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# EVIDENCE OF GENETIC AND ENVIRONMENTAL EFFECTS ON THE DEVELOPMENT OF LOW SELF-CONTROL

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A body of research has revealed that low self-control is one of the strongest and most consistent predictors of antisocial behaviors. As a result, there is great interest in identifying the factors that cause variation in levels of self-control. Much of this work has centered on identifying the effects that social factors, such as parental socialization, have on self-control. More recently, however, there has been research revealing that levels of self-control are scripted by genetic factors as well as environmental factors. The current study examines whether a polymorphism (5HTTLPR) in the serotonin transporter gene and exposure to delinquent peers are associated with levels of self-control. Analysis of the National Longitudinal Study of Adolescent Health indicated that the 5HTTLPR polymorphism interacted with a measure of delinquent peer affiliation to predict variation in self-control during adolescence and adulthood. Implications for theories of crime causation are discussed.

**Keywords:** self-control; 5HTTLPR polymorphism; serotonin; delinquent peers; delinquency

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A long line of empirical research has been conducted examining the genetic and environmental underpinnings to virtually every measurable human phenotype (Plomin, DeFries, McClearn, & McGuffin, 2008). The results of these studies have consistently revealed that genetic factors account for approximately 50% of phenotypic variance, whereas nonshared environmental factors explain most of the remaining variance (Plomin, 1990; Reiss, Neiderhiser, Hetherington, & Plomin, 2000). These findings have laid to waste the antiquated “nature versus nurture” debate, and in general, most scholars in the academic community are now beginning to recognize that both genes and the environment matter to human development (Rutter, 2006). The one major exception is sociological criminologists, who have remained vehemently opposed to conceding that genes have any effect on the development of antisocial phenotypes (Walsh & Ellis, 2004; Wright, Beaver, DeLisi, Vaughn, Boisvert, et al., 2008). They continue to operate under the assumption that antisocial behaviors are not contaminated by genetic influences but instead are affected solely by environmental factors (see, for example, Gottfredson & Hirschi, 1990). This is

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somewhat surprising, given that there have been four meta-analyses (Ferguson, in press; Mason & Frick, 1994; Miles & Carey, 1997; Rhee & Waldman, 2002) and a number of literature reviews (e.g., Moffitt, 2005) revealing that genes explain approximately half of the variance in antisocial outcomes. But perhaps the most compelling evidence linking genetic factors to antisocial phenotypes comes from research that has examined associations between measured genes and measured behavioral outcomes. These studies have revealed that certain genetic polymorphisms—especially those that are involved in neurotransmission—are significantly associated with a range of antisocial outcomes, including aggression, violence, and drug use (Beaver, 2009).

It is important to point out that research showing that genes are influential to human behaviors does not downplay the significance of the environment but, rather, highlights the saliency of environmental factors. The most cutting-edge genetic research, for instance, has drawn attention to the complex interplay between genetic and environmental factors in creating human phenotypic variance (Rutter, 2006). What this necessarily means is that genetic and environmental factors are highly interlocked. Trying to study the effects of one at the expense of the other is completely misguided. We use this logic as a springboard for the current study and examine the development of low self-control—one of the strongest predictors of antisocial behavior (Pratt & Cullen, 2000)—from a biosocial perspective. More specifically, we examine the effects that delinquent peers have on the development of self-control, and we also examine the effects that a serotonin genetic polymorphism (5HTTLPR) has on the development of self-control. Perhaps most importantly, however, we examine whether delinquent peers and the serotonin gene interact to predict variation in levels of self-control.

### GOTTFREDSON AND HIRSCHI'S GENERAL THEORY OF CRIME

In 1990, Gottfredson and Hirschi published *A General Theory of Crime*, which advanced a theory designed to explain all types of crime, delinquency, and analogous behaviors. Instead of explaining crime solely through social factors, as had most sociological criminological theories, Gottfredson and Hirschi argued that crime and other antisocial behaviors were explained by an individual-level trait: low self-control. According to the theory, low self-control is a latent trait that is composed of six different elements. Specifically, persons with low levels of self-control are characterized as being self-centered, being impulsive, being risk seekers, preferring simple activities to complex ones, preferring physical tasks to mental ones, and having a bad temper. These six dimensions, according to the general theory, coalesce to form the unidimensional trait of low self-control.

Gottfredson and Hirschi also argued that for a crime to occur, a criminal opportunity must also be present. For the most part, however, they assumed that crime opportunities were a constant, which resulted in a highly parsimonious and easily testable theory. As a result, Gottfredson and Hirschi's theory has generated a wealth of empirical research testing the proposed link between levels of self-control and antisocial behaviors. The results of these studies have been overwhelmingly supportive of the nexus between low levels of self-control and delinquent or criminal outcomes. Against this backdrop, it was not surprising when a meta-analysis that included 21 studies examining the association between self-control and

delinquency reported that measures of low self-control were among the strongest and most consistent predictors of antisocial behaviors (Pratt & Cullen, 2000).

Because so much empirical research has found a strong association between levels of self-control and crime and delinquency, attention has recently shifted to studying the potential causes of variation in self-control. In advancing their theory, Gottfredson and Hirschi (1990) provided an explanation of the factors associated with the development of self-control. According to their theory, levels of self-control hinged on three interrelated parental management techniques. First, parents must monitor and supervise their children. Second, parents must recognize their children's misbehaviors. Third, parents must consistently correct their children's transgressions. Parents who engage in these three parenting practices will, according to the general theory, raise children with relatively high levels of self-control. In contrast, parents who do not engage in these three parenting practices will, on average, raise children with relatively low levels of self-control. Importantly, parents do not have a long time frame to mold their children's levels of self-control—according to Gottfredson and Hirschi, levels of self-control become relatively fixed by approximately the time children are ages 8 to 10 years old.

A growing body of research has tested Gottfredson and Hirschi's (1990) parental management thesis. The results of these studies have provided some evidence linking different parental socialization tactics to levels of self-control (for an overview, see Cullen, Unnever, Wright, & Beaver, 2008). However, there are at least three important qualifications to this line of research. First, the effect sizes for the parenting measures are relatively modest in magnitude and account for only a fraction of the explained variance. What this means is that although parenting may be related to levels of self-control, there are other, more dominant, factors that Gottfredson and Hirschi did not identify. Second, research testing the parental management thesis has failed to take into account child-driven effects. To elaborate, any association between parenting and self-control could be the result of the child's behaviors' causing negative parental reactions as opposed to negative parenting's affecting the child's level of self-control (Lytton, 1990). Third, and perhaps most importantly, research has generally failed to control for genetic factors when estimating the effect of parenting on levels of self-control (Wright & Beaver, 2005). This leaves open the possibility that the association between parenting and self-control is spurious and really the result of genetic confounding (J. R. Harris, 1995, 1998; Rowe, 1994). The limited research that has controlled for genetic factors has found that the effects of parental socialization dissipate from statistical significance after removing variance attributable to genetics (Wright & Beaver, 2005; Wright, Beaver, DeLisi, & Vaughn, 2008). Taken together, these three limitations cast serious doubt on the likelihood that parental socialization is an influential factor in sculpting levels of self-control.

If parental socialization is not related to levels of self-control, then what factor or factors are responsible for creating variation in low self-control? One possible explanation is that levels of self-control are driven by genetic factors. Although Gottfredson and Hirschi (1990) vehemently argued that genetic factors were unrelated to self-control, behavioral genetic research has shown that problems with impulse control, self-regulation, and attention—all of which overlap considerably with Gottfredson and Hirschi's conceptualization of self-control—are strongly influenced by genetic factors. Behavioral genetic research analyzing kinship pairs to estimate genetic and environmental effects on traits

closely aligned with low self-control have found that more than half of the variance in these phenotypes is attributable to genetic factors (Rietveld, Hudziak, Bartels, van Beijsterveldt, & Boomsma, 2004). These findings point to the very real likelihood that levels of self-control are also affected by genetics.

A limited number of studies have examined the genetic basis to Gottfredson and Hirschi's (1990) conceptualization of self-control. For instance, Beaver, Wright, DeLisi, and Vaughn (2008) analyzed twin pairs drawn from the National Longitudinal Study of Adolescent Health (Add Health) to estimate the extent to which genetic factors shaped levels of self-control. The results of the univariate variance decomposition models indicated that between 53% and 64% of the variance in self-control was explained by genetic factors. Another study, conducted by Beaver, Shutt, et al. (2009), also analyzed twin pairs from the Add Health study. Using a different statistical approach, they found self-control to be between 40% and 56% heritable. In another study, Beaver, DeLisi, Vaughn, Wright, and Boutwell (2008) estimated the heritability of low self-control in a sample of twins from the Early Childhood Longitudinal Survey, Kindergarten Class of 1998-1999 (ECLS-K). The results of their biometric model fitting techniques indicated that genetic factors accounted for 46% to 72% of the variance in self-control.

### **THE GENE-ENVIRONMENT BASIS TO LOW SELF-CONTROL**

The results of the studies presented earlier converge to show that genetic factors account for at least 50% of the variance in self-control. Although this is certainly important to know, these studies are able to reveal only the percentage of variance in self-control accounted for by genetic factors, and they are unable to identify the specific genes that may be involved in the development of self-control. To identify the precise genes that are related to levels of self-control, researchers cannot rely solely on samples of kinship pairs but instead have to analyze samples containing respondents who were genotyped for specific genetic polymorphisms. Recently, there has been an explosion of research analyzing such genetically sensitive samples, and the results of some of these studies have provided insight into the genes that are the most likely to be associated with self-control.

One gene that has been identified as a likely contributor to impulsivity, self-regulation problems, and low self-control is the serotonin transporter gene. This gene is centrally involved in the regulation of serotonin. Additionally, the serotonin transporter gene has a polymorphism in the promoter region (5HTTLPR), and alleles of this polymorphism have been shown to affect the function and the level of serotonin (Heils et al., 1996; Hu et al., 2006; Lesch et al., 1996; Reist, Mazzanti, Vu, Tran, & Goldman, 2001). Serotonin is a neurotransmitter that has inhibitory properties that work to dampen innate drives and to reduce impulsive behaviors. By logical extension, then, low serotonin levels are thought to increase aggression, violence, and other behavioral manifestations of low self-control (Raine, 1993). It stands to reason, therefore, that genes involved in modulating serotonin levels could also be linked to deficiencies with self-control and self-regulation. Although no research has directly tested the association between the 5HTTLPR polymorphism and levels of self-control, research has revealed that the 5HTTLPR polymorphism is associated with closely related phenotypes, including impulsivity and impulsive violence (Lee, Kim, & Hyun, 2003; Retz, Retz-Junginger, Supprian, Thome, & Rösler, 2004).

The 5HTTLPR-impulsivity nexus is far from universal, however, as some studies have failed to find any association (Xu et al., 2008). Whether the 5HTTLPR polymorphism relates to levels of self-control thus remains unclear.

One possible explanation for the inconsistent effects of the 5HTTLPR polymorphism is that most of the extant studies have failed to examine the possibility that certain environments may condition the association between 5HTTLPR and problems with self-control. This is a serious oversight because there is now an abundance of research revealing that the effects that genes have on phenotypes depend on the presence of certain environments—a phenomenon known as a gene–environment interaction (Beaver, 2009; Moffitt, 2005; Rutter, 2006; Walsh, 2002). If an environment does condition the effect of a genetic polymorphism, and if a measure of that environment is not included in the statistical models, then the end result is that the effect of the gene may not be detectable. As a result, more and more research is beginning to examine the interactions between measured genes and measured environments in the prediction of antisocial phenotypes.

A major obstacle in studying gene–environment interactions is identifying the environments that may moderate the effects of the genetic polymorphism. Prior research has revealed that environments found in the family—such as parental maltreatment—are able to condition the effects of certain genes (Caspi et al., 2002), but other environmental stimuli are also likely to interact with certain genes. One potentially important environment is exposure to delinquent peers. Affiliating with antisocial peers is a strong and robust predictor of delinquent behavior (Warr, 2002), and it has also recently been found to be associated with levels of self-control (Burt, Simons, & Simons, 2006). At the same time, exposure to delinquent friends has been shown to interact with certain genetic factors to predict variation in antisocial phenotypes (Harden, Hill, Turkheimer, & Emery, 2008). Against this backdrop, we hypothesize that the effect of 5HTTLPR on low self-control will be conditioned by the respondent's exposure to delinquent peers.

## METHOD

### PARTICIPANTS

Data for this study come from the National Longitudinal Study of Adolescent Health (Add Health), which is a three-wave nationally representative sample of American youths (Udry, 2003). The initial wave of data—known as the Wave 1 in-school data—was collected during 1994-1995 when more than 90,000 students were administered a self-report survey at school. A wide range of questions was asked, including items pertaining to school experiences, family life, and peer relationships. A subsample of respondents was then reinterviewed, along with their primary caregivers (usually their mother), at their home. Detailed questions were asked about the adolescent's involvement in delinquency, their sexual activities, and their use of tobacco, drugs, and alcohol. A total of 20,745 adolescents and 17,700 of their primary caregivers participated in the Wave 1 in-home component of the Add Health study (K. M. Harris et al., 2003).

Approximately one-and-a-half years later, the second wave of data was collected. Because most of the respondents were still adolescents, the questions asked during the Wave 1 in-home interviews were still age appropriate and thus were retained on the Wave

2 surveys. For example, respondents were asked about their delinquent behaviors, their peer relationships, and the functioning of their family. A total of 14,738 adolescents completed Wave 2 survey instruments. Then, between 2001 and 2002, the third round of interviews was completed. Most of the respondents were young adults at Wave 3, and thus the surveys were altered to include more age-appropriate questions. For instance, questions were asked about the respondent's marital status, their childbearing history, and their contact with the criminal justice system. Overall, 15,197 respondents were successfully interviewed at Wave 3 (K. M. Harris et al., 2003).

The Add Health data contain an array of variables measuring various environmental factors. However, unlike most other large social science samples, a subsample of respondents was asked to submit samples of their buccal cells at Wave 3 to be genotyped. Respondents who had a sibling who was also participating in the Add Health study were eligible to be included in the DNA subsample. A total of 2,574 respondents agreed to participate and were genotyped for a number of genetic polymorphisms involved in neurotransmission. Taken together, the Add Health data are one of the richest samples, as the data span 7 years of human development and contain measures of the environment, genes, behaviors, and personality traits.

## MEASURES

*Low-self-control scales.* When detailing their theory, Gottfredson and Hirschi (1990) provided a very specific description of self-control. Specifically, they argued that people with low levels of self-control are characterized as being self-centered, as preferring physical activities to mental ones, as being impulsive, as being risk seekers, as preferring simple tasks, and as having a temper. Although a number of different ways of measuring levels of self-control have been used, the most widely employed method is Grasmick, Tittle, Bursik, and Arneklev's (1993) scale. The Add Health data did not contain the Grasmick et al. scale, and thus we were forced to use a different set of items to measure individual variation in levels of self-control. It should be pointed out, however, that there are at least two reasons that using a scale other than the Grasmick et al. scale will not bias the results. First, research has revealed that the Grasmick et al. scale may not be as reliable and valid as is typically assumed (DeLisi, Hochstetler, & Murphy, 2003; Marcus, 2004). Second, Pratt and Cullen's (2000) meta-analysis revealed that the association between levels of self-control and crime and delinquency was statistically significant regardless of whether the Grasmick et al. scale was used.

Three low-self-control scales were available in the data: one measured at Wave 1, one measured at Wave 2, and one measured at Wave 3. During Wave 1 interviews, respondents were asked questions pertaining to their ability to exercise self-control. The primary caregiver was also asked a number of questions about the adolescent's self-control. For instance, questions were asked about the respondent's temper, decision-making skills, ability to pay attention, and whether he or she has difficulty keeping his or her mind focused. A total of 23 items that tapped self-control were asked. Responses to each of these items were summed together to create the Wave 1 Low Self-Control scale, where higher scores reflect lower levels of self-control ( $\alpha = .76$ ). This scale is identical to the one used previously by Add Health researchers (Beaver, DeLisi, Mears, & Stewart, in press). See the appendix for

a complete listing of all the individual items that are included in the scales used in the current study.

Questions measuring self-control were also asked to respondents (but not their primary caregiver) during Wave 2 interviews. Many of the questions were identical to the ones asked at Wave 1, but some additional items were also included. Adolescents, for example, were asked whether difficult problems make them very upset, whether they like to take risks, and whether they live life without much thought for the future. Responses to the 20 items were then summed together to create the Wave 2 Low Self-Control scale ( $\alpha = .72$ ). Once again, higher values on this scale indicate lower levels of self-control.

During Wave 3 interviews, respondents were also asked 20 items designed to capture individual variation in levels of self-control. Specifically, respondents were asked whether they like to take risks, whether they often do things on the basis of how they feel at the moment, whether they sometimes get so excited that they lose control, and whether they often follow their own instincts without thinking through all of the details. These items were then summed together to create the Wave 3 Low Self-Control scale ( $\alpha = .80$ ), where higher values reflect lower levels of self-control.

*5HTTLPR.* The serotonin transporter gene is located on Chromosome 17 and has a 43 base pair insertion–deletion polymorphism in the 5' regulatory section of the gene (5HTTLPR; Heils et al., 1996). This polymorphism contains two types of alleles: low expressing alleles and high expressing alleles. Compared with the high-expressing alleles, the low-expressing alleles have been found to suppress transcription of the serotonin transporter protein (Hu et al., 2006; Lesch et al., 1996). As a result, the low-expressing alleles could lead to lower levels of serotonin in the brain. Because serotonin has inhibitory properties that work to dampen innate drives and to control impulses, most researchers have hypothesized that the low-expressing alleles confer an increased risk of developing antisocial phenotypes, such as low levels of self-control. To take this possibility into account, 5HTTLPR was coded codominantly, where the value on the 5HTTLPR variable indicates the total number of low-expressing alleles that each respondent possesses.

*Delinquent peers.* To examine whether exposure to antisocial friends moderates the effect of 5HTTLPR on low self-control, we created a three-item Delinquent Peers scale. During Wave 1 interviews, adolescents were asked to indicate how many of their three best friends smoke at least one cigarette each day, smoke marijuana more than once per month, and drink alcohol at least one time a month. The response set for these questions was 0 = zero friends, 1 = one friend, 2 = two friends, and 3 = three friends. All of the responses were summed together to create the Delinquent Peers scale, where higher values indicate greater contact with delinquent peers ( $\alpha = .76$ ). This scale is identical to the one that has been used by previous researchers analyzing the Add Health data (Beaver, 2008; Bellair, Roscigno, & McNulty, 2003).

*Control variables.* To help avoid model misspecification, three control variables were included in the analyses: age, gender, and race. Age was a continuous variable (measured in years), and gender (0 = female, 1 = male) and race (0 = White, 1 = African American) were dichotomous dummy variables.

**TABLE 1: Ordinary Least Squares Regression Models Predicting Low Self-Control at Wave 1**

	<i>Model 1</i>				<i>Model 2</i>			
	<i>b</i>	<i>SE</i>	<i>Beta</i>	<i>p</i>	<i>b</i>	<i>SE</i>	<i>Beta</i>	<i>p</i>
Polymorphism 5HTTLPR	0.20	.27	.02	.457	-0.31	.37	-.03	.396
Socialization variable Delinquent peers	0.99	.08	.31	<.001	0.83	.11	.26	<.001
Gene × Environment interaction 5HTTLPR × Delinquent Peers					0.21	.10	.08	<.050
Control variables								
Age	-0.40	.12	-.08	<.001	-0.40	.12	-.08	<.001
Gender	-0.88	.38	-.05	<.050	-0.83	.38	-.05	<.050
Race	-1.60	.47	-.08	<.001	-1.64	.47	-.08	<.001

Note. Huber-White standard errors.

## ANALYSIS

First, for each low-self-control scale, two models were estimated: a main effects model and an interactive model. The main effects model included 5HTTLPR, delinquent peers, age, gender, and race as independent variables and estimated the independent main effects that each variable has on levels of self-control. The second model included all of the independent variables from the main effects model, but it also contained an interaction term: 5HTTLPR × Delinquent Peers. This model estimated whether 5HTTLPR and delinquent peers interact to predict a significant amount of variation in the low-self-control scales. Ordinary least squares regression was employed because all of the low self-control scales approximated normality. Collinearity diagnostics were calculated and there was no evidence to suggest that collinearity was evident in any of the models.

It is important to note that some of the cases of the DNA subsample of the Add Health were not independent of each other (i.e., more than one adolescent per household was eligible to take part in the study). We addressed this issue in two ways. First, one MZ **IAQ 11** twin from each MZ-twin pair was removed from the analysis (Haberstick et al., 2005). Second, all of the statistical models were estimated using Huber-White standard errors. Using this criteria and after removing data with missing cases, we were left with a sample size that ranged between  $N = 1,631$  and  $N = 1,838$ .

## RESULTS

As Table 1 reveals, 5HTTLPR failed to maintain a direct association with low self-control, but the Delinquent Peers scale exerted a positive and statistically significant effect on levels of self-control. More specifically, respondents who self-reported having more delinquent peers had, on average, relatively low levels of self-control. Model 2 depicts the findings for the interactive model. As can be seen, the interaction between 5HTTLPR and delinquent peers emerged as a statistically significant predictor of low self-control. This statistically significant effect indicates that 5HTTLPR and delinquent peers work synergistically to predict variation in levels of self-control.

**TABLE 2: Ordinary Least Squares Regression Models Predicting Low Self-Control at Wave 2**

	<i>Model 1</i>				<i>Model 2</i>			
	<i>b</i>	<i>SE</i>	<i>Beta</i>	<i>p</i>	<i>b</i>	<i>SE</i>	<i>Beta</i>	<i>p</i>
Polymorphism								
5HTTLPR	−0.03	.25	−.00	.912	−0.51	.34	−.05	.133
Socialization variable								
Delinquent peers	0.82	.07	.29	<.001	0.67	.10	.24	<.001
Gene × Environment interaction								
5HTTLPR × Delinquent Peers					0.20	.09	.09	<.050
Control variables								
Age	−0.66	.11	−.15	<.001	−0.67	.11	−.15	<.001
Gender	0.94	.33	.07	<.010	0.99	.33	.07	<.010
Race	−1.02	.43	−.06	<.050	−1.06	.43	−.06	<.050

Note. Huber-White standard errors.

**TABLE 3: Ordinary Least Squares Regression Models Predicting Low Self-Control at Wave 3**

	<i>Model 1</i>				<i>Model 2</i>			
	<i>b</i>	<i>SE</i>	<i>Beta</i>	<i>p</i>	<i>b</i>	<i>SE</i>	<i>Beta</i>	<i>p</i>
Polymorphism								
5HTTLPR	0.32	.31	.02	.303	−0.49	.43	−.03	.253
Socialization variable								
Delinquent peers	0.64	.09	.16	<.001	0.39	.13	.10	<.010
Gene × Environment interaction								
5HTTLPR × Delinquent Peers				.32	0.12	.10	<.010	
Control variables								
Age	−1.08	.14	−.18	<.001	−1.08	.14	−.18	<.001
Gender	4.62	.46	.23	<.001	4.70	.46	.23	<.001
Race	−0.88	.58	−.03	.132	−0.93	.58	−.04	.112

Note. Huber-White standard errors.

The results thus far provide some evidence that GxEs **TAQ 21** may be important in the etiology of low self-control. However, the models estimated in Table 1 are largely cross-sectional, where both low self-control and delinquent peers were both measured at Wave 1. To provide a more conservative estimate of the interrelationships among 5HTTLPR, delinquent peers, and low self-control, Table 2 used the Wave 2 Low Self-Control scale as the dependent variable. In the main effects model, 5HTTLPR is unrelated to levels of self-control, whereas delinquent peers maintains a positive and statistically significant association with the Wave 2 Low Self-Control scale. For the interactive model, however, 5HTTLPR and delinquent peers interacted to predict a significant amount of variation in the Wave 2 Low Self-Control scale.

The last models to be estimated use the Wave 3 Low Self-Control scale as the dependent variable. Recall that Wave 3 data was collected approximately 7 years after Wave 1. These models, therefore, provide a very rigorous test of the associations among 5HTTLPR, delinquent peers, and low self-control. Table 3 portrays the results of these analyses, which show a pattern of findings that is strikingly similar to those presented in the first two tables. For instance, 5HTTLPR did not maintain a statistically significant association with low self-control, whereas the Delinquent Peers scale was significantly related to levels of self-control.

Perhaps most important was that the interaction between 5HTTLPR and delinquent peers once again emerged as a statistically significant predictor of levels of self-control.

## DISCUSSION

In recent years, there has been growing interest among criminologists in identifying and studying the potential causes of self-control. Research bearing on this topic carried out by sociological criminologists has tended to center almost exclusively on social factors, especially parental socialization techniques, while simultaneously ignoring the possibility that genetic factors matter (Cullen et al., 2008). This is a serious oversight because research from disciplines other than criminology has revealed that self-control, self-regulation, and impulse control are heavily influenced by genetic factors (Wright & Beaver, 2005). Still, the exact genetic polymorphisms that are associated with self-control and self-regulation have remained relatively elusive.

The purpose of the current study was to examine whether the 5HTTLPR polymorphism explained any of the variance in self-control during adolescence and early adulthood. On the basis of prior research (Caspi et al., 2003), we hypothesized that the 5HTTLPR polymorphism would not have a statistically significant additive effect on levels of self-control. The reasoning behind this hypothesis was that behavioral genetic research has demonstrated that genes, in isolation, often do not have strong effects on phenotypes (Walsh, 2002). Instead, genes tend to have their strongest associations with phenotypes when they are paired with certain environments (Caspi et al., 2002). Employing this logic, we hypothesized that the 5HTTLPR polymorphism would have an effect on levels of self-control only when it was paired with a criminogenic environment—delinquent peers.

The results of our multivariate regression models revealed substantial support for our hypotheses. Across three measures of self-control, which spanned approximately 7 years of human development, the 5HTTLPR polymorphism did not have a main effect on low self-control. In the nonadditive models that tested for gene–environment interactions between 5HTTLPR and delinquent peers, a very different set of results surfaced. These models revealed that 5HTTLPR interacted with delinquent peers to predict a significant amount of variation in measures of self-control assessed during adolescence and early adulthood. Recall that the measure of delinquent peers was drawn from data collected during the Wave 1 interviews, whereas the three low-self-control scales were constructed from data collected at Wave 1, data collected at Wave 2, and data collected at Wave 3. What these findings seem to hint at, then, is that contact with delinquent peers in adolescence can condition the effects that genes have on phenotypes more than 6 years into the future. To our knowledge, this is the first time that a gene–environment interaction has been detected on a measure of self-control.

The findings highlight the importance of studying both genetic and environmental influences on antisocial phenotypes. Had we examined only the association between the 5HTTLPR polymorphism and low self-control, it would have appeared as though the two were unrelated. Only when we incorporated a measure of delinquent peers were we able to detect the effect that 5HTTLPR had on levels of self-control. These findings should act as a cautionary tale for researchers who examine only the effects of genes or only the effects of the environment on human phenotypes. Unfortunately, most criminologists do exactly this—they ignore and downplay the possibility that genes could have an effect on

antisocial outcomes and instead examine only environmental effects on crime and delinquency. As our research reveals, however, this approach is completely misguided. There is now a large and rich body of empirical research demonstrating that all antisocial phenotypes are influenced by genetic factors, and some, such as levels of self-control and psychopathy, have strong genetic origins (Viding, Blair, Moffitt, & Plomin, 2005; Wright & Beaver, 2005). Unless sociological criminologists begin to weave these genetic findings into their theories and research, they run the risk of marginalizing criminology from the broader academic community.

Of course, we are not advocating the abandonment of all criminological theories; rather, we are arguing that sociological criminological theories need to be refined and modified to include concepts and research findings from behavioral genetic research (Walsh, 2002). For example, genetic research could easily be integrated into Gottfredson and Hirschi's (1990) theory on the development of self-control. Instead of arguing that only social factors matter in the creation of self-control, their theory could include research from brain science studies, psychology studies, and behavioral genetic studies to provide a very detailed account of how genes, the brain, and the environment work synergistically to produce levels of self-control (DeLisi, 2009). Not only would this type of theoretical modification link Gottfredson and Hirschi's theory to other disciplines, such as psychiatry and neuroscience, it would also make for a much stronger, much more believable, and much more accurate explanation on the etiology of low self-control (Wright & Beaver, 2005). Other mainstream criminological theories could also be altered relatively easily to include genetic factors without seriously distorting the central tenets of the theory (Walsh, 2000).

As with any research, however, there are a number of limitations with our study that need to be addressed by future researchers. First, the Add Health data did not include standardized assessments of low self-control. We were thus forced to use items that had not been created and designed with the intention of measuring variation in self-control. This measurement strategy leaves open the possibility that studies employing other measures of self-control may produce different results. We should note, however, that we calculated a series of psychometric tests (e.g., principal components factor analysis), and the results indicated that the scales were a relatively reliable way to measure self-control. Second, although the Add Health data are nationally representative of American youths, the DNA sample analyzed in the current study is not necessarily nationally representative. Whether the findings reported here would generalize to the larger population or to other samples remains an open empirical question awaiting future investigators. Last, the measure of delinquent peers was based on three items tapping exposure to drug-using peers. We would have preferred to use a measure of delinquent peers that assessed a wider array of peer delinquency. Even so, the measure of delinquent peers continued to have independent and interactive effects on levels of self-control 7 years into the future. In light of these limitations, the results presented here need to be viewed with caution until replication studies are able to substantiate the findings.

We close by pointing out that opponents to genetic explanations of human behavior often argue that genetic research will produce policies that are inhumane and dangerous. Nowhere is this more evident than in criminological research, where biosocial antagonists insist that genetic research will lead to a new eugenics movement or highly punitive crime control policies. This type of argument is grounded in the vacuous argument that because DNA is immutable, so too are genetic effects. This simply is not the case. Genetic effects are modifiable by altering the environment. In the current research, the effect of the 5HTTLPR polymorphism on low self-control changed as contact with delinquent peers changed. As a

result, knowing which environments condition the effects of certain genes can be extremely important in the development of prevention and intervention programs. However, the effectiveness of such programs hinges on the ability to unpack the complex ways in which genes and the environment interlock to produce human phenotypic variance.

## APPENDIX

### Description and Coding of Measures and Scales

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#### 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.
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 our 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.

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*(continued)*

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**APPENDIX (continued)**


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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. **[AQ 3]**
16. You felt happy. **[AQ 4]**
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.
20. You live life without much thought for the future.

**Wave 3 Low Self-Control Scale**

Scale was created by summing responses to the following items:

1. You never get sad or you felt sad. **[AQ 5]**
2. You usually go out of your way to avoid having to deal with problems in your life.
3. When making decisions, you usually go with your "gut feeling" without thinking too much about the consequences of each alternative.
4. You like yourself just the way you are.
5. You feel like you are doing everything just about right.
6. Do you have trouble keeping your mind focused?
7. You enjoyed life. **[AQ 6]**
8. You like to take risks.
9. You live life without much thought for the future.
10. How satisfied are you with your life as a whole?
11. You tend not to follow the crowd, but instead behave in a way that suits your mood at the time.
12. I often try new things just for fun or thrills, even if most people think they are a waste of time. **[AQ 7]**
13. When nothing new is happening, I usually start looking for something exciting.
14. I can usually get people to believe me, even when what I am saying isn't quite true.
15. I often do things based on how I feel at the moment.
16. I sometimes get so excited that I lose control of myself.
17. I like it when people can do whatever they want, without strict rules and regulations.
18. I often follow my instincts, without thinking through all the details.
19. I can do a good job of "stretching the truth" when I am talking to people.
20. I change my interest a lot, because my attention often shifts to something else.

**Delinquent Peers Scale**

Scale was created by summing responses to the following items. Respondents were asked how many of their three closest friends

1. Smoke at least one cigarette each day
  2. Smoke marijuana more than once a month
  3. Drink alcohol at least once a month
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