonczar, Thomas P. 2003. "Prevalence of Imprisonment in the U.S. Population, 1974–2001." Washington, DC: Bureau of Justice Statistics.

Borowsky, Iris W., Majorie Ireland, and Michael D. Resnick. 2001. "Adolescent Suicide Attempts: Risks and Protectors." *Pediatrics* 107:485–93.

Botstein, David and Neal Risch. 2003. "Discovering Genotypes Underlying Human Phenotypes: Past Successes for Mendelian Disease, Future Approaches for Complex Disease." *Nature Genetics* 33:228–37.

Brunner, H. G., M. Nelen, X. O. Breakefield, H. H. Ropers, and B. A. Vanoost. 1993. "Abnormal-Behavior Associated with a Point Mutation in the

Structural Gene for Monoamine Oxidase-A." *Science* 262:578–80.

Bunzow, James R., Herbert H. M. Vantol, David K. Grandy, Paul Albert, John Salon, MacDonald Christie, Curtis A. Machida, Kim A. Neve, and Oliver Civelli. 1988. "Cloning and Expression of a Rat D2 Dopamine Receptor cDNA." *Nature* 336:783–87.

Cases, O., I. Seif, J. Grimsby, P. Gaspar, K. Chen, S. Pournin, U. Muller, M. Aguet, C. Babinet, J. C. Shih, and E. Demaeyer. 1995. "Aggressive-Behavior and Altered Amounts of Brain-Serotonin and Norepinephrine in Mice Lacking *MAOA*." *Science* 268:1763–66.

Caspi, Avshalom, Joseph McClay, Terrie E. Moffitt, Jonathon Mill, Judy Martin, Ian W. Craig, Alan Taylor, and Richie Poulton. 2002. "Role of Genotype in the Cycle of Violence in Maltreated Children." *Science* 297:851–54.

Caspi, Avshalom, Terrie E. Moffit, Phil A. Silva, Magda Stouthamer-Loeber, Robert F. Krueger, and Pamela S. Schmutte. 1994. "Are Some People Crime-Prone?: Replications of the Personality-Crime Relationship across Countries, Genders, Races, and Methods." *Criminology* 32:163–95.

Cervoni, Nadia, Nancy Detich, Sang-Beom Seo, Debabrata Chakravarti, and Moshe Szyf. 2002. "The Oncoprotein set/taf-1 beta, an Inhibitor of Histone Acetyltransferase, Inhibits Active Demethylation of DNA, Integrating DNA Methylation and Transcriptional Silencing." *Journal of Biological Chemistry* 277:25026–31.

Cervoni, Nadia and Moshe Szyf. 2001. "Demethylase Activity Is Directed by Histone Acetylation." *Journal of Biological Chemistry* 276:40778–87.

Chantala, Kim, William D. Kalsbeek, and Eugenio Andraca. 2004. "Non-response in Wave III of the Add Health Study." In *Report on Bias in Wave III sampling in National Longitudinal Survey of Adolescent Health.* Retrieved May 28, 2007 (http://www.cpc.unc.edu/projects/addhealth/pubs/guides).

Christiansen, Karl O. 1977. "A Preliminary Study of Criminality among Twins." Pp. 89–108 in *Biosocial Bases of Criminal Behavior,* edited by S. A. Mednick and K. O. Christiansen. New York: Gardner.

Comings, David E. and James P. MacMurray. 2000. "Molecular Heterosis: A Review." *Molecular Genetics and Metabolism* 71:19–31.

Cook, Edwin H., Mark A. Stein, Matthew D. Krasowski, Nancy J. Cox, Deborah M. Olkon, John E. Kieffer, and Bennett L. Leventhal. 1995. "Association of Attention Deficit Disorder and the Dopamine Transporter Gene." *American Journal of Human Genetics* 56:993–98.

Cornish, K. M., T. Manly, R. Savage, J. Swanson, D. Morisano, N. Butler, C. Grant, G. Cross, L. Bentley, and C. P. Hollis. 2005. "Association of the Dopamine Transporter (DAT1) 10/10-Repeat Genotype with ADHD Symptoms and Response Inhibition in a General Population Sample." *Molecular Psychiatry* 10:686–98.

Corsaro, William A. and Donna Eder. 1990. "Children's Peer Cultures." *Annual Review of Sociology* 16:197–220.

Cusick, Philip A. 1973. *Inside High School.* New York: Holt, Rinehardt, & Winston.

Daly, G., Z. Hawi, M. Fitzgerald, and M. Gill. 1999. "Mapping Susceptibility Loci in Attention Deficit Hyperactivity Disorder: Preferential Transmission of Parental Alleles at *DAT1, DBH* and *DRD5* to Affected Children." *Molecular Psychiatry* 4:192–96.

de Almeida, Rosa M. M., Pier F. Ferrari, Stefan Parmigiani, and Klausen A. Miczek. 2005. "Escalated Aggressive Behavior: Dopamine, Serotonin and Gaba." *European Journal of Pharmacology* 526:51–64.

de Waal, Frans. 2005. *Our Inner Ape.* New York: Riverhead Books.

Doucettestamm, Lynn A., Derron J. Blakely, Jingxiang Tian, Sue Mockus, and Jen-i Mao. 1995. "Population Genetic-Study of the Human Dopamine Transporter Gene (DAT1)." *Genetic Epidemiology* 12:303–08.

Durkheim, Emile. 1897. *Le Suicide.* Paris: Alcan.

Eisenberg, Marla E., Rachel E. Olson, Dianne Neumark-Sztainer, Mary Story, and Linda H. Bearinger. 2004. "Correlations between Family Meals and Psychosocial Well-Being among Adolescents." *Archives of Pediatrics & Adolescent Medicine* 158:792–96.

Francis, Darlene, Josie Diorio, Dong Liu, and Michael J. Meaney. 1999. "Nongenomic Transmission across Generations of Maternal Behavior and Stress Responses in the Rat." *Science* 286:1155–58.

Gill, M., G. Daly, S. Heron, Z. Hawi, and M. Fitzgerald. 1997. "Confirmation of Association between Attention Deficit Hyperactivity Disorder and a Dopamine Transporter Polymorphism." *Molecular Psychiatry* 2:311–13.

Gillman, Matthew W., Sheryl L. Rifas-Shiman, A. Lindsay Frazier, Helaine R. H. Rockett, Carlos A. Camargo, Alison E. Field, Catherine S. Berkey, and Graham A. Colditz. 2000. "Family Dinner and Diet Quality among Older Children and Adolescents." *Archives of Family Medicine* 9:235–40.

Giros, Bruno, Mohamed Jaber, Sara R. Jones, R. Mark Wightman, and Marc G. Caron. 1996. "Hyperlocomotion and Indifference to Cocaine and Amphetamine in Mice Lacking the Dopamine Transporter." *Nature* 379:606–12.

Gottesman, Irving I., Gregory Carey, and Daniel R. Hanson. 1983. "Pearls and Perils in Epigentic Psychopathology." Pp. 287–300 in *Childhood Psychopathology and Development,* edited by

S. B. Guze, E. J. Earls, and J. E. Barrett. New York: Raven Press.

Gottfredson, Michael R. and Travis Hirschi. 1990. *A General Theory of Crime.* Stanford, CA: Stanford University Press.

Guo, Guang, Xiao-ming Ou, Michael E. Roettger, and Jean C. Shih. 2008. "The VNTR 2-Repeat in *MAOA* and Delinquent Behavior in Adolescence and Young Adulthood: Associations and *MAOA* Promoter Activity." *European Journal of Human Genetics.*

Guo, Guang, Michael E. Roettger, and Jean C. Shih. 2007. "Contributions of the DAT1 and DRD2 Genes to Serious and Violent Delinquency among Adolescents and Young Adults." *Human Genetics* 121:125–36.

Guo, Guang and Elizabeth Stearns. 2002. "The Social Influences on the Realization of Genetic Potential for Intellectual Development." *Social Forces* 80:881–910.

Hagan, John and Holly Foster. 2003. "S/he's a Rebel: Toward a Sequential Stress Theory of Delinquency and Gendered Pathways to Disadvantage in Emerging Adulthood." *Social Forces* 82:53–86.

Hannon, Lance. 2003. "Poverty, Delinquency, and Educational Attainment: Cumulative Disadvantage or Disadvantage Saturation?" *Sociological Inquiry* 73:575–94.

Harris, Kathleen M., Greg J. Duncan, and Johanne Boisjoly. 2002. "Evaluating the Role of 'Nothing to Lose' Attitudes on Risky Behavior in Adolescence." *Social Forces* 80:1005–39.

Harris, Kathleen M., Francesca Florey, Joyce Tabor, Peter S. Bearman, Jo Jones, and J. Richard Udry. 2003. "The National Longitudinal Study of Adolescent Health: Research Design." Vol. 2005. Retrieved May 28, 2007 (http://www.Cpc.Unc.Edu/projects/addhealth/design).

Haynie, Dana L. 2001. "Delinquent Peers Revisited: Does Network Structure Matter?" *American Journal of Sociology* 106:1013–57.

———. 2003. "Contexts of Risk? Explaining the Link between Girls' Pubertal Development and their Delinquency Involvement." *Social Forces* 82:355–97.

Heinz, Andreas, David Goldman, Douglas W. Jones, Roberta Palmour, Dan Hommer, Julia G. Gorey, Kan S. Lee, Markku Linnoila, and Daniel R. Weinberger. 2000. "Genotype Influences in Vivo Dopamine Transporter Availability in Human Striatum." *Neuropsychopharmacology* 22:133–39.

Hindelang, Michael J. 1981. "Variations in Sex-Race-Age-Specific Incidence Rates of Offending." *American Sociological Review* 46:461–74.

———. 2001. *Measuring Delinquency.* Thousand Oaks, CA: Sage Publications.

Hindelang, Michael J., Travis Hirschi, and Joseph G. Weis. 1979. "Correlates of Delinquency: Illusion of Discrepancy between Self-Report and Official Measures." *American Sociological Review* 44:995–1014.

Hirschi, Travis. 1969. *Causes of Delinquency.* Los Angeles, CA: University of California Press.

Hood, Roger and Richard Sparks. 1970. *Key Issues in Criminology.* Wallop, NH: BAS.

Hunter, David J. 2005. "Gene-Environment Interactions in Human Diseases." *Nature Reviews Genetics* 6:287–98.

Jacobsen, Leslie K., Julie K. Staley, Sami Zoghbi, John P. Seibyl, Thomas R. Kosten, Robert B. Innis, and Joel Gelernter. 2000. "Prediction of Dopamine Transporter Binding Availability by Genotype: A Preliminary Report." *American Journal of Psychiatry* 157:1700–03.

Khoury, Mulin J., M. J. Adams, and W. Dana Flanders. 1988. "An Epidemiologic Approach to Ecogenetics." *American Journal of Human Genetics* 42:89–95.

Kingon, Yvonne S. and Ann L. O'Sullivan. 2001. "The Family as a Protective Asset in Adolescent Development." *Journal of Holistic Nursing* 19:102–21.

Kornhauser, Ruth. 1978. *Social Sources of Delinquency.* Chicago, IL: University of Chicago Press.

Laruelle, M., J. Gelernter, and R. B. Innis. 1998. "D-2 Receptors Binding Potential Is Not Affected by Taq1 Polymorphism at the D-2 Receptor Gene." *Molecular Psychiatry* 3:261–65.

Lubin, Bernard, Reed M. Larsen, and Joseph D. Matarazzo. 1984. "Patterns of Psychological Test Usage in the United States: 1935–1982." *American Psychologist* 39:451–54.

Malone, Stephen M., Jeanette Taylor, Naomi R. Marmorstein, Matt McGue, and William G. Iacono. 2004. "Genetic and Environmental Influences on Antisocial Behavior and Alcohol Dependence from Adolescence to Early Adulthood." *Development and Psychopathology* 16:943–66.

Marchini, Jonathon, Lon R. Cardon, Michael S. Phillips, and Peter Donnelly. 2004. "The Effects of Human Population Structure on Large Genetic Association Studies." *Nature Genetics* 36:512–17.

Matsueda, Ross L. 1982. "Testing Control-Theory and Differential Association: A Causal-Modeling Approach." *American Sociological Review* 47:489–504.

Matsueda, Ross L. and Kathleen Anderson. 1998. "The Dynamics of Delinquent Peers and Delinquent Behavior." *Criminology* 36:269–308.

Matsueda, Ross L. and Karen Heimer. 1987. "Race, Family-Structure, and Delinquency: A Test of Differential Association and Social-Control Theories." *American Sociological Review* 52:826–40.

Meaney, Michael J., Moshe Szyf, and Jonathon R. Seckl. 2007. "Epigenetic Mechanisms of Perinatal

Programming of Hypothalamic-Pituitary-Adrenal Function and Health." *Trends in Molecular Medicine* 13:269–77.

Miczek, Klaus A. and Eric W. Fish. 2005. "Dopamine, Glutamate, and Aggression." Pp. 237–63 in *Dopamine and Glutamate in Psychiatric Disorders,* edited by J. Schmidt and M. E. A. Reith. Totowa, NJ: Humana Press.

Miczek, Klaus A., Eric W. Fish, Joseph F. de Bold, and Rosa M. M. de Almeida. 2002. "Social and Neural Determinants of Aggressive Behavior: Pharmacotherapeutic Targets at Serotonin, Dopamine and Gamma-Aminobutyric Acid Systems." *Psychopharmacology* 163:434–58.

Moffitt, Terrie E. 1993. "Adolescence-Limited and Life-Course-Persistent Antisocial-Behavior: A Developmental Taxonomy." *Psychological Review* 100:674–701.

Murphy, Fred J., M. M. Shirly, and Helen L. Witmer. 1946. "The Incidence of Hidden Delinquency." *American Journal of Orthopsychiatry* 16:686–96.

Nagin, Daniel S. and Kenneth C. Land. 1993. "Age, Criminal Careers, and Population Heterogeneity: Specification and Estimation of a Nonparametric, Mixed Poisson Model." *Criminology* 31:327–62.

Nagin, Daniel S., Linda Pagani, Richard E. Tremblay, and Frank Vitaro. 2003. "Life Course Turning Points: The Effect of Grade Retention on Physical Aggression." *Development and Psychopathology* 15:343–61.

Neumark-Sztainer, Dianne, Peter J. Hannan, Mary Story, Jillian Croll, and Cheryl Perry. 2003. "Family Meal Patterns: Associations with Sociodemographic Characteristics and Improved Dietary Intake among Adolescents." *Journal of the American Dietetic Association* 103:317–22.

Neumark-Sztainer, Dianne, Mary Story, Diann Ackard, James Moe, and Cheryl Perry. 2000. "The 'Family Meal': Views of Adolescents." *Journal of Nutrition Education* 32:329–34.

Neumark-Sztainer, Dianne, Melanie Wall, Mary Story, and Jayne A. Fulkerson. 2004. "Are Family Meal Patterns Associated with Disordered Eating Behaviors among Adolescents?" *Journal of Adolescent Health* 35:350–59.

Pagani, Linda, Richard E. Tremblay, Frank Vitaro, Bernard Boulerice, and Pierre McDuff. 2001. "Effects of Grade Retention on Academic Performance and Behavioral Development." *Development and Psychopathology* 13:297–315.

Pohjalainen, T., J. O. Rinne, K. Nagren, P. Lehikoinen, K. Anttila, E. K. G. Syvalahti, and J. Hietala. 1998. "The A1 Allele of the Human D-2 Dopamine Receptor Gene Predicts Low D-2 Receptor Availability in Healthy Volunteers." *Molecular Psychiatry* 3:256–60.

Resnick, M. D., P. S. Bearman, R. W. Blum, K. E. Bauman, K. M. Harris, J. Jones, J. Tabor, T. Beuhring, R. E. Sieving, M. Shew, M. Ireland, L. H. Bearinger, and J. R. Udry. 1997. "Protecting Adolescents from Harm: Findings from the National Longitudinal Study on Adolescent Health." *Journal of the American Medical Association* 278:823–32.

Rice, J. A. and L. F. Brown. 1967. "Validity of Peabody Picture Vocabulary Test in a Sample of Low IQ Children." *American Journal of Mental Deficiency* 71:602–15.

Risch, Neil J. 2000. "Searching for Genetic Determinants in the New Millennium." *Nature* 405:847–56.

Robison, Sophia Moses. 1936. *Can Delinquency Be Measured?* New York: Columbia University Press.

Rodgers, Joseph L., Maury Buster, and David C. Rowe. 2001. "Genetic and Environmental Influences on Delinquency: DF Analysis of NLSY Kinship Data." *Journal of Quantitative Criminology* 17:145–68.

Rothstein, Mark A. 2005. "Science and Society: Applications of Behavioural Genetics: Outpacing the Science?" *Nature Reviews Genetics* 6:793–98.

Rowe, David C. and D. Wayne Osgood. 1984. "Heredity and Sociological Theories of Delinquency: A Reconsideration." *American Sociological Review* 49:526–40.

Sabol, Sue Z., Stella Hu, and Dean Hamer. 1998. "A Functional Polymorphism in the Monoamine Oxidase A Gene Promoter." *Human Genetics* 103:273–79.

Sabol, Sue Z., Mark L. Nelson, Craig Fisher, Lorraine Gunzerath, Cindy L. Brody, Stella Hu, Leo A. Sirota, Stephen E. Marcus, Benjamin D. Greenberg, Frank R. Lucas, Jonathan Benjamin, Dennis L. Murphy, and Dean H. Hamer. 1999. "A Genetic Association for Cigarette Smoking Behavior." *Health Psychology* 18:7–13.

Sampson, Robert J. and John H. Laub. 1993. *Crime in the Making: Pathways and Turning Points through Life.* Cambridge, MA: Harvard University Press.

Searle, Shayle R. 1971. *Linear Models.* New York: Wiley and Sons.

Searle, Shayle R., George Casella, and Charles E. McCulloch. 1992. *Variance Components.* New York: Wiley & Sons.

Shih, J. C. and R. F. Thompson. 1999. "Monoamine Oxidase in Neuropsychiatry and Behavior." *American Journal of Human Genetics* 65:593–98.

Stattin, Hakan and Margaret Kerr. 2000. "Parental Monitoring: A Reinterpretation." *Child Development* 71:1072–85.

Sutherland, Edwin H. 1947. *Principles of Criminology.* 4th ed. Philadelphia, PA: J. B. Lippincott.

Tepper, Robin L. 1999. "Parental Regulation and Adolescent Time-Use Decisions: Findings from the NLSY97." Pp. 77–103 in *Social Awakening: Adolescent Behavior as Adulthood Approaches,*

edited by R. Michael. New York: Russell Sage Foundation.

Thornberry, Terence P. and Marvin D. Krohn. 2000. "The Self-Report Method for Measuring Delinquency and Crime." Pp. 33–83 in *Criminal Justice 2000,* Vol. 4. Washington, DC: National Institute of Justice.

Tourangeau, Roger and Tom W. Smith. 1996. "Asking Sensitive Questions: The Impact of Data Collection Mode, Question Format, and Question Context." *Public Opinion Quarterly* 60:275–304.

Tsankova, Nadia, William Renthal, Arvind Kumar, and Eric J. Nestler. 2007. "Epigenetic Regulation in Psychiatric Disorders." *Nature Reviews Neuroscience* 8:355–67.

Turkheimer, Eric, Andreana Haley, Mary Waldron, Brian D'Onofrio, and Irving I. Gottesman. 2003. "Socioeconomic Status Modifies Heritability of IQ in Young Children." *Psychological Science* 14:623–28.

Vandenbergh, David J., Antonio M. Persico, Anita L. Hawkins, Constance A. Griffin, Xiang Li, Ethylin W. Jabs, and George R. Uhl. 1992. "Human Dopamine Transporter Gene (DAT1) Maps to Chromosome-5p15.3 and Displays a VNTR." *Genomics* 14:1104–06.

Videon, Tami M. and Carolyn K. Manning. 2003. "Influences on Adolescent Eating Patterns: The Importance of Family Meals." *Journal of Adolescent Health* 32:365–73.

Waldman, I. D., D. C. Rowe, A. Abramowitz, S. T. Kozel, J. H. Mohr, S. L. Sherman, H. H. Cleveland, M. L. Sanders, J. H. C. Card, and C. Stever. 1998. "Association and Linkage of the Dopamine Transporter Gene and Attention-Deficit Hyperactivity Disorder in Children: Heterogeneity Owing to Diagnostic Subtype and Severity." *American Journal of Human Genetics* 63:1767–76.

Weaver, I. C. G., N. Cervoni, F. A. Champagne, A. C. D'Alessio, S. Sharma, J. R. Seckl, S. Dymov, M. Szyf, and M. J. Meaney. 2004. "Epigenetic Programming by Maternal Behavior." *Nature Neuroscience* 7:847–54.

Weiss, Lauren A., Lin Pan, Mark Abney, and Carole Ober. 2006. "The Sex-Specific Genetic Architecture of Quantitative Traits in Humans." *Nature Genetics* 38:218–22.

Wilson, Edward O. 1975. *Sociobiology: The New Synthesis.* Cambridge, MA: Harvard University Press.

Wilson, Michael L. and Richard W. Wrangham. 2003. "Intergroup Relations in Chimpanzees." *Annual Review of Anthropology* 32:363–92.

Wolin, Steven J. and Linda A. Bennett. 1984. "Family Rituals." *Family Process* 23:401–20.

Wrangham, Richard and Dale Peterson. 1996. *Demonic Males: Apes and the Origins of Human Violence.* New York: Houghton Mifflin Company.

Wright, Debra L., William S. Aquilino, and Andrew J. Supple. 1998. "A Comparison of Computer-Assisted and Paper-and-Pencil Self-Administered Questionnaires in a Survey on Smoking, Alcohol, and Drug Use." *Public Opinion Quarterly* 62:331–53.

Zhu, Quin S., Kevin Chen, and Jean C. Shih. 1994. "Bidirectional Promoter of Human Monoamine-Oxidase-A *(MAO-A)* Controlled by Transcription Factor SP1." *Journal of Neuroscience* 14:7393–7403.

Zhu, Quin S., Joseph Grimsby, Kevin Chen, and Jean C. Shih. 1992. "Promoter Organization and Activity of Human Monoamine-Oxidase *(MAO)* A and B Genes." *Journal of Neuroscience* 12:4437–46.