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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