AN INVESTIGATION OF GENETIC AND ENVIRONMENTAL INFLUENCES ACROSS THE DISTRIBUTION OF SELF-CONTROL

JOSEPH A. SCHWARTZ
University of Nebraska at Omaha

ERIC J. CONNOLLY
Sam Houston State University

JOSEPH L. NEDELEC
University of Cincinnati

KEVIN M. BEAVER
Florida State University
King Abdulaziz University

Previous research illustrating a robust, negative association between self-control and various forms of delinquent and criminal behavior has resulted in a more concentrated focus on the etiological development of self-control. The current study aims to contribute to this literature using a sample of twin and sibling pairs from the National Longitudinal Study of Adolescent to Adult Health (Add Health) to examine genetic and shared environmental influences across levels of self-control. The results of modified DeFries–Fulker (DF) equations revealed that genetic and shared environmental influences were distributed in a nonlinear pattern across levels of self-control. Subsequent biometric quantile regression models revealed that genetic influences on self-control were maximized in the 50th and 60th percentiles, and minimized in the tails of the distribution. Shared environmental influences were nonsignificant at all examined quantiles of self-control with only one exception. The theoretical importance of utilizing genetically informed modeling strategies is discussed in more detail.

Keywords: self-control; nonlinear; quantile regression; DeFries–Fulker analysis

Self-control is one of the most commonly studied phenotypes in the behavioral sciences. Previous research has identified that individuals with higher levels of self-control (or traits directly implicated in the concept of self-control) are less likely to engage in criminal

AUTHORS' NOTE: *This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by Grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due to Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (http://www.cpc.unc.edu/addhealth). No direct support was received from Grant P01-HD31921 for this analysis. Correspondence concerning this article should be addressed to Joseph A. Schwartz, School of Criminology and Criminal Justice, University of Nebraska at Omaha, 310 Nebraska Hall, 901 N. 17th Street, Lincoln, NE 68588-0561; e-mail: jaschwartz@unomaha.edu.*

CRIMINAL JUSTICE AND BEHAVIOR, 201X, Vol. XX, No. X, Month 2017, 1–20.
DOI: 10.1177/0093854817709495

and antisocial behavior (Fine, Steinberg, Frick, & Cauffman, 2016; Goode, 2008; Meier, Slutske, Arndt, & Cadoret, 2008; Pratt & Cullen, 2000), to have fewer physical and mental health problems (Moffitt et al., 2011; Nedelec & Beaver, 2014), to have greater earning potential over the life course (Evans et al., 1997), and to experience greater overall levels of interpersonal success (Tangney, Baumeister, & Boone, 2004). While the behavioral and psychological concept of self-control transcends disciplinary boundaries, a substantial amount of research within the field of criminology has been devoted to this particular topic, with studies focusing on the etiological development of self-control (Cullen, Unnever, Wright, & Beaver, 2008; Hay, 2001; Pratt, Turner, & Piquero, 2004; Turner, Piquero, & Pratt, 2005; Unnever, Cullen, & Pratt, 2003), the association between self-control and criminal or delinquent behavior (Goode, 2008; Pratt & Cullen, 2000), and longitudinal trends in self-control over extended periods of the life course (Hay & Forrest, 2006; Na & Paternoster, 2012; Ray, Jones, Loughran, & Jennings, 2013). Despite this impressive accumulation of studies, much remains unknown.

One particular aspect that has recently come under scrutiny is the functional form of the association between levels of self-control and delinquency, with studies providing compelling evidence of both a linear (Sullivan & Loughran, 2014) and nonlinear (Mears, Cochran, & Beaver, 2013) association. This increased focus on functional form in recent years is understandable, as findings flowing from this line of research have important methodological and theoretical implications for future research centered on the concept of self-control. For example, the detection of a nonlinear association would potentially require different data reduction strategies and analytic techniques relative to a linear association. Similar concerns may also extend to the underlying factors implicated in the development of self-control, wherein some sources of influence may be stronger or weaker at different levels of self-control. This possibility also has important implications for studies attempting to identify factors that explain individual differences in self-control, as each source of influence may contribute in distinct ways at various regions of the distribution. For example, sources of influence may vary based on differences in self-control between individuals, wherein some sources may be implicated for individuals with high levels of self-control but not for those with moderate or low levels of self-control. This possibility has potentially important implications for Gottfredson and Hirschi's (1990) conceptualization of self-control and may imply that the existing theoretical framework may be overly simplistic.

The current study addresses this limitation in the existing literature by using a sample of twin and sibling pairs from the National Longitudinal Study of Adolescent to Adult Health (Add Health; Harris, 2011; Harris et al., 2009; Udry, 2003). Rather than focusing on specific sources of influence, the current study examines latent genetic and environmental influences across different levels of self-control. This approach offers three specific advantages. First, previous research has revealed that genetic and environmental mechanisms are involved in creating individual differences in self-control and related concepts (Anokhin et al., 2011; Friedman et al., 2011; Fulker et al., 1980; Miles et al., 2001; Niv et al., 2012; Wright & Beaver, 2005), indicating that examining both sources of influence will provide a more comprehensive understanding of the etiological development of self-control. Second, examining both sources of influence provides a more comprehensive understanding of the underlying factors that ultimately shape levels of self-control. Rather than identifying a single source of influence (e.g., parental socialization), the approach used in the current study is focused on examining all potential sources of influence (both genetic and

environmental). Third, recent developments in behavior genetics have emphasized the importance of estimating genetic influences on behavioral phenotypes in the interest of isolating *environmental* sources of influence (Johnson et al., 2009; Turkheimer & Harden, 2014). In this way, identifying the pattern of genetic influences contributing to overall variation in self-control can potentially allow for the better identification of meaningful sources of environmental influence at varying levels of self-control.

IMPLICATIONS OF NONLINEAR GENETIC AND ENVIRONMENTAL INFLUENCES ON SELF-CONTROL

When proposing their general theory of crime, Gottfredson and Hirschi (1990) emphasized the importance of parental socialization in the development of self-control over the life course. Subsequent research has found evidence in favor of this "parental socialization hypothesis" (Cullen et al., 2008; Hay, 2001; Hay & Forrest, 2006; Pratt et al., 2004; Unnever et al., 2003), but other studies have also identified additional environmental sources of influence, including neighborhood and school surroundings (Gibson, Sullivan, Jones, & Piquero, 2010; Pratt et al., 2004; Turner et al., 2005). At the same time, a related line of research has emphasized the importance of both genetic and environmental influences in the development of variation in self-control (Anokhin et al., 2011; Friedman et al., 2011; Fulker et al., 1980; Miles et al., 2001; Niv et al., 2012; Yancey, Venables, Hicks, & Patrick, 2013). Collectively, these studies have revealed that, much like any other complex behavioral phenotype, variation in self-control is the result of an intricate combination of both genetic and environmental influences (Polderman et al., 2015). A related line of research has also demonstrated a significant association between self-control and delinquency, even after controlling for genetic factors that collectively influence both phenotypes (Moffitt et al., 2011; Seroczynski et al., 1999). In addition, a recent study by Boisvert, Wright, Knopik, and Vaske (2012) examined genetic influences on the association (or covariance) between self-control and delinquency at different stages of the life course. The results indicated that, depending on the life-course stage examined, common genetic influences explained between 49% and 83% of the covariance between self-control and delinquency, with the remaining covariance explained by nonshared environmental influences.

Taken together, these findings indicate that genetic influences are directly implicated not only in the development of both self-control and delinquency but also in the association between both phenotypes. While these findings, in combination with the larger body of self-control research, further elucidate the etiological development of both self-control and the association between self-control and delinquency, such studies have not yet explored the possibility of nonlinear sources of influence on self-control. Rather, previous genetically informed studies operate under the assumption that meaningful sources of influence are distributed consistently across levels of self-control with the resulting parameter estimates reflecting average genetic, shared environmental, and nonshared environmental influences (Carey, 2003; Neale & Maes, 1992; Plomin et al., 2013). While this aspect of previous research is reasonable if genetic and environmental influences on self-control are distributed consistently, it may be less acceptable when these influences are distributed in a less consistent pattern. This is primarily due to the possibility that genetic and environmental influences may be differentially emphasized at varying levels of self-control. For example, if genetic influences are strongest near the median of the distribution, traditional multivariate biometric

modeling strategies attempting to identify sources of environmental influence on self-control may be more susceptible to Type II error. Stated differently, traditional genetically informed models may be less likely or capable of identifying a meaningful source of environmental influence on self-control, especially if genetic and environmental influences are nonlinear. This is primarily due to the fact that, in the aforementioned example, genetic (or environmental) influences may be stronger around the median, but such influences may be more muted at other regions of the distribution. While an examined source of environmental influence may not significantly contribute to moderate levels of self-control, such a finding would not necessarily indicate that the same environmental influence is not implicated in the development of low and/or high levels of self-control.[1]

While the extent to which genetic and environmental influences vary across levels of self-control remains an open empirical question, there is at least some theoretical evidence suggesting that such influences could be distributed in a nonlinear manner. To illustrate, in a presidential address to the Society for Research in Child Development, Sandra Scarr (1992) underscored the importance of average and expectable environments that are critical to normative human development across the life course. More specifically, when considering the underlying etiological development of behavioral phenotypes, environmental influences within the "species-normal range" (e.g., parent–child bonding during infancy, proper nutrition in childhood, parental support and supervision during adolescence) would be largely indistinguishable, and genetic influences would be more directly implicated. In an effort to better specify the range of average and expectable environments, Scarr noted, "Evolution has not left development of the human species, nor any other, at the easy mercy of variation in their environments. We are robust and able to adapt to wide-ranging circumstances . . ." (p. 15), indicating that such environments are represented with a relatively wide interval. However, environmental influences that fall beyond what is considered average and expectable would be more directly implicated in creating individual differences in a given phenotype, and thus outweigh genetic influences. A similar observation was made by Rowe (2001), who noted the importance of "biological set-points." Rowe argued that while genetic influences are responsible for the possible range of values on a given phenotype within individuals, environmental influences are largely responsible for either pushing or pulling individuals along this range. As such, an individual's absolute maximum and minimum scores on a self-control measure are largely the result of genetic influences, but where an individual falls within the present range is more directly the result of environmental influences.[2]

Taken together, both perspectives imply that biological and environmental influences may be distributed in a nonlinear pattern across levels of self-control, wherein genetic influences may explain more variance in the presence of average or neutral environments (i.e., near the center of the distribution), and environmental influences may explain more variance as such influences begin to fall outside of the normal developmental range and become more potent (i.e., in the tails of the distribution). While previous research has not yet examined whether genetic and environmental influences vary across levels of self-control, previous studies have examined such influences in other related concepts such as attention-deficit hyperactivity disorder (ADHD). Due to a lack of consensus regarding whether ADHD should be considered a categorical disorder or measured continuously (Coghill & Sonuga-Barke, 2012; Willcutt, 2005), previous studies have attempted to examine whether genetic and environmental influences on overall levels of ADHD are distinct from such influences

at more extreme levels of ADHD (Greven et al., 2016; Larsson et al., 2012; Willcutt et al., 2000). The results of these studies are decidedly mixed with some identifying similar heritability estimates for overall and extreme levels of ADHD (Larsson et al., 2012), others reporting patterns emphasizing the importance of genetic influences at one end of the distribution but not the other (Greven et al., 2016), while others report different patterns of genetic and environmental influences based on which symptoms were present (Willcutt et al., 2000).

While the debate surrounding the functional form of genetic and environmental influences on ADHD remains unsettled, the results of this limited literature provide additional evidence suggesting that such influences may be distributed in a nonlinear pattern across other similar phenotypes such as self-control. As mentioned, an impressive number of studies have emphasized the importance of both genetic and environmental influences in shaping overall levels of self-control (e.g., Boisvert et al., 2012; Moffitt et al., 2011; Seroczynski et al., 1999), but studies employing these modeling strategies assume that such influences are fixed and do not vary across levels of self-control. In this way, traditional biometric modeling strategies rely on averages to estimate genetic and environmental influences for examined phenotype(s), potentially masking the varying influence of scores across the examined distribution. Based on this limitation, whether such influences vary across levels of self-control currently remains an open empirical question.

THE CURRENT STUDY

The current study aims to address the abovementioned limitations using a series of modified DeFries–Fulker (DF; DeFries & Fulker, 1985, 1988; Frazier et al., 2014; Knopik, Alarcón, & DeFries, 1997) equations and a sample of twin and sibling pairs from the Add Health. A series of DF equations will be used to determine whether genetic and environmental influences are distributed in a linear or nonlinear pattern across levels of self-control. The estimation of these equations is a necessary first step, as the presence of a linear pattern of influences would provide preliminary evidence, suggesting that results from previous studies examining heritability estimates across the full distribution of self-control may also apply to more specific areas of the distribution. Second, a series of modified quantile regression models will be estimated to provide a more detailed description of the distribution of genetic and environmental influences across levels of self-control. More specifically, the quantile regression models will identify specific regions of the self-control distribution in which genetic (or environmental) influences are maximized and environmental (or genetic) influences are minimized. In this way, the current study provides greater insight into the distribution of genetic and environmental influences across levels of self-control and, in turn, the underlying mechanisms involved in individual differences in self-control during adolescence.

METHOD

DATA

Data for the current study were drawn from the first wave of the Add Health, a nationally representative four-wave prospective study of American youth enrolled in middle or high school between the years of 1994 and 1995 (Udry, 2003). Participants were selected using

a multistage cluster design that resulted in the selection of 132 middle and high schools throughout the country. Youth attending each of the selected schools and present for interviews were asked to participate in the in-school portion of the study. Approximately 90,000 youth completed the in-school survey. A subsample of the youth who completed the in-school survey was selected and asked to participate in the in-home portion of the study in an effort to obtain more detailed information on a wide range of issues related to health, development, and behavior. In total, 20,745 youth aged between 12 and 21 and more than 17,000 of their primary caregivers agreed to participate in the Wave I in-home portion of the study (Harris et al., 2009). Approximately 1 year after the completion of Wave I (1996), the second wave of the study commenced, with close to 15,000 respondents from the first wave participating in Wave II. The third wave of the study was completed between 2001 and 2002, and included a total of 15,197 participants from Wave I. The fourth wave of data collection was carried out between 2007 and 2008 when respondents were between 24 and 34 years old and included 15,701 of the original Wave I participants. The resulting sample covers approximately 14 years of development, and includes information on a wide range of topics central to various stages of the life course (for a more detailed description, see Harris, 2011).

The Add Health data also include a large subsample of twin and sibling pairs. Sibling pairs were identified during the in-home portion of the Wave I interviews by asking respondents if they had a cotwin or any other siblings. Unrelated siblings, cousins, half-siblings, and cotwins were added to the sample with certainty, and a probability sample of full-siblings between the ages of 11 and 20 was also included, resulting in a sample of more than 3,000 sibling pairs (Harris et al., 2006). The analytic sample for the current study included monozygotic (MZ; $n = 285$ pairs) twins, dizygotic (DZ; $n = 445$ pairs) twins, and full-sibling ($n = 1{,}035$ pairs) pairs. Directly in line with previous studies analyzing twin and sibling pairs (Plomin et al., 2013), each sibling was included in the final analytic sample twice, once as a cotwin and again as a proband. This technique is referred to as the "double entry" method and is commonly used when employing regression-based biometric modeling strategies (more detail provided below). Importantly, use of the double-entry method may potentially bias standard errors due to nonindependence in the resulting residuals. In an effort to overcome this limitation, standard errors estimated in the current study were adjusted for within-family clustering using the "cluster" option in Stata MP 13.1 (StataCorp, 2013).

MEASURES

SELF-CONTROL

Previous research has focused on the most appropriate way to measure the concept of self-control as proposed by Gottfredson and Hirschi (1990; Grasmick et al., 1993; Marcus, 2004; Piquero & Bouffard, 2007). Despite considerable debate in this area, results from an influential and comprehensive meta-analysis (Pratt & Cullen, 2000) found that different measurement strategies did not significantly moderate the association between self-control and various forms of offending. In the current study, we follow the lead of previous researchers analyzing the Add Health data and use a multirater scale comprised of 23 items from the Wave I in-home portion of the study to measure self-control (Boisvert et al., 2012; Mears et al., 2013; Yun, Cheong, & Walsh, 2014). The resulting scale includes items

tapping multiple dimensions of self-control, including attentiveness, the ability to focus, and impulsivity. The self-control scale was coded such that higher values represent higher levels of self-control, with scores ranging from 38 to 104 ($\alpha = .75$). As recommended in previous studies using a similar analytic strategy (Frazier et al., 2014; Logan et al., 2012; Petscher & Logan, 2014), the resulting self-control measure was standardized as a *z* score ($M = 0.00$, $SD = 1.00$) within the full Add Health sample from which the final analytic sample of twins and full-sibling pairs was derived. The mean, standard deviation, and other descriptive statistics for the self-control scale and all other measures used in the current study are reported in Table 1.

CONTROLS

Three control measures were used to account for demographic differences within DZ twin and full-sibling pairs. First, age was measured continuously in years at Wave I. Second, gender was coded dichotomously where 0 = *female* and 1 = *male*. Third, race was also measured dichotomously where 0 = *Caucasian* and 1 = *all other races*.[3]

PLAN OF ANALYSIS

The analytic plan for the current study was carried out in three interconnected steps. The first step in the analysis involved the estimation of a series of baseline DF equations for the self-control measure. DF analysis is a regression-based biometric modeling technique appropriate for samples of twin and sibling pairs. The baseline DF equation can be presented as

$$S_2 = \beta_0 + \beta_1(S_1) + \beta_2(R) + \beta_3(S_1 \times R) + \beta_4(Cov_{\text{diff}}) + e, \quad (1)$$

where S_2 is sibling 2's (or the cosibling) score on the measure of interest, S_1 is sibling 1's (commonly referred to as the proband in behavioral genetics) score on the same measure, *R* represents the level of genetic relatedness between sibling pairs (coded 1.00 for MZ twins and 0.50 for DZ twins and full-sibling pairs), and Cov_{diff} represents between-sibling differences on the included covariates and are entered into the equation as difference scores where sibling 2's score on a given covariate (e.g., age) is subtracted from sibling 1's score on the same measure. The resulting regression coefficients can be interpreted such that β_0 is the intercept or the mean cotwin score on the measure of interest, β_1 provides an estimate of the proportion of variance in the measure of interest explained by nongenetic influences that are similar between both siblings (commonly referred to as the shared environment and symbolized as c^2), β_2 provides an estimate of the extent to which the expected value of S_2 varies across levels of genetic relatedness, and β_3 provides an estimate of the proportion of variance in the measure of interest explained by individual-level genetic influences (typically referred to as heritability and symbolized as h^2). β_4 provides an estimate of within-pair differences on a measured covariate on the outcome of interest, and *e* contains all residual variance including nongenetic influences that differ between siblings (commonly referred to as the nonshared environment and symbolized as e^2) and measurement error. While DF analysis represents only one approach in estimating genetic and environmental influences on a given phenotype (Carey, 2003; Neale & Maes, 1992; Plomin et al., 2013), recent simulation studies have revealed an overall convergence in findings between DF analysis and

TABLE 1: Mean, Standard Deviation, and Range for All Study Variables

	MZ twin pairs			DZ twin/full-sibling pairs			Full analytic sample		
Study Variables	*M*/%	*SD*	Range	*M*/%	*SD*	Range	*M*/%	*SD*	Range
Self-control (overall)	0.09	0.97	–3.01 to 2.82	–0.02	0.99	–4.94 to 3.06	0.00	0.99	–4.95 to 3.06
10th percentile	–1.59	0.47	–3.01 to –1.19	–1.74	0.59	–4.95 to –1.19	–1.72	0.58	–4.95 to –1.19
20th percentile	–0.87	0.14	–1.06 to –0.70	–0.87	0.13	–1.06 to –0.70	–0.87	0.13	–1.06 to –0.70
30th percentile	–0.45	0.10	–0.58 to –0.34	–0.46	0.10	–0.58 to –0.34	–0.46	0.10	–0.58 to –0.34
40th percentile	–0.16	0.06	–0.21 to 0.09	–0.15	0.06	–0.21 to –0.09	–0.15	0.06	–0.21 to –0.09
50th percentile	0.08	0.06	0.03 to 0.15	0.09	0.06	0.03 to 0.15	0.09	0.06	0.03 to 0.15
60th percentile	0.33	0.06	0.27 to 0.39	0.34	0.06	0.27 to 0.39	0.33	0.06	0.27 to 0.39
70th percentile	0.62	0.10	0.51 to 0.76	0.63	0.10	0.51 to 0.76	0.63	0.10	0.51 to 0.76
80th percentile	0.97	0.09	0.88 to 1.12	0.99	0.10	0.88 to 1.12	0.99	0.10	0.88 to 1.12
90th percentile	1.64	0.43	1.24 to 2.82	1.62	0.38	1.24 to 3.06	1.63	0.39	1.24 to 3.06
Age	16.17	1.59	13 to 19	16.12	1.69	12 to 21	16.13	1.67	12 to 21
Gender			0.00-1.00			0.00-1.00			0.00-1.00
Male (%)		50.00		50.58			50.54		
Female (%)		50.00		49.42			49.53		
Race			0.00-1.00			0.00-1.00			0.00-1.00
Caucasian (%)	63.83			65.39			65.14		
All other races (%)	36.17			34.58			34.83		
N (pairs)		570 (285)			2,960 (1,480)			3,530 (1,765)	

Note. All measures assessed at Wave I. Self-control and delinquency measures were *z*-transformed within the full Add Health sample (*N* = 20,745). MZ = monozygotic; DZ = dizygotic.

other analytic techniques, such as univariate biometric decomposition models (Smith & Hatemi, 2013).

While DF analysis was originally designed to estimate genetic and environmental influences on groups of individuals that fall at the extreme ends of a given distribution (sometimes referred to as DF extremes analysis), the resulting parameter estimates are confined to a specific region of the examined distribution (see Plomin & Kovas, 2005 for a more detailed overview). One of the primary limitations of this approach is identifying appropriate cut-points that specify extreme values on the examined phenotype (Plomin et al., 2013). This problem is further magnified when the examined phenotype is measured continuously and there is no consensus regarding a diagnostic cutoff, as is the case with self-control. Despite this limitation, traditional DF analysis can be used to estimate the amount of variance in a given measure that can be explained by genetic and shared environmental influences at a specific point of the distribution but cannot provide estimates across different levels of the measure being examined. Additional modifications to the traditional DF equation have been made to address this limitation. The second step in the analysis for the current study was the estimation of specially tailored equations developed by Cherny, Cardon, Fulker, and DeFries (1992) aimed at assessing the functional form of genetic and shared environmental influences across the distribution of self-control. The proposed method involves the estimation of two separate regression equations aimed at assessing linear and nonlinear (i.e., quadratic) changes in heritability and shared environmental influences over the distribution of a given phenotype, and can be estimated as

$$\begin{aligned} S_2 = {} & \beta_0 + \beta_1 (S_1) + \beta_2 (R) + \beta_3 (S_1 \times R) + \beta_4 (Cov_{diff}) \\ & + \beta_5 (S_1 \times S_1) + \beta_6 (S_1 \times S_1 \times R) + e, \end{aligned} \tag{2}$$

$$\begin{aligned} S_2 = {} & \beta_0 + \beta_1 (S_1) + \beta_2 (R) + \beta_3 (S_1 \times R) + \beta_4 (Cov_{diff}) \\ & + \beta_5 (S_1 \times S_1) + \beta_6 (S_1 \times S_1 \times R) + \beta_7 (S_1 \times S_1 \times S_1) \\ & + \beta_8 (S_1 \times S_1 \times S_1 \times R) + e. \end{aligned} \tag{3}$$

Equation 2 modifies the baseline equation with the addition of interaction terms that test for linear trends in shared environmental (β_5) and genetic (β_6) influences. While β_1 and β_3 provide estimates of shared environmental and genetic influences (respectively) on the examined phenotype (e.g., self-control), a single estimate for each variance component is provided. When the examined phenotype is measured continuously, the resulting estimates reflect average levels of shared environmental and genetic influences. The additional interaction terms included in Equation 2 directly test the assumption that the resulting influences are distributed in a linear or consistent pattern across the distribution of the examined phenotype. For example, a significant β_6 coefficient would indicate that genetic influences on self-control are linearly or consistently distributed across the range of scores (e.g., $h^2 = .50$ at all examined levels), but, alternatively, a nonsignificant coefficient would provide preliminary evidence that genetic influences are distributed in a nonlinear manner.[4] In the event that Equation 2 yields nonsignificant linear estimates, Equation 3 allows for the estimation of potential nonlinear trends in h^2 and c^2 with the inclusion of two additional interaction terms. Significant coefficients for β_7 or β_8 would indicate that shared environmental

and genetic influences are distributed across the examined outcome in a nonlinear manner (for a more detailed explanation, see Cherny et al., 1992).

Despite the obvious advantages of these modified equations, this particular method does not provide precise estimates of h^2 and c^2 at various levels of the examined measure; rather, the generated coefficients only provide information regarding the significance and direction of any observed change. These limitations have been recently addressed with the use of *biometric quantile regression* (Logan et al., 2012). In more traditional applications, quantile regression offers a distinct advantage over linear regression (i.e., ordinary least squares [OLS]), in that it allows for the estimation of the association between a key independent variable (X) and dependent variable (Y) across specified quantiles or percentiles of Y. Where OLS regression provides an estimate of the change in the mean score of Y based on a one-unit increase or decrease in X, quantile regression presents unique estimates of the association between X and Y at each of the examined quantiles of Y. Rather than relying on the minimization of squared residuals, quantile regression allows for the estimation of the association between X and Y at a given quantile of Y by minimizing the sum of absolute residuals within each examined quantile (for a more technical explanation, see Petscher & Logan, 2014), which can be presented as

$$\sum \theta \left| y_t - x_t \beta \right| + \sum (1-\theta) \left| y_t - x_t \beta \right|, \tag{4}$$

where y_t is the dependent variable, x_t is the independent variable of interest, β is the estimated coefficient, and θ specifies the estimated quantile.

This minimization procedure expressed in Equation 4 is carried out for each examined quantile and allows for the inclusion of all nonmissing cases in the analytic sample, as opposed to only cases that fall within the examined quantile, by assigning a weight of θ to quantiles > θ and a weight of $(1-\theta)$ to quantiles < θ. The alternative technique of creating specified groupings within the examined phenotype (e.g., quartiles or deciles) and estimating separate models for each specified group suffers from a number of limitations: (a) the resulting groups likely suffer from selection bias (Heckman, 1979), (b) subsequent statistical models are likely underpowered due to sample limitation, and (c) grouping procedures likely result in nonnormal error terms, effectively violating a basic OLS assumption. Due to the examination of the full analytic sample and the lack of assumptions regarding the underlying distribution of error terms, quantile regression effectively overcomes these limitations. In addition, due to the weighting procedure described above, traditional quantile regression allows for the examination of associations between *X* and *Y* at any point in the distribution of Y, providing a more precise description of the resulting association.

The third and final step in the plan of analysis for the current study involved the estimation of a biometric quantile regression equation that allows for the estimation of h^2 and c^2 parameter estimates across the distribution of the examined variable. This procedure can be carried out by nesting the baseline DF equation (Equation 1) within the quantile minimization procedure (Equation 4; for a more detailed description, see Logan et al., 2012). The resulting equation can be presented as

$$\begin{aligned} &\sum \theta \left| +\mathrm{S}_2 - (\beta_0 + \beta_1(\mathrm{S}_1) + \beta_2(\mathrm{S}_1 \times R) + \beta_3(Cov_{diff})) \right| \\ &+ \sum (1-\theta) \left| \mathrm{S}_2 - (\beta_0 + \beta_1(\mathrm{S}_1) + \beta_2(\mathrm{S}_1 \times R) + \beta_3(Cov_{diff})) \right| \end{aligned} \tag{5}$$

TABLE 2: Results From DeFries–Fulker Models Examining Linear and Nonlinear Change

	Baseline model		Linear change		Quadratic change	
Study measures	Estimate	*SE*	Estimate	*SE*	Estimate	*SE*
Self-control						
h^2	.34**	0.10	.33**	0.10	.61**	0.14
c^2	.04	0.06	.06	0.06	–.05	0.09
Linear change in h^2			.00	0.07	.05	0.06
Linear change in c^2			.01	0.04	–.03	0.04
Quadratic change in h^2					–.10**	0.03
Quadratic change in c^2					.04*	0.02
Covariates						
Age	–.01	0.01	–.01	0.01	–.01	0.01
Gender	–.03	0.03	–.03	0.03	–.03	0.03
Race	.12	0.10	.12	0.10	.12	0.10
N (pairs)	1,370 (685)		1,370 (685)		1,370 (685)	

Note. Standard errors corrected for within-family clustering.
*$p \leq .05$. **$p \leq .01$.

where θ specifies the estimated quantile, S_2 is sibling 2's score on the measure of interest, S_1 is sibling 1's score on the same measure, R represents the level of genetic relatedness, and Cov_{diff} represents between-sibling differences on the included covariates. β_1 provides an estimate of c^2 at the specified quantile, and β_2 provides an estimate of h^2 at the specified quantile. In this way, the results of the biometric quantile regression equation will provide estimates of h^2 and c^2 at each of the examined quantiles, effectively providing detailed information regarding the shape of the underlying distribution of such influences across levels of self-control.

RESULTS

The first step in the analysis involved estimating baseline DF models (Equation 1) for the self-control and delinquency measures, the results of which are presented in the first column of Table 2. The results revealed that approximately 34% of the variance in self-control was explained by genetic influences, while shared environmental influences did not explain any variance, leaving the residual variance to be explained by nonshared environmental influences and measurement error. The second step in the analysis involved the estimation of a modified DF model to assess whether changes in h^2 and c^2 were distributed linearly across self-control (β_5 and β_6, respectively, in Equation 2). The resulting estimates of linear change in h^2 and c^2 were nonsignificant, providing preliminary evidence that both sets of influences may be distributed in a nonlinear pattern. The final model assessed whether changes in h^2 and c^2 were nonlinear across the distribution of self-control (β_7 and β_8 in Equation 3). The results revealed nonlinear patterns of influence across the distribution of self-control, where h^2 decreased ($b = -.10$, $p < .001$) and c^2 increased ($b = .04$, $p = .02$) as levels of self-control increased.

Based on these results, the next step in the analysis involved the estimation of a biometric quantile regression equation (Equation 5) to estimate h^2 and c^2 across various levels of self-control. The results of the quantile regression analysis are presented in

TABLE 3: Biometric Quantile Regression Results for Self-Control

Self-control quantiles	Biometric components		Statistical covariates		
	h^2	c^2	Age	Gender	Race
10th percentile	.06	.23	−.01	−.09	.01
	[−.33, .45]	[.00, .46]	[−.05, .03]	[−.21, .03]	[−.37, .38]
20th percentile	.37*	.05	−.02	−.02	.14
	[.05, .68]	[−.14, .23]	[−.11, .07]	[−.11, .07]	[−.11, .40]
30th percentile	.37**	.03	−.02	.00	.19*
	[.12, .63]	[−.12, .19]	[−.04, .01]	[−.07, .08]	[.01, .37]
40th percentile	.43**	.00	−.03*	.00	.01
	[.19, .68]	[−.15, .14]	[−.05, −.00]	[−.07, .07]	[−.19, .21]
50th percentile	.52**	−.06	−.02	.10	.10
	[.28, .76]	[−.21, .08]	[−.05, .00]	[−.04, .10]	[−.11, .30]
60th percentile	.54**	−.07	−.01	.02	.10
	[.31, .77]	[−.21, .07]	[−.03, .01]	[−.04, .09]	[−.09, .28]
70th percentile	.47**	−.04	−.01	−.01	.11
	[.25, .69]	[−.17, .09]	[−.03, .01]	[−.08, .06]	[−.09, .32]
80th percentile	.28*	.09	−.01	−.07	.09
	[.07, .50]	[−.05, .23]	[−.03, .02]	[−.14, .01]	[−.03, .20]
90th percentile	.21	.12	−.01	−.10	.05
	[−.12, .55]	[−.08, .32]	[−.04, .02]	[−.19, .00]	[−.16, .27]
N (pairs)			1,370 (685)		

Note. 95% confidence intervals corrected for within-family clustering and presented in brackets.
*$p \leq .05$. **$p \leq .01$.

Table 3. In total, nine quantiles were examined, ranging from .10 to .90 in increments of .10.[5] The results revealed that changes in h^2 did indeed follow a nonlinear pattern across levels of self-control, with heritability estimates ranging between 6% and 54%. Genetic influences on self-control appear to be strongest in the middle of the distribution, peaking at 54% (60th percentile). Conversely, genetic influences were weakest in the tails of the distribution with h^2 estimates of only 6% and 21% for the 10th and 90th percentiles, respectively. The quantile regression equations also examined changes in c^2 across levels of self-control; however, all of the estimates were nonsignificant aside from the estimate for the 10th percentile which was significant ($p = .051$) and indicated that 23% of the variance in self-control was explained by shared environmental influences.

In an effort to provide a visual presentation of the quantile regression results, h^2 and c^2 estimates (along with the accompanying 95% confidence intervals) are plotted for each examined quantile in Figure 1. Importantly, it is not possible to compare coefficients across quantiles of self-control using conventional methods such as nonoverlapping 95% confidence intervals or Wald's tests as the entire sample is used to estimate each coefficient, resulting in interdependence among the resulting estimates (Logan et al., 2012; Petscher & Kim, 2011). Rather, the confidence intervals that accompany each coefficient simply indicate whether they significantly differ from 0. For these reasons, the estimates presented in the figure are intended to provide a visual illustration of the patterns of genetic and shared environmental influences across the distribution of self-control, not aid in the comparison between specific coefficients.

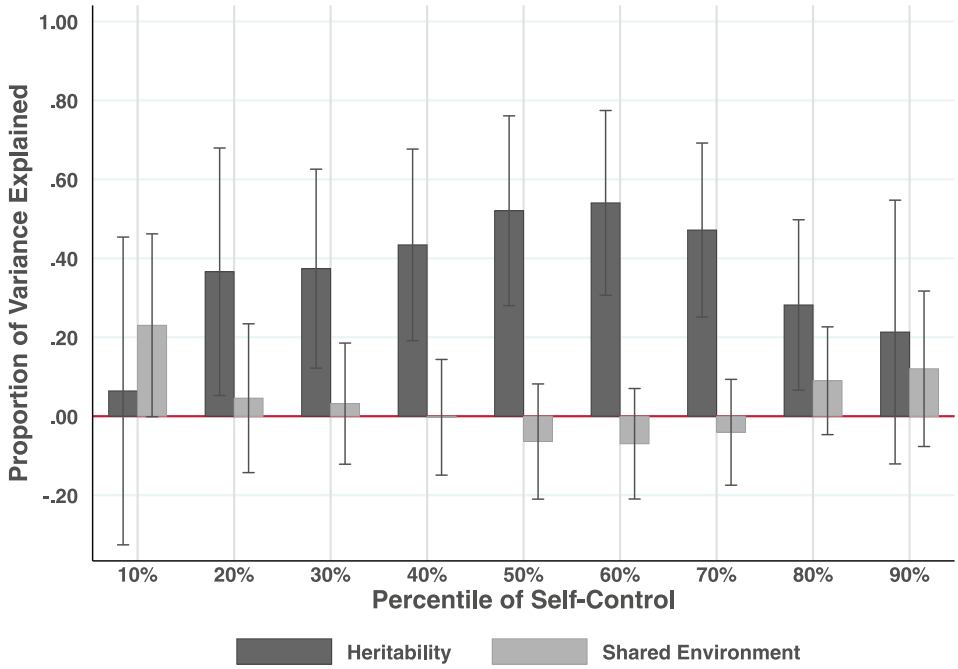

Figure 1: Results of the Biometric Quantile Regression Models
Note. Error bars represent corrected 95% confidence intervals for the corresponding heritability and shared environmental estimates. Estimates with error bars that do not cross the horizontal red reference line (.00) are significant at the $p < .05$ level. All estimates adjusted for age, gender, and race.

DISCUSSION

Despite the overwhelming number of studies examining various aspects of the concept of self-control and how it ultimately manifests as behavioral variation, much remains unknown. For example, while previous studies have identified self-control as one of the most robust and consistent correlates of delinquent behavior (Fine et al., 2016; Goode, 2008; Meier et al., 2008; Pratt & Cullen, 2000), only recently have studies begun to examine the functional form of this association (Mears et al., 2013; Sullivan & Loughran, 2014). The evidence flowing from this developing literature remains mixed, but this line of inquiry raises important questions regarding the more intricate features of the association between self-control and delinquency. Directly in line with these observations, the current study aimed to contribute to the existing literature by providing a more nuanced investigation of the genetic and environmental influences on the etiological development of self-control. More specifically, the current study made use of biometric quantile regression equations to estimate genetic and environmental influences across the distribution of self-control. The results revealed three primary findings, all of which will be discussed in more detail.

First, the results of the modified DF equations and biometric quantile regression models revealed that genetic influences are distributed across levels of self-control in a nonlinear pattern. More specifically, genetic influences explained the greatest amount of variance around the median (50th-60th percentile) of the distribution, accounting for between 52%

and 54% of the variance. Conversely, genetic influences were weakest in the tails of the distribution and were particularly weak at the lowest examined levels of self-control (bottom 10th percentile). Collectively, this pattern of findings provides preliminary evidence suggesting that the factors that ultimately contribute to the development of self-control may vary across the distribution. Based on these observations, sources of environmental influence that have been previously implicated in the development of self-control require additional inquiry. These findings do not necessarily indicate that such sources of influence are any less meaningful, but, rather, that such factors may be more intricately involved with the etiological development of self-control than previously assumed. For example, previous studies have identified school- and neighborhood-level factors as meaningful influences that contribute to the development of self-control (Gibson et al., 2010; Pratt et al., 2004; Turner et al., 2005). The results of the current study do not undermine this pattern of results, but instead indicate that such influences may account for more variance among adolescents with relatively high and low overall levels of self-control. Future research would benefit from a more thorough investigation of the manner in which previously identified sources of influence differentially contribute to self-control across the distribution.

The maximization of genetic influences around the median of self-control also has implications for genetically informed studies interested in isolating measured sources of environmental influence on self-control. As the analytic techniques typically used in such studies assume a flat distribution of genetic and environmental influences (Carey, 2003; Neale & Maes, 1992; Plomin et al., 2013), it remains possible that such studies may be less likely to effectively identify specific sources of environmental influence. Stated differently, environmental influences more directly involved in the development of relatively high or low levels of self-control may be less influential around the median, where genetic influences are maximized. Directly in line with this observation, previous studies have failed to find a significant association between parental socialization and the development of self-control after controlling for genetic influences (Wright & Beaver, 2005; Wright et al., 2008). While these findings have offered greater insight into the development of self-control, the observed null findings may be confined to specific areas of the distribution in which genetic influences are greatest (i.e., the median). While genetically informed research designs represent some of the most powerful quasi-experimental analytic tools available (Johnson et al., 2009; Larsson, 2016; Turkheimer & Harden, 2014), the underlying functional form of the examined phenotypes has a direct bearing on the results flowing from such designs.

The third and final finding to emerge from the current study was that while genetic influences are maximized in the middle of the distribution of self-control scores, environmental influences appear to be maximized in the tails. Importantly, the estimated quantile regression models also estimated shared environmental influences across the specified quantiles. The results revealed that shared environmental influences were largely inconsequential, with the only exception being the c^2 estimate for the 10th percentile of self-control (but at $p = .051$). The overall lack of shared environmental influence is a common occurrence in biometric modeling strategies. Directly in line with this observation, the results of a recent comprehensive meta-analysis of virtually all twin studies over the past 50 years examining more than 17,000 phenotypes from nearly 3,000 studies and including more than 14 million twin pairs revealed that nearly half of the overall variance in the observed phenotypes was explained by genetic influences and shared environmental influences explained little to no variance (Polderman et al., 2015). While shared environmental influences on self-control

and related concepts have been found to be relatively limited (Anokhin et al., 2011; Friedman et al., 2011; Fulker et al., 1980; Miles et al., 2001; Niv et al., 2012; Wright & Beaver, 2005), such sources of influence continue to be emphasized in studies examining the etiological development of self-control (Cullen et al., 2008; Hay, 2001; Pratt et al., 2004; Turner et al., 2005; Unnever et al., 2003). These findings further emphasize the need to consider additional sources of influence that ultimately contribute to the development of self-control.

The lack of shared environmental influences does not indicate that self-control is free from environmental influences altogether; rather, these findings indicate that environments shared between siblings from the same household that work to make them more similar have little to no influence. In turn, these findings indicate that nonshared environmental influences seem to be centrally implicated in the development of self-control, particularly for individuals who fall within the extreme tails of the distribution. The importance of nonshared environmental influences has been noted in behavior genetics for quite some time (Turkheimer & Waldron, 2000), and the identification of specific nonshared environmental influences continues to occupy a sizable portion of the contemporary literature (Turkheimer & Harden, 2014). Unfortunately, the employed DF equations (including the biometric quantile regression equation) do not directly model nonshared environmental influences as in other biometric techniques (such as biometric regression analyses; Turkheimer & Harden, 2014). Rather, all residual variance in the examined phenotype that is not explained by h^2 or c^2 is lumped into the resulting error term, which includes nonshared environmental influences and measurement error. This is an important limitation, as nonshared environmental influences have been found to be highly influential on a wide range of phenotypes (Turkheimer, 2000), including self-control (Anokhin et al., 2011; Friedman et al., 2011; Fulker et al., 1980; Miles et al., 2001; Niv et al., 2012; Wright & Beaver, 2005). In this way, future research would benefit from a more directed effort toward identifying specific aspects of nonshared environmental influences that shape self-control, particularly at the extremes of the distribution.

These findings also have direct implications for both prevention and intervention efforts moving forward, in that such patterns suggest programming aimed at modifying environmental influences experienced by children and adolescents with relatively low levels of self-control may result in increased levels of success, even after taking into account genetic influences. These findings highlight the continued use of intervention programs involving elements of cognitive behavioral therapy (CBT; Vaske, Galyean, & Cullen, 2011) or other components aimed at minimizing automatic associations, changing attentional biases, and modifying approach tendencies (for more information, see Friese, Hofmann, & Wiers, 2011). While previous research has evaluated the potential impact of intervention programming aimed at improving self-control (for example, see Na & Paternoster, 2012), future research would benefit from continuing these efforts within the confines of a genetically informed research design and with a careful eye toward targeting those groups of individuals with the greatest levels of responsivity toward such programs.

Despite these contributions to the existing literature, the current study is not without limitation. First, the current study relies on a sample of high school students (the Add Health), and as Sullivan and Loughran (2014) pointed out the "lower ranges of the self-control distribution may not be well populated" (p. 710). While quantile regression allows for the isolation of specific quantiles within the distribution, the underrepresentation of individuals with extremely low levels of self-control remains a limitation inherent to the Add Health sample. Future

research would benefit from the investigation of genetic influences across the distribution of self-control within more high-risk samples that also contain twins and/or sibling pairs. Second, the current study relied on a single measure of self-control. While the same measure has been used previously (Boisvert et al., 2012; Mears et al., 2013; Yun et al., 2014), and studies have revealed that both attitudinal and behavioral measures of self-control tend to yield similar results (Pratt & Cullen, 2000), other studies have revealed additional measurement strategies. For example, studies have proposed multifaceted measurement strategies that identify multiple dimensions of self-control including impulse control and sensation seeking (Whiteside & Lynam, 2001), while other studies have employed experimental measures of self-control (Bechara et al., 2000; Brand et al., 2005). Future research would benefit from an attempt to triangulate the findings from the current study using additional measures of self-control. Third, the genetic and environmental influences estimated in the current study are latent and do not provide insight into the specific genetic polymorphisms or nonshared environmental influences that may be implicated in the observed patterns of influence. Recent developments in behavior genetics such as genome-wide association studies (GWAS) and genetic complex trait analysis (GCTA) are rapidly advancing and may assist in identifying specific genes implicated in the association between self-control and delinquency. Fourth, and as mentioned above, the employed DF equations do not allow for the direct estimation of nonshared environmental influences. Future research would benefit from the development of additional modeling strategies that more directly assess nonshared environmental influences, particularly based on the findings from the current study that further underscore the importance of the nonshared environment in the development of self-control.

The results of the current study demonstrate not only the importance of taking both genetic and environmental influences into account but also making use of both sets of influences to gain a better understanding of the underlying mechanisms involved in phenotypic development and well-established associations between phenotypes that are common to the field of criminology. Recently, behavioral geneticists have made calls for behavioral scientists to move "beyond heritability" and maximize the potential of twin-based methodologies (Johnson et al., 2009; Turkheimer & Harden, 2014). In this way, the importance of heritability studies is no longer to determine whether genetic influences play a role in the development of behavioral phenotypes, but, rather, to use such estimates in a manner that provides a more comprehensive understanding of such phenotypes. The "First Law of Behavior Genetics" (Turkheimer, 2000) and the results of the recent comprehensive meta-analysis (Polderman et al., 2015) indicate that all behavioral phenotypes are the result of genetic and environmental influences. This finding underlines the importance of estimating genetically informed models in an effort to minimize model misspecification stemming from genetic confounding in the interest of better isolating environmental influences (Turkheimer & Harden, 2014), while also extending traditional twin-based modeling strategies in an effort to better understand the manner in which both sets of influences ultimately shape phenotypes and associations. The current study provides an additional example of the manner in which such strategies can be used to thoroughly examine concepts and associations directly related to theoretical perspectives common within the behavioral sciences.

NOTES

1. This observation would be similar to the concept of a gene–environment interaction (G × E), in which genetic influences on a given phenotype are effectively moderated by an observed environmental influence (or vice versa). In this way, genetic

influences on the examined phenotype may fluctuate across levels of the examined environmental influence. While G × E offers one potential explanation for variability of genetic and environmental influences across levels of a given phenotype, such variability does not require G × E. For example, children who ultimately develop overall lower levels of self-control may be less likely to have experienced adequate levels of parental attachment, while children with greater overall levels of self-control may have been more likely to be enrolled in schools with more prosocial peers. In this way, different aspects of environmental influence may be implicated or more potent at different points in the distribution of self-control. While such differences may be the result of G × Es, such an assumption is not necessary in all cases. As h^2 and c^2 are estimated as proportions of overall variance in the examined phenotype, the differential emphasis on one component will effectively change the other; however, such patterns of results do not necessarily indicate that genetic influences are moderated, as environmental influences may simply manifest at different levels of the examined phenotype. In addition, the statistical equations employed in the current study are not equipped to examine G × Es. While further modification of these equations is possible, previous studies have noted the difficulty of modeling G × Es within a DeFries–Fulker (DF) framework (Purcell, 2002).

2. We thank one of the anonymous reviewers for providing a similar illustrative example when describing Rowe's (2001) set point theory.

3. Race was dichotomized in the current study because of the limited amount of variability in the other race categories observed in the Add Health twin subsample. The models estimated in the primary analysis were reestimated with race measured as a series of dummy variables (with Caucasian as the reference category), and the overall pattern of results did not differ from those presented in the current study. For these reasons, we have retained the dichotomized race variable in the current study.

4. In Equation 2, β_5 and β_6 look similar to quadratic interaction terms included in traditional regression models to examine nonlinear trends. However, as DF analysis regresses a cosibling's score on a given phenotype on their proband's score on the same phenotype, the resulting interpretation is different. More specifically, the resulting coefficients can be interpreted as the extent to which shared environmental and genetic influences are a linear function of the proband's score on the examined phenotype. Additional information regarding the estimation of the modified DF equations and the interpretation of the resulting coefficients can be found elsewhere (Cherny et al., 1992).

5. Due to a lack of theoretical development regarding an appropriate number of quantiles to examine, additional models examining fewer and greater numbers of quantiles were also estimated. For example, additional models examining 17 quantiles ranging between .10 and .90 in increments of .05, and four quantiles ranging between .20 and .80 in increments of .20 were estimated. All of the additional models produced results that fell directly in line with the models presented in the main analysis and did not diverge in any meaningful way. In an effort to balance model parsimony and comprehensiveness, we ultimately settled on the estimation of nine quantiles.

REFERENCES

Anokhin, A. P., Golosheykin, S., Grant, J. D., & Heath, A. C. (2011). Heritability of delay discounting in adolescence: A longitudinal twin study. *Behavior Genetics*, *41*, 175-183. doi:10.1007/s10519-010-9384-7

Bechara, A., Tranel, D., & Damasio, H. (2000). Characterization of the decision-making deficit of patients with ventromedial prefrontal cortex lesions. *Brain*, *123*, 2189-2202. doi:10.1093/brain/123.11.2189

Boisvert, D., Wright, J. P., Knopik, V., & Vaske, J. (2012). Genetic and environmental overlap between low self-control and delinquency. *Journal of Quantitative Criminology*, *28*, 477-507. doi:10.1007/s10940-011-9150-x

Brand, M., Fujiwara, E., Borsutzky, S., Kalbe, E., Kessler, J., & Markowitsch, H. J. (2005). Decision-making deficits of korsakoff patients in a new gambling task with explicit rules: Associations with executive functions. *Neuropsychology*, *19*, 267-277. doi:10.1037/08944105.19.3.267

Carey, G. (2003). *Human genetics for the social sciences*. Thousand Oaks, CA: SAGE.

Cherny, S. S., Cardon, L. R., Fulker, D. W., & DeFries, J. C. (1992). Differential heritability across levels of cognitive ability. *Behavior Genetics*, *22*, 153-162.

Coghill, D., & Sonuga-Barke, E. J. (2012). Categories versus dimensions in the classification and conceptualisation of child and adolescent mental disorders: Implications of recent empirical study. *Journal of Child Psychology and Psychiatry*, *53*, 469-489. doi:10.1111/j.1469-7610.2011.02511.x

Cullen, F. T., Unnever, J. D., Wright, J. P., & Beaver, K. M. (2008). Parenting and self-control. In E. Goode (Ed.), *Out of control: Assessing the general theory of crime* (pp. 61-76). Stanford, CA: Stanford University Press.

DeFries, J. C., & Fulker, D. W. (1985). Multiple regression analysis of twin data. *Behavior Genetics*, *15*, 467-473.

DeFries, J. C., & Fulker, D. W. (1988). Multiple regression analysis of twin data: Etiology of deviant scores versus individual differences. *Acta Geneticae Medicae et Gemellologiae: Twin Research*, *37*, 205-216.

Evans, T. D., Cullen, F. T., Burton, V. S., Dunaway, R. G., & Benson, M. L. (1997). The social consequences of self-control: Testing the general theory of crime. *Criminology*, *35*, 475-504. doi:10.1111/j.1745-9125.1997.tb01226.x

Fine, A., Steinberg, L., Frick, P. J., & Cauffman, E. (2016). Self-control assessments and implications for predicting adolescent offending. *Journal of Youth and Adolescence*, *45*, 701-712. doi:10.1007/s10964-016-0425-2

Frazier, T. W., Thompson, L., Youngstrom, E. A., Law, P., Hardan, A. Y., Eng, C., & Morris, N. (2014). A twin study of heritable and shared environmental contributions to autism. *Journal of Autism and Developmental Disorders*, *44*, 2013-2025. doi:10.1007/s10803-014-2081-2

Friedman, N. P., Miyake, A., Robinson, J. L., & Hewitt, J. K. (2011). Developmental trajectories in toddlers' self-restraint predict individual differences in executive functions 14 years later: A behavioral genetic analysis. *Developmental Psychology*, *47*, 1410-1430. doi:10.1037/a0023750

Friese, M., Hofmann, W., & Wiers, R. W. (2011). On taming horses and strengthening riders: Recent developments in research on interventions to improve self-control in health behaviors. *Self and Identity*, *10*, 336-351. doi:10.1080/15298868.2010.536417

Fulker, D. W., Eysenck, S. B., & Zuckerman, M. (1980). A genetic and environmental analysis of sensation seeking. *Journal of Research in Personality*, *14*, 261-281.

Gibson, C. L., Sullivan, C. J., Jones, S., & Piquero, A. R. (2010). "Does it take a village?" Assessing neighborhood influences on children's self-control. *Journal of Research in Crime and Delinquency*, *47*, 31-62.

Goode, E. (2008). *Out of control: Assessing the general theory of crime*. Stanford, CA: Stanford University Press.

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

Grasmick, H. G., Tittle, C. R., Bursik, R. J., & Arneklev, B. J. (1993). Testing the core empirical implications of Gottfredson and Hirschi's general theory of crime. *Journal of Research in Crime & Delinquency*, *30*, 5-29. doi:10.1177/0022427893030001002

Greven, C. U., Merwood, A., Meer, J. M., Haworth, C., Rommelse, N., & Buitelaar, J. K. (2016). The opposite end of the attention deficit hyperactivity disorder continuum: Genetic and environmental aetiologies of extremely low ADHD traits. *Journal of Child Psychology and Psychiatry*, *57*, 523-531. doi:10.1111/jcpp.12475

Harris, K. M. (2011). *Design features of Add Health*. Chapel Hill: Carolina Population Center, University of North Carolina.

Harris, K. M., Florey, F., Tabor, J., Bearman, P. S., Jones, J., & Udry, J. R. (2009). *The National Longitudinal Study of Adolescent Health: Research design*. Chapel Hill: Carolina Population Center, University of North Carolina, 2003. Retrieved from http://www.cpc.unc.edu/projects/addhealth/design

Harris, K. M., Halpern, C. T., Smolen, A., & Haberstick, B. C. (2006). The National Longitudinal Study of Adolescent Health (Add Health) twin data. *Twin Research and Human Genetics*, *9*, 988-997.

Hay, C. (2001). Parenting, self-control, and delinquency: A test of self-control theory. *Criminology*, *39*, 707-736. doi:10.1111/j.1745-9125.2001.tb00938.x

Hay, C., & Forrest, W. (2006). The development of self-control: Examining self-control theory's stability hypothesis. *Criminology*, *44*, 739-774. doi:10.1111/j.1745-9125.2006.00062.x

Heckman, J. J. (1979). Sample selection bias as a specification error. *Econometrica*, *47*, 153-161.

Johnson, W., Turkheimer, E., Gottesman, I. I., & Bouchard, T. J. (2009). Beyond heritability twin studies in behavioral research. *Current Directions in Psychological Science*, *18*, 217-220. doi:10.1111/j.1467-8721.2009.01639.x

Knopik, V. S., Alarcón, M., & DeFries, J. C. (1997). Comorbidity of mathematics and reading deficits: Evidence for a genetic etiology. *Behavior Genetics*, *27*, 447-453. doi:10.1023/A:1025622400239

Larsson, H. (2016). Commentary: How can family-based quasi-experimental designs and national registers be used to address confounding in risk factor studies of psychopathology? A reflection on Obel et al. (2016). *Journal of Child Psychology and Psychiatry*, *57*, 538-539. doi:10.1111/jcpp.12519

Larsson, H., Anckarsater, H., Råstam, M., Chang, Z., & Lichtenstein, P. (2012). Childhood attention-deficit hyperactivity disorder as an extreme of a continuous trait: A quantitative genetic study of 8,500 twin pairs. *Journal of Child Psychology and Psychiatry*, *53*, 73-80. doi:10.1111/j.1469-7610.2011.02467.x

Logan, J. A., Petrill, S. A., Hart, S. A., Schatschneider, C., Thompson, L. A., Deater-Deckard, K., . . . Bartlett, C. (2012). Heritability across the distribution: An application of quantile regression. *Behavior Genetics*, *42*, 256-267. doi:10.1007/s10519-011-9497-7

Marcus, B. (2004). Self-control in the general theory of crime: Theoretical implications of a measurement problem. *Theoretical Criminology*, *8*, 33-55. doi:10.1177/1362480604039740

Mears, D. P., Cochran, J. C., & Beaver, K. M. (2013). Self-control theory and nonlinear effects on offending. *Journal of Quantitative Criminology*, *29*, 447-476. doi:10.1007/s10940-012-9187-5

Meier, M. H., Slutske, W. S., Arndt, S., & Cadoret, R. J. (2008). Impulsive and callous traits are more strongly associated with delinquent behavior in higher risk neighborhoods among boys and girls. *Journal of Abnormal Psychology*, *117*, 377-385. doi:10.1037/0021-843X.117.2.377

Miles, D. R., van den Bree, M. B., Gupman, A. E., Newlin, D. B., Glantz, M. D., & Pickens, R. W. (2001). A twin study on sensation seeking, risk taking behavior and marijuana use. *Drug and Alcohol Dependence*, *62*, 57-68. doi:10.1016/S0376-8716(00)00165-4

Moffitt, T. E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R. J., Harrington, H., . . . Caspi, A. (2011). A gradient of childhood self-control predicts health, wealth, and public safety. *Proceedings of the National Academy of Sciences*, *108*, 2693-2698. doi:10.1073/pnas.1010076108

Na, C., & Paternoster, R. (2012). Can self-control change substantially over time? Rethinking the relationship between self- and social control. *Criminology*, *50*, 427-462. doi:10.1111/j.1745-9125.2011.00269.x

Neale, M. C., & Maes, H. H. M. (1992). *Methodology for genetic studies of twins and families*. Dordrecht, The Netherlands: Kluwer Academic.

Nedelec, J. L., & Beaver, K. M. (2014). The relationship between self-control in adolescence and social consequences in adulthood: Assessing the influence of genetic confounds. *Journal of Criminal Justice*, *42*, 288-298. doi:10.1016/j.jcrimjus.2014.02.002

Niv, S., Tuvblad, C., Raine, A., Wang, P., & Baker, L. A. (2012). Heritability and longitudinal stability of impulsivity in adolescence. *Behavior Genetics*, *42*, 378-392. doi:10.1007/s10519-011-9518-6

Petscher, Y., & Kim, Y. S. (2011). The utility and accuracy of oral reading fluency score types in predicting reading comprehension. *Journal of School Psychology*, *49*, 107-129. doi:10.1016/j.jsp.2010.09.004

Petscher, Y., & Logan, J. A. (2014). Quantile regression in the study of developmental sciences. *Child Development*, *85*, 861-881. doi:10.1111/cdev.12190

Piquero, A. R., & Bouffard, J. A. (2007). Something old, something new: A preliminary investigation of Hirschi's redefined self-control. *Justice Quarterly*, *24*, 1-27. doi:10.1080/07418820701200935

Plomin, R., DeFries, J. C., Knopik, V. S., & Neiderhiser, J. M. (2013). *Behavioral genetics* (6th ed.). New York, NY: Worth Publishers.

Plomin, R., & Kovas, Y. (2005). Generalist genes and learning disabilities. *Psychological Bulletin*, *131*, 592-617. doi:10.1037/0033-2909.131.4.592

Polderman, T. J., Benyamin, B., de Leeuw, C. A., Sullivan, P. F., van Bochoven, A., Visscher, P. M., & Posthuma, D. (2015). Meta-analysis of the heritability of human traits based on fifty years of twin studies. *Nature Genetics*, *47*, 702-709. doi:10.1038/ng.3285

Pratt, T. C., & Cullen, F. T. (2000). Empirical status of Gottfredson and Hirschi's General Theory of Crime: A meta-analysis. *Criminology*, *38*, 931-964. doi:10.1111/j.1745-9125.2000.tb00911.x

Pratt, T. C., Turner, M. G., & Piquero, A. R. (2004). Parental socialization and community context: A longitudinal analysis of the structural sources of low self-control. *Journal of Research in Crime & Delinquency*, *41*, 219-243. doi:10.1177/0022427803260270

Purcell, S. (2002). Variance components models for gene-environment interaction in twin analysis. *Twin Research*, *5*, 554-571.

Ray, J. V., Jones, S., Loughran, T. A., & Jennings, W. G. (2013). Testing the stability of self-control: Identifying unique developmental patterns and associated risk factors. *Criminal Justice and Behavior*, *40*, 588-607. doi:10.1177/0093854812464222

Rowe, D. C. (2001). Do people make environments or do environments make people? *Annals of the New York Academy of Sciences*, *935*, 62-74. doi:10.1111/j.1749-6632.2001.tb03471.x

Scarr, S. (1992). Developmental theories for the 1990s: Development and individual differences. *Child Development*, *63*, 1-19. doi:10.1111/j.1467-8624.1992.tb03591.x

Seroczynski, A. D., Bergeman, C. S., & Coccaro, E. F. (1999). Etiology of the impulsivity/aggression relationship: Genes or environment? *Psychiatry Research*, *86*, 41-57. doi:10.1016/S0165-1781(99)00013-X

Smith, K. B., & Hatemi, P. K. (2013). OLS is AOK for ACE: A regression-based approach to synthesizing political science and behavioral genetics models. *Political Behavior*, *35*, 383-408. doi:10.1007/s11109-012-9192-2

StataCorp. (2013). Stata statistical software: Release 13. College Station, TX: StataCorp LP.

Sullivan, C. J., & Loughran, T. (2014). Investigating the functional form of the self-control-delinquency relationship in a sample of serious young offenders. *Journal of Quantitative Criminology*, *30*, 709-730. doi:10.1007/s10940-014-9220-y

Tangney, J. P., Baumeister, R. F., & Boone, A. L. (2004). High self-control predicts good adjustment, less pathology, better grades, and interpersonal success. *Journal of Personality*, *72*, 271-324. doi:10.1111/j.0022-3506.2004.00263.x

Turkheimer, E. (2000). Three laws of behavior genetics and what they mean. *Current Directions in Psychological Science*, *9*, 160-164. doi:10.1111/1467-8721.00084

Turkheimer, E., & Harden, K. P. (2014). Behavior genetic research methods: Testing quasi-causal hypotheses using multivariate twin data. In H. T. Reis & C. M. Judd (Eds.), *Handbook of research methods in personality and social psychology* (pp. 159-187). New York, NY: Cambridge University Press.

Turkheimer, E., & Waldron, M. (2000). Nonshared environment: A theoretical, methodological, and quantitative review. *Psychological Bulletin*, *126*, 78-108. doi:10.1037/0033-2909.126.1.78

Turner, M. G., Piquero, A. R., & Pratt, T. C. (2005). The school context as a source of self-control. *Journal of Criminal Justice*, *33*, 327-339. doi:10.1016/j.jcrimjus.2005.04.003

Udry, J. R. (2003). *The National Longitudinal Study of Adolescent Health (Add Health), Waves I and II, 1994-1996; Wave III, 2002-2002*. Chapel Hill: Carolina Population Center, University of North Carolina at Chapel Hill.

Unnever, J. D., Cullen, F. T., & Pratt, T. C. (2003). Parental management, ADHD, and delinquent involvement: Reassessing Gottfredson and Hirschi's general theory. *Justice Quarterly*, *20*, 471-500. doi:10.1080/07418820300095591

Vaske, J., Galyean, K., & Cullen, F. T. (2011). Toward a biosocial theory of offender rehabilitation: Why does cognitive-behavioral therapy work? *Journal of Criminal Justice*, *39*, 90-102. doi:10.1016/j.jcrimjus.2010.12.006

Whiteside, S. P., & Lynam, D. R. (2001). The Five Factor Model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences*, *30*, 669-689. doi:10.1016/S0191-8869(00)00064-7

Willcutt, E. G. (2005). The etiology of ADHD: Behavioral and molecular genetic approaches. In D. Barch (Ed.), *Cognitive and affective neuroscience of psychopathology* (pp. 2-34). Oxford, UK: Oxford University Press.

Willcutt, E. G., Pennington, B. F., & DeFries, J. C. (2000). Etiology of inattention and hyperactivity/impulsivity in a community sample of twins with learning difficulties. *Journal of Abnormal Child Psychology*, *28*, 149-159.

Wright, J. P., & Beaver, K. M. (2005). Do parents matter in creating self-control in their children? A genetically informed test of Gottfredson and Hirschi's theory of low self-control. *Criminology*, *43*, 1169-1202. doi:10.1111/j.1745-9125.2005.00036.x

Wright, J. P., Beaver, K. M., Delisi, M., & Vaughn, M. (2008). Evidence of negligible parenting influences on self-control, delinquent peers, and delinquency in a sample of twins. *Justice Quarterly*, *25*, 544-569. doi:10.1080/07418820701864599

Yancey, J. R., Venables, N. C., Hicks, B. M., & Patrick, C. J. (2013). Evidence for a heritable brain basis to deviance-promoting deficits in self-control. *Journal of Criminal Justice*, *41*, 309-317. doi:10.1016/j.jcrimjus.2013.06.002

Yun, I., Cheong, J., & Walsh, A. (2014). The relationship between academic achievement and likelihood of police arrest among delinquents. *International Journal of Offender Therapy and Comparative Criminology*, *58*, 607-631. doi:10.1177/0306624X13482882

Joseph A. Schwartz is an assistant professor in the School of Criminology and Criminal Justice at the University of Nebraska Omaha. His research interests include behavior genetics, developmental/life-course criminology, and additional factors involved in the etiology of criminal behavior. He is also a co-founder and the current Treasurer/Secretary of the Biosocial Criminology Association (www.biosocialcrim.org).

Eric J. Connolly is an assistant professor in the Department of Criminal Justice and Criminology at Sam Houston State University. His research interests include biosocial criminology, criminological theory, and developmental/life-course criminology. He is president of the Biosocial Criminology Association (Website: www.biosocialcrim.org; Twitter: @biosocialcrim).

Joseph L. Nedelec is an assistant professor in the School of Criminal Justice at the University of Cincinnati. His primary research interests lie within biosocial criminology, evolutionary psychology, behavioral genetics, and cybercrime. He is co-founder and vice president of the Biosocial Criminology Association (www.biosocialcrim.org).

Kevin M. Beaver is the Judith Rich Harris Professor of Criminology at Florida State University and visiting distinguished research professor in the Center for Social and Humanities Research at King Abdulaziz University.