THE ASSOCIATION BETWEEN PARENTING AND LEVELS OF SELF-CONTROL

A Genetically Informative Analysis

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A growing body of criminological research has tested Gottfredson and Hirschi’s parental management thesis that highlights the causal role that parents play in shaping their child’s level of self-control. Although the results of these studies appear to provide support for the parental management thesis, in general, they all fail to adequately control for genetic factors and child-driven effects, which may result in biased findings. The current study addresses these limitations by analyzing a sample of twin pairs drawn from the National Longitudinal Study of Adolescent Health. Following quantitative genetic analysis, the results revealed that after taking into account genetic factors and child-driven effects, none of the covariance between parental management techniques and levels of low self-control was explained by parental socialization. The importance of these findings for criminological research examining the influence of parents on self-control specifically and antisocial behaviors generally is discussed.

Keywords: Add Health; genetics; low self-control; parenting; twins

Gottfredson and Hirschi’s (1990) general theory of crime has left an indelible mark on the study of crime, delinquency, and antisocial behavior in general. Their theory, which identified individual differences in levels of self-control as the cause of crime, has guided a wealth of criminological scholarship during the past 20 years. Out of this line of literature, low levels of self-control have been found to be predictive of criminal and delinquent involvement across a wide array of heterogeneous studies (Gottfredson, 2006; Hirschi & Gottfredson, 2008). Perhaps the most impressive evidence linking levels of self-control to antisocial behavior came from a meta-analysis that found measures of self-control...
to be among the strongest, most consistent, and most robust predictors of a range of delinquent and criminal acts (Pratt & Cullen, 2000). As a result of the strong connection between self-control and crime, criminologists have recently begun to examine the factors that might be responsible for causing variation in levels of self-control (Cullen, Unnever, Wright, & Beaver, 2008). To do so, they have turned their attention to what is widely known as the parental management thesis (Gottfredson & Hirschi, 1990).

Central to Gottfredson and Hirschi’s (1990) parental management thesis is the family, especially ineffective parental socialization tactics. According to Gottfredson and Hirschi, parents who wish to raise children with high levels of self-control must engage in at least three main parental management techniques. First, parents must supervise their children. Second, during their supervision, parents must recognize when their children are misbehaving. Third, parents must appropriately punish and correct their children’s transgressions. All else equal, parents who dutifully follow these parental management techniques will, on average, raise children with relatively high levels of self-control. Parents, in contrast, who are not as adept at these three parenting techniques will tend to raise children with comparatively lower levels of self-control.

An emerging line of research has analyzed an array of samples and employed various measures of parenting to examine the veracity of the parental management thesis. For example, studies have examined whether measures of parental monitoring and supervision, authoritative parenting, consistency in parenting, parental attachment, and parental efficacy are predictive of levels of self-control (Cullen et al., 2008). The findings flowing from these studies have provided evidence purportedly supporting the parental management thesis (for a general overview of findings, see Cullen et al., 2008); however, when examined through a biosocial lens, the results of these studies may not be as unambiguous as they initially appear. Of particular importance is that studies testing the parental management thesis fail to account for the role of genetic factors in the etiology of self-control. This is a serious oversight, because genetic factors could be driving the covariation between parenting and levels of self-control. The only way to test this possibility is by analyzing genetically informative samples that include kinship pairs of varying levels of genetic relatedness (J. R. Harris, 1998; Wright & Beaver, 2005). Unfortunately, criminological research rarely uses genetically sensitive research designs.

The current study addresses this gap in the criminological literature by examining the association between parenting and levels of self-control in a genetically sensitive sample. Specifically, our research is guided by two interrelated research issues. First, we examine whether there is a significant association between parenting measures and measures of low self-control. Second, we decompose the covariation between parenting and low self-control using quantitative genetic analyses to evaluate three different explanations for this association. By doing so, we will be able to examine whether the nexus between parental management techniques and variation in self-control is attributable to genetic factors, environmental factors, or some combination of the two.

EXPLAINING THE ASSOCIATION BETWEEN PARENTING AND LOW SELF-CONTROL

The most common way to test the parental management thesis is by examining the statistical association between measures designed to tap parental management techniques and
measures designed to tap individual variation in levels of low self-control. Any statistically significant association between the parenting measures and the measures of low self-control is frequently interpreted as evidence in support of the parental management thesis. Although this is certainly a plausible interpretation, the data employed in these studies and the statistical techniques used to analyze these data are ill suited to test the parental management thesis. The overarching problem with the methodologies used by criminologists to test the parental management thesis is that they are unable to rule out the possibility that other processes might be driving the association between parenting and levels of self-control. In general, and as detailed below, there are at least three mechanisms that could account for the relationship between parenting and self-control: a causal mechanism, a spurious mechanism, and a child-driven mechanism.

The first mechanism that can account for the association between parenting and levels of self-control is referred to as the causal mechanism. This mechanism is in line with Gottfredson and Hirschi's (1990) parental management thesis, which states that the way in which parents socialize their children will determine their offspring’s level of self-control. For the most part, the causal mechanism (and, by implication, the parental management thesis) is tested by conducting some type of multivariate correlational analysis, where a low self-control scale is used as the dependent variable and parenting measures are entered into the equation as independent variables. Evidence supporting the causal mechanism is assumed to be found when the parenting measures emerge as statistically significant predictors of low self-control. Most studies testing the parental management thesis have used this type of methodology and, in doing so, have found that measures tapping various dimensions of the parental management techniques predict a significant amount of variation in measures of low self-control. At least 12 studies have found evidence in favor of the causal mechanism (see Cullen et al., 2008); however, these correlational studies, in general, are unable to rule out the possibility that other mechanisms, especially the spurious mechanism, might be driving this association (J. R. Harris, 1998; Rowe, 1994; Wright & Beaver, 2005).

The logic of the spurious mechanism holds that the association between parenting and levels of self-control is not causal but is accounted for by a third, unmeasured variable. This unmeasured variable causes variation in both parenting and levels of self-control, which makes it appear as though parenting is causing self-control when, in fact, the two are unrelated. Typically, spurious factors are assumed to be demographic characteristics, such as gender or age, or other criminogenic environments, such as residing in a disadvantaged neighborhood. Criminologists are very meticulous about trying to include all potential confounding variables, especially in research testing the parental management thesis. The one main exception—and one that could seriously bias the results of studies testing the parental management thesis—is that criminologists rarely analyze samples that allow them to control for the effects of genetic factors.

The overarching way in which genes could render the relationship between parenting and levels of self-control spurious is through a process known as a passive gene-environment correlation. Passive gene-environment correlations occur because parents pass along two elements to their children: genetic material and an environment (Plomin, DeFries, & Loehlin, 1977). Because genetic material and the environment are supplied by the same source—that is, parents—they are likely to be correlated. For example, a parent who is highly aggressive will likely transmit to their child the genetic propensity to be aggressive. At the same time, aggressive and violent parents are also likely to engage in ineffective
child-rearing techniques (Farrington & Welsh, 2007). The end result is that the child’s genetic propensities to be aggressive are correlated with his or her (criminogenic) rearing environment. Without controlling for genetic factors in this scenario, any association that was detected between ineffective child rearing and childhood aggression could incorrectly be interpreted as evidence in favor of the causal mechanism when it may really be attributable to a spurious mechanism (J. R. Harris, 1998; Wright & Beaver, 2005).

There are at least four reasons to believe that passive gene-environment correlations are likely to account for at least part of the association between parenting and levels of self-control. First, a number of criminological studies have found measures of low self-control to have a significant genetic component to them (Beaver, DeLisi, Mears, & Stewart, 2009; Beaver, DeLisi, Vaughn, Wright, & Boutwell, 2008; Wright, Beaver, DeLisi, & Vaughn, 2008). Second, there is some evidence indicating that parents who have relatively low levels of self-control engage in ineffective parental management techniques (Boutwell & Beaver, in press; Nofziger, 2008). As a result, it is quite possible that in addition to transmitting genetic propensities for low levels of self-control, parents with low levels of self-control also provide criminogenic home environments for their children. Third, a recent review of behavioral genetic research revealed that parental socialization tactics are modestly influenced by genetic factors (Kendler & Baker, 2007). What this means is that the genes that influence the child’s level of self-control could also be the same genes that are associated with variation in parenting techniques—a classic example of a confounding variable. Fourth, and perhaps most compelling, a handful of studies have revealed that after controlling for genetic factors, the association between parenting and levels of self-control dissipates from statistical significance or is reduced considerably (Wright & Beaver, 2005; Wright et al., 2008).

Taken together, the available evidence tentatively suggests that passive gene-environment correlations may account for part of the covariation between parenting and levels of self-control and may render this association spurious.

The third mechanism that can account for the association between parenting and levels of self-control is a child-driven mechanism (J. R. Harris, 1998; Lytton, 1990; Patterson, 1982). The child-driven mechanism is the exact opposite of the causal mechanism, wherein the child’s level of self-control is posited to be causing parental management techniques rather than vice versa. Prior research exploring the association between antisocial behaviors and parenting have found that this association can be almost entirely accounted for by child-driven processes (Beaver & Wright, 2007; Huh, Tristan, Wade, & Stice, 2006). Criminological research testing the parental management thesis, however, has generally failed to completely account for child-driven effects. Although a number of studies have attempted to control for child-driven effects by controlling for prior levels of self-control or childhood measures of antisocial propensities (e.g., Hay, 2001; Hay & Forrest, 2006), this analytical approach cannot rule out the possibility of child-driven effects. For example, controlling for prior levels of self-control provides information as to whether changes in parenting correspond to changes in self-control, but covariation in these change measures could be the result of the causal mechanism (i.e., changes in parenting techniques produce changes in levels of self-control), the child-driven mechanism (i.e., changes in levels of self-control produce changes in parenting), or some combination of the two.

Child-driven effects can also be the result of genetic factors in a process known as an evocative gene-environment correlation. Evocative gene-environment correlations capture the process by which genetic propensities elicit certain responses from the environment.
(Plomin et al., 1977; Walsh, 2002). For example, compare a child who is aggressive, a trait
that is under partial genetic control (DiLalla, 2002), with his sibling, who is relatively doc-
ile and obedient. In this family, the aggressive child would be more likely than the sibling
to elicit negative reactions from their parents. Statistically speaking, there would be a cor-
relation between parenting and child behavior, and without controlling for genetic predis-
positions, it would have appeared as though the negative parenting style was causing the
child to act aggressively. As such, the possible presence of evocative gene-environment
correlations necessitates controlling for genetic factors when testing the parental manage-
ment thesis. However, because the extant parental management literature does not control
for genetic factors, these studies are unable to rule out the possibility that the covariation
between parenting and levels of self-control is attributable to evocative gene-environment
correlations. Although some of the parental management research has attempted to control
for time-stable differences (e.g., Hay & Forrest, 2006), this analytical approach does not
take into account the fact that genetic effects ebb and flow over time (Ferguson, 2010). As
a result, it remains unknown whether the association between parental management tech-
niques and levels of self-control is driven by evocative gene-environment correlations.

TESTING THE CAUSAL MECHANISM, THE SPURIOUS
MECHANISM, AND THE CHILD-DRIVEN MECHANISM

Criminologists testing the parental management thesis usually employ social science
data sets that are unable to control for genetic factors. Consequentially, it is nearly impos-
sible to disentangle the effects of the causal mechanism from the effects of the spurious and
child-driven mechanisms. To provide a reliable and valid test of these three mechanisms,
criminologists need to follow the lead of behavioral geneticists and analyze samples that
include kinship pairs, especially twin pairs. By analyzing twin pairs, it is possible to esti-
mate the effect of genes and the environment on low self-control, making it possible to test
directly the three mechanisms discussed above. To understand how this is possible, con-
sider that monozygotic (MZ) twins share 100% of their DNA, whereas dizygotic (DZ)
twins share, on average, 50% of their DNA. The only reason that MZ twins should be more
similar to each other (on a measure of low self-control) than DZ twins is because they share
twice as much genetic material.

In general, genetic effects are detected on a phenotype when the similarity of MZ twins
exceeds the similarity of DZ twins. The variance not explained by genetic factors is attrib-
utable to the environment. However, behavioral geneticists make the distinction between
two different types of environments: shared environments and nonshared environments.
Shared environments are environments that are the same between siblings, such as family-
wide parenting techniques and the socioeconomic status of the family. Nonshared environ-
ments, in contrast, are environments that are different between siblings, such as child-specific
parenting techniques and unique peer groups. Shared environments work to make siblings
similar to each other on phenotypes (e.g., low self-control), and nonshared environments
work to make siblings dissimilar to each other on phenotypes. The cumulative effects of
genetic factors, shared environmental factors, and nonshared environmental factors account
for 100% of the variance in the phenotype of interest.

Behavioral genetic research has moved beyond simply estimating the genetic, shared,
and nonshared environmental effects on a single phenotype and instead has also employed
bivariate genetic analysis to examine the relative effects of these three components on the covariation between measures. By using bivariate genetic analysis, it is possible to discern whether genetic factors, shared environmental factors, nonshared environmental factors, or some combination of the three are driving the covariation between the measures.

Bivariate genetic analysis is perhaps the most appropriate methodology that is available to disentangle, whether the covariation between parenting and levels of self-control is attributable to a causal mechanism, a spurious mechanism, or a child-driven mechanism. To do so, the first step is to estimate the covariation between a parenting measure and a measure of low self-control. Figure 1 provides a schematic depiction of the covariation between parenting and self-control, with the overlapping areas representing the covariation. This overlapping area (i.e., the covariation) is then treated as the “measure” of interest and can be subjected to genetic analysis to determine the extent to which genetic, shared environmental, and nonshared environmental factors account for the covariation.

The estimates of genetic, shared environmental, and nonshared environmental effects can then be used to test the three mechanisms that can explain the parenting–low self-control link. Specifically, the shared environmental effect corresponds to the causal mechanism because familywide parental influences would be captured by the shared environmental estimate. The genetic effect corresponds to both the spurious mechanism and the child-driven mechanism because genes are involved in both passive and evocative gene-environment correlations. Unfortunately, the methodology that will be employed in the current study cannot distinguish whether the genetic estimate is more in line with a spurious mechanism or a child-driven mechanism. Last, the nonshared environment corresponds to the child-driven mechanism because the way in which children act differently will produce differences in the way parents react, a process captured by the nonshared environmental component.

THE CURRENT STUDY

The purpose of the current study was twofold. First, we examined the degree to which measures of maternal negativity and paternal negativity correlate with measures of their offspring’s level of low self-control. The findings generated from this set of analyses
represent where most criminological research ends. However, the second goal of our study moved beyond prior tests of the parental management thesis and examined the genetic, shared environmental, and nonshared environmental effects on the covariation between the parenting measures and the low self-control scales. The results culled from these analyses provide specific information as to whether the association between parenting and levels of self-control is attributable to the causal mechanism, the spurious mechanism, or the child-driven mechanism.

**METHOD**

**DATA**

Data for this study come from the National Longitudinal Study of Adolescent Health (Add Health), which is a four-wave, longitudinal, and nationally representative sample of American youths enrolled in 7th through 12th grades during the 1994-1995 school year (Udry, 2003). The Add Health study employed a school-based research design, where a total of 132 middle and high schools were selected for inclusion in the study. Then, all of the students attending these schools were administered a self-report survey during a specified school day. Questions were asked about the student’s home life, demographics, and behaviors. More than 90,000 students participated in this wave of data collection. To gain more detailed information about some of the youths, a subsample of respondents, along with their primary caregivers (usually their mother), was selected to participate in follow-up interviews administered at the adolescent’s home (i.e., the Wave 1 in-home component of the Add Health study). A total of 20,745 adolescents and approximately 17,700 of their primary caregivers were successfully interviewed and were asked questions pertaining to the adolescents’ social relationships, their involvement in risky behaviors, and their personality (K. M. Harris et al., 2003).

Approximately 1.5 years after the Wave 1 data were collected, the second round of questionnaires was administered. Because relatively little time had passed since the Wave 1 interviews and because most of the respondents were still adolescents, the questions asked at Wave 1 were still age appropriate. As a result, the questions asked at the two waves were very similar. For example, adolescents were asked about their participation in acts of delinquency, they were asked about their family and peer relationships, and they were asked about their victimization experiences. A total of 14,738 respondents participated in the Wave 2 surveys. The third wave of interviews was completed between 2001 and 2002. Because most of the respondents were young adults at Wave 3, the questions asked at the previous two waves were changed to include items that were pertinent to young adults. For example, respondents were asked about their marital status, their lifetime contact with the criminal justice system, and their employment history. In total, 15,197 participants were interviewed at Wave 3. Finally, between 2007 and 2008, the fourth wave of data was collected from 15,701 respondents (K. M. Harris et al., 2003).

One of the unique features of the Add Health data is that twin and sibling pairs were oversampled. During Wave 1 interviews, respondents were asked to indicate whether they resided with a cotwin, a half-sibling, a stepsibling, or a cousin. If their sibling was between the ages of 11 and 20 years old, then they were recruited to participate in the study. A probability sample of biological siblings was also nested in the data (Jacobson & Rowe, 1999).
Overall, more than 3,000 sibling pairs were embedded in the Add Health data (K. M. Harris, Halpern, Smolen, & Haberstick, 2006); however, for reasons to be discussed momentarily, the analytical sample for the current study is confined to MZ and same-sex DZ twin pairs.

MEASURES

Low self-control. There is a great deal of debate concerning the most reliable and valid way to measure individual variation in levels of self-control (e.g., DeLisi, Hochstetler, & Murphy, 2003; Marcus, 2004). Despite divergences in measurement strategies, a meta-analysis conducted by Pratt and Cullen (2000) revealed that the association between levels of self-control and antisocial behaviors was not contingent on the way in which self-control was measured. In other words, the connection between levels of self-control and crime and delinquency was detected regardless of how self-control was measured. Although the Add Health data did not ask questions that were originally designed to measure self-control, the data contain a number of items, garnered at each data collection wave, that are closely in line with Gottfredson and Hirschi’s (1990) description of low self-control. During Wave 1 interviews, adolescents were asked 19 questions that indexed variation in levels of self-control. For example, respondents were asked to indicate whether they have trouble paying attention at school, whether they go with their “gut feeling” when making decisions, and whether they argue with people. In addition, primary caregivers were asked 4 questions about the adolescent’s self-control, including whether their child is trustworthy and whether their child has a bad temper. Principal components factor analysis indicated that all of the items could be accounted for by a common factor, and confirmatory factor analysis indicated that all of the items loaded on a unitary construct. The responses to these 23 items were summed together to create the Wave 1 Low Self-Control Scale (alpha = .74). Higher values on this scale reflect lower levels of self-control. Prior researchers have employed this same low self-control scale in their analysis of the Add Health data (Beaver et al., 2009). The appendix contains all of the individual items used to create the low self-control scales.

During Wave 2 interviews, adolescents were asked a very similar set of questions that tapped their levels of self-control. For example, respondents were asked whether they have trouble keeping their mind focused, whether they like to take risks, and whether they are sensitive to other people’s feelings. The primary caregivers were not interviewed at Wave 2, and thus all of the items included in the Wave 2 Low Self-Control Scale were reported on by the adolescent. Psychometric analyses revealed that the 20 items could be accounted for by a single factor, and additional analyses revealed that removing any of the items from the scale would not significantly increase the internal reliability of the scale. As a result, responses to the 20 questions were summed together to create the Wave 2 Low Self-Control Scale, where higher values indicate lower levels of self-control (alpha = .72).

Maternal negativity. The Add Health data included three maternal parenting scales at Wave 1 and the same three maternal parenting scales at Wave 2. First, a maternal attachment scale was available in the Add Health data. During Wave 1 interviews, adolescents were asked to indicate how close they feel to their mother and how much they think their mother cares about them. Responses to these items were based on a 5-point scale, where higher values indicated more maternal attachment. The responses to both of these items were summed together to create the Wave 1 Maternal Attachment scale (alpha = .70). The
same two items were asked during Wave 2 interviews; thus the Wave 2 Maternal Attachment scale (alpha = .51) is a duplicate of the Wave 1 Maternal Attachment scale. These maternal attachment scales have been used by previous Add Health researchers (Haynie, 2001; Schreck, Fisher, & Miller, 2004).

Second, the Add Health data also contain a 10-item maternal involvement index. During Wave 1 interviews, respondents were presented with a list of 10 different activities and asked which they had done with their mothers during the past month. Youths were asked, for instance, whether they had played a sport with their mother, whether they had gone shopping with their mother, and whether they had seen a movie with their mother. Items were coded dichotomously, where 0 = did not engage in that activity and 1 = did engage in that activity. Responses to the 10 items were summed together to create the Wave 1 Maternal Involvement Index (alpha = .53). The same items were also included in the Wave 2 surveys. As a result, an identical Wave 2 Maternal Involvement Index was also created (alpha = .53). These indexes are similar to ones that have been used previously (Crosnoe & Elder, 2004).

Third, a maternal disengagement scale was also available in the Add Health data. During Wave 1 interviews, adolescents were asked five questions that tapped the degree to which their mothers were disconnected from their child. Specifically, respondents were asked to indicate how frequently they speak with their mother and the overall quality of their relationship with their mother. Responses were coded so that higher values on the Wave 1 Maternal Disengagement scale indicated greater disengagement (alpha = .83). During Wave 2 interviews, a similar set of seven items was included that measured maternal disengagement. As a result, a seven-item Wave 2 Maternal Disengagement scale was also constructed (alpha = .86). These scales are identical to the ones that have been used previously (Beaver, 2008).

The parental socialization research provides some evidence indicating that individual dimensions of parenting do not have as strong effects on adolescent development as global measures of parenting (Wright & Cullen, 2001). Against this backdrop, the possibility that the three scales could be combined to create a more global measure of maternal parenting was explored by calculating a principal components factor analysis with varimax rotation. Before doing so, the Maternal Attachment and Maternal Involvement scales were reverse coded such that higher values indicated less maternal attachment and less maternal involvement. The three maternal parenting scales were then subjected to a factor analysis, and the results revealed evidence consistent with a single-factor solution. The same results were garnered when the Wave 2 maternal parenting scales were factor analyzed. As a result, weighted factor scores were estimated to create the Wave 1 Maternal Negativity scale and the Wave 2 Maternal Negativity scale.

Paternal negativity. When detailing their theory on the development of self-control, Gottfredson and Hirschi (1990) were very clear to note that two-parent households, in comparison with single-parent households, are more likely to instill high levels of self-control in their offspring. Most of the research testing their theory, however, has relied on measures that tap maternal socialization. Fortunately, the Add Health study includes two paternal parenting scales at each wave. First, items measuring paternal attachment were collected. During Wave 1 interviews, respondents were asked to indicate how close they feel to their father and how much they think their father cares about them. Responses were scored such that higher values reflected more paternal attachment. The same two items were also
available at Wave 2. As a result, the Wave 1 Paternal Attachment scale (alpha = .69) and the Wave 2 Paternal Attachment scale (alpha = .68) were both created from the same items.

Similar to the maternal scales, a **paternal involvement** index was included in the Add Health data. During Wave 1 interviews, youths were presented with a list of 10 activities, and they were asked to indicate which, if any, they had done with their father during the past month. For example, respondents were asked whether they had gone shopping with their father, whether they had played a sport with their father, and whether they had worked on a project for school with their father. These items were coded dichotomously, where 0 = no and 1 = yes. The same items were also asked at Wave 2. Consequently, responses to the Wave 1 items were summed together to create the Wave 1 Paternal Involvement index (alpha = .57), and responses to the Wave 2 items were summed together create the Wave 2 Paternal Involvement index (alpha = .61).

To stay consistent with the measurement of maternal negativity, the paternal scales were subjected to a factor analysis. Once again, the scales were recoded such that higher values reflected less paternal attachment and less paternal involvement. The results of the principal components factor analysis with varimax rotation indicated that the Wave 1 Paternal Attachment scale and the Wave 1 Paternal Involvement index loaded on a unitary factor. The same pattern of results was garnered for the Wave 2 Paternal Attachment scale and the Wave 2 Paternal Involvement index. As a result, weighted factor scores were estimated to create the Wave 1 Paternal Negativity scale and the Wave 2 Paternal Negativity scale.

**Negative parenting.** To capture the effects of parenting in general on the adolescent, two negative parenting scales were created that included both maternal and paternal parental socialization. To create the Wave 1 Negative Parenting Scale, the Wave 1 Maternal Negativity scale and the Wave 1 Paternal Negativity scale were summed together (alpha = .63). To create the Wave 2 Negative Parenting Scale, the Wave 2 Maternal Negativity scale and the Wave 2 Paternal Negativity scale were summed together (alpha = .53). For both scales, higher values represent greater exposure to negative parenting.

**ANALYSIS**

The analysis for this study was conducted in two steps. First, bivariate correlations were calculated between the parenting measures and the low self-control scales. The information gleaned from these statistics provided a baseline estimate of the covariation between parenting and levels of self-control. The second step in the analysis was to decompose the percentage of the covariance between parenting and self-control into a genetic component, a shared environmental component, and a nonshared environmental component. To do so, a modified version of the widely used DeFries-Fulker (DF) analysis was employed (DeFries & Fulker, 1985).

DF analysis is a regression-based statistic that is used to analyze samples of sibling pairs to estimate the percentage of variance in a measure that is attributable to genetic factors, shared environmental factors, and nonshared environmental factors. Although the original DF equation was designed to be used with samples where at least one twin had some type of disorder, the equation has been modified so that it can be used with samples drawn from the general population. The baseline DF formula that can be employed with nonclinical samples takes the following form:
\[ K_1 = b_0 + b_1 K_2 + b_2 R + b_3 (R \times K_2) + e, \]  

(1)

where \( K_1 \) is the score on the outcome measure for one twin (e.g., low self-control), \( K_2 \) is the score on the same outcome measure for the cotwin, \( R \) measures genetic similarity (\( R = 1.0 \) for MZ twins and \( R = .5 \) for DZ twins), and \( R \times K_2 \) is a multiplicative interaction term between \( R \) and \( K_2 \). In this equation, \( b_0 \) = the constant, \( b_1 \) = the proportion of variance in \( K_1 \) that is explained by shared environmental factors, \( b_2 \) is not usually interpreted in DF analysis, \( b_3 \) = the proportion of variance in \( K_1 \) that is attributable by genetic factors (i.e., heritability), and \( e \) = the proportion of variance in \( K_1 \) that is results of nonshared environmental factors. Importantly, \( e \) also includes the effects of measurement error.

Rodgers and Kohler (2005) have modified the DF formula presented in Equation 1. This new DF equation takes the following form:

\[ K_1 = b_0 + b_1 (K_2 - K_m) + b_2 [R \times (K_2 - K_m)] + e, \]  

(2)

where \( b_0 \) remains the same, \( K_1 \) remains the score on the outcome measure for one twin, \( K_2 \) remains the value on the same outcome measure for the cotwin, and \( R \) remains the measure of genetic similarity. Note, however, that the new DF equation includes an additional term, \( K_m \). \( K_m \) = the mean value for \( K_2 \). The DF formula presented in Equation 2 shows that \( K_2 \) is being mean centered, whereas in Equation 1, \( K_2 \) was not mean centered. In addition, the main effect of \( R \) has also been removed from Equation 2, but it is still included as part of an interaction term. The interpretation of the coefficients is as follows: \( b_1 \) = the proportion of variance in \( K_1 \) that is explained by the shared environment, \( b_2 \) = the proportion of variance in \( K_1 \) explained by genetic factors, and \( e \) = the proportion of variance in \( K_1 \) explained by nonshared environmental factors plus error.

The two equations discussed above are considered univariate equations because they estimate genetic and environmental influences on only one variable at a time. Some research questions, however, are more complex than can be answered with a univariate equation. For instance, in the current study, we were interested in estimating the percentage of the covariance between parenting and self-control that was attributable to genetic and environmental factors. In this case, the univariate DF equation would not have been appropriate and needed to be replaced with what is known as a bivariate equation. Bivariate genetic modeling is frequently estimated with structural equation modeling programs, where Cholesky decomposition models or correlated factors models are calculated (Neale & Cardon, 1992). Research carried out by Rodgers, Kohler, Kyvik, and Christensen (2001) revealed that the univariate DF model could be transformed into a cross-variable DF model to estimate bivariate genetic analyses. The cross-variable DF model that can be used for bivariate genetic analyses takes the following form:

\[ K_{1lsc} = b_0 + b_1 (K_{2parenting} - K_m) + b_2 [R \times (K_{2parenting} - K_m)] + e, \]  

(3)

where \( K_{1lsc} \) is the value for the Low Self-Control Scale (i.e., either the Wave 1 or Wave 2 Low Self-Control Scale) for one twin, \( K_{2parenting} \) is the score on one of the parenting measures for the cotwin, and \( R \) is the measure of genetic relatedness. Note that all of the variables are standardized prior to being entered into Equation 3. Equation 3 also reveals that \( K_{2parenting} \) is mean centered. The interpretation of the coefficients contained in Equation 3
does, however, vary from those of the previous two equations. In the cross-variable DF model, \( b_1 \) is the proportion of the covariance between the two measures (e.g., the Low Self-Control Scale and the Negative Parenting Scale) that is attributable to shared environmental effects, whereas \( b_2 \) is the proportion of the covariance between measures that is due to common genetic factors. The variance in the covariance between the two measures that is not explained by \( b_1 \) and \( b_2 \) is attributable to common nonshared environmental factors plus error and is captured by the error term, \( e \).

When estimating DF models, it is permissible to double enter twins, such that each twin is entered into the data twice: once as the dependent variable and once as the independent variable. With double entry, the observations lack independence and the standard errors must be corrected to take into account the clustering of cases. Instead of using double entry, the current study randomly selected one twin from each twin pair to be used as the dependent variable, and his or her cotwin was entered as the independent variable. As a result, the twin pair—not the twin—was the unit of analysis, and the observations were independent of each other. However, the models were recalculated using double entry, and the pattern of results was virtually identical to those reported when each twin was entered into the data only once.

### RESULTS

The analysis for this study began by estimating bivariate correlations between the parenting measures and the low self-control scales. The results of these analyses provide information as to the magnitude of the association between parental socialization and levels of self-control. As Table 1 shows, bivariate correlations were calculated cross-sectionally (Wave 1 parenting measures and Wave 1 low self-control and Wave 2 parenting measures and Wave 2 low self-control) as well as longitudinally (Wave 1 parenting measures and Wave 2 low self-control). Consistent with Gottfredson and Hirschi’s (1990) parental management thesis, all of the bivariate correlations are statistically significant and positive, indicating that negative parenting is related to lower levels of self-control. It should be noted that all of the correlations are relatively moderate in magnitude, which is consistent with much of the past literature evaluating the parental management thesis (Cullen et al., 2008).

Recall that the current study was interested in determining which of the three mechanisms was responsible for the covariation between the parenting scales and the low self-control scales. The next step in examining this issue is to estimate cross-twin, cross-trait correlations, where the parenting scales for one twin are correlated with the low self-control

### TABLE 1: Bivariate Associations Between Parenting Measures and Levels of Self-Control

<table>
<thead>
<tr>
<th>Measure</th>
<th>Low Self-Control (W1)</th>
<th>Low Self-Control (W2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal negativity (W1)</td>
<td>.36*</td>
<td>.32*</td>
</tr>
<tr>
<td>Maternal negativity (W2)</td>
<td>—</td>
<td>.42*</td>
</tr>
<tr>
<td>Paternal negativity (W1)</td>
<td>.28*</td>
<td>.28*</td>
</tr>
<tr>
<td>Paternal negativity (W2)</td>
<td>—</td>
<td>.30*</td>
</tr>
<tr>
<td>Negative parenting (W1)</td>
<td>.21*</td>
<td>.20*</td>
</tr>
<tr>
<td>Negative parenting (W2)</td>
<td>—</td>
<td>.21*</td>
</tr>
</tbody>
</table>

*Note. W1 = Wave 1; W2 = Wave 2.

*p < .05, two-tailed tests.*
scales for his or her cotwin. These cross-twin, cross-trait correlations are estimated separately for DZ twin pairs and MZ twin pairs. For genetic factors to be involved in the association between parenting and levels of self-control, the MZ cross-twin, cross-trait correlations must be larger than the DZ cross-twin, cross-trait correlations. As indicated in Table 2, which contains the results of the cross-twin, cross-trait correlations for the parenting scales and low self-control scales, results appear consistent with this pattern. For example, for DZ twins, only two cross-twin, cross-trait correlations are statistically significant. The MZ cross-twin, cross-trait correlations are all statistically significant, and they are all larger than the DZ cross-twin, cross-trait correlations. This pattern of results for the cross-twin, cross-trait correlations indicates that genetic factors could explain part of the covariance between the parenting and low self-control scales. As a result, the next set of analyses estimate bivariate genetic analyses to provide precise estimates of the percentage of covariance between parenting and low self-control that is attributable to genetic, shared environmental, and nonshared environmental factors.

Figure 2 contains the results of the bivariate genetic analyses for the maternal parenting scales and the low self-control scales. Recall that Equation 3 presented above was used to estimate these parameters, and to facilitate interpretation, the parameter estimates were plotted in a series of bar charts. The first bar chart indicates that 63% of the covariance between maternal negativity at Wave 1 and low self-control at Wave 1 was the result of nonshared environmental effects, and the remaining 37% of the covariance was accounted for by genetic factors. Shared environmental factors explained none of the covariance between these two scales. The middle bar chart in Figure 2 examines the genetic and environmental effects on the covariance between maternal negativity at Wave 1 and low self-control at Wave 2. The results are strikingly similar to those reported in the first bar chart; nonshared environmental factors account for 65% of the covariance, genetic factors account for 35% of the covariance, and shared environmental factors account for none of the covariance. The last bar chart in Figure 2 examined the genetic and environmental effects on the covariance between maternal negativity at Wave 2 and low self-control at Wave 2. As can be seen, nonshared environmental effects accounted for 44% of the covariance, genetic factors accounted for 56% of the covariance, and shared environmental effects accounted for none of the covariance.

The next set of analyses examined the genetic, shared environmental, and nonshared environmental effects on the association between the paternal negativity scales and the low self-control scales. Figure 3 contains the results of these analyses. The far-left-hand bar

<table>
<thead>
<tr>
<th>Measure</th>
<th>Low Self-Control for Dizygotic Twins</th>
<th>Low Self-Control for Monozygotic Twins</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>W1</td>
<td>W2</td>
</tr>
<tr>
<td>Maternal negativity (W1)</td>
<td>.06</td>
<td>.04</td>
</tr>
<tr>
<td>Maternal negativity (W2)</td>
<td>—</td>
<td>.34*</td>
</tr>
<tr>
<td>Paternal negativity (W1)</td>
<td>-.08</td>
<td>.04</td>
</tr>
<tr>
<td>Paternal negativity (W2)</td>
<td>—</td>
<td>.21*</td>
</tr>
<tr>
<td>Negative parenting (W1)</td>
<td>.10</td>
<td>.10</td>
</tr>
<tr>
<td>Negative parenting (W2)</td>
<td>—</td>
<td>.04</td>
</tr>
</tbody>
</table>

Note. W1 = Wave 1; W2 = Wave 2.
*p < .05, two-tailed tests.
Figure 2: The Percentage of Covariance Between Maternal Negativity and Low Self-Control Explained by Genetic and Environmental Factors

Figure 3: The Percentage of Covariance Between Paternal Negativity and Low Self-Control Explained by Genetic and Environmental Factors
The last set of analyses examined the extent to which genetic and environmental factors were implicated in the covariance between the global parental negativity scales and the low self-control scales. Figure 4 contains the findings from these analyses. The first bar chart reveals that 77% of the covariance between parental negativity at Wave 1 and low self-control at Wave 1 was attributable to nonshared environmental effects, and the remaining 23% of the covariance was the result of genetic factors. Shared environmental factors were uninvolved in explaining the covariance between these two scales. The middle column contains the results of the bivariate genetic analysis for parental negativity at Wave 1 and low self-control at Wave 2. As can be seen, nonshared environmental effects explained 63% of the covariance, genetic factors explained 37% of the covariance, and shared environmental factors explained none of the covariance. Last, Figure 4 reveals that 37% of the covariance between parental negativity at Wave 2 and low self-control at Wave 2 was...
attributable to nonshared environmental factors, and 63% of the covariance was the result of genetic factors. Shared environmental effects explained none of the covariance between these two scales.

DISCUSSION

Given that levels of self-control are strongly related to involvement in antisocial behaviors, criminologists have begun to search for the factors that are responsible for causing variation in self-control. Most of this research has been guided by Gottfredson and Hirschi’s (1990) parental management thesis, which identifies different elements of parenting as the causes of self-control. The results of these studies appear to show empirical support for the parental management thesis wherein measures of parental management have been found to be predictive of levels of self-control (Cullen et al., 2008). However, the studies testing the parental management thesis are all host to one major shortcoming: They all fail to control for genetic factors. Failure to take into account genetic factors could upwardly bias parenting coefficients and thus provide artificial support for the role of parents in the etiology of self-control (J. R. Harris, 1998; Rowe, 1994). The current study addressed this limitation by employing quantitative genetic analysis to examine three different mechanisms that could account for the association between parenting and levels of self-control.

Analysis of twin pairs drawn from the Add Health data revealed three broad findings in respect to the association between parental management techniques and levels of self-control. First, in most of the models, nonshared environmental factors explained a majority of the covariance between the parenting scales and low self-control scales. What this finding can be interpreted to mean is that a large part of the reason for the statistical association between parenting and levels of self-control is attributable to a child-driven mechanism, where the child elicits certain responses from the parents. The second key finding to emerge from the analyses was that genetic factors explained a moderate degree of covariance between the parenting measures and low self-control scales. These results provide support for the spurious mechanism and the child-driven mechanism. Third, shared environmental influences accounted for none of the covariance between the parenting measures and low self-control scales. Recall that the shared environmental component indexed the degree of support for the causal mechanism, where parenting causes levels of self-control. The null results for the shared environment across various measures of parenting indicate that parents do not have a causal effect on shaping and molding their offspring’s level of self-control when assessed with the parenting measures available in the Add Health data.

The results generated from our analysis offer a challenge to the results generated from prior tests of the parental management thesis. Specifically, most prior tests have reported a statistically significant association between parenting and self-control, which is typically interpreted as support for the parental management thesis. Our analysis also revealed statistically significant associations between parenting measures and measures of low self-control (see Table 1), with effect sizes similar to those found in previous studies. However, it was only after we employed an appropriate methodology that controlled for genetic factors and child-driven effects that the association between parenting and self-control dropped from statistical significance.
Generally speaking, our findings draw attention to the importance of controlling for genetic and child-driven effects when examining the issue of self-control. More critically, the findings suggest the value of exploring the relationship that child-driven factors may have on other antisocial phenotypes. Criminologists exploring the parental management thesis have been too quick to employ methodologies that are useful for some social science questions but are inappropriate when testing the causal view that parents influence their child’s level of self-control (J. R. Harris, 1998; Pinker, 2002). A study germane to criminology, published by Wright and Beaver (2005), examined the effect that parents had on their children’s self-control. They first calculated their analysis without controlling for genetic factors and found various measures of parenting to predict levels of self-control. They next estimated the same models but controlled for genetic factors. The results of these models revealed that the parenting effects were no longer statistically significant, indicating that the association between parenting and self-control was spurious. Still, and even with empirical evidence showing that genes can act as confounders, criminologists continue to produce studies examining the effects that parents have on their offspring without controlling for genetic factors. The end result is that the research testing the parental management is misinformed, is misguided, and may have come to the wrong conclusions. Only by employing genetically sensitive research designs will the true association between parenting and self-control be revealed. We speculate that if prior tests of the parental management thesis included appropriate controls for genetic factors and child-driven effects, the “causal” effect of parenting on self-control in these studies would prove to be nonsignificant or, at a minimum, substantially attenuated.

It is important to point out, however, that the results of the DF models do not necessarily indicate that parenting has no effect on the development of self-control; rather, the results indicate that parenting likely has no effect on explaining variance in levels of self-control among twins from the Add Health. Parental socialization could affect mean levels of self-control without affecting variance in self-control. Future research needs to explore this issue in much greater detail to determine the extent to which parenting is involved in determining mean levels of self-control. As for now, the results of our analysis provide very little support for Gottfredson and Hirschi’s (1990) claim that variation in parenting tactics correspond to variation in levels of self-control.

The findings reported here need to be interpreted with caution in light of a number of limitations. The most serious shortcoming is that although the full sample of the Add Health is nationally representative, the twin sample drawn from the Add Health may not be, as twin pairs are not necessarily representative of the general population. Consequently, the results reported here may not be generalizable to the larger population of American adolescents. Prior researchers analyzing twins from the Add Health data have explored this issue in detail. Their analyses did not reveal any differences on demographic variables, parenting variables, and measures of antisocial behavior, including low self-control, between the nationally representative sample and the sample of twins (Beaver, 2008; Jacobson & Rowe, 1998). Although the results of these analyses seem to indicate that there are not any major differences in the samples, it is still possible that they differ in ways that have yet to be identified. In addition, the Add Health data did not include measures that are able to capture every dimension of parenting, and thus we were forced to create global measures of parental negativity. This measurement limitation has been faced by most prior
tests of the parental management thesis (e.g., Hay, 2001; Unnever, Cullen, & Pratt, 2003; Wright & Beaver, 2005), and it necessarily precluded us from testing all of the parental management techniques that Gottfredson and Hirschi (1990) argued were important to the development of self-control. In a similar vein, the parenting measures were based on the perception of parenting as reported on by adolescents. As a result, it could be the case that our results would not hold when parenting is measured by an independent rater. Even so, our results do provide some evidence regarding the nature of the association between parenting and levels of self-control. Future researchers need to employ different samples and different measures of parental management techniques to determine the robustness of the findings reported here.

Criminological research has largely been conducted through a sociological lens, whereby social factors are thought to cause crime and delinquency and biological and genetic factors are thought to be unimportant (Walsh & Ellis, 2004; Wright et al., 2008). But a rich line of empirical research has revealed that genetic factors are involved in all human behaviors, including antisocial behaviors. Criminologists, however, have been slow to recognize these findings and, for the most part, have proceeded without testing for genetic factors. Excluding genetic factors has come at a price: The studies produced by criminologists—especially those that investigate the role of parents—are likely to be misspecified. This problem can easily be overcome if criminologists begin to incorporate genetically sensitive research designs into their studies. Failure to do so will result in the continued production of research findings that are biased, incomplete, and therefore inconsequential to understanding the root causes of antisocial phenotypes, including low levels of self-control.

APPENDIX
ITEMS USED TO CREATE THE THREE LOW SELF-CONTROL SCALES

WAVE 1 LOW SELF-CONTROL SCALE

Scale was created by combining summing responses to the following items:

1. All things considered, how is your child’s life going?
2. You get along well with your child.
3. You can trust your child.
4. Does your child have a bad temper?
5. You never argue with anyone.
6. When you get what you want, it is usually because you worked hard for it.
7. You never get sad or you felt sad.
8. You never criticize other people.
9. You usually go out of your way to avoid having to deal with problems in you life.
10. Difficult problems make you very upset.
11. When making decisions, you usually go with your “gut feeling” without thinking too much about the consequences of each alternative.
12. When you have a problem to solve, one of the first things you do is get as many facts about the problem as possible.
13. When attempting to find a solution to a program, you usually try to think of as many different ways to approach the problem as possible.
APPENDIX (continued)

14. When making decisions, you generally use a systematic method for judging and comparing alternatives.
15. After carrying out a solution to a problem, you usually try to analyze what went right and what went wrong.
16. You like yourself just the way you are.
17. You feel like you are doing everything just about right.
18. You feel socially accepted.
19. Do you have trouble getting along with your teachers?
20. Do you have trouble paying attention in school?
21. Do you have trouble keeping your mind focused?
22. Do you have trouble getting your homework done?
23. Do you have trouble getting along with other students?

WAVE 2 LOW SELF-CONTROL SCALE

Scale was created by summing responses to the following items:

1. When you get what you want, it’s usually because you worked hard for it.
2. You never get sad or you felt sad.
3. You usually go out of your way to avoid having to deal with problems in your life.
4. Difficult problems make you very upset.
5. When making decisions, you usually go with your “gut feeling” without thinking too much about the consequences of each alternative.
6. After carrying out a solution to a problem, you usually try to analyze what went right and what went wrong.
7. You like yourself just the way you are.
8. You feel like you are doing everything just about right.
9. You feel socially accepted.
10. Do you have trouble getting along with your teachers?
11. Do you have trouble paying attention in school?
12. Do you have trouble getting your mind focused?
13. Do you have trouble getting your homework done?
14. Do you have trouble getting along with other students?
15. You enjoyed life.
17. You like to take risks.
18. You are sensitive to other people’s feelings.
19. You can pretty much determine what will happen in your life.

REFERENCES


**Kevin M. Beaver** is an associate professor in the College of Criminology and Criminal Justice. His research examines the ways in which the environment intersects with biological and genetic factors to produce antisocial outcomes. He is a past recipient of the American Society of Criminology’s Ruth Shonle Cavan Young Scholar Award and the National Institute of Justice’s Graduate Research Fellowship.

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