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GENETIC AND ENVIRONMENTAL INFLUENCES ON LEVELS OF SELF-CONTROL AND DELINQUENT PEER AFFILIATION

Results From a Longitudinal Sample of Adolescent Twins

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Despite the fact that low self-control and exposure to delinquent peers are two of the most robust and consistent predictors of crime, delinquency, and antisocial behavior, much remains unknown about what causes self-control to develop and what causes youths to befriend antisocial peers. This study estimated the relative effects of environmental and genetic factors on levels of self-control and contact with delinquent peers in a sample of twins from the National Longitudinal Study of Adolescent Health (Add Health). DeFries-Fulker analysis of the Add Health data revealed that both self-control and contact with drug-using friends were influenced by genetic factors and the nonshared environment, whereas the shared environment exhibited relatively small and inconsistent effects. Implications for self-control theory and social learning theory are discussed.

Keywords: Add Health; delinquent peers; genetics; self-control; twins

Numerous criminological studies have tested the central propositions of self-control and social learning theories. The results of these studies, which have analyzed hundreds of different samples and used very different analytical strategies, have converged to reveal that low self-control and self-regulation and exposure to delinquent peers are among the strongest predictors of conduct disorders, aggression, and criminal and delinquent involvement (Akers & Jensen, 2006; Morgan & Lilienfeld, 2002; Pratt & Cullen, 2000; Séguin, Boulerice, Harden, Tremblay, & Pihl, 1999; Séguin, Nagin, Assaad, & Tremblay, 2004; Warr, 2002). At the same time, much less empirical attention has been devoted to identifying what causes variation in levels of self-control and what causes adolescents to befriend delinquent peers.

Most criminological research examining the antecedent causes of self-control and delinquent peer associations has identified social and environmental factors as particularly

important. For example, empirical work has revealed that parental socialization, neighborhoods, and schools can all contribute to variation in levels of self-control (Gibbs, Giever, & Higgins, 2003; Gibbs, Giever, & Martin, 1998; Pratt, Turner, & Piquero, 2004; Turner, Piquero, & Pratt, 2005). Similar conclusions have been reached in regard to the correlates to delinquent peer associations (Akers, 1998; Cairns & Cairns, 1994; Warr, 2002). Although criminologists from a sociological perspective have, in general, dismissed the importance of genetics (Robinson, 2004), other research has indicated that a complex arrangement of social *and* genetic factors are involved in the etiology of self-control and self-regulation (Lahey & Waldman, 2003; Nigg & Huang-Pollock, 2003; Rhee & Waldman, 2003) as well as the formation of delinquent peer groups (Beaver, Wright, & DeLisi, 2008; DiLalla, 2002). Against this backdrop, the purpose of the current article is to examine the relative roles of genetic and environmental factors in the development of self-control and in exposure to delinquent peers.

THE DEVELOPMENT OF SELF-CONTROL

In their book *A General Theory of Crime*, Gottfredson and Hirschi (1990) argued that low self-control was the unitary cause of crime, delinquency, and analogous behaviors. Although this proposition formed the crux of their theory, they also advanced an explanation regarding the development of self-control. According to Gottfredson and Hirschi, individual levels of self-control are determined mainly through three different parental management techniques. Specifically, parents who supervise their children, who recognize their child's misbehavior, and who punish or correct their child's misconduct will, on average, raise children with high levels of self-control. In contrast, parents who fail to engage in these three parenting tactics will, on average, raise children with low levels of self-control.

While Gottfredson and Hirschi highlighted the importance of socialization effects on the emergence of self-control, they simultaneously rejected the possibility that levels of self-control were influenced by genetic and biological factors. In the words of Gottfredson and Hirschi (1990), "the magnitude of the 'genetic effect' is near zero" (p. 60). Given that most tests of the parental management thesis have failed to estimate genetic effects, it is difficult to assess whether the "magnitude of the 'genetic effect' is near zero." However, there are at least three reasons to suspect that low self-control is at least partially explained by biogenic factors.

First, in a recent study, J. P. Wright and Beaver (2005) analyzed a sample of twins from the Early Childhood Longitudinal Study–Kindergarten Class data to examine parental socialization effects on self-control. In their first set of analyses, they did not control for

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genetic influences, and the results of their models revealed that the parenting measures had statistically significant effects on measures of self-control. They then recalculated the models and controlled for genetic effects. Once genetic influences were held constant, most of the parenting measures were reduced to statistical insignificance, indicating that the relationship between parenting and self-control is accounted for by shared genetic factors. The results of this study thus suggested that genetic influences are important in the development of self-control.

Second, behavioral genetic research has examined the genetic and environmental influences on impulsivity, hyperactivity, attention deficit hyperactivity disorder, and other attention deficiencies. Behavioral genetic research typically decomposes variance into three different components: a heritability component, a shared environmental component, and a nonshared environmental component. Heritability captures the proportion of variance accounted for by genetic factors. Shared environments refer to environments that are the same between siblings from the same household, such as economic well-being and parental socialization techniques. Nonshared environments, in contrast, capture any environment that is different between siblings from the same household. It is also important to point out that measurement error is subsumed within nonshared environmental effects. Shared environments work to make siblings similar to each other, whereas nonshared environments work to make siblings dissimilar to each other. Behavioral geneticists typically analyze samples of monozygotic (MZ) and dizygotic (DZ) twins to estimate genetic, shared environmental, and nonshared environmental effects on a broad range of behaviors, and they have also used twin-based research designs to estimate the relative importance of genetic factors and environmental factors in the formation of delinquent peer groups.

The results of behavioral genetic studies have been quite consistent and have revealed that genetic factors account for between 50% and 90% of the variance in self-control, self-regulation, and impulsivity, whereas the remaining variance is attributable to the nonshared environment and measurement error (Barkley, 1997; Price, Simonoff, Waldman, Asherson, & Plomin, 2001; Reiss, Neiderhiser, Hetherington, & Plomin, 2000; Rietveld, Hudziak, Bartels, van Beijsterveldt, & Boomsma, 2003; J. P. Wright, Beaver, DeLisi, & Vaughn, 2008). Findings garnered from these studies stand in direct contradiction to Gottfredson and Hirschi's (1990) claim that self-control is insulated against biogenic effects (J. P. Wright & Beaver, 2005).

Third, research has revealed that levels of self-control are influenced, in part, by the structure and functioning of the prefrontal cortex of the brain (Barkley, 1997; Beaver, Wright, & DeLisi, 2007; Cauffman, Steinberg, & Piquero, 2005; Ishikawa & Raine, 2003; Raine, 2002). The formation of the brain, as well as brain functioning, is due in large part to genetic influences (Pfefferbaum, Sullivan, Swan, & Carmelli, 2000; Thompson et al., 2001; Toga & Thompson, 2005). Thus, variations in brain structure and functioning, which reflect genetic differences, could translate into variations in individual levels of self-control. If this is the case, then genes may have an effect on self-control by producing variability in regions of the brain that are tied to impulse control, aggression, and self-regulation (Meyer-Lindenberg et al., 2006).

It is important to point out that a behavioral genetic explanation of self-control is not necessarily incompatible with an environmental/socialization explanation of self-control. Indeed, researchers working from a behavioral genetic perspective have argued that a combination of biogenic factors and social factors act independently and interactively to produce antisocial dispositions (Caspi et al., 2002; Moffitt, 1993; Robinson, 2004; Rutter, 2006). Even so, the construct of low self-control, as defined by criminologists, has been

subjected to very little behavioral genetic analysis. As a result, much remains unknown about what role, if any, genetic influences play on the development of self-control (but see J. P. Wright et al., 2008; J. P. Wright & Beaver, 2005). The current article seeks to shed some light on this issue.

DELINQUENT PEER GROUP FORMATION

Although a long line of literature has examined the effects that associating with antisocial friends has on delinquent involvement (Akers, 1998; Akers & Jensen, 2006; Warr, 2002), there is a rather limited amount of research that has explored the correlates to delinquent peer group formation. Much of this research has investigated the role that the social context has on adolescent contact with deviant friends. Results from this area of empirical work have revealed that one of the most important predictors of antisocial friendship formation is proximity to delinquent peers. All else equal, adolescents who are in close contact with antisocial youths will be more apt to befriend delinquent others (Cairns & Cairns, 1994; Warr, 2002). Parents, too, are often hypothesized to affect their child's choice of peer groups, either directly through close supervision and location of residence (J. R. Harris, 1998) or indirectly through the development of socioemotional attachment (Hirschi, 1969). Taken together, the common theme cutting across this body of criminological research is that adolescents become embedded in antisocial friendship networks mainly because of social factors.

An additional explanation to the formation of antisocial peer networks has been offered by some behavioral geneticists (Cleveland, Wiebe, & Rowe, 2005; DiLalla, 2002; Scarr, 1992; Scarr & McCartney, 1983; Walsh, 2002b). According to this line of reasoning, adolescents actively seek out certain friends, such as delinquent friends, because of genetic propensities. Youths who are genetically predisposed to be impulsive, to be risk seekers, and to be antisocial will tend to select peer groups that reinforce these propensities. As a result, genetic influences can partially explain why individuals sort themselves into one particular peer group over another. Behavioral geneticists refer to the close nexus between genetic tendencies and the environment (in this case the environment is delinquent peers) as a Gene $\times$ Environment correlation.

A Gene $\times$ Environment correlation explanation of delinquent peer associations is very similar to self-selection arguments. Advocates of a self-selection perspective (e.g., Gottfredson & Hirschi, 1990) argue that adolescents choose to associate with antisocial friends because of some underlying propensity (e.g., low self-control). Advocates of a Gene $\times$ Environment correlation perspective use similar logic, except that instead of focusing on a particular trait they focus on genetic effects. The difference, then, is the unit of analysis. But it should be pointed out that behavioral geneticists do not think that there is a gene for delinquent peers. Instead, behavioral geneticists take a more realistic and nuanced approach and argue that personality traits (e.g., low self-control) are heavily influenced by genetic factors (J. P. Wright & Beaver, 2005). Thus, whereas sociologically oriented criminologists explore self-selection effects by examining personality factors and underlying propensities (Baron, 2003; Chapple, 2005; Gottfredson & Hirschi, 1990; B. R. E. Wright, Caspi, Moffitt, & Silva, 1999), behavioral geneticists reduce the unit of analysis to the genetic level (Cleveland et al., 2005).

To illustrate, Iervolino et al. (2002) analyzed data from the Nonshared Environment in Adolescent Development (NEAD) research and from the Colorado Adoption Project (CAP) to estimate the relative importance of genes and the environment on deviant peer affiliations.

The results of the biometric model-fitting techniques revealed two somewhat contradictory findings. For the NEAD sample, Iervolino et al. found that almost all of the variance in the measure of peer delinquency was accounted for by the shared environment (20%) and the nonshared environment (77%), whereas genetic effects were close to zero (3%). Remarkably dissimilar results were garnered when the CAP data were analyzed. For this analysis, genetic factors accounted for 65% of the variance in peer delinquency, the nonshared environment explained 35% of the variance, and the shared environment explained none of the variance.

In a similar vein, Cleveland and his colleagues (2005) analyzed a sample of sibling pairs from the National Longitudinal Study of Adolescent Health (Add Health) to estimate genetic and environmental effects on substance-using friends. Analysis of the data revealed that 64% of the variance in delinquent peer affiliations was accounted for by genetic factors, 36% of the variance was attributable to the nonshared environment, and the shared environment explained 0% of the variance. In the most recent study, Kendler et al. (2007) analyzed a sample of male twin pairs from the Virginia Twin Registry and found that genetic factors account for between 30% and 50% of the variance in peer-group deviance. Most of the remaining variance was due to nonshared environmental factors. Collectively, the results of these studies provide mixed evidence on the extent to which genetic factors influence the formation of antisocial friendship networks. These divergent results are perhaps due to differences in sample characteristics, the operationalization of delinquent peers, or some other unidentified factor or factors. Regardless, these studies implicate genetic factors—to varying degrees—in the befriending of antisocial peers.

THE CURRENT STUDY

The goal of the current study was to determine the relative effects of genetic and environmental factors in the development of self-control and in delinquent peer associations. It is important to note that prior research by Beaver (2008) and J. P. Wright et al. (2008) also used twins drawn from the Add Health data to examine issues related to the development of self-control and the formation of antisocial peer groups. However, their studies differ from the present one in three ways. First, Beaver examined difference scores only in the sample of MZ twins to estimate the effects that specific nonshared environments had on differences in low self-control and delinquent peers. His research did not estimate the heritability of these outcomes. Second, J. P. Wright and his colleagues examined the genetic and environmental effects on low self-control and delinquent peers, but they used a different modeling strategy than the one used in the present study. The different modeling strategy utilized herein helps determine whether the findings are robust or whether they may be explained away as statistical artifacts. Third, and unlike these other two studies, our research simultaneously estimates the effects of genetic and shared environmental factors and then estimates the effects of specific nonshared environments in a sample of MZ and DZ twins.

METHOD

DATA

Data for this study come from the Add Health (Udry, 2003). The Add Health used a school-based research design to select a nationally representative sample of adolescents enrolled in

7th through 12th grade. Using stratified sampling techniques, a total of 80 high schools and 52 middle schools were included in the study. On a specified day in 1994, all students attending these schools were administered a self-report survey that asked a variety of questions about their lives, their daily activities, and their peer and familial relationships. Overall, more than 90,000 adolescents submitted completed questionnaires (K. M. Harris et al., 2003).

A subsample of adolescents was then chosen to be interviewed in their homes along with their primary caregiver (usually their mother). The in-home questionnaires included more sensitive items that tapped the adolescents' involvement in delinquent and criminal behaviors, their involvement in risky behaviors, and the quality of their relationships with their parents and friends, among others (K. M. Harris et al., 2003). Questions were also asked that measured certain dimensions of the respondents' temperament, such as whether they have a temper, whether they are impulsive, and whether they are risk seekers. In addition, their primary caregivers also provided detailed information about various aspects of the adolescent's life. Altogether, 20,745 adolescents and 17,700 of their primary caregivers took part in the Wave 1 in-home survey (K. M. Harris et al., 2003).

Approximately 1 to 2 years after the Wave 1 in-home survey, respondents were administered a second wave of questionnaires. Given that relatively little time had lapsed since Wave 1, and given that most of the respondents were still adolescents, many of the same questions asked at Wave 1 were retained on the Wave 2 surveys. For example, adolescents were asked about their relationships with family and friends, their involvement in delinquency, and their involvement in sexual behaviors. Interviews were completed with 14,738 respondents at Wave 2, for a response rate of about 71%. Nearly 7 years after the first wave of data was collected, the third round of interviews was conducted. Most of the participants were between the ages of 18 and 26 years old; thus, the questionnaires had to be redesigned to include more age-appropriate items. The surveys now included questions that were geared toward indexing the respondents' childbearing history, their marital status, and their lifetime contact with the criminal justice system. At Wave 3, 15,197 of the original Wave 1 participants were reinterviewed, which corresponds to a response rate of about 73% (K. M. Harris et al., 2003).

Embedded within the entire sample of adolescents is a subsample of sibling pairs. During Wave 1 interviews, respondents were asked to indicate whether they were part of a twin pair. If the adolescent indicated he or she was a twin, then the co-twin was added to the sample. In addition, half siblings, genetically unrelated siblings (e.g., stepsiblings), and cousins were oversampled for inclusion in the study. A probability sample of full siblings was also retained in the sample. Subsequent analyses testing for potential selection biases in the sibling-pairs sample did not reveal any significant differences in the characteristics between the nationally representative sample and the sample of siblings (Jacobson & Rowe, 1998).

To calculate conservative parameter estimates, we included only MZ twin pairs and same-sex DZ twin pairs. Because the items assessing delinquent peer associations were not collected at Wave 3, and because the low self-control items were changed at Wave 3, our analysis was restricted to the first two waves of data. With these criteria in place, and once missing cases were removed and after deleting twins whose zygosity was undetermined, we were left with a final analytical sample size that ranged between $N = 662$ and $N = 914$ twins.

DEPENDENT VARIABLES

Low self-control. Even though Gottfredson and Hirschi's (1990) general theory has been one of the most empirically scrutinized theories in recent years, there is still a considerable

amount of disagreement concerning the most reliable and valid way to measure self-control (DeLisi, Hochstetler, & Murphy, 2003; Longshore, Stein, & Turner, 1998; Longshore, Turner, & Stein, 1996; Marcus, 2003, 2004; Piquero & Rosay, 1998). Although Gottfredson and Hirschi are quite clear that people with low levels of self-control are risk seekers, are impulsive, are self-centered, prefer physical activities to mental ones, prefer simple tasks, and have a temper, there is wide variability in the scales that have been used to tap self-control. Perhaps the most frequently used measurement strategy is the scale developed by Grasmick, Tittle, Bursik, and Arneklev (1993). Unfortunately, the Add Health surveys did not include items that could be used to construct the Grasmick et al. scale.

There are, however, a series of items available in the Add Health data that researchers have used to create a composite measure of self-control. Respondents were asked questions that tapped whether they have trouble paying attention, problems finishing their homework, troubled relationships with their teachers, and a difficult time staying focused. Perrone, Sullivan, Pratt, and Margaryan (2004) argued that "these questions tap into the simple tasks, physical activities, and impulsivity components of self-control" (p. 302). Last, respondents were asked whether they felt they do everything just right. This item indexed the self-centeredness dimension of self-control (Perrone et al., 2004). The exact same items were available at Wave 1 and Wave 2. Responses to each of the items were summed together to form the Wave 1 Low Self-Control scale ($\alpha = .65$) and the Wave 2 Low Self-Control scale ($\alpha = .62$). Higher values on both scales reflected lower levels of self-control.

We calculated a series of additional statistical specifications to examine the psychometric properties of the low self-control scales. We conducted principal components analysis, and only one component was present. Structural equation models were also calculated to determine whether the observable indicators all loaded on the same factor. Again, the results suggested that all of the items were significant indicators of one unobservable construct. In addition, we examined the predictive validity of the self-control scales. To do so, we created a Wave 1 and a Wave 2 Delinquency scale. The correlations between the Wave 1 Low Self-Control scale and the two delinquency scales ($r = .29, p < .05$ for the Wave 1 Delinquency scale; $r = .24, p < .05$ for the Wave 2 Delinquency scale) were statistically significant, and the correlation between the Wave 2 Low Self-Control scale and the Wave 2 Delinquency scale ($r = .32, p < .05$) was statistically significant. The effect sizes for the two self-control scales were very similar to those reported in Pratt and Cullen's (2000) meta-analysis. Finally, the two self-control scales were correlated, $r = .50, p < .05$, suggesting that the two scales have wave-to-wave consistency and are measuring the same latent construct over time. Taken together, there is reason to believe that the low self-control scales available in the Add Health study are valid and reliable measures of individual variation in self-control.

Drug-using peers. The Add Health data contain a number of questions that index each respondent's contact with, and exposure to, drug-using peers. At Wave 1 and Wave 2 interviews, respondents were asked three questions about their friends' drug and alcohol use. Specifically, adolescents were asked how many of their three closest friends smoked at least one cigarette each day, smoked marijuana at least once a month, and got drunk at least once per month. Responses to each question were coded as follows: 0 = 0 friends, 1 = 1 friend, 2 = 2 friends, and 3 = 3 friends. The three items were then summed together to create the Wave 1 Drug-Using Peers scale ($\alpha = .76$) and the Wave 2 Drug-Using Peers scale ($\alpha = .77$). Higher scores indicated more contact with drug-using peers.

Similar to the low self-control scales, we also subjected the drug-using peers scales to additional statistical testing to examine the psychometric properties of the scales. First, we calculated principal components analyses, and the results revealed that the covariance structure of the three items was accounted for by a single component. Second, structural equation models were estimated, and once again the results confirmed the presence of one latent factor. Third, and in line with prior research examining stability in drug-using friends over time (Warr, 1993), the wave-to-wave stability in drug-using peers was high ($r = .60$, $p < .05$). Finally, it should also be pointed out that researchers analyzing the Add Health data have used identical scales and found them to be some of the strongest correlates to delinquent involvement (Beaver & Wright, 2005a; Bellair, Roscigno, & McNulty, 2003).

SOCIALIZATION VARIABLES

Maternal disengagement. Adolescents who have cold and withdrawn parents are at risk for engaging in delinquency, using drugs and alcohol, and affiliating with delinquent peers (Gottfredson & Hirschi, 1990; Loeber & Stouthamer-Loeber, 1986; Patterson, 1982). To take these findings into account, we created a Maternal Disengagement scale. During Wave 1 interviews, adolescents were asked five different questions that measured maternal disengagement. For example, respondents were asked whether they are satisfied with their relationship with their mother, whether their mother is warm and loving toward them, and whether they are satisfied with the way their mother communicates with them. Responses to the items were added together, and higher scores indicated more maternal disengagement ($\alpha = .83$).

Maternal attachment. Research has indicated that parents who are emotionally connected and attached to their children are more apt to raise prosocial offspring (Sampson & Laub, 1993). In line with previous researchers analyzing the Add Health data (Haynie, 2001; Schreck, Fisher, & Miller, 2004), we included a two-item measure of maternal attachment. During Wave 1 interviews, adolescents were asked how much they thought their mother cares about them and how close they felt to their mother. Responses to these two questions were added to create the Maternal Attachment scale ($\alpha = .70$). Higher scores indicated greater maternal attachment.

Maternal involvement. Parents who are heavily involved in their children's lives act as a protective factor against antisocial outcomes (Loeber & Stouthamer-Loeber, 1986). As a result, and similar to the work of Crosnoe and Elder (2004), we developed a 10-item measure of maternal involvement from Wave 1 interviews with the adolescent. Specifically, participants were presented with a series of activities and asked to indicate which ones they had taken part in with their mother in the past 4 weeks. Items that were endorsed were assigned a value of 1; otherwise, they were coded 0. The items were then summed to form the Maternal Involvement index, where higher scores indicated more maternal involvement ($\alpha = .53$).

Parental permissiveness. Parents who fail to monitor and supervise their children or who fail to set appropriate boundaries for their children are more likely to raise disruptive and troublesome offspring (Gottfredson & Hirschi, 1990; Loeber & Stouthamer-Loeber, 1986). The Add Health data contain seven different items that index parental permissiveness. During Wave 1 interviews, adolescents were asked whether their parents let them make their own decisions about their bedtimes, curfews, friends, clothes, and what they eat. All

of the items were coded dichotomously (0 = *no*, 1 = *yes*). Responses to the questions were summed to create the Parental Permissiveness scale, where higher scores reflected more parental permissiveness ($\alpha = .65$).

CONTROL VARIABLES

Age. Levels of self-control are age graded, and contact with drug-using peers varies as a function of age (Gottfredson & Hirschi, 1990; Warr, 1993). As a result, and to help prevent misspecification of the statistical models, we included age as a continuous variable measured in years.

Gender. To control for potential gender differences in levels of self-control and exposure to drug-using friends, gender was included as a dichotomous dummy variable in all of the statistical models (0 = *female*, 1 = *male*).

Race. We included race as a control variable to account for potential racial differences in self-control and drug-using peers (0 = *White*, 1 = *non-White*).

ANALYSIS

To estimate genetic and environmental effects on low self-control and on drug-using peers, we use DeFries-Fulker (DF) analysis. DF analysis was originally developed by DeFries and Fulker (1985) to be used when one twin had an extreme score on some measure (e.g., having severe language deficits). The original DF formula, however, has been adjusted and extended, and now it can be used with samples drawn from the general population (Rodgers & Kohler, 2005; Rodgers, Rowe, & Li, 1994). The newly amended DF analysis, referred to as the "augmented" DF model, has been used quite frequently by a range of researchers from different disciplines including psychology, behavioral genetics, and criminology (Cyphers, Phillips, Fulker, & Mrazek, 1990; Haynie & McHugh, 2003; McCartan, 2007; Rodgers, Buster, & Rowe, 2001; Rodgers et al., 1994).

DF analysis is a regression-based analytical approach designed to analyze data consisting of sibling pairs. The basic DF equation takes the following form:

$$K_1 = b_0 + b_1K_2 + b_2R + b_3(R * K_2) + e, \quad (1)$$

where K_1 is the score for one twin on a particular measure, K_2 is his or her co-twin's score on that same measure, R is the coefficient of genetic relatedness ($R = 1.0$ for MZ twins, $R = .5$ for DZ twins), and $R * K_2$ is the multiplicative interaction term between R and K_2 . For this equation, b_0 is the constant, b_1 is the proportion of variance in K_1 explained by shared environmental influences (c^2), b_2 is not usually interpreted in the DF model, and b_3 is the proportion of variance in K_1 explained by genetic effects (h^2). The effects of the nonshared environment (e^2) and measurement error are captured by the error term, e .

Recently, Rodgers and Kohler (2005) reformulated the DF model presented in Equation 1 to help overcome some inconsistencies inherently built into the DF equation. Their newly transformed DF equation is

$$K_1 = b_0 + b_1(K_2 - K_m) + b_2(R * (K_2 - K_m)) + e, \quad (2)$$

where K_1 is still the score for one twin on a particular measure, K_2 is still the co-twin's score on that same measure, and R is still the coefficient of genetic relatedness. The main difference is that in this equation the term K_m is introduced into the model. K_m is the mean of the measure for K_2 . In contrast to Equation 1, K_2 is being centered on its mean, whereas in Equation 1 it was left untransformed. The main effect of R is also removed in Equation 2. The interpretation of the coefficients, however, remains unchanged. For example, b_1 is the shared environmental effects and b_2 is the genetic effects. The unexplained variance is attributable to measurement error and to nonshared environmental effects.¹

Equation 2 effectively removes the variance in K_1 that is accounted for by the shared environment and by genetic factors. The terms b_1 and b_2 are in many ways like latent factors because it is not possible to determine which shared environments are important or which genetic factors are important. Although some researchers have introduced shared environmental variables into DF models to try to identify salient shared environments, this procedure is probably unadvisable because, as Turkheimer, D'Onofrio, Maes, and Eaves (2005) argue, "by adding the shared family-level covariate to this model [i.e., the DF model], one is attempting to predict shared twin variability in a model that already has a term specifically designed to account for all of it" (p. 1224). However, it is possible to introduce measures of the nonshared environment into the equation in order to determine their effects on K_1 once the effects of the shared environment and genetic influences are held constant.

Rodgers et al. (1994) presented an expanded DF equation to demonstrate how to include nonshared environmental effects. This expanded DF equation can be used in conjunction with Equation 2 to yield the following formula:

$$K_1 = b_0 + b_1(K_2 - K_m) + b_2(R * (K_2 - K_m)) + b_3\text{ENVDIF} + e. \quad (3)$$

This equation introduces one additional term, ENVDIF. ENVDIF is the difference score on a particular environmental measure between two twins from the same twin pair. For example, Twin 2's score on the Maternal Disengagement scale can be subtracted from Twin 1's score on the Maternal Disengagement scale to produce a difference score in maternal disengagement. In the above equation, this difference score could be entered as the ENVDIF variable. Essentially, ENVDIF captures differences between twins and uses those difference scores to tap the nonshared environment. Although Equation 3 shows the inclusion of only one ENVDIF, more than one ENVDIF variable can be entered into the model without biasing the coefficients. All of the remaining regression coefficients can be interpreted the same way that they were in Equation 2.²

Although it might seem that nonshared environmental effects are detected if the ENVDIF coefficient is statistically significant, such a conclusion would be too hasty because the possibility exists that the ENVDIF coefficient may be estimating nonshared genetic influences rather than nonshared environmental effects. To determine whether a significant ENVDIF coefficient is being driven by the nonshared environment or by nonshared genetic effects, an additional term needs to be introduced into Equation 3. The inclusion of this term results in the following equation:

$$K_1 = b_0 + b_1(K_2 - K_m) + b_2(R * (K_2 - K_m)) + b_3\text{ENVDIF} + b_4(\text{ENVDIF} * R) + e. \quad (4)$$

The only difference between this equation and Equation 3 is the inclusion of the interaction term, ENVDIF * R . If the coefficient for ENVDIF * R is significant, then the effect of

ENVDIF (b_3) is attributable to nonshared genetic effects. On the other hand, if the ENVDIF * R coefficient is not statistically significant, then the effect of ENVDIF (b_3) is attributable to nonshared environmental effects. It is important to note that Equation 4 is estimated only if the ENVDIF variable is statistically significant in Equation 3.

Researchers using DF analysis are confronted with the decision of which twin from each twin pair should be selected to be used as the dependent variable (K_1) and which twin should be selected to be used as the independent variable (K_2). This problem is often resolved by "double entering" the twins, where each twin is entered into the data twice: One time the twin is the dependent variable and the co-twin is the independent variable, and one time the twin is the independent variable and the co-twin is the dependent variable. Indeed, this is the most common approach when using the augmented DF equations, and we use double entry in the current study (Haynie & McHugh, 2003; Kohler & Rodgers, 2001; Rodgers et al., 2001; Rodgers & Rowe, 1987). Although double entering twins is advantageous, it also violates one of the main assumptions of ordinary least squares regression—namely, the observations are no longer independent from each other. Although nonindependence does not bias the regression slopes, it does deflate the standard errors and thus biases tests of statistical significance for the coefficients (Hanushek & Jackson, 1977). To take the clustering of observations into account, all of the standard errors were calculated using Huber/White variance estimators.

DF analyses were calculated for the Wave 1 and Wave 2 Low Self-Control scales and for the Wave 1 and Wave 2 Drug-Using Peers scales. All four of the parental socialization scales were transformed into difference scores by subtracting the two twins' scores on each measure. These difference scores were then entered into the DF equation as measures of the nonshared environment. Any nonshared environmental measures that were statistically significant using Equation 3 were then reexamined using Equation 4 to determine whether nonshared genetic factors or nonshared environment influences were driving this effect.

RESULTS

We begin our analyses by examining cross-twin correlations for the low self-control scales and the delinquent peers scales. Zero-order cross-twin correlations allow for an initial estimation of whether genetic influences are potentially important to self-control and to associating with drug-using peers. In general, if the MZ cross-twin correlation is significantly larger than the DZ cross-twin correlation, then there is at least a minimal genetic effect on the measure. Table 1 reveals the cross-twin correlations for the entire sample of twins and separately for MZ and DZ twins. As can be seen, all of the cross-twin correlations were statistically significant, but the MZ cross-twin correlations were all larger than the DZ cross-twin correlations. These findings provide initial evidence that low self-control and contact with drug-using peers may be at least partially genetically influenced. However, to provide a more rigorous examination of the relative effects of environmental influences and genetic forces on the self-control and drug-using peers scales, we next turn to the results generated from DF analysis.

Table 2 presents the results of the DF equations using the low self-control scales as the dependent variable. Model 1 displays the results of a baseline DF model that estimated genetic and shared environmental effects on the Wave 1 Low Self-Control scale with race,

TABLE 1: Cross-Twin Correlations for the Low Self-Control Scales and the Delinquent Peers Scales

	<i>All Twins</i>	<i>MZ Twins</i>	<i>DZ Twins</i>
Low Self-Control (Wave 1)	.290*	.406*	.122*
Low Self-Control (Wave 2)	.232*	.316*	.118*
Drug-Using Peers (Wave 1)	.588*	.666*	.435*
Drug-Using Peers (Wave 2)	.551*	.674*	.416*

Note. MZ = monozygotic; DZ = dizygotic.

* $p < .05$, two tailed.

TABLE 2: DeFries-Fulker (DF) Analysis of Low Self-Control at Waves 1 and 2

	<i>Low Self-Control at Wave 1</i>				<i>Low Self-Control at Wave 2</i>			
	<i>Model 1</i>		<i>Model 2</i>		<i>Model 3</i>		<i>Model 4</i>	
	b	SE	b	SE	b	SE	b	SE
DF analysis components								
Heritability	.56*	.13	.52*	.14	.40*	.15	.44*	.15
Shared environment	-.16	.11	-.11	.12	-.09	.12	-.09	.12
Nonshared environments								
Maternal disengagement			.14*	.03			.11*	.03
Maternal attachment			-.03	.08			.12	.10
Maternal involvement			.05	.05			.09	.05
Parental permissiveness			.05	.06			.14*	.06
Control variables								
Age	.01	.06	.00	.06	-.03	.07	-.03	.07
Gender	.38	.20	.46*	.20	.19	.21	.21	.21
Race	-.08	.20	-.05	.21	-.27	.22	-.18	.22

Note. Huber/White standard errors are presented.

* $p < .05$, two tailed.

age, and gender introduced as control variables. Remember the slope of $(R^* (K_2 - K_m))$ is interpreted as the proportion of variance accounted for by genetic factors (i.e., heritability) and the slope of $(K_2 - K_m)$ is interpreted as the proportion of variance accounted for by the shared environment. To facilitate readability, these two terms have been labeled as “heritability” and “shared environment” in Table 2. The results of this model revealed that genetic influences accounted for 56% of the variance in low self-control. The coefficient for the shared environment was negative, but this does not mean that the shared environment explained a negative amount of variance in low self-control. Instead, because the slope for the shared environment is not statistically significant, the results simply mean that the confidence interval for the slope of the shared environment includes the value of 0. When a coefficient is not significantly different from 0, the sign and the value of the coefficient should be ignored and subsequently equated with 0. In this case, the slope of the shared environment ($b = -.16$, $p > .05$) should really be interpreted as $b = 0$. Applying this logic to the current analyses means that the proportion of variance in self-control that was accounted for by the shared environment is 0. The remaining variance (44%) in low self-control was explained by the nonshared environment and measurement error.

Recall that if the nonshared environment accounts for a significant amount of variance, then it is possible to estimate the effects of specific nonshared environments. We next turn our attention to Model 2, which introduces four different nonshared environmental variables into the DF equation. The reader is reminded that the nonshared environmental variables were constructed by calculating difference scores. Three findings warrant attention in this model. First, genetic influences accounted for more than one half (52%) of the variance in low self-control. Second, the shared environment accounted for none (0%) of the variance in low self-control. Third, of the four measures of the nonshared environment, only maternal disengagement had a statistically significant effect on low self-control.³ The association between maternal disengagement and low self-control was positive, meaning that the twin who received more maternal disengagement had lower levels of self-control. This effect was observed even after holding genetic effects constant.

The next set of analyses is identical to those previously calculated, except that the Wave 2 Low Self-Control scale was used as the dependent variable. As shown in Model 3, genetic effects accounted for 40% of the variance in low self-control, the shared environment had no effect on low self-control (0% of the variance), and the nonshared environment and measurement error accounted for the remaining 60% of the variance.

In Model 4, the four nonshared environmental variables (all measured at Wave 1) were used to predict variance in the Wave 2 Low Self-Control scale. After partitioning out the effects of genetic influences, two nonshared environmental variables were statistically significant: maternal disengagement and parental permissiveness. The association for both of these variables was positive, meaning that the twin who received more maternal disengagement or more parental permissiveness had lower levels of self-control. It is important to note that in Model 4, genetic effects accounted for 44% of the variance in low self-control, whereas the remaining 56% of variance was attributable to the nonshared environment and measurement error. Shared environmental effects were zero. Taken together, the DF models calculated for the low self-control scales indicated that genetic and nonshared environmental effects accounted for 100% of the variance, whereas the shared environment had no effect on individual levels of self-control.

The last set of analyses used the drug-using peers scales as dependent variables in the DF equations controlling for the effects of age, gender, and race. Model 1 of Table 3 contains the findings for the baseline DF model where the Wave 1 Drug-Using Peers scale is the outcome measure. Results gleaned from this model indicated the genetic effects accounted for 37% of the variance, the shared environment accounted for 27% of the variance, and the nonshared environment and measurement error accounted for the remaining 36% of variance. As with the low self-control scales, the environment and genes both contributed to the explained variance in drug-using peers; however, and in contrast to the low self-control models, the shared environment explained a statistically significant amount of variance (27%).

Because a significant portion of the variance in the Wave 1 Drug-Using Peers scale was accounted for by the nonshared environment, we also calculated an additional equation where the four nonshared environmental variables were entered into the equation. As shown in Model 2, the introduction of the nonshared environmental variables increased the percentage of explained variance attributable to genetic effects to 40%, whereas the percentage of variance attributable to the shared environment decreased to 22%. Most important,

TABLE 3: DeFries-Fulker (DF) Analysis of Drug-Using Peers at Waves 1 and 2

	<i>Drug-Using Peers at Wave 1</i>				<i>Drug-Using Peers at Wave 2</i>			
	<i>Model 1</i>		<i>Model 2</i>		<i>Model 3</i>		<i>Model 4</i>	
	b	SE	b	SE	b	SE	b	SE
DF analysis components								
Heritability	.37*	.12	.40*	.13	.53*	.12	.62*	.13
Shared environment	.27*	.10	.22*	.11	.12	.11	.02	.11
Nonshared environments								
Maternal disengagement			.03	.02			.03	.02
Maternal attachment			-.16*	.07			-.09	.06
Maternal involvement			.04	.04			.02	.04
Parental permissiveness			-.01	.04			.01	.05
Control variables								
Age	.18*	.04	.20*	.05	.14*	.05	.18*	.05
Gender	.12	.15	.13	.15	.28	.16	.37*	.17
Race	-.30	.16	-.37*	.16	-.38*	.17	-.39*	.18

Note. Huber/White standard errors are presented.

* $p < .05$, two tailed.

however, was that only one of the nonshared environmental variables, maternal attachment, was significantly associated with the Wave 1 Drug-Using Peers scale. The effect of the maternal attachment variable was negative, meaning that the twin who received more maternal attachment had fewer drug-using peers. The remaining three nonshared environmental variables were not related to the Drug-Using Peers scale in the DF equation.

Models 3 and 4 of Table 3 were estimated using the Wave 2 Drug-Using Peers scale as the dependent variable. The results presented in Model 3 revealed that 53% of the variance in the Drug-Using Peers scale was accounted for by genetic factors, 0% of the variance was attributable to the shared environment, and 47% of the variance was accounted for by the nonshared environment and measurement error.

Last, we entered the four nonshared environmental variables into the DF equation to determine whether they were associated with the Wave 2 Drug-Using Peers scale. In this model, 62% of the variance in the Drug-Using Peers scale was due to genetic influences, 0% of the variance was due to the shared environment, and 38% of the variance was attributable to the nonshared environment. Of particular importance are the results for the nonshared environmental variables, which indicated that none of the four variables was significantly associated with the Wave 2 Drug-Using Peers scale. The results presented in Table 3 revealed the importance of both genetic and environmental effects on the two drug-using peers scales.

DISCUSSION

Social learning theory and low self-control theory are two of the most empirically supported criminological theories (Pratt & Cullen, 2000). Extant empirical tests of these perspectives have focused on linking low levels of self-control to delinquent involvement and linking contact with delinquent peers to antisocial behaviors. Comparatively less research

has attempted to identify the causes of low self-control and the forces that lead youths to befriend delinquent peers. The current article addressed this gap in the literature by examining whether environmental and genetic factors are associated with individual levels of self-control and with the formation of drug-using friendship networks. To address these issues, a sample of MZ and DZ twins from the Add Health study was analyzed by using DF regression techniques. The results of the DF models revealed three broad findings.

First, once genetic effects were held constant, the shared environment had no effect on either of the two low self-control scales and no effect on the Wave 2 Drug-Using Peers scale. Indeed, the shared environment had a significant effect on only the Wave 1 Drug-Using Peers scale, where it accounted for between 22% and 27% of the variance. Second, and relatedly, across all of the models, the nonshared environment accounted for a large proportion of variance in the dependent variables. However, the specific measures of the nonshared environment—that is, the ones that were tapping differential parental treatment—had small and relatively inconsistent effects. The one exception was maternal disengagement, which was significantly associated with the Wave 1 and Wave 2 Low Self-Control scales. However, the only other two nonshared environmental measures that were significant were parental permissiveness and maternal attachment, both of which were statistically significant in only one of the four equations.

Third, the Wave 1 and Wave 2 Low Self-Control scales and the Wave 1 and Wave 2 Drug-Using Peers scales were under substantial genetic influence. Genetic factors accounted for between 40% and 56% of the variance in low self-control and between 37% and 62% of the variance in drug-using peers. Taken together, analysis of the Add Health data suggests that contact with drug-using peers and levels of self-control are strongly affected by the nonshared environment and genetic factors, whereas the shared environment is relatively unimportant.

The results garnered from the DF models have important implications for Gottfredson and Hirschi's (1990) parental management thesis and for social learning theory. Recall that Gottfredson and Hirschi argued that levels of self-control were determined largely by the way in which parents socialize their children. Analysis of the Add Health data revealed a very different picture—namely, that once genetic effects were removed, the shared environment did not exert any effect on the development of self-control. We also found very limited evidence that differential parental treatment (i.e., the nonshared environment) was associated with low self-control. The skeptical reader will be quick to point out that there is a body of research that has revealed some evidence linking parental socialization to individual levels of self-control (Gibbs et al., 1998; Gibbs et al., 2003; Polakowski, 1994; Unnever, Cullen, & Pratt, 2003). Unfortunately, none of these studies has controlled for genetic influences, raising the very real possibility that the models are misspecified and the findings are spurious (J. P. Wright & Beaver, 2005).

Our findings also add to an emerging body of empirical research revealing that biogenetic factors are a cause—perhaps even the dominant cause—of problems with self-control and self-regulation (Beaver & Wright, 2005b; Beaver et al., 2007; Tremblay, 2003; J. P. Wright & Beaver, 2005). We are, of course, not saying that the environment is unimportant. Rather, we are pointing to the likelihood that the environments that are most likely to be linked to self-control are rarely studied by criminologists. For example, there is now convincing research revealing that prenatal exposure to toxins, such as drugs, alcohol, nicotine, and lead, interferes with normal brain development (Karr-Morse & Wiley, 1997; Sood

et al., 2001; Yolton, Dietrich, Auinger, Lanphear, & Hornung, 2005). Given that levels of self-control are closely tied to the structure and functioning of the brain, any event or pathogen that may lead to neurological deficits may also reduce levels of self-control (Barkley, 1997; Beaver & Wright, 2005b; Beaver et al., 2007). Future research should begin to unravel the complex ways that these nonshared environmental effects may ultimately lead to maladaptive behaviors and antisocial traits.

Our findings also partially stand in direct contrast to arguments that parents are able to influence and affect their child's choice of peer groups. As our findings revealed, most of the variance in exposure to drug-using peers was accounted for by genetic factors. Behavioral geneticists have long theorized that individuals create their own environments based in large part on genotype—a phenomenon that is referred to as a Gene $\times$ Environment correlation (Scarr, 1992; Scarr & McCartney, 1983). The logic of Gene $\times$ Environment correlations can easily be applied to delinquent peer group formation (Cleveland et al., 2005). For example, individuals with a genetic propensity to engage in delinquency, or to use illegal substances, are likely to seek out peers with these same preferences. In this case, genes are the driving force behind why some youths associate with antisocial friends whereas other adolescents associate with prosocial peers. From a theoretical standpoint, these findings would be in line with a self-selection argument and would cast doubt on a purely social causation explanation to the delinquent peers–delinquent involvement nexus.

Before concluding, it is important to touch on three limitations of the analysis. First, there were only a limited number of parenting measures available in the Add Health, which hampered our ability to provide a more stringent test of Gottfredson and Hirschi's (1990) parental socialization thesis. For example, there were no questions that measured parental recognition of deviance or parental punishment. This problem, however, is not unique to the current study; many prior studies examining the effect parents have on the development of self-control did not include all of the parenting dimensions outlined by Gottfredson and Hirschi (Unnever et al., 2003; J. P. Wright & Beaver, 2005). Second, the Add Health data did not contain items that could be used to construct Grasmick et al.'s (1993) Low Self-Control scale. Even so, we were able to reconstruct the Low Self-Control scale used by Perrone and her colleagues (2004). Third, the analysis was based on a sample of MZ and DZ twins, leaving open the possibility that the findings reported here may not generalize to the larger population.

Nonetheless, the results of our study provide a serious challenge to some of the central tenets from low self-control theory and from social learning theory. But does this mean that we should abandon these theories and start afresh? Of course not, nor are we advocating such a radical position. Instead, we are in agreement with Walsh (2000, 2002a), who has recently argued that many of the dominant criminological theories should be revised to incorporate findings from biology and genetics. Not only would this type of theoretical integration link criminology with other disciplines, but also it would provide a much more complete explanation of crime, delinquency, and antisocial behavior.

NOTES

1. In describing the use of Equation 2, Rodgers and Kohler (2005) suggest that "in cases where trait means significantly differ across kinship categories (e.g., across MZ [monozygotic] and DZ [dizygotic] twins), K_m should be calculated separately by kinship category, and the reformulated DeFries-Fulker [DF] analysis presented here [i.e., Equation 2] based on centered scores should use category-specific means" (p. 212). We calculated independent-samples *t* tests to examine whether there were mean differences between MZ and DZ twins on the low self-control scales and the drug-using peers scales. The results

of the t tests did not reveal any average differences in the dependent variables between MZ and DZ twins. As a result, in all of our analyses K_m is the average combined score for MZ and DZ twins.

2. Model misspecification is not a problem with DF analysis. That is, the omission of variables will not bias the results because all shared environmental effects (e.g., neighborhood conditions and family influences) are captured by the shared environmental term (b_1) and the omission of nonshared environmental effects is captured by the error term (e). Including particular measures of the nonshared environment helps to remove some of the unexplained variance, but there is no reason to include measures of the shared environment. Thus, the lack of control variables in the models will not affect the substantive findings. We do, however, include control variables for age, gender, and race. All of the models were recalculated without these control variables, and the results remained virtually identical.

3. It is important to note that for all models estimated, any time that a nonshared environmental variable was statistically significant, we reestimated the models using Equation 4. Recall that Equation 4 is calculated to determine whether nonshared environmental effects are really capturing nonshared genetic effects. The results of the models revealed that the effects were due to the nonshared environment, not to genetic differences. Therefore, we do not present the results garnered from using Equation 4.

REFERENCES

- Akers, R. L. (1998). *Social learning and social structure: A general theory of crime and deviance*. Boston: Northeastern University Press.
- Akers, R. L., & Jensen, G. F. (2006). The empirical status of social learning theory of crime and deviance: The past, present, and future. In F. T. Cullen, J. P. Wright, & K. R. Blevins (Eds.), *Taking stock: The status of criminological theory* (Advances in Criminological Theory Series Vol. 15, pp. 37-76). New Brunswick, NJ: Transaction Publishers.
- Barkley, R. A. (1997). *ADHD and the nature of self-control*. New York: Guilford.
- Baron, S. W. (2003). Self-control, social consequences, and criminal behavior: Street youth and the general theory of crime. *Journal of Research in Crime and Delinquency*, 40, 403-425.
- Beaver, K. M. (2008). Nonshared environmental influences on adolescent delinquent involvement and adult criminal behavior. *Criminology*, 46, 341-370.
- Beaver, K. M., & Wright, J. P. (2005a). Biosocial development and delinquent involvement. *Youth Violence and Juvenile Justice*, 3, 168-192.
- Beaver, K. M., & Wright, J. P. (2005b). Evaluating the effects of birth complications on low self-control in a sample of twins. *International Journal of Offender Therapy and Comparative Criminology*, 49, 450-471.
- Beaver, K. M., Wright, J. P., & DeLisi, M. (2007). Self-control as an executive function: Reformulating Gottfredson and Hirschi's parental socialization thesis. *Criminal Justice and Behavior*, 34, 1345-1361.
- Beaver, K. M., Wright, J. P., & DeLisi, M. (2008). Delinquent peer group formation: Evidence of a gene X environment correlation. *Journal of Genetic Psychology*, 169, 227-244.
- Bellair, P. E., Roscigno, V. J., & McNulty, T. L. (2003). Linking local labor market opportunity to violent adolescent delinquency. *Journal of Research in Crime and Delinquency*, 40, 6-33.
- Cairns, R. B., & Cairns, B. D. (1994). *Lifelines and risks: Pathways of youth in our time*. Cambridge, UK: Cambridge University Press.
- Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., et al. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, 297, 851-854.
- Cauffman, E., Steinberg, L., & Piquero, A. R. (2005). Psychological, neuropsychological and physiological correlates of serious antisocial behavior in adolescence: The role of self-control. *Criminology*, 43, 133-175.
- Chapple, C. L. (2005). Self-control, peer relations, and delinquency. *Justice Quarterly*, 22, 89-106.
- Cleveland, H. H., Wiebe, R. P., & Rowe, D. C. (2005). Sources of exposure to smoking and drinking friends among adolescents: A behavioral-genetic evaluation. *Journal of Genetic Psychology*, 166, 153-169.
- Crosnoe, R., & Elder, G. H., Jr. (2004). Family dynamics, supportive relationships, and educational resilience during adolescence. *Journal of Family Issues*, 25, 571-602.
- Cyphers, L. H., Phillips, K., Fulker, D. W., & Mrazek, D. A. (1990). Twin temperament during the transition from infancy to early childhood. *Journal of the American Academy of Child and Adolescent Psychiatry*, 29, 392-397.
- DeFries, J. C., & Fulker, D. W. (1985). Multiple regression analysis of twin data. *Behavior Genetics*, 15, 467-473.
- DeLisi, M., Hochstetler, A., & Murphy, D. S. (2003). Self-control behind bars: A validation study of the Grasmick et al. scale. *Justice Quarterly*, 20, 241-263.
- DiLalla, L. F. (2002). Behavior genetics of aggression in children: Review and future directions. *Developmental Review*, 22, 593-622.
- Gibbs, J. J., Giever, D., & Higgins, G. E. (2003). A test of Gottfredson and Hirschi's general theory using structural equation modeling. *Criminal Justice and Behavior*, 30, 441-458.

- Gibbs, J. J., Giever, D., & Martin, J. S. (1998). Parental management and self-control: An empirical test of Gottfredson and Hirschi's general theory. *Journal of Research in Crime and Delinquency*, 35, 40-70.
- Gottfredson, M., & Hirschi, T. (1990). *A general theory of crime*. Palo Alto, CA: Stanford University Press.
- Grasmick, H. G., Tittle, C. R., Bursik, R. J., Jr., & Arneklev, B. J. (1993). Testing the core empirical implications of Gottfredson and Hirschi's general theory of crime. *Journal of Research in Crime and Delinquency*, 30, 5-29.
- Hanushek, E. A., & Jackson, J. E. (1977). *Statistical methods for social scientists*. San Diego, CA: Academic Press.
- Harris, J. R. (1998). *The nurture assumption: Why children turn out the way they do*. New York: Free Press.
- Harris, K. M., Florey, F., Tabor, T., Bearman, P. S., Jones, J., & Udry, J. R. (2003). *The National Longitudinal Study of Adolescent Health: Study design*. Chapel Hill, NC: Carolina Population Center. Available at <http://www.cpc.unc.edu/projects/addhealth/design>
- Haynie, D. L. (2001). Delinquent peers revisited: Does network structure matter? *American Journal of Sociology*, 106, 1013-1057.
- Haynie, D. L., & McHugh, S. (2003). Sibling deviance: In the shadows of mutual and unique friendship effects. *Criminology*, 41, 355-392.
- Hirschi, T. (1969). *Causes of delinquency*. Berkeley: University of California Press.
- Iervolino, A. C., Pike, A., Manke, B., Reiss, D., Hetherington, E. M., & Plomin, R. (2002). Genetic and environmental influences in adolescent peer socialization: Evidence from two genetically sensitive designs. *Child Development*, 73, 162-174.
- Ishikawa, S. S., & Raine, A. (2003). Prefrontal deficits and antisocial behavior. In B. B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 277-304). New York: Guilford.
- Jacobson, K., & Rowe, D. C. (1998). Genetic and shared environment influences on adolescent BMI: Interaction with race and sex. *Behavior Genetics*, 28, 265-278.
- Karr-Morse, R., & Wiley, M. S. (1997). *Ghosts from the nursery: Tracing the roots of violence*. New York: Atlantic Monthly.
- Kendler, K. S., Jacobson, K. C., Gardner, C. O., Gillespie, N., Aggen, S. A., & Prescott, C. A. (2007). Creating a social world: A developmental twin study of peer-group deviance. *Archives of General Psychiatry*, 64, 958-965.
- Kohler, H. P., & Rodgers, J. L. (2001). DF-analysis of heritability with double-entry twin data: Asymptotic standard errors and efficient estimation. *Behavior Genetics*, 31, 179-191.
- Lahey, B. B., & Waldman, I. D. (2003). A developmental propensity model of the origins of conduct problems during childhood and adolescence. In B. B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 76-117). New York: Guilford.
- Loeber, R., & Stouthamer-Loeber, M. (1986). Family factors as correlates and predictors of juvenile conduct problems and delinquency. In M. Tonry & N. Morris (Eds.), *Crime and justice: A review of research* (Vol. 7, pp. 29-149). Chicago: University of Chicago Press.
- Longshore, D., Stein, J. A., & Turner, S. (1998). Reliability and validity of a self-control measure: Rejoinder. *Criminology*, 36, 175-182.
- Longshore, D., Turner, S., & Stein, J. A. (1996). Self-control in a criminal sample: An examination of construct validity. *Criminology*, 34, 209-228.
- Marcus, B. (2003). An empirical examination of the construct validity of two alternative self-control measures. *Educational and Psychological Measurement*, 63, 674-706.
- Marcus, B. (2004). Self-control in the general theory of crime: Theoretical implications of a measurement problem. *Theoretical Criminology*, 8, 33-55.
- McCartan, L. M. (2007). Inevitable, influential, or unnecessary? Exploring the utility of genetic explanation for delinquent behavior. *Journal of Criminal Justice*, 35, 219-233.
- Meyer-Lindenberg, A., Buckholtz, J. W., Kolachana, B., Hariri, A. R., Pezawas, L., Blasi, G., et al. (2006). Neural mechanisms of genetic risk for impulsivity and violence in humans. *Proceedings of the National Academy of Sciences of the United States of America*, 103, 6269-6274.
- Moffitt, T. E. (1993). Adolescence-limited and life-course persistent antisocial behavior: A developmental taxonomy. *Psychological Review*, 100, 674-701.
- Morgan, A. B., & Lilienfeld, S. O. (2002). A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clinical Psychology Review*, 20, 113-136.
- Nigg, J. T., & Huang-Pollock, C. L. (2003). An early-onset model of the role of executive functions and intelligence in conduct disorder/delinquency. In B. B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 227-553). New York: Guilford.
- Patterson, G. R. (1982). *Coercive family process*. Eugene, OR: Castilia.
- Perrone, D., Sullivan, C. J., Pratt, T. C., & Margaryan, S. (2004). Parental efficacy, self-control, and delinquency: A test of a general theory of crime on a nationally representative sample of youth. *International Journal of Offender Therapy and Comparative Criminology*, 48, 298-312.
- Pfefferbaum, A., Sullivan, E. V., Swan, G. E., & Carmelli, D. (2000). Brain structure in men remains highly heritable in the seventh and eighth decades of life. *Neurobiology of Aging*, 21, 63-74.
- Piquero, A. R., & Rosay, A. B. (1998). The reliability and validity of Grasmick et al.'s self-control scale: A comment on Longshore et al. *Criminology*, 35, 157-174.

- Polakowski, M. (1994). Linking self- and social control with deviance: Illuminating the structure underlying a general theory of crime and its relation to deviant identity. *Journal of Quantitative Criminology*, 10, 41-78.
- Pratt, T. C., & Cullen, F. T. (2000). The empirical status of Gottfredson and Hirschi's general theory of crime: A meta-analysis. *Criminology*, 38, 931-964.
- 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 and Delinquency*, 41, 219-243.
- Price, T. S., Simonoff, E., Waldman, I., Asherson, P., & Plomin, R. (2001). Hyperactivity in preschool children is highly heritable. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40, 1362-1364.
- Raine, A. (2002). Annotation: The role of prefrontal deficits, low autonomic arousal, and early health factors in the development of antisocial and aggressive behavior in children. *Journal of Child Psychology and Psychiatry*, 43, 417-434.
- Reiss, D., Neiderhiser, N. M., Hetherington, E. M., & Plomin, R. (2000). *The relationship code: Deciphering the genetic and social influences on adolescent development*. Cambridge, UK: Cambridge University Press.
- Rhee, S. H., & Waldman, I. D. (2003). Testing alternative hypotheses regarding the role of development on genetic and environmental influences underlying antisocial behavior. In B. B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 305-318). New York: Guilford.
- Rietveld, M. J. H., Hudziak, J. J., Bartels, M., van Beijsterveldt, C. E. M., & Boomsma, D. I. (2003). Heritability of attention problems in children: Cross-sectional results from a study of twins, age 3 to 12 years. *Neuropsychiatric Genetics*, 1176, 102-113.
- Robinson, M. (2004). *Why crime? An integrated systems theory of antisocial behavior*. Upper Saddle River, NJ: Pearson Prentice Hall.
- Rodgers, J. L., Buster, M., & Rowe, D. C. (2001). Genetic and environmental influences on delinquency: DF analysis of NLSY kinship data. *Journal of Quantitative Criminology*, 17, 145-168.
- Rodgers, J. L., & Kohler, H. P. (2005). Reformulating and simplifying the DF analysis model. *Behavior Genetics*, 35, 211-217.
- Rodgers, J. L., & Rowe, D. C. (1987). IQ similarity in twins, siblings, half-siblings, cousins, and random pairs. *Intelligence*, 11, 199-206.
- Rodgers, J. L., Rowe, D. C., & Li, C. (1994). Beyond nature versus nurture: DF analysis of nonshared influences on problem behaviors. *Developmental Psychology*, 30, 374-384.
- Rutter, M. (2006). *Genes and behavior: Nature-nurture interplay explained*. Malden, MA: Blackwell.
- Sampson, R. J., & Laub, J. H. (1993). *Crime in the making: Pathways and turning points through life*. Cambridge, MA: Harvard University Press.
- Scarr, S. (1992). Developmental theories for the 1990s: Development and individual differences. *Child Development*, 63, 1-19.
- Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype → environment effects. *Child Development*, 54, 424-435.
- Schreck, C. J., Fisher, B. S., & Miller, J. M. (2004). The social context of violent victimization: A study of the delinquent peer effect. *Justice Quarterly*, 21, 23-47.
- Séguin, J. R., Boulerice, B., Harden, P. W., Tremblay, R. E., & Pihl, R. O. (1999). Executive functions and physical aggression after controlling for attention deficit hyperactivity disorder, general memory, and IQ. *Journal of Child Psychology and Psychiatry*, 40, 1197-1208.
- Séguin, J. R., Nagin, D., Assaad, J. M., & Tremblay, R. E. (2004). Cognitive-neuropsychological function in chronic physical aggression and hyperactivity. *Journal of Abnormal Psychology*, 113, 603-613.
- Sood, B., Delaney-Black, V., Covington, C., Nordstrom-Klee, B., Ager, J., Templin, T., et al. (2001). Prenatal alcohol exposure and childhood behavior at age 6 to 7 years: I. Dose-response effect. *Pediatrics*, 108, 34-43.
- Thompson, P. M., Cannon, T. D., Narr, K. L., van Erp, T., Poutanen, V., Huttunen, M., et al. (2001). Genetic influences on brain structure. *Nature Neuroscience*, 4, 1-6.
- Toga, A. W., & Thompson, P. M. (2005). Genetics of brain structure and intelligence. *Annual Review of Neuroscience*, 28, 1-23.
- Tremblay, R. E. (2003). Why socialization fails: The case of chronic physical aggression. In B. B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 182-224). New York: Guilford.
- Turkheimer, E., D'Onofrio, B. M., Maes, H. H., & Eaves, L. J. (2005). Analysis and interpretation of twins studies including measures of the shared environment. *Child Development*, 76, 1217-1233.
- 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.
- Udry, J. R. (2003). The National Longitudinal Study of Adolescent Health (Add Health), Waves I and II, 1994-1996; Wave III, 2001-2002 [Machine-readable data file and documentation]. 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.
- Walsh, A. (2000). Behavior genetics and anomie/strain theory. *Criminology*, 38, 1075-1107.
- Walsh, A. (2002a). *Biosocial criminology: Introduction and integration*. Cincinnati, OH: Anderson.
- Walsh, A. (2002b). Essay review: Companions in crime: A biosocial perspective. *Human Nature Review*, 2, 169-178.
- Warr, M. (1993). Age, peers, and delinquency. *Criminology*, 31, 17-40.

- Warr, M. (2002). *Companions in crime: The social aspects of criminal conduct*. New York: Cambridge University Press.
- 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.
- Wright, J. P., Beaver, K. M., DeLisi, M., & Vaughn, M. G. (2008). Evidence of negligible parenting influences on self-control, delinquent peers, and delinquency in a sample of twins. *Justice Quarterly*, 25, 544-569.
- Wright, B. R. E., Caspi, A., Moffitt, T. E., & Silva, P. A. (1999). Low self-control, social bonds, and crime: Social causation, social selection, or both? *Criminology*, 37, 479-514.
- Yolton, K., Dietrich, K., Auinger, P., Lanphear, B. P., & Hornung, R. (2005). Exposure to environmental tobacco smoke and cognitive abilities among U.S. children and adolescents. *Environmental Health Perspectives*, 113, 98-103.

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