EXAMINING THE GENETIC UNDERPINNINGS TO MOFFITT’S DEVELOPMENTAL TAXONOMY: A BEHAVIORAL GENETIC ANALYSIS*

J.C. BARNES  
School of Economic, Political and Policy Sciences  
The University of Texas at Dallas

KEVIN M. BEAVER  
College of Criminology and Criminal Justice  
Florida State University

BRIAN B. BOUTWELL  
College of Criminal Justice  
Sam Houston State University

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In recent years, criminological research has observed an increase in studies examining different offending trajectories. Much of this research has been guided by Moffitt’s (1993) developmental taxonomy of life-course persistent offenders, adolescence-limited offenders, and abstainers. Moffitt (1993) argued that the etiologies of these different pathways could be traced to several biosocial factors, including perhaps genetic factors. To date, research has failed to address this possibility directly. The current study addressed this gap in the literature by examining the extent to which genetic factors explain variance in different offending

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patterns. Analysis of sibling pairs (N = 2,284; ages spanned between 11 and 27 years) drawn from the National Longitudinal Study of Adolescent Health (Add Health) revealed that genetic factors contributed significantly to being classified in each of the different offending patterns. Specifically, genetic factors explained between 56 and 70 percent of the variance in being classified as a life-course persistent offender across different coding strategies, 35 percent of the variance in being classified as an adolescence-limited offender, and 56 percent of the variance in being classified as an abstainer. We discuss the importance of integrating genetics into future studies examining offending trajectories.

Moffitt’s (1993) developmental taxonomy is one of the most prominent explanations of antisocial behavior, and it has generated an impressive body of research (see for a review Moffitt, 2006). One feature of the theory that has sparked much interest among criminologists is whether the age–crime curve masks different offending patterns. Moffitt (1993) suggested that two types of offenders and one group of nonoffenders are identifiable in the population. The two groups of offenders are referred to as life-course persistent (LCP) offenders and as adolescence-limited (AL) offenders, whereas the group of nonoffenders is referred to as abstainers. According to Moffitt (1993), LCP offenders begin to manifest antisocial behavior in early childhood, their behavioral problems carry over into adolescence, and these problems remain relatively stable throughout adulthood. Although only approximately 5–10 percent of the population fits this pattern (Wolfgang, Figlio, and Sellin, 1972), they are responsible for most of the crimes committed (DeLisi, 2005). In contrast to LCP offenders, AL offenders limit their offending to the period of adolescence. These individuals do not engage in serious misbehavior in childhood, and they desist from delinquent activity in early adulthood. Finally, a small portion of the population abstains from delinquent activity in childhood, in adolescence, and in adulthood (Wolfgang, Figlio, and Sellin, 1972).

Researchers have identified myriad factors that can predict the different offending patterns (i.e., LCP, AL, and abstainer), many of which are environmental influences (Bersani, Nieuwbeerta, and Laub, 2009; Piquero, Farrington, and Blumstein, 2003; Walsh and Beaver, 2009; Wright, Tibbetts, and Daigle, 2008). The current study extends this line of research by analyzing the extent to which genetic and environmental influences are responsible for explaining variance in the etiology of the different offending patterns. Moffitt’s (1993) theory hints at the possibility that genetic factors partially influence the development of each offending pattern, and findings from behavioral genetic research have consistently shown that genetic factors generally underlie the etiology of antisocial behavior (Moffitt, 2005). No research, however, has examined the genetic underpinnings to Moffitt’s (1993) typology of offending patterns.
ETIOLOGY OF ANTISOCIAL BEHAVIOR

During the past 20 years, a large literature examining the genetic and environmental influences on the etiology of antisocial behavior has developed. Four meta-analyses and several literature reviews are available that summarize the extant research estimating the heritability of antisocial behaviors (Ferguson, 2010; Fishbein, 1990; Harris, 1995, 1998; Mason and Frick, 1994; Miles and Carey, 1997; Moffitt, 2005; Raine, 1993; Rhee and Waldman, 2002; Rowe, 1990, 2002; Turkheimer and Waldron, 2000). Mason and Frick (1994) conducted one of the first meta-analyses and reported an average heritability ($h^2$) estimate of .48 for antisocial behavior, meaning that 48 percent of the variance in antisocial behaviors was explained by genetic factors. Other meta-analyses also have emerged that provide similar estimates with $h^2$ ranging between .41 and .56 (Ferguson, 2010; Miles and Carey, 1997; Rhee and Waldman, 2002). Taken together, these studies indicate that approximately half of the variance in antisocial behavior is attributable to genetic influences with the remaining variance being attributable to environmental influences.

The empirical evidence indicating that antisocial behavior is partially influenced by genetic factors has spawned some theorists to advocate for an integration of behavioral genetic findings into mainstream criminological theories (Robinson and Beaver, 2010; Walsh, 2002). Walsh (2002), for example, argued that many of the most popular criminological theories, such as strain theory, can be revamped to incorporate the results from genetic research. Moffitt’s (1993) developmental taxonomy is perhaps one of the most obvious candidates for this type of integration, in large part, because of studies revealing links between genetic influences and many of the risk factors highlighted in the theory. The following sections will outline the etiologies of the LCP pathway, the AL pathway, and the abstainer pathway, as hypothesized by Moffitt (1993), and then discuss the various ways in which these offending patterns might be influenced by genetic factors.

THE LIFE-COURSE PERSISTENT PATHWAY

According to Moffitt (1993), LCP offenders are hit with a “double whammy” of criminogenic risk factors that are present in early childhood. First, LCP offenders suffer from neuropsychological deficits that make offending more likely (Beaver et al., 2010; Cauffman, Steinberg, and Piquero, 2005; Moffitt, 1990). Neuropsychological deficits can result from factors ranging from genetic to postnatal influences that undermine normal brain functioning (McGloin, Pratt, and Piquero, 2006; Raine, 2008). Second, and in addition to their cognitive deficits, LCPs are born into adverse rearing environments. These rearing environments tend to exacerbate the difficult temperaments that covary with neuropsychological deficits. As a result, the
environment cannot respond to the child in an appropriate and prosocial fashion. Over time, the interactions between neuropsychological deficits and a criminogenic environment begin to increase the odds that an individual will follow the LCP pathway.

A line of research examining the etiological background of LCP offenders has emerged, and the theory has some support (Blokland, Nagin, and Nieuwbeerta, 2005; Cauffman, Steinberg, and Piquero, 2005; Gibson and Tibbetts, 2000; McGlone, Pratt, and Piquero, 2006; Tibbetts and Piquero, 1999). For example, Turner, Hartman, and Bishop (2007) found that neuropsychological deficits interacted with a disadvantaged home environment to predict LCP offending. In another study, Raine et al. (2005) divided their sample of young males into the following groups according to their pattern of offending: a control group, a childhood-limited group, an AL group, and an LCP group. Their analyses indicated that boys identified as LCPs had greater deficits in spatial and memory functions as compared with boys identified as ALs or controls. These findings provide partial support for Moffitt’s (1993) hypothesis that a combination of neuropsychological factors and an adverse rearing environment contribute to the development of LCP offending. Importantly, however, the amount of variance explained by these two factors is relatively moderate, suggesting that other factors, including genetic influences, also might be involved.

At least three lines of research indicate that genetic factors are influential for the development of LCP offenders. First, an impressive amount of research indicates that variance in one of the purported causes of LCP offending—neuropsychological deficits—is explained by genetic factors (Bergen, Gardner, and Kendler, 2007; Bouchard and McGue, 1981; Burdick et al., 2007; Dick et al., 2007; Kebir et al., 2009; Loehlin, 1989; Plomin, 1990b; Plomin et al., 2001; Rowe, Jacobson, and Van den Oord, 1999; Savitz, Solms, and Ramesar, 2006; Waldman et al., 2006). For example, researchers have reported that heritability estimates for cognitive abilities range between .40 and .80 (Devlin, Daniels, and Roeder, 1997). From this perspective, genetic factors would not directly cause someone to become an LCP offender, but they would operate indirectly by affecting levels of neuropsychological deficits.

The second reason to suspect that genes influence LCP offending comes from studies that have examined the etiology of serious antisocial behavior in childhood. Given that LCP offenders begin to display signs of serious antisocial conduct in childhood, it stands to reason that the factors that contribute to childhood misbehavior also might be causally related to the development of LCP offenders. The results flowing from behavioral genetic research have revealed that childhood antisocial behavior is under substantial genetic influence (DiLalla and Gottesman, 1989; Jaffee et al., 2005; Taylor, Iacono, and McGue, 2000; Thapar et al., 2005). For example, Viding
et al. (2005) examined childhood misconduct in 7-year-olds and reported heritability estimates that ranged from .30 to .81 (see also Arseneault et al., 2003; van Beijsterveldt et al., 2003; Van Hulle et al., 2009). These results tend to suggest that the early developmental origins of LCP offending are scripted in part by genetic factors.

The third line of research suggesting that genetic factors might be related to LCP offending comes from studies examining the stability of antisocial behaviors across the life course. One hallmark of LCP offenders is that they display extremely high levels of stability in antisocial behavior from childhood through adulthood. Identifying the underlying factors that account for such stability would provide some insight into the potential causes of LCP offending. If genetic factors, for example, explained part of the stability in antisocial behavior, then that would represent additional evidence that the etiology of LCP offending has roots in genetic influences. Several behavioral genetic studies have explored the extent to which genetic factors are involved in producing stability in antisocial behaviors. Overall, the results of these studies have revealed that a significant amount of covariance in antisocial behaviors over time is explained by genetic factors (Dick et al., 2009; Haberstick et al., 2006; Lyons et al., 1995; Malone et al., 2004; van Beijsterveldt et al., 2003; Van Hulle et al., 2009). For example, Reiss and colleagues (2000) analyzed data drawn from a sample of adolescent siblings who were interviewed at two separate points in time. The results of their bivariate Cholesky decomposition models revealed that 69 percent of the stability in antisocial behavior was explained by genetic influences. Similarly, Eley, Lichtenstein, and Moffitt (2003) reported that the stability of aggression from childhood to adolescence was almost entirely a result of genetic influences ($h^2 = .84$). The results of these and other studies indicate that stability in antisocial behavior is governed by genetic factors (Haberstick et al., 2006; Lyons et al., 1995), a finding that points to the possibility that the etiology of LCP offending also might be tied to genetic factors.

THE ADOLESCENCE-LIMITED PATHWAY

Unlike LCP offending, whose etiology can be traced to early childhood, the etiology of AL offending is restricted solely to the adolescent years (Moffitt, 1993). Specifically, Moffitt (1993) argued that AL offenders commit delinquent acts because of their frustration with the maturity gap. The maturity gap, which refers to the distance between an adolescent’s biological maturity and his or her social maturity, is a frustrating influence that leads the adolescent to engage in delinquency (Agnew, 2003). Moffitt (1993) argued that adolescents physically resemble adults (e.g., they are biologically capable of reproduction), but they lack the social maturity that
is afforded to adults (e.g., unlike adults, adolescent youth are restricted from many activities such as drinking and smoking and they are rarely allotted important decision-making power). During the time period when adolescents are experiencing the maturity gap, they are likely to witness their LCP counterparts engaging in a range of adult-like behaviors such as drinking alcohol and having sex. AL offenders mimic these behaviors in an attempt to alleviate the dissonance between their biological maturity and their social immaturity. As AL offenders age, they are granted more rights and privileges, which begins to bring their social maturity in line with their biological maturity. Eventually, the maturity gap is erased and AL offending ceases—barring any snares that propel the offender onto the LCP pathway (Hussong et al., 2004).

In comparison with research conducted on the etiology of LCP offending, much less criminological research has examined Moffitt’s (1993) hypotheses regarding the etiology of AL offending (Moffitt, 2006). Indeed, only two studies have directly analyzed the effect of the maturity gap on AL offending (Barnes and Beaver, 2010; Galambos, Barker, and Tilton-Weaver, 2003). Galambos, Barker, and Tilton-Weaver (2003) identified the following groups of adolescents in their sample: pseudomature adolescents, immature adolescents, and mature adolescents. Pseudomature adolescents were the most advanced physically, and they reported a strong desire to be older than their current age, a hallmark of being caught in the maturity gap. Supporting Moffitt’s (1993) hypotheses, the pseudomature adolescents (i.e., those caught in the maturity gap) were more involved in crime and delinquency than were adolescents in either of the other two groups (i.e., those not caught in the maturity gap). Similar findings were reported by Barnes and Beaver (2010).

Circumstantial evidence tying genetics to AL offending comes from behavioral genetic research that has estimated genetic influences on delinquency scales that capture many of the types of behaviors that typify AL offenders. The results of these studies were reviewed by Raine (1993), who concluded that delinquency committed during the adolescent years was under genetic influence. Raine, however, also noted that, because adolescent delinquency is normative, environmental factors are likely to be more influential for youthful offending as compared with adult offending. Findings from quantitative genetic research have supported this claim by showing that heritability estimates are larger for adult offending as compared with adolescent delinquency (Lyons et al., 1995; Miles and Carey, 1997; Raine, 1993).

Another line of evidence suggesting that genetic factors might be involved in AL offending comes from research examining factors that account for behavioral change. Recall that one of the main characteristics of AL offending is that, as adolescence ends and early adulthood emerges,
AL offenders begin to cease their delinquent involvement. Identifying the factors that promote behavioral change thus might shed some light on the etiological factors of AL offending. Several studies have examined genetic influences on behavioral change, the results of which have consistently suggested that genetic factors account partially for changes in and desistance from antisocial activity (Collins, 2004; Eley, Lichtenstein, and Moffitt, 2003; Reiss et al., 2000; van Beijsterveldt et al., 2003). To illustrate, Reiss et al. (2000) reported that genetic factors were the predominate influence on changes in antisocial behavior during a 3-year time span ($h^2 = .67$). Because AL offenders desist from crime in early adulthood, and because desistance and behavioral change have been shown to be affected by genetic factors, it is possible that AL offending is partially influenced by genetic factors.

Juxtaposing these lines of research suggests that AL offending might be partly under genetic control. In keeping with Raine’s (1993) early assessment, however, it is likely that the genetic component to AL offending will be smaller than the environmental component. In other words, genetic factors might influence the etiology of AL offending, but environmental influences are likely to explain a larger proportion of the variance.

THE ABSTAINER PATHWAY

Because involvement in delinquent activity during adolescence is statistically normative (Elliott et al., 1983), Moffitt (1993) argued that abstinence from delinquency is abnormal and thus has its own unique causal factors. Specifically, she claimed that abstainers avoid the maturity gap, are likely to display undesirable personality traits (from the perspective of their peers), and lack contact with delinquent peer groups (often as a result of their personality traits). To date, no criminological research has directly examined the hypothesis linking the maturity gap to delinquency abstention. However, a handful of studies have identified other factors that can predict delinquency abstention (Boutwell and Beaver, 2008; Brezina and Piquero, 2007; Farrington, Ttofi, and Coid, 2009; Moffitt et al., 1996; Pulkkinen, Lyyra, and Kokko, 2009; Shedler and Block, 1990; White, Bates, and Buyske, 2001), and at least three reasons are available that explain why genes might be related to delinquency abstention.

First, recall that the maturity gap is an important influence on delinquency abstention and that the maturity gap is tied to pubertal development. Pubertal development is a biological process that is influenced by genetic factors (Beaver and Wright, 2005; Walsh, 2009; Wright, Tibbetts, and Daigle, 2008). Genes regulate many biological functions, and scholars have begun to identify certain genes that might trigger pubertal onset (Seminara et al., 2003; Sisk and Foster, 2004). Because pubertal
development is governed by genetic factors, it is possible that abstaining behaviors are indirectly influenced by these same factors.

Second, Moffitt (1993) linked undesirable personality traits (from the peers’ point of view) to abstaining behaviors. An impressive body of research has underscored the importance of genetic factors on personality development (Caspi, Roberts, and Shiner, 2005; Rowe, 1990). Indeed, Bouchard and Loehlin (2001: 243) explained that, “all human psychological traits are influenced by genetic factors to a significant degree.” Drawing on this literature, we expect abstainers to be influenced by genetic factors via the genetic influence on undesirable personality traits.

Third, Moffitt (1993) argued that abstainers do not associate with delinquent peer groups. Although historically viewed as a social phenomenon, recent behavioral genetic research has identified a significant genetic component to peer associations (Beaver et al., 2009; Cleveland, Wiebe, and Rowe, 2005; Iervolino et al., 2002; Kendler et al., 2007). Kendler et al. (2007), for example, reported that genes were important influences on peer group selection, with their models indicating that the heritability of peer group deviance ranged between .30 and .50.

Taken together, these three lines of research suggest that abstaining behaviors might be partially scripted by genetic factors. Importantly, one study offers some strong support for the role of genetic factors on delinquency abstention. Boutwell and Beaver (2008) analyzed data drawn from the National Longitudinal Study of Adolescent Health (Add Health) to examine whether two dopaminergic genes (DRD2 and DRD4) were associated with the odds of abstaining from delinquency. The results of their study revealed that both of these genes significantly affected the odds of delinquency abstention. Given that this study is the only one to examine the genetic foundation to abstention, the extent to which genetic factors influence delinquency abstention begs further investigation.

THE CURRENT STUDY

Converging lines of research suggest that Moffitt’s (1993) explanation of the factors that propel an individual onto the LCP pathway, the AL pathway, or the abstainer pathway could be improved by integrating findings from behavioral genetic research. Whether such integration is needed hinges on whether the etiology of these offending groups is indeed differentially affected by genetic factors; however, research has not yet examined this possibility directly. The current study seeks to fill this gap in the literature by examining whether and to what extent genetic influences can explain variance in being identified as an LCP offender, an AL offender, and an abstainer.
DATA

Data from the National Longitudinal Study of Adolescent Health (Udry, 1998) were analyzed in the current study. Add Health provides a longitudinal and nationally representative sample of adolescents who were enrolled in grades 7–12 in 1995. A stratified random sampling procedure ultimately resulted in 132 schools being included in the study (see Harris et al., 2009, or Kelly and Peterson, 1997, for an overview of the Add Health data and the research design). All students attending these 132 schools were asked to complete a self-report questionnaire during a designated class session. This round of data collection netted information from more than 90,000 students (i.e., the in-school survey).

A subsample of the students who completed the in-school surveys were contacted and asked to complete a follow-up interview, along with their primary caregiver, in their homes. Information from 20,745 adolescents and 17,700 primary caregivers was gathered during this round of data collection (i.e., wave I in-home interviews). The wave I in-home surveys lasted approximately 90 minutes and were designed to gain more detailed information about the adolescent, his or her experiences, and his or her rearing environment. For example, information was gathered about the respondent’s personality traits, his or her social relationships, and his or her behaviors.

Approximately 1 year after the wave I in-home interviews were completed, 14,738 of the wave I respondents were again interviewed in their homes (i.e., wave II in-home interviews). Because only a short amount of time elapsed between wave I and wave II, and because most of the respondents were still adolescents, the questionnaires remained very similar. For instance, respondents were asked about their social relationships and about their behaviors. Primary caregivers were not interviewed at wave II.

Nearly 6 years after wave I interviews were conducted (and nearly 5 years after wave II), a third round of interviews took place with 15,197 respondents (i.e., wave III in-home interviews). During this data collection period, most of the respondents were young adults, not adolescents. As a result, the surveys were changed to include more age-appropriate questions. Respondents, for instance, were asked about their employment history and their marital status.

Nested within the Add Health data is a subsample of sibling pairs who resided in the same household at wave I. This subsample of sibling pairs is used in the current analysis. During wave I in-home interviews, all respondents who lived with an identical twin (monozygotic [MZ]), a fraternal twin (dizygotic [DZ]), a half-sibling, or a step-sibling were identified, and their
Table 1. Levels of Genetic Relatedness for Add Health Pairs

<table>
<thead>
<tr>
<th>Pair Type (Percent Genetic Similarity)</th>
<th>Individuals (Pairs)</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>MZ twin (1.00)</td>
<td>578 (289)</td>
<td>12.65</td>
</tr>
<tr>
<td>DZ twin/full sibling (.50)</td>
<td>2,972 (1,486)</td>
<td>65.06</td>
</tr>
<tr>
<td>Half-sibling (.25)</td>
<td>750 (375)</td>
<td>16.42</td>
</tr>
<tr>
<td>Cousin (.125)</td>
<td>268 (134)</td>
<td>5.87</td>
</tr>
<tr>
<td>Total</td>
<td>4,568 (2,284)</td>
<td>100.00</td>
</tr>
</tbody>
</table>

**ABBREVIATIONS:** DZ = dizygotic; MZ = monozygotic.

A sibling was automatically included in the study. Additionally, full siblings were included in the sample, but they were selected probabilistically. In all, information from more than 3,000 sibling pairs was collected. The sibling-pair subsample was compiled in a fashion that allowed more than one pair of siblings to be included per household. This meant that more than two siblings living in the same household were sometimes interviewed. To eliminate any possible biases, the current study restricted the sample to two children per household. This process of excluding certain siblings from the sample was carried out using a semistructured selection method where all MZ and DZ twins were selected with certainty. In other words, if a household had three respondents and two of them were DZ twins, then the DZ twins were automatically included and the third sibling was removed. Full siblings, half-siblings, and cousins were chosen at a rate that would ensure sample sizes large enough to perform statistical analyses. Table 1 presents a breakdown of the sample that will be used for the current study.

MEASURES

*Life-Course Persistent Offenders*

Because of the difficulty of properly identifying LCP offenders (DeLisi, 2005), we elected to use two different approaches for classifying respondents as an LCP offender. For both approaches, it was first necessary to create a scale of each respondent’s involvement in delinquency at waves I, II, and III. During wave I interviews, respondents were asked to indicate the extent to which they had been involved in 17 different delinquent activities during the past 12 months. Specifically, respondents were asked how often they had painted graffiti, damaged property, lied to their parents, stolen from a store, gotten into a serious fight, hurt someone badly enough to require medical attention, run away from home, stolen a car, stolen something worth more than $50, broken into a house, committed an armed robbery, sold drugs, stolen something worth less than $50, taken part in a group fight, acted loud or unruly in a public place, carried a weapon to school, and used a weapon in a fight (responses were coded as 0 = never, 1 = one or two times, 2 = three or four times, and 3 = five or more times).
The wave I scale was created by summing each respondent’s answers to these 17 questions ($\alpha = .85$). These same questions were asked at wave II, allowing for the calculation of a wave II delinquency scale by summing across the 17 items ($\alpha = .82$).

During wave III interviews, respondents were asked about the frequency with which they had engaged in 12 different criminal behaviors during the past 12 months. Specifically, respondents were asked to indicate how often they had deliberately damaged property, stolen something worth less than $50, stolen something worth more than $50, broken into a house, committed an armed robbery, sold drugs, taken part in a group fight, bought or sold stolen property, committed identity theft, written a bad check, used a weapon in a fight, and carried a gun to school or work (responses were coded as $0$ = never, $1$ = one or two times, $2$ = three or four times, and $3$ = five or more times). As before, each respondent’s answers to the 12 questions were summed together to create the wave III delinquency scale ($\alpha = .69$).

The first approach for identifying LCPs (labeled as “LCP offending pattern (top 20 percent)” in the tables) began by creating a variable in which $1$ was assigned to all respondents who scored a $1$ or higher on each of the three delinquency scales. Respondents who did not score a $1$ or higher on all three delinquency scales were assigned a value of $0$. For example, a respondent who indicated delinquent involvement at wave I and wave II but not at wave III was coded as $0$. Respondents with a missing value on any of the three delinquency scales were assigned a missing value and thus excluded from the analyses. As is shown in table 2, this approach led to roughly 19 percent of the sample being identified as LCP offenders.

The second approach for identifying LCP offenders (labeled as “LCP offending pattern (top 10 percent)” in the tables) used the same delinquency
scales outlined previously. The only difference was that respondents were only given a 1 on the LCP offender variable if they scored a 2 or higher on each of the three delinquency scales.\(^1\) Respondents who did not score a 2 or higher on all three delinquency scales were assigned a value of 0, and respondents with a missing value on any of the three delinquency scales were assigned a missing value. This second approach identified a much smaller proportion of the sample as LCP offenders (\(\sim 9\) percent). Both LCP offender variables are used in the following analyses.

**Adolescence-Limited Offenders**

To identify respondents who offended only during their adolescent years, a three-step process was used. First, respondents who scored a value of 1 or higher on the wave I delinquency scale or on the wave II delinquency scale and scored a 0 on the wave III delinquency scale were assigned a value of 1 for the AL offender variable. Second, respondents who had a 0 at wave I and wave II but a nonzero score at wave III, a 0 on all three scales, or a 1 on all three scales were coded as 0. Third, respondents with missing values on the wave I, wave II, or the wave III delinquency scales were deleted from the final analytical sample.

**Abstainers**

Respondents were identified as abstainers if they reported no involvement in delinquency across all three waves. In other words, a respondent who scored a 0 on the wave I delinquency scale, a 0 on the wave II delinquency scale, and a 0 on the wave III delinquency scale was assigned a 1 on the abstainer variable. Respondents who reported at least one delinquent act at wave I, wave II, or wave III, were assigned a 0 on the abstainer variable. Respondents who were missing data for any of the three delinquency scales were assigned a missing value for the abstainer variable.\(^2\)

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1. Note that a 2 could mean that the respondent chose “1” (meaning the respondent committed the act in question one or two times) on two different questions or that the respondent chose “2” (meaning the respondent committed the act in question three or four times) on a single question.

2. It is important to note that the three offending patterns (i.e., LCP, AL, and abstainer) are mutually exclusive, but not all respondents fit into one of the three groups. For example, some respondents offended at waves II and III, but not at wave I, whereas other respondents offended only at wave III. These cases do not fall into any of the three offending patterns described previously and, therefore, only appear in the reference category for each group. It is for this reason that the total percentage of respondents identified as LCP, AL, and abstainer do not add up to 100 percent. Also note that some respondents were assigned a 1 for both LCP offending variables (i.e., some respondents appear in “LCP offending pattern (top 20 percent)” and in “LCP offending pattern (top 10 percent)”).
ANALYSIS PLAN

The analysis proceeded in two stages. The first stage determined whether genetic factors influence the different offending patterns by considering the following statistics: the cross-sibling odds ratio, the cross-sibling concordance rate, and the cross-sibling tetrachoric correlation. The term “cross-sibling” indicates that each respondent is being compared with his or her sibling. To estimate these statistics, the data were formatted so that each row corresponded to a sibling pair.

The cross-sibling odds ratio is a statistic that indicates the odds of sibling 1 having a 1 on the target variable if sibling 2 has a 1 on that same variable. To estimate this statistic, a logistic regression model is employed (Cho et al., 2006). The logistic model takes the following form:

\[
\log_e \left[ \frac{P(K_1)}{1 - P(K_1)} \right] = b_0 + \exp[b_1(K_2)]
\]

where \(K_1\) refers to sibling 1’s value on the target variable and \(K_2\) refers to sibling 2’s value on the same variable (respondents were randomly assigned to appear as sibling 1 or sibling 2). As is shown, the coefficient \(b_1\) represents the estimated odds that sibling 1 will have a 1 on the target variable if sibling 2 has a 1 on that same variable.

The cross-sibling concordance rate is a statistic that represents the risk of sibling 1 having a 1 on the outcome variable if sibling 2 has a 1 on that same variable. The cross-sibling concordance rate is estimated by applying the following formula (Plomin, 1990a):

\[
\text{Cross–sibling Concordance} = \frac{2C}{2C + D}
\]

where \(C\) represents the number of concordant pairs (i.e., both siblings have a 1 on the outcome) and \(D\) denotes the number of discordant pairs (i.e., pairs where only one member has a 1 on the outcome). The number of concordant pairs is multiplied by two and divided by the sum of the number of concordant pairs (multiplied by two) and the number of discordant pairs.

Finally, the cross-sibling tetrachoric correlation coefficient is a measure of the degree to which family members are concordant on an either/or trait (i.e., a dichotomous variable). The tetrachoric correlation incorporates more information than the cross-sibling concordance rate. The cross-sibling concordance rate, for example, only incorporates information from pairs in which at least one sibling is affected (Plomin, 1990a). Instead of limiting the focus in this way, the tetrachoric correlation accounts for population incidence. In other words, the tetrachoric correlation uses information from all cases in the data set. For this reason, the tetrachoric correlation is a useful statistic for behavioral geneticists examining dichotomous variables (Lyons et al., 1995).
If genetic factors influence any of the three offending patterns, then the cascade of sibling similarity for the cross-sibling odds ratio, concordance rate, and tetrachoric correlation will reveal that MZ twins are more similar to one another than DZ twins and full siblings, DZ twins and full siblings are more similar to one another than half-siblings, and half-siblings are more similar to one another than cousins (i.e., MZ > DZ and full siblings > half-siblings > cousins).

None of the aforementioned statistics, however, provide an estimate of the degree to which genetic factors influence the different outcomes. Because it is unlikely that any of the three offending patterns are completely explained by genetic factors, it is informative to consider the relative influence of genetic and environmental factors simultaneously. This leads to the second stage of the analysis. To analyze the proportion of variance in the etiology of each offending pattern that is attributable to genetic factors, it is necessary to estimate an ACE decomposition model. The ACE model provides estimates of the amount of variance in each offending pattern that is explained by genetic factors ($h^2$), shared environmental factors ($c^2$), and nonshared environmental factors ($e^2$). The ACE model is presented in figure 1.

Several pieces of information are crucial to understanding how the ACE model produces estimates of $h^2$, $c^2$, and $e^2$. First, note that the A corresponds to $h^2$ (genetic factors), the C corresponds to $c^2$ (shared environmental factors), and the E corresponds to $e^2$ (nonshared environmental factors and measurement error). Second, the model contains two observed
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indicators (i.e., an observation for sibling 1 and an observation for sibling 2). Thus, the ACE model includes information from both sibling 1 and sibling 2 on the same measure. Third, A, C, and E are estimated as latent factors that cumulatively explain 100 percent of the variance in the variable of interest. The path coefficients leading from these latent factors (i.e., a1, c1, e1, a2, c2, and e2) to the observed variables will provide estimates of h², c², and e². Fourth, note that the A factors have a correlation (i.e., the double-headed arrow) that can vary from .125 to 1.00. The value for the correlation between the A factors corresponds to the level of genetic relatedness between the two siblings providing data (see table 1 for the levels of genetic relatedness). Thus, when MZ twins are being observed, the correlation is set to 1.00; when DZ twins are being observed, the correlation is set to .50; and so on. Fifth, the C factors are also correlated, but this correlation is always set to 1.00. The C factor captures the variance that is a result of shared environmental influences. Because, by definition, shared environments are always identical between two siblings, the correlation is set to 1.00 for all sibling pairs. Finally, the E factors are left free to vary. By definition, nonshared environmental factors are not shared between two siblings; thus, no correlation exists between two siblings on nonshared environmental influences.

When analyzing the ACE model, researchers typically estimate several hierarchical (or constrained) models and compare model fit statistics. Specifically, the ACE model is estimated first, an AE model is estimated next, followed by a CE model, and finally an E model. A chi-square difference test indicates whether any of the constrained models produced an equivalent fit (i.e., the chi-square difference test reveals a nonsignificant difference of fit). If a constrained model produces equivalent fit, then that model is chosen over the ACE model. In the analyses that follow, the best-fitting model is always presented. The statistical package, Mx, was used to perform all of the ACE model-fitting analyses.

FINDINGS

Table 3 presents the cross-sibling odds ratio, the cross-sibling concordance rate, and the cross-sibling tetrachoric correlation statistics. Each statistic is presented separately according to the type of sibling pair under consideration. Genetic influences are evident to the extent that the estimates increase as a function of genetic similarity. A review of table 3 reveals that this pattern of findings emerges, with a few exceptions, across all three statistics for all of the offending patterns. The exceptions concern the similarity of cousins and half-siblings. Counter to what would be expected if genetic influences were the only operative influence, cousins resembled each other more closely than half-siblings on one of the LCP
### Table 3. Cross-Sibling Odds Ratios, Concordance Rates, and Tetrachoric Correlations for the Different Offending Patterns

<table>
<thead>
<tr>
<th>Offending Pattern</th>
<th>LCP Offending Pattern (Top 20 Percent)</th>
<th>LCP Offending Pattern (Top 10 Percent)</th>
<th>AL Offending Pattern</th>
<th>Abstainer Offending Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cross-sibling odds ratio</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MZ</td>
<td>5.36*</td>
<td>23.06*</td>
<td>3.20*</td>
<td>4.98*</td>
</tr>
<tr>
<td>95 percent CI</td>
<td>(2.15–13.38)</td>
<td>(5.27–100.88)</td>
<td>(1.67–6.12)</td>
<td>(1.64–15.13)</td>
</tr>
<tr>
<td>DZ/FS</td>
<td>2.38*</td>
<td>2.49*</td>
<td>1.50*</td>
<td>3.22*</td>
</tr>
<tr>
<td>95 percent CI</td>
<td>(1.63–3.48)</td>
<td>(1.30–4.76)</td>
<td>(1.14–1.97)</td>
<td>(2.01–5.14)</td>
</tr>
<tr>
<td>HS</td>
<td>1.55</td>
<td>4.10*</td>
<td>1.11</td>
<td>.65</td>
</tr>
<tr>
<td>95 percent CI</td>
<td>(.76–3.19)</td>
<td>(1.48–11.37)</td>
<td>(.62–2.00)</td>
<td>(.08–5.20)</td>
</tr>
<tr>
<td>Cousin</td>
<td>4.13*</td>
<td>3.80</td>
<td>2.01</td>
<td>—a</td>
</tr>
<tr>
<td>95 percent CI</td>
<td>(1.13–15.12)</td>
<td>(.33–43.63)</td>
<td>(.69–5.83)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cross-sibling Concordance Rate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MZ</td>
<td>22/56 (.39)</td>
<td>10/23 (.43)</td>
<td>182/243 (.75)</td>
<td>12/39 (.31)</td>
</tr>
<tr>
<td>DZ/FS</td>
<td>104/346 (.30)</td>
<td>26/157 (.17)</td>
<td>806/1,203 (.67)</td>
<td>62/233 (.27)</td>
</tr>
<tr>
<td>HS</td>
<td>30/97 (.31)</td>
<td>14/47 (.30)</td>
<td>150/242 (.62)</td>
<td>2/34 (.06)</td>
</tr>
<tr>
<td>Cousin</td>
<td>12/28 (.43)</td>
<td>2/10 (.20)</td>
<td>62/87 (.71)</td>
<td>0/8 (.00)</td>
</tr>
<tr>
<td></td>
<td>Cross-sibling Tetrachoric Correlation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MZ</td>
<td>.51*</td>
<td>.74*</td>
<td>.42*</td>
<td>.46*</td>
</tr>
<tr>
<td>DZ/FS</td>
<td>.28*</td>
<td>.24*</td>
<td>.15*</td>
<td>.35*</td>
</tr>
<tr>
<td>HS</td>
<td>.15</td>
<td>.42*</td>
<td>.04</td>
<td>−.10</td>
</tr>
<tr>
<td>Cousin</td>
<td>.47</td>
<td>.35</td>
<td>.26</td>
<td>−1.00</td>
</tr>
</tbody>
</table>

**ABBREVIATIONS**: CI = confidence interval; DZ = dizygotic; FS = full sibling; HS = half-sibling; MZ = monozygotic.

*a* Model could not produce a solution because of the perfect prediction of the outcome.

*p < .05* (two-tailed).

offending patterns (top 20 percent) and on the AL offending pattern. Note, however, that in only one of these instances (the cross-sibling odds ratio for cousins on the top 20 percent LCP offending pattern) was the cousins statistic significantly different from zero. Also, when analyzing the second LCP offending pattern (top 10 percent), half-siblings and cousins were more similar to one another than were DZ/full siblings.³

The statistics presented in table 3 indicate that genetic factors likely underlie each of the different offending patterns. MZ twins resembled one another more closely than did DZ twins and full siblings, which suggests

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³. An anonymous reviewer pointed out that a discrepancy existed between the pattern of results gleaned from the concordance rates and those gleaned from the tetrachoric correlations. Specifically, the concordance rates suggest a more substantial environmental influence as compared with the tetrachoric correlations. The differences between these two statistics are most likely rooted in the fact that tetrachoric correlations take into account population incidence, whereas concordance rates are limited to twin pairs where at least one twin is “affected.”
that genetic influences explain a portion of the variance in each of the three offending patterns. As was noted, however, cousins who lived in the same household tended to resemble one another more closely than half-siblings who lived together. This finding suggests that environmental influences are also important in explaining variance in the different offending patterns.

Although the cross-sibling odds ratio, concordance rate, and tetrachoric correlation are informative, the degree to which genetic and environmental influences explain variance in the etiology of the different offending patterns cannot be gleaned from these analyses. To garner these estimates, the ACE model was estimated, and the results can be found in table 4. As is shown, genetic factors accounted for 56 percent of the variance in the first measure of LCP offending (top 20 percent), whereas the nonshared environment accounted for the remaining portion of the variance ($e^2 = .44$). The shared environment failed to explain any of the variance. As for the second measure of LCP offending (top 10 percent), genetic factors accounted for the largest portion of the variance (70 percent). As before, the nonshared environment accounted for the remainder of the variance and the shared environment accounted for none of the variance. Variance in the AL offending pattern also was explained by a combination of genetic and nonshared environmental influences. Specifically, genetic factors accounted for 35 percent of the variance, nonshared environmental factors accounted for the remaining 65 percent, and shared environmental influences explained none of the variance. Finally, genetic factors accounted

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4. The ACE models were fit to the raw data using the threshold model suggested by Neale and Maes (2004).
for 56 percent of the variance in the abstainer offending pattern. The remaining 44 percent was attributable to nonshared environmental influences. Again, the shared environment did not explain any of the variance.

A coefficient comparison test was conducted to determine whether the heritability estimates presented in table 4 significantly differed across the different offending groups. The AL offender group was used as the reference category because Moffitt’s (1993) theory suggests this group will be least influenced by genetic factors (see also Raine, 1993). Thus, the heritability estimate for the two LCP groups (top 20 percent and top 10 percent) and the abstainer group were compared with the heritability estimate for the AL group. The p value for the coefficient difference tests are presented in the last column of table 4. As is shown, when the heritability estimate for the LCP (top 20 percent) group was compared with the heritability estimate for the AL group, the difference was statistically significant ($z = 1.88$), meaning that the heritability estimate for the LCP (top 20 percent) group is significantly higher than the heritability estimate for the AL group. When the heritability estimate for the LCP (top 10 percent) group was compared with the heritability estimate for the AL group, the difference was statistically significant ($z = 2.93$). Finally, when the heritability estimate for the abstainer group was compared with the heritability estimate for the

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5. An anonymous reviewer suggested that we conduct a coefficient comparison test. In response, the test suggested by Paternoster et al. (1998) was carried out. The test can be expressed algebraically as follows:

$$z = \frac{b_1 - b_2}{\text{SE}b_1 + \text{SE}b_2}$$

where the coefficient estimates (i.e., $b$) are the heritability estimates presented in table 4 and the standard errors (i.e., $\text{SE}$) are the standard errors of the heritability estimates. To get the standard error of the heritability estimate for each offending pattern, we first subtracted the heritability estimate from the upper bound of the 95 percent confidence interval (CI). Next, we divided the difference between the heritability estimate and the upper bound estimate by 1.96 (the pattern of results were substantively identical when the lower bound estimate was used to generate the standard error). It is important to note that this method of retrieving the standard error, although suitable for standard regression techniques, might not be suitable for the current purposes. To be specific, $Mx$ does not generate typical standard error statistics. Instead, $Mx$ estimates 95 percent CIs based on an optimization algorithm (Neale and Miller, 1997). These likelihood-based confidence intervals have several desirable properties that are better suited to behavioral genetic modeling as compared with the typical method for estimating standard errors. For example, standard errors assume a normal distribution of the parameter estimate. This assumption, however, might not hold for behavioral genetic modeling strategies (Neale and Miller, 1997). Thus, the estimate for the standard error that was used to perform these tests might not be directly translated to a normal standard error produced by a regression-based procedure. In light of these issues, the results of the coefficient comparison tests should be interpreted with caution.
AL group, the difference was statistically significant ($z = 1.82$). In short, the heritability estimates for both LCP offending patterns (top 20 percent and top 10 percent) were significantly higher than the heritability estimate for the AL group. The heritability estimate gleaned for the abstainer group was also higher than the estimate for the AL group. Note, however, that certain limitations might preclude any firm conclusions being drawn from the results of these tests (see footnote 5).

**DISCUSSION**

Moffitt’s (1993) developmental taxonomy has spurred a large body of research, most of which has focused on identifying environmental risk factors for LCP offending and AL offending (Moffitt, 2006). As a result, the role that genetics play in the etiologies of different offending patterns has been overlooked. This study tested the hypothesis that genetic factors can explain variance in membership in two different groups of LCP offenders, in a group of AL offenders, and in a group of delinquency abstainers. The results were consistent with the hypothesis. Specifically, genetic factors played a role in the etiology of the two LCP offending groups, in the AL offending group, and in the group of abstainers. The amount of variance explained by genetic factors, however, was not uniform across the different offending groups, suggesting that the causal processes vary across offending patterns. This latter point begs more attention.

Genetic influences accounted for a larger proportion of the variance for the LCP offender groups (both the top 20 percent grouping and the top 10 percent grouping) as compared with the AL offender group (but see footnotes 5 and 6). The proportion of variance explained by genetic factors for abstaining behaviors was also larger than the proportion of variance explained by genetic factors for AL offending. These findings are in line with Moffitt’s (1993) theory and suggest that genetic factors might underlie some of the “risk factors” for LCPs, whereas genetic factors might underlie some of the “protective factors” for abstainers (see also Belsky and Beaver, 2011, for a discussion of “plasticity alleles,” or the idea that genetic markers can influence susceptibility to both positive and negative outcomes). These

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6. An alternative strategy for testing the equality of the heritability estimates was to analyze the different groups together in a “full” model and equate the heritability estimates. Next, models where the parameter estimates are free to vary are estimated. The final step is to observe/compare model fit statistics across the different specifications. We explored these analyses and the pattern of results was consistent with those presented in the text (i.e., the results from the Paternoster et al. [1998] test). In other words, when the heritability estimates were equated with one another, the model fit statistics indicated a statistically significant worsening of fit, suggesting that the heritability estimates differ significantly across the different groupings.
findings also are consistent with Raine’s (1993) hypothesis that genetic factors have less of an influence on AL offending as compared with LCP offending. It bears repeating, however, that future research into this area is still necessary.

Recall that Moffitt (1993) pointed primarily to environmental influences in her explanation of the etiology of AL offending. For example, she noted the importance of peers and of the maturity gap. Although self-selection undoubtedly occurs (Gottfredson and Hirschi, 1990), the influence of delinquent peers might appear in behavioral genetic analyses as an environmental factor (Harris, 1998). The maturity gap also entails environmental influences; it reflects a disjuncture between biological maturity and social maturity (i.e., privileges allotted by society, parents, teachers, etc.). Thus, maturity gap influences might be partially captured by environmental factors in a behavioral genetic analysis of AL offending. Also, the sheer fact that delinquency is statistically normative during the adolescent years (Farrington, 1983) dovetails with the finding that AL offending is influenced more by environmental factors than genetics.

One possible explanation for the finding that genetic factors explained more variance for LCP offending patterns than for the AL offending pattern can be found in the literature linking neuropsychological deficits with LCP offending. It is useful to think of a neuropsychological deficit as any influence that undermines normal brain functioning (Moffitt, 1993). Certainly, environmental influences affect the presence of neuropsychological dysfunction. Notable examples are prenatal exposure to nicotine, alcohol, or drugs (McGloin, Pratt, and Piquero, 2006). Under normal conditions, however, brain formation and function is largely scripted by genetic factors (Raine, 2008). Indeed, current estimates suggest that 60 percent of the human genome codes for brain structure and functioning (Pinker, 2002; Rutter, 2006). This leads to the possibility that many neuropsychological deficits are controlled by genetic influences. Because Moffitt (1993) centered much of her discussion of LCP offenders around the effects of neuropsychological deficits, it is not surprising that genetic factors explained more than 50 percent of the variance in the etiology of this group.

The absence of a shared environmental effect for all of the offending patterns is notable. The shared environment captures any environmental influence that makes siblings more alike. Sources of shared environmental influence are often described as exposure to poverty, neighborhood conditions, and similarity in parental treatment. This finding is, however, consistent with a large line of research that suggests the shared environment is of little consequence when considering personality development (Harris, 1998) and involvement in antisocial behavior (Moffitt, 2005). Also, research has revealed that the effects of the shared environment fade over time (Ferguson, 2010). In other words, it seems that the shared environment
might have an influence early in the life course (e.g., during early childhood), but this influence becomes overshadowed by genetic and nonshared environmental factors as the individual ages and nonshared events begin to accumulate (Harris, 2006). In short, the finding of a negligible shared environmental effect underscores the importance of differentiating shared and nonshared environmental influences in criminological research.

Indeed, the nonshared environment explained a significant proportion of the variance for each offending pattern. Interpreting this effect requires an elaboration of the factors that are considered a nonshared environmental influence. By definition, the nonshared environment captures any influence that is nongenetic in origin and that differs between siblings. Siblings often associate with different peer groups, they have different teachers, and they might even be exposed to different parenting strategies (Harris, 1998). These influences, to the extent that they affect one’s offending pattern, will be captured by the nonshared environment. Because nonshared environmental influences were the only operative environmental effects identified by this analysis, future research should seek to identify the factors that differ between siblings that can account for between-sibling differences in offending patterns (see, for example, Beaver, 2008).

Limitations to the current study must be noted. First, the measurement strategy employed to identify the different offending patterns made use of data that were collected in adolescence and early adulthood. Ideally, information from childhood, adolescence, and late adulthood would have been incorporated to help make more accurate assessments about membership in the different offending groups. Unfortunately, the Add Health data do not include assessments at all of these different sections of the life course. Future research should attempt to replicate our analysis with samples that span a longer period of time.

Second, the results from the current study might not be generalizable to singletons (Falbo and Polit, 1986). This limitation is not specific to the current study, however. To be sure, it is unclear whether and to what extent the results gleaned from behavioral genetic research can inform scholars about the processes underlying personality development for children who do not live with siblings. A third limitation is that heritability coefficients might be inflated as a function of gene–environment (GxE) interactions (e.g., Caspi et al., 2002). Specifically, to the extent that genetic factors interact with shared environmental influences, the heritability coefficient will be inflated. It must be noted, however, that heritability coefficients can be deflated as a function of GxE interactions when the environmental influence is a nonshared environment (Purcell, 2002). When a GxE interaction occurs with a nonshared environmental influence, the result is that the nonshared environmental estimate is inflated, and thus, the heritability estimate is deflated. Along these lines, it is possible—but outside the scope of the current study—that the various offender types are differentially affected by GxE.
interactions. Indeed, it is consistent with Moffitt’s (1993) theory to expect such a finding; recall that LCP offenders suffer from neuropsychological deficits and adverse rearing environments.

With the recent mapping of the human genome, researchers are beginning to pull back the “heritability curtain” and identify links between measured genes and phenotypic outcomes. This line of research—referred to as molecular genetics—has already produced a wealth of knowledge. Certain genetic polymorphisms have been linked to well-known predictors of antisocial behaviors such as attention deficit hyperactivity disorder (Faraone et al., 2001) and conduct disorder (Caspi et al., 2002). The current study, however, does not allow for the direct identification of which genes are involved in the etiology of offending groups. Instead, the current study represents a first step toward understanding the genetic and environmental factors that underlie different offending patterns. Building on the current work, future research would benefit by examining whether certain genotypes can differentiate LCP offenders, AL offenders, and abstainers (Boutwell and Beaver, 2008).

In closing, we turn our attention to the theoretical implications that stem from this study. In the nearly 20 years that have passed since Moffitt (1993) penned her theory, remarkable advances have been made in both criminological and behavioral genetic research. Theorists have begun to highlight the ways in which these two lines of research might be integrated, and the current study evinces how Moffitt’s (1993) theory might benefit from such an interdisciplinary focus. It is our hope that integrating behavioral genetic findings into mainstream criminological theories will elucidate the factors, both genetic and environmental, that ultimately lead to antisocial behavior (Rutter, 2006).

REFERENCES


Plomin, Robert, Linzy Hill, Ian W. Craig, Peter McGuffin, Shaun Purcell, Pak Sham, David Lubinski, Lee A. Thompson, Paul J. Fisher, Dragana


J.C. Barnes is an assistant professor in the Criminology program at The University of Texas at Dallas. His primary research interests focus on the biosocial underpinnings to antisocial, aggressive, and violent behaviors. He is also interested in research that blends behavioral genetic modeling with mainstream criminological theory. Recent works can be found in *Aggressive Behavior, Criminal Justice and Behavior*, and *Journal of Criminal Justice*.

Kevin M. Beaver is an associate professor in the College of Criminology and Criminal Justice at Florida State University. He is the past recipient of the American Society of Criminology’s Ruth Shonle Cavan Young Scholar Award and the National Institute of Justice’s Graduate Research Fellowship. His research focuses on the genetic basis to behavioral phenotypes, and he has been published in a range of journals, including *Addiction, Biological Psychiatry, Intelligence, Journal of Adolescent Health, Journal of Child Psychology and Psychiatry, Journal of Criminal Justice, International Journal of Environmental Health Research, Criminal Justice and Behavior, and Criminal Behaviour and Mental Health*, among others.

Brian B. Boutwell received his PhD from Florida State University in 2010 and is currently an assistant professor in the College of Criminal Justice at Sam Houston State University. His research interests include behavioral genetics, gene–environment interactions, evolutionary psychology, and life-course criminology. His work has appeared in such journals as *Criminology, Journal of Research in Crime and Delinquency, Journal of Criminal Justice, International Journal of Environmental Health Research, Criminal Justice and Behavior, and Criminal Behaviour and Mental Health*, among others.