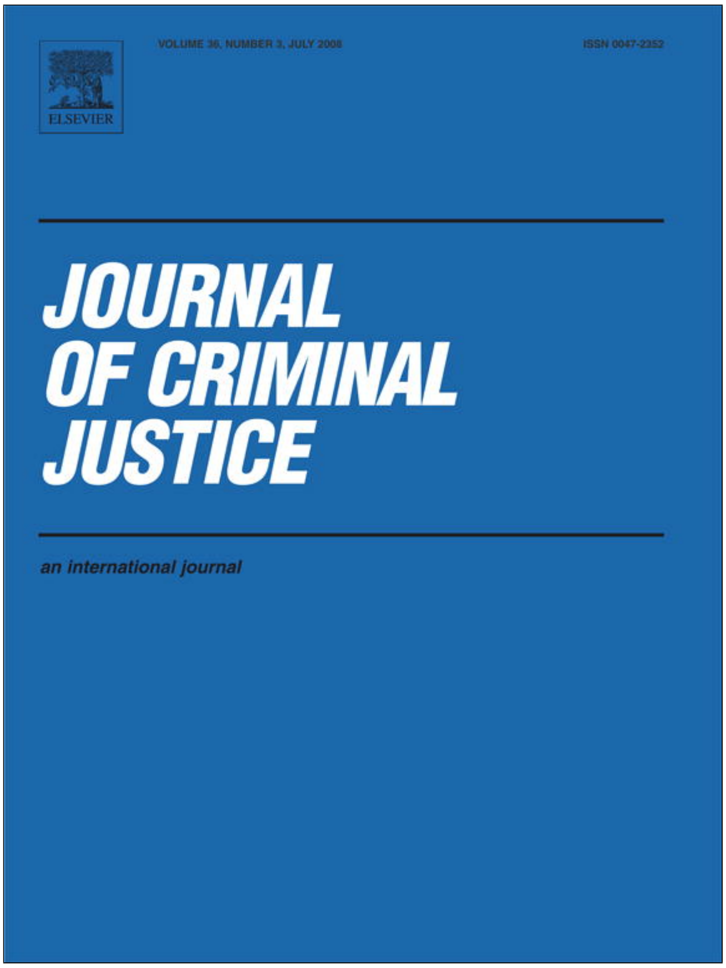
VOLUME 36, NUMBER 3, JULY 2008
ISSN 0047-2352
ELSEVIER
JOURNAL
OF CRIMINAL
JUSTICE
an international journal

Journal of Criminal Justice 36 (2008) 217–223

Contents lists available at ScienceDirect

Journal of Criminal Justice

The etiology of criminal onset: The enduring salience of nature *and* nurture

Matt DeLisi [a,*], Kevin M. Beaver [b], John Paul Wright [c], Michael G. Vaughn [d]

[a] *Department of Sociology, Iowa State University, 203A East Hall, Ames, IA 50011-1070*
[b] *College of Criminology and Criminal Justice, Florida State University, Tallahassee, FL 32306-1127*
[c] *Division of Criminal Justice, University of Cincinnati, Cincinnati, OH 45221-0389*
[d] *School of Social Work, St. Louis University, St. Louis, MO 63101*

ARTICLE INFO

ABSTRACT

Based on data from the National Longitudinal Study of Adolescent Health (Add Health), the current study was the first to use measures of genetic polymorphisms (DRD2 and DRD4) to empirically examine the onset of crime. Net of the effects of race, age, gender, and low self-control, genetic polymorphisms explained variation in police contacts and arrest, but only among youths in low risk family environments. Moreover, youths with genetic risk factors experienced a *later* onset than youths without these risk factors. Borrowing from the behavioral and molecular genetics literatures, various interpretations of the findings are discussed as well as a call for increasingly interdisciplinary perspectives in criminology that encompass both sociological and biosocial frameworks.

Introduction

The timing of the onset of antisocial behavior is one of the most critical pieces of information in understanding maladaptive behaviors, substance use, alcoholism, delinquency, and criminal justice system involvement. Antisocial behaviors that emerge during early and middle childhood are often harbingers of sustained antisocial behavior that persist through adolescence and endure into adulthood (Loeber & Dishion, 1983; Robins, 1978). For example, based on data from the National Epidemiologic Survey on Alcohol and Related Conditions, 47 percent of persons who began drinking alcohol before age fourteen will become dependent on alcohol at some point during their lifetime—a prevalence estimate that is five times greater than among persons whose onset of alcohol use occurs at the legal age of twenty-one (Hingson, Heeren, & Winter, 2006). For delinquent and criminal behavior, the effects of early onset are especially deleterious. Persons with an early onset of delinquency tend to have more chronic offending careers, tend to engage in more serious forms of crime, tend to be more violent, tend to be more criminally versatile, and tend to show sustained continuity in criminal behavior (Blumstein, Cohen, Roth, & Visher, 1986; DeLisi, 2005, 2006; Farrington et al., 1990; Piquero, Farrington, & Blumstein, 2003; Wolfgang, Figlio, & Sellin, 1972). The delineation of early onset as a powerful predictor of various antisocial outcomes appears in the American Psychiatric Association's *Diagnostic and Statistical Manual-IV*, numerous theories of delinquency in the social sciences, and summary reports or fact sheets produced by the National Institute of Mental Health, United States Surgeon General, World Health Organization, National Institutes of Health, and others (Cullen, Wright, & Blevins, 2006).

Conversely, antisocial behaviors that emerge during middle to late adolescence usually dissipate as abruptly as they emerge. Persons with a "late" onset tend to have short-lived or largely nonexistent criminal careers, to engage in benign even trivial forms of delinquency, to avoid involvement in the most serious forms of criminal violence, and to, almost effortlessly, desist from crime (Lahey, Moffitt, & Caspi, 2003). To illustrate, in their review of nineteen criminal career studies conducted between 1940 and 1999, Krohn, Thornberry, Rivera, and Le Blanc (2001, pp. 92–93) found that early onset offenders were *forty times* more likely than late onset offenders to become habitual criminals and committed between 40 and 700 percent more criminal acts.

Literature review

Onset and offending careers

Beyond its empirical relationship to antisocial behavior, onset is also implicated in theoretical accounts for the etiology of delinquency and related antisocial outcomes even though scholars readily admit that much is unknown about its causes (Farrington et al., 1990). For the normative, late onset pathway, the emergence of antisocial behavior is theorized to be caused by mostly sociological factors that speak to the uneven transition from child to adult status. The ambiguity in status acquisition for assorted behaviors, such as obtaining a driver's license, purchasing tobacco, using alcohol, engaging in romantic relationships, entering the work force, and deciding on an educational career, confers a great deal of uncertainty in the lives of teenagers. Moffitt (2003, pp. 50–51) calls this period the "maturity gap" whereby adolescents experience dissatisfaction with their dependent

* Corresponding author. Tel.: +1 515 294 8008; fax: +1 515 297 2303.
E-mail address: delisi@iastate.edu (M. DeLisi).

0047-2352/$ – see front matter © 2008 Elsevier Ltd. All rights reserved.
doi:10.1016/j.jcrimjus.2008.04.001

status as a child and impatience for what they anticipate are the privileges and rights of adulthood. By engaging in antisocial behavior and mimicking the behavior of antisocial peers, youths are simply asserting autonomy and attempting to hasten their social maturation (Moffitt, 1993). Again, these processes are not only normal, but the delinquents acts produced are normative. Empirically there is ample support for this late-starter or "adolescence-limited" prototype of delinquency (Moffitt, 1993, 2003; Patterson, 1995; Piquero & Brezina, 2001; Simons, Wu, Conger, & Lorenz, 1994).

Although sociological processes or factors are also invoked to explain early onset, its etiology is believed to be more dependent on biological, genetic, and biosocial variables. Indeed, a variety of criminological explanations view recurrent problem behavior as manifestations of some individual-level pathology that remains stable within an individual across social settings and circumstances. More pointedly, this theoretical perspective asserts that serious offenders have been problematic since early childhood and their multifaceted acts of wayward behavior are, quite simply, demonstrative of their psychopathology (DeLisi, 2005, pp. 79–80; Gottfredson & Hirschi, 1990).

The predominant theoretical account of early onset criminality is arguably Moffitt's (1993, 2003) life-course persistent offender prototype. According to Moffitt's (2003) perspective:

> The child's risk emerges from inherited or acquired deficits, difficult temperament, or hyperactivity. The environment's risk comprises factors such as inadequate parenting, disrupted family bonds, and poverty. The environmental risk domain expands beyond the family as the child ages, to include poor relations with people such as peers and teachers. Opportunities to learn prosocial skills are lost. Over the first two decades of development, transactions between the individual and the environment gradually construct a disordered personality with hallmark features of physical aggression and antisocial behavior persisting to midlife…the life-course persistent pattern of antisocial behavior appears to have substantial heritable liability. (pp. 50–53)

Empirically, the early onset life-course persistent offender has been differentially predicted by individual-level, biosocial factors, including neurological abnormalities, autonomic hypoactivity (e.g., low resting heart rate), low cognitive ability, low birth weight, maternal cigarette use, perinatal complications, and neuropsychological deficits (DeLisi, 2001; Gibson & Tibbetts, 2000; Moffitt & Caspi, 2001; Moffitt, Caspi, Rutter, & Silva, 2001; Moffitt, Lynam, & Silva, 1994; Piquero, 2001; Raine, Brennan, Mednick, & Mednick, 1996; Raine, Brennan, & Mednick, 1997; Tibbetts & Piquero, 1999). The essential conclusion to distill from this area of research is the combinatory importance of biological, sociological, and biosocial variables that *interact and mutually reinforce* to produce antisocial behavior (Caspi, Henry, McGee, Moffitt, & Silva, 1995).

Onset and genetics

Given the empirical salience of biosocial factors in predicting assorted outcomes of early onset antisocial behavior, behavioral genetics researchers had recently begun to investigate the role of biological and genetic factors in explaining the initiation of antisocial behaviors. The bulk of this extant research employed twin studies to indirectly assess genetic effects. Silberg et al. (1996) examined conduct disorder and hyperactivity among 265 MZ and 163 DZ male-male, 347 MZ and 160 DZ female-female, and 262 male-female twin pairs between the ages of eight and sixteen selected from the Virginia Twin Study of Adolescent Behavioral Development (VTSABD). Using structural equation modeling, Silberg et al. (1996) found that genetic and shared environmental factors were significantly related to both hyperactivity and conduct disorder. The covariation between hyperactivity and conduct problems was almost entirely attributable to genetic factors, however (also see, Eaves et al., 1997). Based on retrospective analyses of 2,682 adult twin pairs selected from the Australian Twin Register, Slutske et al. (1997) estimated that 71 percent of the variance in the etiology of conduct disorder was attributable to genetic factors. Drawing on data from the E-Risk Study, a nationally representative cohort of 1,116 five-year-old British twin pairs and their families, Arseneault et al. (2003) found that 82 percent of the etiology of childhood antisocial behavior was explained by genetic factors. Moreover, genetic vulnerabilities interact with criminogenic environments to produce antisocial behavior. To illustrate, children who were victims of maltreatment were significantly likely to present conduct problems. The effect of maltreatment on conduct problems was *twelve times* stronger among youths with genetic risk factors than those without genetic risks (Jaffee et al., 2005).

Taylor, Iacono, and McGue (2000) conducted the most direct examination of the genetic determinants of early onset using data from participants in the Minnesota Twin Family Study, an ongoing, community-based study of same-sexed, reared together twins (aged ten to twelve) and their parents. Taylor et al. (2000) examined potential behavioral differences among thirty-six early starters, eighty-six late starters, and twenty-five nondelinquent controls. They expected that the genetic component of early onset antisocial behavior centers on problems with inhibition, such that early onset offenders would present more impulsivity, lower constraint, more negative emotionality, greater levels of ADHD, CD, and ODD, and lower skin conductivity than those who are not early starters. Taylor et al. (2000) hypothesized that the early onset trajectory would be higher among MZ than DZ twins and that there would not be differences among MZ and DZ twins for late onset delinquency. Higher rates of antisocial behavior were expected among MZ co-twins of early starters. Compared to nondelinquent controls and late starters, early starters had lower verbal and spatial memory functioning; more psychological, emotional, and behavioral problems related to inhibition; higher negative emotionality; earlier and more persistent association with antisocial peers; higher familial transmission of antisocial behavior; and greater genetic influence on antisocial phenotypes (Taylor et al., 2000, p. 641).

Also based on data from the Minnesota Twin Family Study, Blonigen, Hicks, Krueger, Patrick, and Iacono (2005) found that adolescent psychopathic traits were also differentially predicted by genetic factors. Specifically, fearless dominance, which is marked by social potency, stress immunity, and fearlessness, was associated with reduced genetic risk for internalizing psychopathology. Impulsive antisociality, which is marked by negative emotionality (aggression and alienation) and low behavioral constraint (impulsivity and sensation seeking), was associated with increased genetic risk for externalizing psychopathology. For both males and females, the heritability estimates for fearless dominance (h^2 = .45), impulsive antisociality (h^2 = .49), internalizing psychopathology (h^2 = .36), and externalizing psychopathology (h^2 = .73) were moderate to high.

Overall, these studies provided convincing evidence for the place of biological and genetic factors and their interaction with environmental characteristics in explaining the onset of antisocial behavior. Using data from the National Longitudinal Study of Adolescent Health (Add Health), the current study sought to take the next step: empirically examine the etiology of early onset using genetic measures. With measures of genetic polymorphisms, the current study could directly assess the role of biological factors in predicting criminal onset that are more precise than indirect measures used by twin studies. Moreover, the current study could explore the environmental conditions that serve to amplify or suppress the expression of genetic risk factors.

Methods

Sample

Data for this study came from the National Longitudinal Study of Adolescent Health (Add Health). The Add Health is a large, prospective sample of American adolescents in seventh through twelfth grade (Harris et al., 2003; Udry, 1998, 2003). Multistage stratified sampling techniques were employed to obtain a nationally representative sample of 132 middle and high schools (Chantala, 2003; Harris et al., 2003; Udry, 1998, 2003). In 1994, students attending these schools were administered a self-report survey asking them questions about their interests, their behaviors, their friends, and their family. In total, over 90,000 adolescents completed the Wave I in-school questionnaire. The first wave of data also included a subsample of respondents who, along with their primary caregiver (typically the mother), were re-interviewed in their homes. The Wave I in-home survey covered topics related to delinquent involvement, romantic relationships, and victimization experiences, among others. Altogether, 20,745 respondents and 17,700 of their primary caregivers participated in the Wave I in-home component of the Add Health study (Harris et al., 2003).

Approximately one to two years after the initial wave of data was collected, 14,738 of the original respondents participated in the second round of interviews (Harris et al., 2003). The Wave II questionnaires contained many of the same questions that were asked at Wave I including items tapping drug and alcohol use, criminal activities, sexual history, and relationship patterns. The third and final wave of data was collected in 2001–2002 when the respondents were between the ages of eighteen and twenty-six years old (N = 15,197). Given that respondents were, on average, seven years older at Wave III than they were at Wave I, the survey instrument was altered to include more age-appropriate topics. At Wave III, Add Health participants were asked an extensive series of questions about their past contact with the criminal justice system, about their use of drugs and alcohol, about their sexual history, and about other topics germane to young adults (Harris et al., 2003).

One of the unique features of this data set was that the Add Health study was one of the largest, longitudinal studies to include DNA markers. At Wave III, a subset of 3,787 respondents was asked to submit buccal cells for genetic typing and analysis (Add Health Biomarker Team, n.d.). Only those respondents that had a sibling or co-twin who was also participating in the Add Health study were eligible to be part of the DNA subsample (Add Health Biomarker Team, n.d.). Overall, 2,574 Add Health respondents agreed to participate and were genotyped for the following genetic polymorphisms: a dopamine transporter gene (DAT1), two dopamine receptor genes (DRD2 and DRD4), a serotonin transporter gene (5HTT), and a gene that codes for the production of the enzyme, monoamine oxidase A (MAOA).

In line with prior research using the Add Health data (Haberstick et al., 2005), the current authors removed one MZ twin from each MZ dyad. In addition, only respondents who indicated that they had had contact with the police were retained. With this selection criteria in place, and after missing cases were deleted, the final analytical sample consisted of n = 324 subjects.

Measures

Dependent variables

Criminal onset was measured in two ways: age of first contact with police and age of first criminal arrest. At Wave III, Add Health respondents were asked whether they had ever been stopped and questioned by the police for any reason other than a traffic violation. Respondents who replied affirmatively were then asked to indicate the age that their first encounter with the police had occurred. Responses to this item were used to construct the age of first contact with police variable. Follow-up questions were also asked to determine whether the encounter with the police resulted in an arrest. If the respondent indicated that they had been arrested (n = 181), they were then prompted to specify how old they were at the time they were first arrested. Responses to this item thus reflected the age at which the respondent was first arrested.

Genetic polymorphisms

Two dopamine receptor polymorphisms—DRD2 and DRD4—were included to examine the genetic effects on age of criminal onset. The dopamine D2 receptor gene (DRD2) codes for the production of the D2 receptor protein and is found throughout the body, but especially in the striatum, the pituitary gland, the amygdala, the caudatus, the putamen, and other regions of the brain (Marino et al., 2004). Different variants of the DRD2 gene have been linked to certain personality styles (Munafò et al., 2003), to criminal victimization (Beaver, Wright, DeLisi, Daigle, et al., 2007), to age of first sexual intercourse (Miller et al., 1999), and to visuospatial performance (Berman & Noble, 1995).

The dopamine D4 receptor gene (DRD4) codes for the manufacturing of the D4 dopamine receptor protein. The D4 dopamine receptor protein is found in areas of the brain that are responsible for the expression of emotions and for the stimulation of cognitive faculties (Schmidt, Fox, Perez-Edgar, Hu, & Hamer, 2001). Moreover, the DRD4 gene, like other genes in the dopaminergic system, regulates attention processes, promotes motivation, and has been linked to exploratory behaviors (Schmidt et al., 2001). Molecular genetic research reveals that different DRD4 polymorphisms may actually demonstrate unique pharmacological properties that may affect a wide range of behaviors and personality traits (Van Tol et al., 1992).

Given that DRD2 and DRD4 have been found to be related to a number of phenotypes, there is reason to believe they may also contribute to an early contact with the criminal justice system. To explore this possibility, the DRD2 and DRD4 variables were constructed to indicate the number of risk alleles that each person possessed. For DRD2, the A1 allele was coded as the risk allele and assigned a value of "1" and the A2 allele was assigned a value of "0." For DRD4, alleles that had repeat sequences less than seven were assigned a value of "0"; alleles that had repeat sequences greater than or equal to seven were assigned a value of "1" (Hopfer et al., 2005). Scores for each allele (i.e., the maternal allele and the paternal allele) were then added together to form the DRD2 risk allele scale and the DRD4 risk allele scale.

Family risk

Prior research revealed that dimensions of the family environment interact with certain genetic polymorphisms to predict variation in antisocial behaviors (Caspi et al., 2002; Foley et al., 2004; Rutter, 2006). To take this possibility into account, three different family risk scales were developed. First, and following prior research using the Add Health data, a two-item maternal attachment scale was created (Haynie, 2001; Schreck, Fisher, & Miller, 2004). During Wave I interviews, respondents were asked how close they felt to their mother and how much they thought their mother cared about them. Responses to these questions were then added together to form the maternal attachment scale (α = .64).

Second, a maternal involvement scale was developed to index how frequently the mother and her child engaged in a variety of activities. At Wave I, each respondent was presented with a series of different activities including shopping, talking about a personal problem, and going to a movie, play, or sporting event. They were then asked to specify those that they had completed with their mother in the past month. Each item was coded dichotomously (0 = no, 1 = yes). Similar to the scale used by Crosnoe and Elder (2004), the maternal involvement scale was created by summing together responses to ten different activities to index how involved the mother was with their adolescent (α = .55).

Third, a maternal disengagement scale was created from five different questions tapping whether the adolescent's mother was cold and withdrawn (α = .84). For example, at Wave I, adolescents were asked whether they were satisfied with the way that their mother communicated with them. The three scales were recoded so that higher scores reflected less maternal attachment, less maternal involvement, and greater maternal disengagement. The three scales were then factor analyzed. The analysis revealed that maternal attachment, maternal involvement, and maternal disengagement could be accounted for by a unitary factor. The regression factor scores were thus used to create a global measure of family risk.

Control variables

Four control variables were also included in the analyses to help rule out the possibility that any significant findings were due to an unmeasured confounding effect. Population genetic research indicates that the distribution of alleles varies across different racial categories (Kang, Palmatier, & Kidd, 1999; Mountain & Risch, 2004; Shields et al., 2005). Quantitative genetic research that fails to control for race/ethnicity may upwardly bias the parameter estimates of the genetic polymorphisms (Cardon & Palmer, 2003). As a result, race was coded as a dichotomous dummy variable (0 = White, 1 = Black). Age (measured in years) and gender (0 = female, 1 = male) were included as statistical controls.

Last, measures of low self-control have been found to be strong and robust predictors of a range of antisocial behaviors, and research that does not control for the effects of low self-control may be misspecified (Pratt & Cullen, 2000). The current authors followed the lead of previous researchers analyzing the Add Health data and include a five-item measure of low self-control (Perrone, Sullivan, Pratt, & Margaryan, 2004). Items comprising the low self-control scale tapped whether the respondent had a difficult time keeping their mind focused and whether they had trouble paying attention at school. Responses to these items were then added together with higher scores representing lower levels of self-control (α = .68).

Analysis plan

The analyses proceeded in a series of stages. To begin, the current authors calculated an ordinary least squares (OLS) regression model to examine the direct effects that the two genetic polymorphisms had on age of first contact with police. In this model, the family risk scale was included as a predictor variable. In subsequent models, however, the current authors tested for gene x environment interactions by examining whether the genetic effects are conditioned by the level of family risk. Specifically, the family risk scale was dichotomized by dividing it at the mean. Those respondents who scored less than or equal to the mean were coded “0” and respondents who scored above the mean were assigned a value of “1.” Respondents with a score of “0” were placed into the low risk families group and respondents with a score of “1” were placed into the high risk families group. Separate analyses were then calculated for the low risk sample and for the high risk sample. Gene x environment interactions will be detected if the coefficients for the genetic polymorphisms are significant for one sample, but not for the other sample. The exact same process is used for the OLS models employing the age of first criminal arrest as the dependent variable. OLS regression is appropriate for analyzing the dependent variables, age of first contact with the police (mean = 17.99, median = 18, mode = 18, standard deviation = 2.66) and age of first criminal arrest (mean = 18.03, median = 18, mode = 18, standard deviation = 2.54), because they are interval level measures that are normally distributed.

Results

As shown in the first model in Table 1, the DRD2 and DRD4 polymorphisms did not exert a significant direct effect on police contacts, however, research indicates that genetic effects often are only observed when they are paired with certain types of environments (Caspi et al., 2002; Foley et al., 2004; Rutter, 2006). To take this possibility into account, the models for the low risk sample were

Table 1
OLS regression equations predicting age of first contact with police (N = 324)

	Full sample		Low risk sample		High risk sample	
	b	Beta	b	Beta	b	Beta
Genetic polymorphisms						
Dopamine D2 receptor (DRD2)	.36 (.23)	.08	.75 (.29)	.19**	−.12 (.36)	−.03
Dopamine D4 receptor (DRD4)	−.11 (.23)	−.03	−.06 (.30)	−.01	−.26 (.37)	−.05
Socialization variable						
Family risk	−.22 (.14)	−.09				
Control variables						
Race	−.01 (.35)	−.00	−.28 (.43)	−.05	.42 (.59)	.06
Age	.57 (.08)	.378**	.56 (.10)	.39**	.59 (.12)	.35**
Gender	−1.19 (.32)	−.19**	−.66 (.43)	−.11	−1.64 (.47)	−.26**
Low self-control	−.14 (.04)	−.18**	−.17 (.06)	−.21**	−.12 (.06)	−.14
R-squared	.18		.20		.19	

Note: Standardized errors in parentheses.
**p<.05, two-tailed.

estimated. The middle column of Table 1 reveals that the DRD2 gene had a significant and positive effect on age of first contact with police. Specifically, as the number of DRD2 risk alleles that a person possesses increased, so did their age of first contact with the police. The DRD4 polymorphism failed to reach statistical significance for the low risk sample. The equation calculated with the high risk sample is presented in the last column of Table 1 and shows that both the DRD2 gene and the DRD4 gene were insignificant.

The results of the analysis thus far suggested that there was a significant gene x environment interaction between DRD2 and family risk in the prediction of age of first contact with the police. In *low risk families*, and somewhat contrary to expectations, the DRD2 gene was associated with an *increase* in the age of contact with the police.

Next, attention was paid to the models predicting age of first criminal arrest. Table 2 shows that the DRD4 gene had a significant direct effect on the dependent variable, whereas DRD2 was statistically insignificant. The next two models were calculated separately for the low risk family sample and for the high risk family sample to determine whether the effects of the two genes were conditioned by the environment. As shown in the middle column of Table 2, both the DRD2 gene and the DRD4 gene exerted statistically significant and positive effects on age of first criminal arrest. For high risk families, however, the genetic effects diminished from statistical significance.

The results of these models indicated that the DRD2 gene and the DRD4 gene interacted with family risk to predict variation in the age of first criminal arrest. The effects of both the DRD2 and the DRD4 polymorphisms were observed only in low risk families, indicating a significant gene x environment interaction. Interestingly, for both dependent variables, the genetic polymorphisms *increased* the age of criminal onset. The potential reasons for these findings were explored next.

Discussion

The current findings were noteworthy on two fronts. First, they represented the first study in the behavioral sciences to use measured genes, not indirect measures employed by twin studies, to estimate the emergence of criminal behavior as measured by police contacts and arrest. For both measures of criminal onset, genetic polymorphisms were significantly predictive and there was significant evidence of gene x environment interactions. Genetic risk factors only

Table 2
OLS regression equations predicting age of first criminal arrest (N=181)

	Full sample		Low risk sample		High risk sample	
	b	Beta	b	Beta	b	Beta
Genetic polymorphisms						
Dopamine D2 receptor (DRD2)	.46 (.30)	.11	.87 (.36)	.24**	.02 (.46)	.01
Dopamine D4 receptor (DRD4)	.61 (.30)	.14**	.80 (.37)	.22**	.22 (.50)	.05
Socialization variable						
Family risk	−.22 (.18)	−.09				
Control variables						
Race	−.48 (.43)	−.08	−.87 (.49)	−.18	.25 (.75)	.03
Age	.63 (.10)	.43**	.52 (.13)	.38**	.70 (.15)	.44**
Gender	−.48 (.43)	−.08	−.29 (.59)	−.05	−.83 (.66)	−.12
Low self-control	−.05 (.05)	−.06	−.07 (.07)	−.10	−.02 (.08)	−.03
R-squared	.21		.28		.20	

Note: Standardized errors in parentheses.
**p<.05, two-tailed.

manifested among youths in low risk social settings. Second, significant effects for the DRD2 and DRD4 genes were positive, that is, they increased onset. That risk alleles actually increased the age of onset was counterintuitive, but not entirely unexpected in behavioral genetics research. In discussing these findings, the current authors were mindful of limitations of the current effort and ways that the current research design could inform subsequent research.

Although the Add Health data set was among the most methodologically impressive efforts in the social sciences, the current biosocial analyses looked at a criminal subsample within the Add Health. This subsample was truncated for first police contact (n=324) and first arrest (n=181). Essentially the current authors restricted the amount of variation that could be explained by the polymorphisms because the goal was to attempt to explain onset with a gene in the criminal population. The risk alleles may have propelled some individuals into crime, but the same alleles may have differential effects on the age of onset. Moreover, the current authors were only looking at respondents from the Add Health *who had been caught offending*. It was unknown whether the genetic polymorphisms were impacted by the filtering processes of the juvenile justice system and an inherent limitation of using official criminal justice statuses as outcome variables.

Recall that for DRD2, the A1 allele was coded as the risk allele and assigned a value of "1" and the A2 allele was assigned a value of "0." For DRD4, alleles that had repeat sequences less than seven were assigned a value of "0"; alleles that had repeat sequences greater than or equal to seven were assigned a value of "1." This measurement approach was consistent with prior research (see Hopfer et al., 2005). Scores for each allele (i.e., the maternal allele and the paternal allele) were then added together to form the DRD2 risk allele scale and the DRD4 risk allele scale. Unfortunately, it was known from prior research (e.g., Caspi et al., 2002; Haberstick et al., 2005) that meaningful variation is usually found at the extreme end of the polymorphic distribution. For example, in exploratory analyses (not shown but provided upon request), youths that had two risk alleles for DRD4 and were reared in high-risk family environments had an earlier onset of police contact than youths with zero or one risk allele. Regrettably, there were insufficient degrees of freedom to expand on these exploratory analyses, but doing so constitutes a pressing avenue for subsequent research.

As discussed by Rutter (2006; also see Blonigen et al., 2005), risk alleles do not unilaterally predict behavioral outcomes. Sometimes risk alleles "flip" their effects depending on the outcome measure. So, while the A1 allele of DRD2 had been shown to be the risk allele for some forms of pathology, it did not seem to impact criminal onset as measured in the current study. For example, Beaver, Wright, DeLisi, Walsh, et al. (2007) examined genetic effects on antisocial behavior among 872 males from the Add Health study. Although they found that neither DRD2 nor DRD4 significantly predicted criminal outcomes, the *interaction* between DRD2 and DRD4 significantly predicted CD and adult crime.

Although the inverse relationship between onset and severity of antisocial career is well established, there are important exceptions in the literature that have shown that some individuals have chronic offending careers but somehow avoid the criminal justice system until adulthood. To illustrate, Eggleston and Laub (2002) explicated the criminal careers literature and discovered that approximately 50 percent of the offenders in large-scale studies of adults actually had an arrest onset of age eighteen or older. Based on data derived from the 1942 and 1949 birth cohorts selected from Racine, Wisconsin (see Shannon, 1988), Eggleston and Laub also found that many of the same correlates of early onset offending were also meaningful predictors of adult onset offending. Similarly, among a sample of five hundred adult offenders with a minimum of thirty career arrests, DeLisi (2006) found that 62 percent of the sample was not first arrested until adulthood. Related to this issue, Elander, Rutter, Simonoff, and Pickles (2000, pp. 506–507) found that serious offenders with delayed or adult onset also frequently suffer from major mental health disorders. In this sense, the comorbidity implicated by psychiatric problems seemingly insulates seriously disturbed offenders from the criminal justice system.

Concomitantly, the current authors did not control for similar comorbidity issues among youths in the Add Health. Children with risk alleles for DRD2 and DRD4 could also suffer from medical problems or psychiatric problems that significantly reduce the opportunities to engage in antisocial behavior and be detected by the police. For example, the DRD2 gene is associated with comorbid conditions, such as depression, anxiety, and social dysfunction (Lawford, Young, Noble, Kann, & Ritchie, 2006) that can significantly curtail normal social functioning. Physical therapy and treatment for various medical problems and developmental disabilities are extremely intensive and circumscribe the "normal" socialization processes of adolescence that theoretically engender crime or at least provide opportunities for delinquency (Moffitt, 1993). In other words, although the current study used controls for race, age, gender, low self-control, family risk, two genetic polymorphisms, and disaggregated the analyses by risk environment, there are, of course, many covariates that would be useful to more intensively evaluate genetic effects on delinquent careers.

The current findings build on a burgeoning literature that has used the Add Health data set to investigate antisocial behavior from interdisciplinary or biosocial perspectives. Prior investigators have shown the interconnections between biological and sociological constructs in explaining victimization (Haynie & Piquero, 2006), suicide (Cho, Guo, Iritani, & Hallfors, 2006), delinquency among males and females (Beaver & Wright, 2005), and gender-specific delinquent involvement (Felson & Haynie, 2002). The common thread in these prior studies was the significant relationship between pubertal development; the ways that this development, particularly early pubertal development, enhanced opportunities for antisocial behavior; and delinquency. In other words, there is increasing awareness in sociology, criminology, and the social sciences generally, that human behavior is not bound by artificial disciplinary boundaries (also see Freese, Li, & Wade, 2003; Plomin & Asbury, 2005; Udry, 1988, 1995). In fact, in his presidential address to the American Sociological Association, Massey (2002) exhorted sociologists to learn, appreciate, and incorporate biological constructs into the discipline as a way to *enhance* the sociological imagination. Most phenotypes, conventional or criminal, have multiple etiological bases that span biological, psychological, sociological, and in the current study, genetic perspectives. Today, behavioral scientists are best equipped to employ phenomena from diverse academic disciplines to arrive at the most scientifically-sound conclusions. The current effort found that genetic risk factors affect the timing of criminal onset, but only under certain environmental conditions and in counterintuitive ways. Finally, the current authors urge other researchers to replicate the current study in hopes of moving to a broader understanding of the emergence of antisocial behavior, an event that is rife with protean negative consequences for healthy social development.

Acknowledgements

This research used 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 seventeen other agencies. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Persons interested in obtaining data files from Add Health should contact Add Health, Carolina Population Center, 123 West Franklin Street, Chapel Hill, North Carolina 27516–2524 (addhealth@unc.edu). We thank Tom Blomberg and the College of Criminology and Criminal Justice at Florida State University for providing the financial support needed to obtain the restricted-use version of the Add Health data.

References

Add Health Biomarker Team. (n.d.). *Biomarkers in Wave III of the Add Health Study.* Chapel Hill: University of North Carolina, Carolina Population Center.

Arseneault, L., Moffitt, T. E., Caspi, A., Taylor, A., Rijsdijk, F. V., Jaffee, S. R., et al. (2003). Strong genetic effects on cross-situational antisocial behavior among 5-year old children according to mothers, teachers, examiner-observers, and twins' self-reports. *Journal of Child Psychology and Psychiatry*, *44*, 832−848.

Beaver, K. M., & Wright, J. P. (2005). Biosocial development and delinquent involvement. *Youth Violence and Juvenile Justice*, *3*, 168−192.

Beaver, K. M., Wright, J. P., DeLisi, M., Daigle, L. E., Swatt, M. L., & Gibson, C. L. (2007). Evidence of a gene x environment interaction in the creation of victimization: Results from a longitudinal sample of adolescents. *International Journal of Offender Therapy and Comparative Criminology*, *51*, 620−645.

Beaver, K. M., Wright, J. P., DeLisi, M., Walsh, A., Vaughn, M. G., Boisvert, D., et al. (2007). A gene x gene interaction between DRD2 and DRD4 is associated with conduct disorder and antisocial behavior in males. *Behavioral and Brain Functions*, *3*, 3−30.

Berman, S. M., & Noble, E. P. (1995). Reduced visuospatial performance in children with the D2 dopamine receptor A1 allele. *Behavior Genetics*, *25*, 45−58.

Blonigen, D. M., Hicks, B. M., Krueger, R. F., Patrick, C. J., & Iacono, W. G. (2005). Psychopathic personality traits: Heritability and genetic overlap with internalizing and externalizing psychopathology. *Psychological Medicine*, *35*, 637−648.

Blumstein, A., Cohen, J., Roth, J., & Visher, C. (Eds.). (1986). *Criminal careers and "career criminals".* Washington, DC: National Academy Press.

Cardon, L. R., & Palmer, L. J. (2003). Population stratification and spurious allelic association. *Lancet*, *361*, 598−604.

Caspi, A., Henry, B., McGee, R. O., Moffitt, T. E., & Silva, P. A. (1995). Temperamental origins of child and adolescent behavior problems: From age three to age fifteen. *Child Development*, *66*, 55−68.

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., et al. (2002). Role of the genotype in the cycle of violence in maltreated children. *Science*, *297*, 851−854.

Chantala, K. (2003). *Introduction to analyzing Add Health data.* Chapel Hill: University of North Carolina, Carolina Population Center.

Cho, H., Guo, G., Iritani, B. J., & Hallfors, D. D. (2006). Genetic contribution to suicidal behaviors and associated risk factors among adolescents in the U.S. *Prevention Science*, *7*, 303−311.

Crosnoe, R., & Elder, G. H. (2004). Family dynamics, supportive relationships, and educational resilience during adolescence. *Journal of Family Issues*, *25*, 571−602.

Cullen, F. T., Wright, J. P., & Blevins, K. R. (Eds.). (2006). *Taking stock: The status of criminological theory: Advances in criminological theory, Vol. 15.* New Brunswick, NJ: Transaction.

DeLisi, M. (2001). Scaling archetypal criminals. *American Journal of Criminal Justice*, *26*, 77−92.

DeLisi, M. (2005). *Career criminals in society.* Thousand Oaks, CA: Sage.

DeLisi, M. (2006). Zeroing in on early arrest onset: Results from a population of extreme career criminals. *Journal of Criminal Justice*, *34*, 17−26.

Eaves, L., Silberg, J. L., Meyer, J., Maes, H., Simonoff, E., Pickles, A., et al. (1997). Genetics and developmental psychopathology: 2. The main effects of genes and environment on behavioral problems in the Virginia Twin Study of Adolescent Behavioral Development. *Journal of Child Psychology and Psychiatry*, *38*, 965−980.

Eggleston, E. P., & Laub, J. H. (2002). The onset of adult offending: A neglected dimension of the criminal career. *Journal of Criminal Justice*, *30*, 603−622.

Elander, J., Rutter, M., Simonoff, E., & Pickles, A. (2000). Explanations for apparent late onset criminality in a high-risk sample of children followed up in adult life. *British Journal of Criminology*, *40*, 497−509.

Farrington, D. P., Loeber, R., Elliott, D. S., Hawkins, J. D., Kandel, D. B., Klein, M. W., et al. (1990). Advancing knowledge about the onset of delinquency and crime. In B. Lahey & A. Kazdin (Eds.), *Advances in clinical child psychology* (Vol. 13, pp. 283–342). New York: Plenum.

Felson, R. B., & Haynie, D. L. (2002). Pubertal development, social factors, and delinquency among adolescent boys. *Criminology*, *40*, 967−988.

Foley, D. L., Eaves, L. J., Wormley, B., Silberg, J. L., Maes, H., Kuhn, J., et al. (2004). Childhood adversity, monoamine oxidase A genotype, and risk for conduct disorder. *Archives of General Psychiatry*, *61*, 738−744.

Freese, J., Li, J., & Wade, L. D. (2003). The potential relevances of biology to social inquiry. *Annual Review of Sociology*, *29*, 233−256.

Gibson, C. L., & Tibbetts, S. G. (2000). A biosocial interaction in predicting early onset of offending. *Psychological Reports*, *86*, 509−518.

Gottfredson, M. R., & Hirschi, T. (1990). *A general theory of crime.* Stanford, CA: Stanford University Press.

Haberstick, B. C., Lessem, J. M., Hopfer, C. J., Smolen, A., Ehringer, M. A., Timberlake, D., et al. (2005). Monoamine oxidase A (MAOA) and antisocial behaviors in the presence of childhood and adolescent maltreatment. *American Journal of Medical Genetics*, *135B*, 59−64.

Harris, K. M., Florey, F., Tabor, J., Bearman, P. S., Jones, J., & Udry, J. R. (2003). *The National Longitudinal Study of Adolescent Health: Research design* [Data file]. Retrieved from http://www.cpc.unc.edu/projects/addhealth/design

Haynie, D. L. (2001). Delinquent peers revisited: Does network structure matter? *American Journal of Sociology*, *106*, 1013−1057.

Haynie, D. L., & Piquero, A. R. (2006). Pubertal development and physical victimization in adolescence. *Journal of Research in Crime and Delinquency*, *43*, 3−35.

Hingson, R. W., Heeren, T., & Winter, M. R. (2006). Age at drinking onset and alcohol dependence. *Archives of Pediatrics and Adolescent Medicine*, *160*, 739−746.

Hopfer, C. J., Timberlake, D., Haberstick, B., Lessem, J. M., Ehringer, M. A., Smolen, A., et al. (2005). Genetic influences on quantity of alcohol consumed by adolescents and young adults. *Drug and Alcohol Dependence*, *78*, 187−193.

Jaffee, S. R., Caspi, A., Moffitt, T. E., Dodge, K. A., Rutter, M., Taylor, A., et al. (2005). Nature x nurture: Genetic vulnerabilities interact with physical maltreatment to promote conduct problems. *Development and Psychopathology*, *17*, 67−84.

Kang, A. M., Palmatier, M. A., & Kidd, K. K. (1999). Global variation of a 40-bp VNTR in the 3'-untranslated region of the dopamine transporter gene (SLC6A3). *Biological Psychiatry*, *46*, 151−160.

Krohn, M. D., Thornberry, T. P., Rivera, C., & Le Blanc, M. (2001). Later delinquency careers. In R. Loeber & D. P. Farrington (Eds.), *Child delinquents: Development, intervention, and service needs* (pp. 67−93). Thousand Oaks, CA: Sage.

Lahey, B. B., Moffitt, T. E., & Caspi, A. (Eds.). (2003). *Causes of conduct disorder and juvenile delinquency.* New York: Guilford Press.

Lawford, B., Young, R., Noble, E. P., Kann, B., & Ritchie, T. (2006). The DRD2 gene is associated with comorbid depression, anxiety, and social dysfunction in untreated veterans with PTSD. *European Psychiatry*, *21*, 180−185.

Loeber, R., & Dishion, T. (1983). Early predictors of male delinquency: A review. *Psychological Bulletin*, *94*, 68−99.

Marino, C., Vanzin, L., Giorda, R., Frigerio, A., Lorusso, M. L., Nobile, M., et al. (2004). An assessment of transmission disequilibrium between quantitative measures of childhood problem behaviors and DRD2/TaqI and DRD4/48bp-repeat polymorphisms. *Behavior Genetics*, *34*, 495−502.

Massey, D. S. (2002). A brief history of human society: The origin and role of emotion in social life. *American Sociological Review*, *67*, 1−29.

Miller, W. B., Pasta, D., MacMurray, J., Chiu, C., Wu, H., & Comings, D. E. (1999). Dopamine receptor genes are associated with age at first sexual intercourse. *Journal of Biosocial Science*, *31*, 43−54.

Moffitt, T. E. (1993). 'Life-course persistent' and 'adolescence-limited' antisocial behavior: A developmental taxonomy. *Psychological Review*, *100*, 674−701.

Moffitt, T. E. (2003). Life-course-persistent and adolescence-limited antisocial behavior: A 10-year research review and a research agenda. In B. B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 49−75). New York: Guilford Press.

Moffitt, T. E., & Caspi, A. (2001). Childhood predictors differentiate life-course persistent and adolescence-limited pathways among males and females. *Development and Psychopathology*, *13*, 355−375.

Moffitt, T. E., Caspi, A., Rutter, M., & Silva, P. A. (2001). *Sex differences in antisocial behavior: Conduct disorder, delinquency, and violence in the Dunedin Longitudinal Study.* New York: Cambridge University Press.

Moffitt, T. E., Lynam, D., & Silva, P. A. (1994). Neuropsychological tests predict persistent male delinquency. *Criminology*, *32*, 101−124.

Mountain, J. L., & Risch, N. (2004). Assessing genetic contributions to phenotypic differences among "racial" and "ethnic" groups. *Nature Genetics*, *36*, 48−53.

Munafò, M. R., Clar, T., Moore, L., Payne, E., Walton, R., & Flint, J. (2003). Genetic polymorphisms and personality in healthy adults: A systematic review and meta-analysis. *Molecular Psychiatry*, *8*, 471−484.

Patterson, G. R. (1995). Coercion as a basis for early age of onset for arrest. In J. McCord (Ed.), *Coercion and punishment in long-term perspectives* (pp. 81−105). New York: Cambridge University Press.

Perrone, D., Sullivan, C. J., Pratt, T. C., & Margaryan, S. (2004). Parental efficacy, self-control, and delinquency: A test of a general theory of crime on a nationally representative sample of youth. *International Journal of Offender Therapy and Comparative Criminology*, *48*, 298−312.

Piquero, A. (2001). Testing Moffitt's neuropsychological variation hypothesis for the prediction of life-course persistent offending. *Psychology, Crime and Law*, *7*, 193−215.

Piquero, A. R., & Brezina, T. (2001). Testing Moffitt's account of adolescence-limited delinquency. *Criminology*, *39*, 353−370.

Piquero, A. R., Farrington, D. P., & Blumstein, A. (2003). The criminal career paradigm: Background and recent developments. In M. Tonry (Ed.), *Crime and justice: A review of research* (Vol. 30, pp. 359–506). Chicago: University of Chicago Press.

Plomin, R., & Asbury, K. (2005). Nature and nurture: Genetic and environmental influences on behavior. *Annals of the American Academy of Political and Social Science*, *600*, 86−98.
Pratt, T. C., & Cullen, F. T. (2000). The empirical status of Gottfredson and Hirschi's general theory of crime: A meta-analysis. *Criminology*, *38*, 931−964.
Raine, A., Brennan, P., Mednick, B., & Mednick, S. A. (1996). High rates of violence, crime, academic problems, and behavioral problems in males with both early neuromotor deficits and unstable family environments. *Archives of General Psychiatry*, *53*, 544−549.
Raine, A., Brennan, P., & Mednick, S. A. (1997). Interaction between birth complications and early maternal rejection in predisposing individuals to adult violence: Specificity to serious, early-onset violence. *American Journal of Psychiatry*, *154*, 1265−1271.
Robins, L. N. (1978). Sturdy childhood predictors of adult antisocial behavior: Replications from longitudinal studies. *Psychological Medicine*, *8*, 611−622.
Rutter, M. (2006). *Genes and behavior: Nature-nurture interplay explained.* Malden, MA: Blackwell.
Schmidt, L. A., Fox, N. A., Perez-Edgar, K., Hu, S., & Hamer, D. H. (2001). Association of DRD4 with attention problems in normal childhood development. *Psychiatric Genetics*, *11*, 25−29.
Schreck, C. J., Fisher, B. S., & Miller, J. M. (2004). The social context of violent victimization: A study of the delinquent peer effect. *Justice Quarterly*, *21*, 23−47.
Shannon, L. W. (1988). *Criminal career continuity: Its social context.* New York: Human Services Press.
Shields, A. E., Fortun, M., Hammonds, E. M., King, P. A., Lerman, C., Rapp, R., et al. (2005). The use of race variables in genetic studies of complex traits and the goal of reducing health disparities: A transdisciplinary perspective. *American Psychologist*, *60*, 77−103.
Silberg, J., Rutter, M., Meyer, J., Maes, H., Hewitt, J., Simonoff, E., et al. (1996). Genetic and environmental influences on the covariation between hyperactivity and conduct disturbance in juvenile twins. *Journal of Child Psychology and Psychiatry*, *37*, 803−816.
Simons, R. L., Wu, C., Conger, R. D., & Lorenz, F. O. (1994). Two routes to delinquency: Differences between early and late starters in the impact of parenting and deviant peers. *Criminology*, *32*, 247−275.
Slutske, W. S., Heath, A. C., Dinwiddie, S., Madden, P., Bucholz, K., Dunne, M., et al. (1997). Modeling genetic and environmental influences in the etiology of conduct disorder: A study of 2,682 adult twin pairs. *Journal of Abnormal Psychology*, *106*, 266−279.
Taylor, J., Iacono, W. G., & McGue, M. (2000). Evidence for a genetic etiology of early-onset delinquency. *Journal of Abnormal Psychology*, *109*, 634−643.
Tibbetts, S. G., & Piquero, A. R. (1999). The influence of gender, low birth weight, and disadvantaged environment in predicting early onset of offending: A test of Moffitt's interactional hypothesis. *Criminology*, *37*, 843−878.
Udry, J. R. (1988). Biological predispositions and social control in adolescent sexual behavior. *American Sociological Review*, *53*, 709−722.
Udry, J. R. (1995). Sociology and biology: What biology do sociologists need to know? *Social Forces*, *73*, 1267−1278.
Udry, J. R. (1998). *The national longitudinal sample of adolescent health.* Chapel Hill: University of North Carolina, Carolina Population Center.
Udry, J. R. (2003). *The National Longitudinal Study of Adolescent Health (Add Health), waves I and II, 1994–1996; wave III, 2001–2002* [Data file]. Chapel Hill: University of North Carolina, Carolina Population Center.
Van Tol, H., Wu, C., Guan, H., Ohara, K., Bunzow, J., Civelli, J., et al. (1992). Multiple dopamine D4 receptor variants in the human population. *Nature*, *358*, 149−152.
Wolfgang, M. E., Figlio, R. M., & Sellin, T. (1972). *Delinquency in a birth cohort.* Chicago: University of Chicago Press.