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On the relationship of past to future involvement in crime and delinquency: A behavior genetic analysis☆,☆☆

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ABSTRACT

Purpose: Criminologists have devoted much attention to identifying the factors that drive stability in antisocial behavior. This body of research has, however, overlooked the contributions of behavior genetic research. This study sought to blend behavior genetics with the different perspectives used by criminologists to explain stability.
Methods: Employing a behavioral genetic research design, the current study analyzed the correlation between adolescent and adulthood crime (a 13 year time span was covered between the two time points) among a sample of sibling pairs drawn from the National Longitudinal Study of Adolescent Health (Add Health).
Results: The findings revealed that genetic factors accounted for nearly all of the stability in offending behavior from adolescence to adulthood. Environmental factors (particularly, of the nonshared variety) accounted for the majority of the changes in offending.
Conclusions: The implications of these results for criminological research and theory are discussed.

Introduction

Biosocial criminology is an emerging paradigm that holds considerable promise for increasing scholars' understanding of the origins of antisocial behavior (DeLisi & Piquero, 2011; Piquero, 2011). Broadly, biosocial criminology seeks to blend biological, genetic, sociological, and environmental explanations of human behavior into a single analytic focus. This body of literature has shown that genetic and biological factors are significant influences on the development of maladaptive traits (Moffitt, 2005; Raine, 1993). Results from four recent meta-analyses, for example, suggest that about 50 percent of the variance in antisocial behaviors is attributable to genetic factors (Ferguson, 2010; Mason & Frick, 1994; Miles & Carey, 1997; Rhee & Waldman, 2002). The remaining 50 percent is divided among shared environmental influences (i.e., environments that operate to make siblings more similar to one another) and nonshared environmental influences (i.e., environments that operate to make siblings different from one another).

Despite the vast literature, biosocial criminology lacks a unified theoretical framework. To date, there is no single theory that incorporates all of the findings from biosocial research into a succinct set of propositions and theoretical axioms. This does not mean that theorists have not proffered biosocial theories. To be sure, there are a number of theories that incorporate biosocial arguments into their original hypotheses (Barnes, Beaver, & Boutwell, 2011). Notable examples are the theories set forth by Ellis (2005), Moffitt (1993), and Robinson (2004; Robinson & Beaver, 2010). Given the wealth of criminological theorizing (Lilly, Cullen, & Ball, 2011), however, some scholars have argued that extant theories should be revamped to incorporate evidence from biosocial research (Rowe & Osgood, 1984). Along these lines, Walsh (2002) showed that biosocial inquiry may allow researchers to fill in some of the gaps left by contemporary criminological research.

One remaining gap, for example, concerns the identification of the various factors underlying stability in antisocial behavior over the life course. Indeed, a great deal of theorizing (Gottfredson & Hirschi, 1990; Sampson & Laub, 1993; Wilson & Herrnstein, 1985) and empirical work (see below) has been extended to explain the well-known finding that past behavior is one of the best predictors of future behavior (Robins, 1966). The state dependence argument suggests that past criminality increases the probability of future offending due to the effects/outcomes of past behavior (e.g., cumulative continuity). Sampson and Laub (1993) argued that prior involvement in crime and delinquency

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causes future involvement in crime and delinquency because of opportunities that are lost as a consequence of past behavior. In short, opportunities for a prosocial lifestyle are knifed-off due to earlier delinquent activity; the result being that future delinquency becomes more likely.

Population heterogeneity, conversely, argues that an individual's unique propensity toward offending (i.e., their level of criminality) accounts for behavioral stability. Gottfredson and Hirschi's (1990) theory of low self-control offers a primary example of the population heterogeneity perspective. These authors explained that persons will differ in their level of delinquent behavior as a result of their different levels of self-control. Individuals who have lower levels of self-control will be more likely to offend as compared to individuals who have higher levels of self-control. According to Gottfredson and Hirschi (1990), levels of self-control remain relatively stable over time (at least after adolescence; Hay & Forrest, 2006) and, therefore, account for the correlation between past and future involvement in delinquency.

Despite numerous empirical tests, scholars remain divided in their interpretations of the influences that drive behavioral stability (Nagin & Paternoster, 2000). As is discussed shortly, this body of research has overlooked the possible contributions from biosocial research. Behavior genetics is *one* area of biosocial criminology that offers a unique opportunity to analyze the genetic and environmental influences on behavioral stability. The goal of this paper, therefore, is to analyze stability in antisocial behavior through the lens of behavior genetics. The following sections review the relevant literature bearing on the stability of antisocial behavior. Attention is first given to evidence gleaned from criminological studies. Second, relevant findings produced by behavior genetic research are presented.

Findings from criminological research

Criminologists have long been interested in the stability of criminal behavior over different periods of the life course (Glueck & Glueck, 1950; Loeber, 1982; Robins, 1966; West & Farrington, 1973; White, Moffitt, Earls, Robins, & Silva, 1990). In two early examples, Olweus (1979) equated the stability of aggression with that of intelligence, while Robins (1978:611) famously concluded that "adult antisocial behavior virtually requires childhood antisocial behavior." Building on this body of research, more recent investigations have sought to uncover the factors that influence behavioral stability. This has propelled scholars to both theoretically (e.g., Gottfredson & Hirschi, 1990; Sampson & Laub, 1993) and empirically examine the competing explanations of state dependence and population heterogeneity. Interestingly, there has been a wealth of evidence supporting both state dependence (Laub, Sampson, & Sweeten, 2006) and population heterogeneity (Gottfredson, 2008; Pratt & Cullen, 2000).

One set of findings indicates that state dependence processes are the primary influence on behavioral stability. In one of the first papers to pit the two arguments against one another, Nagin and Paternoster (1991) found that state dependence processes overshadowed population heterogeneity (modeled as the correlation between the error terms of time 1 delinquency and time 2 delinquency) as an explanatory influence on behavioral stability. Specifically, their probit model estimates indicated that the latent term tapping population heterogeneity was reduced to zero when an observed indicator of prior delinquency (i.e., state dependence) was entered into the model. Paternoster and Brame (1997) reached similar conclusions using a more representative sample. These authors reported that dynamic variables (such as peer relationships) were strong predictors of offending even after accounting for persistent heterogeneity.

Other studies, however, have reported evidence inconsistent with Nagin and Paternoster's (1991) conclusions (Nagin & Farrington, 1992; Nagin & Paternoster, 2000). Nagin and Farrington (1992) analyzed a sample of males from London over a 20 year period. Their analyses (similar to those performed by Nagin & Paternoster (1991)) revealed that population heterogeneity was the most powerful predictor of individual criminality. Paternoster, Brame, and Farrington (2001), drawing on data from the Cambridge Study, found evidence that variation in adult criminal convictions was the result of random processes after controlling for adolescent conviction history. In other words, state dependence processes could not explain variation in adult offending since criminal arrests were best modeled by a Poisson (i.e., random) process after adolescent deviance had been accounted for. Piquero, Brame, and Moffitt (2005) replicated these results using the Dunedin data and showed similar effects for both males and females.

Another line of research has indicated that both state dependence and population heterogeneity are necessary to explain peoples' diverse pathways to crime (Moffitt, 1993; Paternoster & Brame, 1997; Piquero & Moffitt, 2005). Moffitt's (1993) dual taxonomy identified two groups of offenders: life-course-persistent (LCP) offenders and adolescence-limited (AL) offenders. To briefly summarize, Moffitt (1993) hypothesized that LCP offenders are more likely to be influenced by population heterogeneity processes. For example, LCP offenders suffer from neuropsychological deficits and are reared in adverse home environments. The combination of these risk factors—which occur early in development—predicts antisocial behavior across the entire life course. Recently, scholars have shown that Moffitt's hypotheses about LCP offenders are consistent with a biosocial focus (Barnes et al., 2011). The AL offenders, on the other hand, are more likely to be influenced by state dependence processes. AL offenders limit their delinquent behavior to the period of adolescence (i.e., the teen years). These individuals are motivated to offend due to the coalescence of different cultural, social, and biological factors (Barnes & Beaver, 2010). With the passing of adolescence, AL offenders generally cease their involvement in crime. Moffitt notes, however, that AL offenders can sometimes be diverted to a life of crime if they are caught by a "snare" (Hussong, Curran, Moffitt, Caspi, & Carrig, 2004). The concept of a snare—often defined as a criminal conviction or a drug addiction—represents the state dependence portion of Moffitt's (1993) theory.

To summarize, an ongoing debate regarding the relative importance of state dependence and population heterogeneity abounds (Nagin & Paternoster, 2000). Some researchers have reported evidence supporting state dependence processes/theories (Nagin & Paternoster, 1991), while others have produced results supportive of a population heterogeneity explanation (Nagin & Paternoster, 2000). Importantly, however, this body of research has overlooked the potential role that genetic factors play in explaining behavioral stability.

Findings from behavior genetic research

Behavior genetic research offers a unique opportunity to examine the various factors that influence behavioral stability by decomposing the correlation between behavior at time 1 and behavior at time 2 into three components: a genetic component, a shared environmental component, and a nonshared environmental component. To the extent that genetic factors influence behavioral stability, evidence for population heterogeneity can be inferred. Prior scholars have noted that genetic influences on stability are consistent with population heterogeneity explanations (Rowe, Osgood, & Nicewander, 1990). Because a person's DNA remains unchanged throughout the life course (Carey, 2003),[1] any genetic influence on behavioral and personality stability (McGue, Bacon, & Lykken, 1993) must be taken as support for the population heterogeneity explanation.

Shared environmental influences may also be consistent with a population heterogeneity explanation of behavioral stability. Shared environments operate to make siblings more similar to one another and are typically identified as within-the-home influences such as parental rearing strategies and exposure to poverty (Harris, 1998). Recall that Gottfredson and Hirschi's (1990) theory of self-control is often cited as an example of population heterogeneity (Paternoster

& Brame, 1997; Wright, Tibbetts, & Daigle, 2008). Gottfredson and Hirschi (1990) explained that self-control is developed primarily via parental influences. Thus, a stable personality trait may be the result of early rearing environmental factors that are unalterable later in life. To the extent that shared environmental influences impact behavioral stability, therefore, population heterogeneity arguments will be supported. This viewpoint is consistent with criminological research (see for example, Paternoster et al. (2001)).

Nonshared environments are influences that make siblings different from one another, though it is not necessary to assume that nonshared environments are experienced separately for both siblings. Put another way, the exact same environmental experience can cause differences to emerge between siblings if their individual perceptions of the event are different (Torgersen & Janson, 2002). Nonshared environmental influences on behavioral stability are, thus, most consistent with the state dependence hypothesis. State dependent effects arise as a result of prior behavior and as a result of factors that are beyond the individual's control. Since exposure to life-course transitions are experiences that are not shared between siblings (e.g., siblings marry different spouses, work different jobs, are punished at different times or are punished for different behaviors), these influences *may* fall under the rubric of nonshared environments. The conditional term *may* is used here in recognition that nonshared environmental factors could also reflect population heterogeneity to the extent that gene-environment correlations play a role in nonshared environmental experiences (Purcell, 2002).

A growing body of literature has examined the impact of genetic and environmental influences on the stability of antisocial behavior over time and, in general, the findings suggest that genetic (Burt, McGue, Carter, & Iacono, 2007) and shared environmental factors are most salient (Eley, Lichtenstein, & Moffitt, 2003; Forsman, Lichtenstein, Andershed, & Larsson, 2008; Haberstick, Schmitz, Young, & Hewitt, 2006; Kendler & Prescott, 2006; Lyons et al., 1995; Malone, Taylor, Marmorstein, McGue, & Iacono, 2004; Reiss, Neiderhiser, Hetherington, & Plomin, 2000; van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003; Van Hulle et al., 2009). For instance, Lyons et al. (1995) analyzed data drawn from male twin pairs that served in the military during the Vietnam War. The authors estimated the effect of genetic and environmental factors on stability in antisocial personality disorder from adolescence to middle adulthood and found that genetic factors explained 28 percent of the stability, the shared environment explained 35 percent, and the nonshared environment explained 37 percent.

More recent efforts have revealed similar findings. Eley et al. (2003) reported that 84 percent of the stability in aggressive behavior over a five-year span (respondents were aged 8–9 at wave 1 and 13–14 at wave 2) was attributable to genetic influences. Reiss et al. (2000) found that genetic and shared environmental factors accounted for approximately 90 percent of the stability in antisocial behavior over a three-year time period for their sample of adolescents. Interestingly, however, genetic factors also explained the largest portion of variance in *changes* in antisocial behavior from time 1 to time 2. Approximately 60 percent of the changes in antisocial behavior were the result of genetic influences. Thus, it is clear that genetic factors can affect both stability and change in antisocial behavior over time.

In sum, the extant evidence indicates that both genetic and environmental factors influence behavioral stability (Forsman et al., 2008; Haberstick et al., 2006; Malone et al., 2004; van Beijsterveldt et al., 2003; Van Hulle et al., 2009). Some researchers have found that genetic and shared environmental factors combine to account for the largest portion of the variance (e.g., Eley et al., 2003; Reiss et al., 2000), lending support for population heterogeneity arguments. Others, however, have reported a statistically significant influence of nonshared environmental factors on behavioral stability (Hopwood et al., 2011; Lyons et al., 1995; Tuvblad, Raine, Zheng, & Baker, 2009). Hopwood et al. (2011) recently reported that genetic and nonshared environmental factors explained the majority of the variance in stability in negative emotionality across 12 years of development from adolescence to adulthood.

The current study

The current study seeks to integrate criminological and behavior genetic research regarding the factors that contribute to stability in antisocial behavior. Along these lines, this study informs the criminological literature in two ways. First, stability and changes in criminal/delinquent behavior among a sample of sibling pairs is analyzed. Offending behavior will be measured at two time points: first when the respondents are adolescents and again when respondents have reached adulthood (a time span of approximately 13 years is covered). This analysis will be guided by the following research questions:

Research Question 1: To what extent do genetic factors influence stability and change in antisocial behavior from adolescence to adulthood?

Research Question 2: To what extent do environmental factors influence stability and change in antisocial behavior from adolescence to adulthood?

The second way that the current study informs the criminological literature is by blending behavioral genetic modeling strategies with criminological theorizing. After estimating various statistical models to address the above research questions, a discussion regarding the relevance of the findings for criminological theory is offered.

Methods

Sample

Data for the current study were drawn from the National Longitudinal Study of Adolescent Health (Add Health; Harris, 2009). The Add Health is a nationally representative and longitudinal study of American adolescents who attended middle and high school between 1994 and 1995 (Harris et al., 2009; Kelly & Peterson, 1997). The initial round of data collection (i.e., wave 1) for the Add Health began in September 1994. During wave 1, all students who were attending 132 selected schools were asked to complete a self-report questionnaire. Individual schools were selected using stratified random sampling techniques and information from more than 90,000 students was obtained. Following the completion of data collection within schools, a subset of the original 90,000 students were asked to complete a more in-depth interview that took place in the respondents' homes. A total of 20,745 adolescents were interviewed during the in-home portion of data collection. Respondents ranged between 11 and 21 years of age at wave 1.

The second wave of data collection (i.e., wave 2) began approximately one year after the wave 1 in-home interviews were completed. Wave 2 interviews were conducted with 14,738 of the respondents who completed in-home interviews at wave 1. Between 2001 and 2002—approximately six years after wave 1 interviews were conducted—a third round of interviews took place with 15,197 respondents who completed wave 1 in-home interviews (i.e., wave 3). By this time, most of the respondents had reached young adulthood. As a result, the surveys were modified in order to include more age appropriate items for the participants. Most recently, a fourth round of interviews were conducted (i.e., wave 4) with 15,701 respondents from the wave 1 in-home portion of the study. These interviews took place between 2007 and 2008—approximately 13 years after wave 1 interviews—when the respondents were

between 24 and 34 years old. The questionnaires were similar to those used at wave 3.

A final important feature of the Add Health that bears mentioning concerns the oversampling of sibling pairs among participants during wave 1. To compile the sibling subsample, the Add Health researchers followed one general selection criterion: if a respondent identified himself or herself as a twin, a half-sibling, or a step-sibling during the first wave of interviews, their sibling was automatically added to the sample. All other sibling/relative pairs entered the sample due to chance. These selection procedures netted information from more than 3,000 sibling pairs (Rowe & Jacobson, 1998). Due to the selection procedure outlined above, the sibling subsample included a few cases where more than one set of siblings living in the same household were interviewed. In order to eliminate any possible biases from including more than one sibling pair per household, the current study restricted the sample to two children per home. The final sample consisted of 2,267 sibling pairs (289 monozygotic [MZ] twin pairs; 450 dizygotic [DZ] twin pairs; 1,036 full-sibling pairs; 358 half-sibling pairs; and 134 pairs of cousins).

Measures

Delinquency Wave 1

Respondents were asked a host of questions during the wave 1 in-home interviews that referenced delinquent behavior. Each respondent was asked to indicate whether and how often they had been involved in 17 different delinquent activities within the last year. 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 $50, taken part in a group fight, and acted loud or unruly in a public place (responses were coded: 0 = never, 1 = one or two times, 2 = three or four times, and 3 = five or more times). Two questions asked whether the respondent had carried a weapon to school and whether the respondent had used a weapon in a fight. These were coded dichotomously where 1 indicated that the event had occurred and a 0 indicated that the event had not occurred. Factor analysis indicated that a single latent construct best explained the covariance of the 17 items. As a result, the wave 1 delinquency scale was created by summing the responses to all 17 items ($\alpha = .85$). Higher values indicated a greater involvement in delinquency. Prior Add Health researchers have utilized similar delinquency scales (Boisvert, 2009).

Criminal Behavior Wave 4

During wave 4 interviews, respondents were asked 11 questions that referenced criminal activity that had occurred within the last 12 months. Specifically, respondents were asked to indicate whether and how often they had deliberately damaged property, stolen something worth less than $50, stolen something worth more than $50, broken into a house, committed an armed robbery, sold drugs, taken part in a group fight, gotten into a serious physical fight, bought or sold stolen property, committed credit card fraud, and written a bad check. Each of the 11 items were coded on a scale ranging from 0 (i.e., never) to 3 (i.e., five or more times). Factor analysis indicated that a single underlying construct best explained the structure of the items. To create the wave 4 criminal behavior scale, responses to each of the 11 items were summed together so that higher values reflected more involvement in criminal activity ($\alpha = .69$).

Analysis plan

The analysis proceeds in three steps. The first step estimates the genetic and environmental influences on wave 1 delinquency and separately on wave 4 criminal behavior. In order to do so, an analytic technique that decomposes variance into three components (a genetic component, a shared environment component, and a nonshared environment component) is employed. A structural equation method of estimation known as the ACE model (see Fig. 1) is estimated (Neale & Maes, 2004). In the ACE model, the A parameter captures genetic influences (h^2), C captures shared environmental influences (c^2), and E captures nonshared environmental influences (e^2) and measurement error. The ACE model analyzes variance in each of the outcomes and the coefficients provided by the ACE model are standardized to reflect the proportion of the variance explained by each factor. The ACE model parameters, therefore, explain 100 percent of the variance in the measure of focus.

There are several elements of the ACE model that must be noted. First, the model contains two observed variables; sibling 1's offending score and sibling 2's offending score (respondents were randomly sorted as sibling 1 or sibling 2). Behavior genetic methods compare siblings on an outcome measure to determine whether siblings who share more genetic material resemble each other more closely than siblings who share less genetic material. The simplest case is when MZ twins are compared against DZ twins. MZ twins share 100 percent of their DNA and they also share their environment. DZ twins share (on average) 50 percent of their distinguishing DNA, but they also share their environment. Based on this information, if MZ twins are more similar to one another on some outcome measure (say, delinquency) than are DZ twins, genetic influences are operative. It is for this reason that the ACE model includes information from both sibling 1 and sibling 2 on the same measure.

Second, notice that A, C, and E are estimated as latent factors that explain the variance in the measure of interest. The path coefficients leading from these latent factors to the observed measures are used to garner estimates of h^2, c^2, and e^2, respectively. The A factors have a fixed correlation that varies according to the level of genetic relatedness for the sibling type that is providing data (i.e., MZ twins = 1.00; DZ twins and full-siblings = .50; half-siblings = .25; and cousins = .125). The C factors are correlated, but their correlation is fixed at 1.00. The C factor captures the variance that is due to shared environmental influences. The E factors are free to vary since nonshared environmental influences are unique to each sibling.

The second step of the analysis will examine the extent to which genetic and environmental factors influence *stability and change* in offending behaviors from wave 1 to wave 4. A variant of the ACE model is necessary to perform these analyses. Presented in Fig. 2 is a graphical depiction of the bivariate Cholesky model that will be used at this stage of the analysis. The Cholesky model is considered bivariate because it incorporates information from two measures per sibling; in this case, wave 1 delinquency and wave 4 criminal behavior. Using information from the two measures, the Cholesky

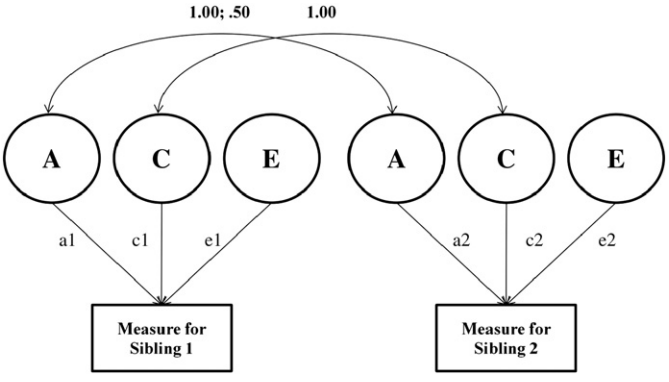

Fig. 1. The ACE Model.

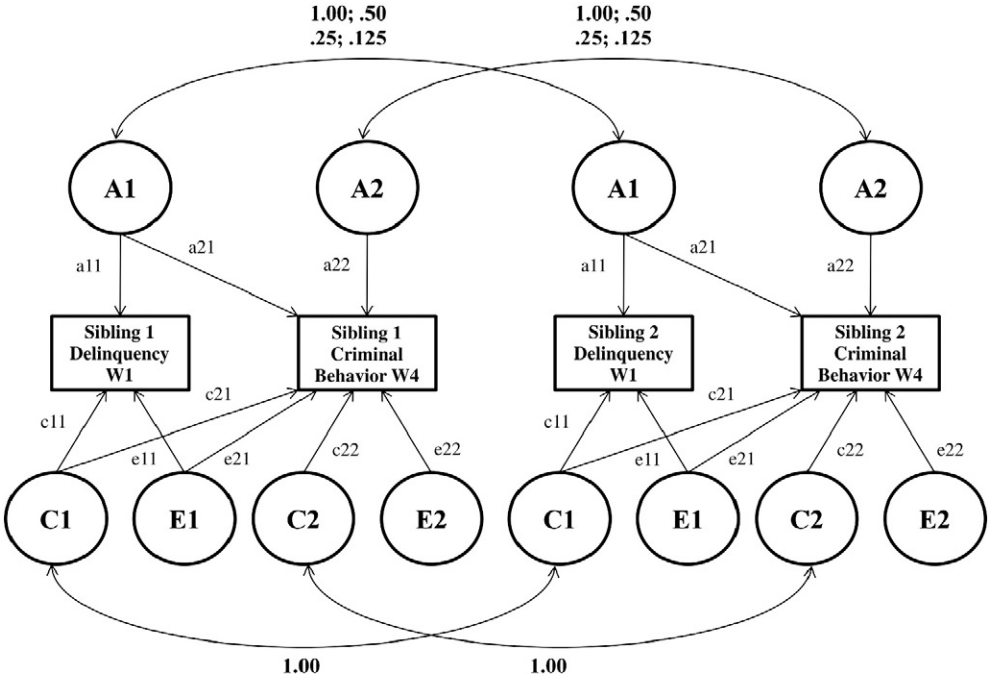

Fig. 2. The Bivariate Cholesky Model.

model will estimate the genetic and environmental influences on the *covariation* between wave 1 and wave 4 offending behaviors.

In general, the points made above about the ACE model carry-over to the Cholesky model. There is, however, one additional point that must be mentioned about the Cholesky model: there are two versions of A, C, and E. The Cholesky model incorporates estimates of A1, C1, E1, A2, C2, and E2. The difference between the factors with a *1* and those with a *2* is important. Factors A1, C1, and E1 provide estimates of the genetic and environmental influences on the *covariance* between the two measures (i.e., wave 1 delinquency and wave 4 criminal behavior). For instance, A1 provides an estimate of the amount of genetic influence that is shared between wave 1 and wave 4 criminal behavior. For the present purposes, it will be useful to think of the factors A1, C1, and E1 as providing estimates of the proportion of the stability in offending from wave 1 to wave 4 that is due to genetic (A1), shared environmental (C1), and nonshared environmental (E1) influences.

Factors A2, C2, and E2, in contrast, provide estimates of the proportion of the variance unique to wave 4 criminal behavior (i.e., variance that is not shared with wave 1 delinquency) that is due to genetic influences (A2), shared environmental influences (C2), and nonshared environmental influences (E2). The E2 factor, for example, captures the effect of nonshared environmental influences on variance that is unique to wave 4 criminal behavior. For the present purposes, it will be useful to think of these factors as providing estimates of the genetic and environmental influences on *changes* in criminal behavior.

A word about model fit is necessary. When estimating the ACE and Cholesky models, it is customary to also estimate a series of supplemental or nested 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 is conducted each time and any supplemental model that is equivalent to the ACE model (i.e., the chi square difference test reveals a non-significant difference of fit) is chosen over the ACE model. In the tables that follow, the best-fitting model is always presented. All analyses were estimated using the structural equation modeling program, *Mx*. *Mx* is a statistical program that was developed specifically to perform behavior genetic modeling (Neale & Maes, 2004).

The third and final step to the analysis will estimate the effect of wave 1 delinquency on wave 4 criminal behavior after accounting for genetic influences on the latter. This portion of the analysis will utilize the DF regression model so that a measured nonshared environmental influence can be directly estimated (Rodgers, Rowe, & Li, 1994):

$$K_1 = b_0 + b_1(K_2 - K_m) + b_2(R^*[K_2 - K_m]) + b_3(ENVDIF) + e$$

In the equation above, K_1 refers to sibling 1's score on the wave 4 criminal behavior scale, K_2 refers to sibling 2's score on the wave 4 criminal behavior scale, K_m is used to mean center sibling 2's wave 4 criminal behavior score, R is a coefficient of genetic relatedness (ranging from .125 for cousins to 1.00 for MZ twins), and $R^*[K_2 - K_m]$ is an interaction term that captures genetic influences on wave 4 criminal behavior. Importantly, *ENVDIF* is calculated by subtracting sibling 2's score on the wave 1 delinquency scale from sibling 1's score on the same measure. The regression coefficient for the *ENVDIF* variable (i.e., b_3) captures the effect of between-sibling differences in wave 1 delinquency on wave 4 criminal behavior after controlling for genetic (i.e., b_2) and shared environmental effects (i.e., b_1). Put differently, if prior involvement in delinquency (at wave 1) has a causal impact on later criminality (i.e., wave 4) after controlling for two sources of population heterogeneity (i.e., genetic factors and shared environmental factors), then the regression coefficient for the *ENVDIF* variable (i.e., b_3) will emerge as statistically significant.

Findings

Because the current analysis was motivated to unpack the stability of delinquent behavior over time, it was first important to observe the correlation between wave 1 delinquency and wave 4 criminal behaviors. The zero-order correlation between wave 1 and wave 4 criminal behavior was .21 and was statistically significant ($p<.05$, two-tailed test). The correlation indicated that respondents showed a moderate degree of stability in criminal behavior from adolescence (wave 1) to adulthood (wave 4). At the same time, however, a good deal of change occurred between the two observation periods—as evidenced by the correlation coefficient being below unity.

Table 1
Univariate ACE Model Parameter Estimates

	Genetic Factors (A)	Shared Environment (C)	Nonshared Environment (E)
Wave 1 Delinquency	.45 (.39-.51)	.00 (.00-.00)	.55 (.49-.61)
Wave 4 Criminal Behavior	.36 (.23-.49)	.00 (.00-.00)	.64 (.51-.77)

Note: 95% confidence interval in parentheses.

Table 1 presents the parameter estimates from the ACE models that analyzed the wave 1 and the wave 4 criminal behavior measures separately. The table is split into two sections with the top section presenting the estimates for wave 1 delinquency and the bottom section presenting estimates for wave 4 criminal behavior. Looking first at the parameter estimates for the wave 1 delinquency measure, the table reveals that genetic influences explained a significant portion of the variance ($h^2 = .45$). As for environmental influences, the shared environment did not have a significant impact on wave 1 delinquency ($c^2 = .00$). Finally, the nonshared environment explained the majority of the variance in wave 1 delinquency ($e^2 = .55$). Taken together, these findings indicate that genetic and nonshared environmental factors are important influences on wave 1 delinquency.

Turning to the ACE model results for the wave 4 criminal behavior variable (bottom portion of Table 1) we see a pattern of results that is similar to the wave 1 results. Specifically, genetic ($h^2 = .36$) and nonshared environmental factors ($e^2 = .64$) combined to explain all of the variance in wave 4 offending. The shared environment did not explain any of the variance in wave 4 criminal behavior ($c^2 = .00$).

Thus far, the results have indicated that genetic and nonshared environmental factors underlie delinquent behavior reported at wave 1 and criminal behavior reported at wave 4. These results are an important first step in establishing the relationship between wave 1 and wave 4 criminal behavior, but they do not reveal whether genetic and environmental factors underlie stability and changes in behavior over time. As was previously noted, there was a moderate degree of stability in delinquency from adolescence to adulthood, but there was also a high degree of change. The degree to which stability and changes in offending are influenced by genetic and environmental influences has yet to be determined.

Table 2 presents parameter estimates gleaned from the bivariate Cholesky models. Parameter estimates for the stability in criminal behavior from wave 1 to wave 4 are presented separately from parameter estimates for changes in criminal behavior from wave 1 to wave 4. Upon observation of Table 2, one point is immediately obvious: genetic factors accounted for nearly all of the variance in the stability in offending. Specifically, genetic factors accounted for 97 percent of the stability in criminal behavior between wave 1 and wave 4.[2] This means that criminal behavior that remained stable from wave 1 to wave 4 was almost completely due to genetic factors that influenced criminal behavior at both time points. In other words, the genetic factors that influenced wave 1 delinquency were also operating on wave 4 criminality.[3]

Table 2
Bivariate Cholesky Model Parameter Estimates for Stability and Change in Criminal Behavior from Wave 1 to Wave 4

	Genetic Factors (A)	Shared Environment (C)	Nonshared Environment (E)
Factors Accounting for Stability	.97 (.65-1.00)	.00 (.00-.00)	.03 (.00-.34)
Factors Accounting for Change	.36 (.24-.49)	.00 (.00-.02)	.64 (.51-.76)

Note: 95% confidence interval in parentheses.

Table 3
Results from the DF Model

	Wave 4 Criminal Behavior	
	b	*SE*
Genetic Factors	.25*	.06
Nonshared Environment - Wave 1 Delinquency	.02*	.01

*$p < .05$, two-tailed.

Table 2 also reveals an interesting pattern of findings concerning the factors that account for changes in behavior over time. Specifically, genetic influences explained a significant portion of the changes in criminal behavior ($h^2 = .36$). At the same time, nonshared environmental influences accounted for the majority of changes in criminal behavior ($e^2 = .64$).[4]

The final step of the analysis examined whether cross-sibling differences in wave 1 delinquency predicted wave 4 criminal behavior after controlling for genetic influences. The results from this analysis are presented in Table 3. After controlling for sources of population heterogeneity (i.e., genetic factors), cross-sibling differences in wave 1 delinquency were positively and significantly related to wave 4 criminal behavior. In other words, after controlling for genetic influences on wave 4 criminal behavior, the sibling who exhibited more delinquent behavior at wave 1 tended to report more criminal behavior at wave 4. This result is consistent with the Cholesky model results which revealed that the nonshared environment accounted for a portion (albeit, a small portion) of the stability in delinquent/criminal behavior.

Discussion

Two decades ago, a group of criminologists noted the potential importance of genetic influences for explaining stability in criminal behavior over time (Rowe et al., 1990). Rowe et al. (1990:244) stated that, "Genetic influences on illegal behavior are relevant to our purposes because they are consistent with the assumptions of a latent trait model: namely, that stable causes of crime, which are not directly observable, produce individual differences." The authors concluded that a latent trait model of offending was the most accurate approach to modeling long-term offending patterns. In line with the conclusion drawn by Rowe et al., the current study revealed that stable between-individual influences accounted for the majority of the stability in criminal behavior observed from adolescence to adulthood. To be specific, the current results indicated that genetic factors accounted for the majority of the stability in criminal behavior over time.

Although the current study was unable to directly test the competing hypotheses of state dependence and population heterogeneity, the findings do warrant close attention by scholars on both sides of the debate. In short, the current findings indicated that population heterogeneity explanations may be most salient for explaining behavioral stability, but state dependence arguments should not be dismissed. The population heterogeneity approach is centered on the key argument that stable individual differences explain stability in behavior over time. One of the most stable individual-level factors is a person's unique suite of DNA (i.e., their genotype). As scholars have noted in the past, a person takes their genotype with them everywhere they go, throughout the entire life span. For this reason, it makes sense that genetic factors should explain stability in behavior over time (see Harris, 1998; Pinker, 2002).

But how do genetic factors influence behavioral stability? One of the most consistent findings to emerge from behavior genetic research is that nearly every aspect of personality is under genetic influence (Harris, 1998; Raine, 1993; Rutter, 2006). Research has revealed that personality traits such as self-control, which are linked with

criminality, are under significant genetic influence (Beaver et al., 2009; Boisvert, 2009). Thus, the current finding that genetic factors influence behavioral stability is likely the result of an indirect causal pathway that is mediated by personality development. Genes influence personality development (Harris, 1998) which, in turn, drives behavioral stability.

So where does this leave the state dependence perspective? Recall that the findings from the DF analysis (Table 3) revealed that state dependence may play a role in behavioral stability: after controlling for genetic factors (population heterogeneity), the sibling who was more involved in delinquent behavior at wave 1 was also more involved in criminal activities at wave 4. Perhaps more in line with the state dependence literature was the finding that the nonshared environment had a large (and statistically significant) influence on *changes* in behavior from wave 1 to wave 4. This is important for state dependence arguments since most scholars identify state dependent factors as influencing stability and changes in behavior (Sampson & Laub, 1993). In short, the current findings suggest that state dependence processes may not be the primary force underlying behavioral stability, but that state dependence processes may be a viable explanation of changes in offending behaviors over time.[5]

Another curious result to emerge from the analysis was that genetic influences affected *changes* in behavior from wave 1 to wave 4. This finding may at first appear inconsistent with traditional conceptions of genetic effects. It is commonly known that a person's genotype does not change over time. Thus, it is often assumed that genetic factors only influence stability in behavior. This assumption, however, overlooks the possibility that the effects of genes can change without altering a person's DNA. Though a person's genotype is generally unalterable over their life span (except through events like mutation), the *effects* of genes are malleable through the process of epigenetic alterations (Walsh, 2009) and/or gene-environment interaction (Caspi et al., 2002). Epigenetic mechanisms are responsible for turning genes "on" and "off." Interestingly, scientists have recently discovered that this process can be influenced by environmental exposures. Whether this line of inquiry can inform criminology is yet to be realized (Walsh, 2009). As for gene-environment interaction, Caspi et al. (2002) reported that the effect of MAOA (a gene that impacts neurotransmission in the brain) on antisocial behavior was contingent upon the individual's home experiences. Only for respondents raised in adverse home conditions was the MAOA gene predictive of antisocial behavior.

The two lines of research mentioned above (i.e., epigenetics and gene-environment interaction) are beginning to reveal myriad ways in which genes and environments can interact to impact behavior (Dick, 2011). To the extent, then, that environmental exposures change over time we might expect to identify a genetic influence on changes in behavior.[6] Along these lines, exploring the genetic origins of change may open doors to treatment and rehabilitation opportunities for individuals ensnared in antisocial lifestyles. A polymorphism in the gene that encodes for the μ-opioid receptor (OPRM1), for example, has been linked to success in treatment for alcoholism (Oslin et al., 2003). Individuals in one study conducted by Oslin et al. (2003) were most responsive to pharmacological treatments when they carried at least one copy of the Asp40 allele. Relapse rates were higher, however, for individuals carrying two copies of an alternative allele (e.i., subjects homozygous for the Asn40 allele). Ultimately, work in this area points towards two intriguing possibilities: 1) genetic factors may help to shed light on why, and under what circumstances, behaviors can change, and 2) assessing the presence of measured genes may represent avenues for increasing treatment efficacy for certain individuals in the population.

Several limitations of this analysis should be mentioned. First, although the Add Health data currently span a 13 year time period, future work should seek to uncover the factors that explain stability and changes in behavior over much longer periods of the life course. For example, much of the criminological literature (e.g., Moffitt, 1993; Sampson & Laub, 1993) discusses the importance of examining behavioral stability and change from childhood to late adulthood (Cullen, 2011). Unfortunately, the current study was restricted to the time period spanning between adolescence and young/middle adulthood. This represents a primary area for future research to expand on the current findings. A second limitation was that the current study was unable to determine *which* genetic and environmental influences are important for explaining stability and change. Behavior genetic research is quite informative when the goal is to determine whether and how much genes and environments contribute to certain outcomes. When the questions become more specific (i.e., which genes matter – or – which environments matter?), behavior genetic research is less informative. Thus, future research should expand on this study by identifying which genes impact behavioral stability and which nonshared environments impact changes in behavior. Extant life-course theories such as Sampson and Laub's (1993) are likely to be good starting points for identifying the latter.

A third limitation was that myriad forms of delinquent and criminal behavior were collapsed into a single scale of delinquency/criminal behavior. Recent research has suggested that different forms of antisocial behavior may be differentially influenced by genetic factors (Burt, 2009). Burt (2009) reported that aggressive behaviors were under more genetic influence (65%) as compared to non-aggressive antisocial behavior (48%). As a result, an important follow-up to the current study might separately explore the genetic and environmental factors that underlie stability and changes in violent and non-violent behavior.

Although behavior genetic research has increasingly focused on the genetic and environmental underpinnings to stability and change in behavior over time, very little thought has been given to whether the findings from these studies are compatible with criminological theories. At the same time, criminological scholars have overlooked the importance of behavior genetic research for explaining behavioral stability and change. The result has been that these two lines of research have operated in isolation of one another. The current effort was to integrate two lines of research that are compatible with one another and that, together, provide a more in-depth explanation of the stability in offending behavior over time. The findings drawn from this analysis underscore the importance of considering both genetic and environmental influences when analyzing stability and changes in antisocial behavior.

Notes

1. Though genetic *expression* changes over time via epigenetic regulation (Walsh, 2009).

2. After controlling for the effect of age, the heritability estimate for stability was .98 with a 95% confidence interval of .67-1.00.

3. To account for the non-normality of the scales, both the wave 1 delinquency scale and the wave 4 criminal behavior scale were recoded into quartile measures and the ACE and Cholesky models were estimated using threshold techniques. The threshold model has been shown to provide more accurate parameter estimates in the face of non-normality (Derks, Dolan, & Boomsma, 2004). The parameter estimates and the substantive conclusions from these models were virtually identical to those presented in the text. The parameter estimates were as follows: ACE for wave 1 delinquency A = .58, C = .00, E = .42; ACE for wave 4 criminal behavior A = .48, C = .00, E = .52; Cholesky parameters for stability A = .75, C = .00, E = .25; Cholesky parameters for change A = .47, C = .00, E = .53.

4. As a sensitivity check, the Cholesky model was re-estimated using only MZ twins, DZ twins, and full siblings. The substantive findings were identical to those presented in the text. Cholesky parameters for stability A = .88, C = .00, E = .12; Cholesky parameters for change A = .28, C = .00, E = .72.

5. Again, however, it is worth noting that the nonshared environment *may* include genetic influences if gene-environment correlations are at play (Purcell, 2002).

6. This point is especially relevant for studies that employ behavioral genetic modeling (as the current study did). Gene-environment interactions will show up as genetic effects in behavioral genetic studies when the environmental influence is a shared environment. When the environmental influence is a nonshared environment, the gene-environment interaction will show up as a nonshared environmental effect.

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