

# The Effects of Genetics, the Environment, and Low Self-Control on Perceived Maternal and Paternal Socialization: Results from a Longitudinal Sample of Twins

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Published online: 9 July 2010  
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**Abstract** The association between parental socialization and antisocial behavior is central to much criminological theory and research. For the most part, criminologists view parental socialization as reflecting a purely social process, one that is not influenced by genetic factors. A growing body of behavioral genetic research, however, has cast doubt on this claim by revealing that environments are partially shaped by genetic factors. The current study used these findings as a springboard to examine the genetic and environmental underpinnings to various measures of perceived paternal and maternal parenting. Analysis of twin pairs drawn from the National Longitudinal Study of Adolescent Health revealed that between 16 and 31% of the variance in perceptions of maternal attachment, maternal involvement, maternal disengagement, and maternal negativity was the result of genetic factors. Additionally, between 46 and 63% of the variance in perceptions of paternal attachment, paternal involvement, and paternal negativity was accounted for by genetic factors. The implications that these results have for criminologists are explored.

**Keywords** Add Health · Genetics · Low self-control · Parenting

## Introduction

A large body of empirical research has examined the effects that parental socialization has on childhood and adolescent development. For the most part, this line of research has found consistent links between various measures of parenting and an assortment of behaviors, personality traits, and later-life outcomes. For example, measures of parental negativity have been found to predict a range of maladies, including depression (Shah and Waller 2000), school failure (Astone and McLanahan 1991), teenage pregnancy (Miller et al. 2001), substance use (Simons et al. 2004), and delinquency in general (Loeber and Stouthamer-Loeber 1986; Unnever et al. 2006). The reason for the nexus between parental

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socialization and the development of antisocial behaviors seems obvious and straightforward: through parental socialization tactics, parents mold and shape their children's behavioral patterns and personalities (Pinker 2002). From this perspective, parenting is seen as a purely social variable that tends to have unidirectional effects (Neiderhiser et al. 2004).

This explanation, while certainly plausible, is only one of a number of different perspectives that could be employed to explicate the association between parental socialization and antisocial behavior (Harris 1995, 1998; Rowe 1994). An equally possible, yet very different, explanation has been proposed by some behavioral geneticists. According to them, variation in parental socialization techniques, such as parental supervision and parental attachment, may be explained, in part, by genetic factors (Harris 1995, 1998; Rowe 1994; Wright and Beaver 2005). A growing line of research testing this proposition has revealed that variation in a wide range of environments, including family environments, is partially the result of genetic factors (Kendler and Baker 2007). The current study adds to this body of research and explores the extent to which variance in perceptions of maternal attachment, maternal involvement, maternal disengagement, maternal negativity, paternal attachment, paternal involvement, paternal negativity, and parental supervision is due to genetic factors and the extent to which environmental factors play a role. To do so, a sample of twin pairs drawn from the National Longitudinal Study of Adolescent Health is analyzed.

### **The Importance of Parental Socialization to Criminology**

The overwhelming majority of all criminological research attempts to identify the various factors that are causes or correlates of crime, delinquency, or some other form of antisocial behavior. In these studies, the dependent variable is typically some measure of delinquent or criminal involvement, while the independent variable consists of putative criminogenic risk factors, such as exposure to delinquent peers or levels of self-control. The findings flowing from this body of research have produced a significant knowledge base revealing a host of risk factors that are associated with antisocial behaviors. As a result, criminologists have begun to examine the causes of some well-established criminogenic risk factors.

The question, however, is which criminogenic risk factors should be examined. While not an exclusive set of criteria, there are at least three factors that should inform this decision. First, empirical research should consistently tie the criminogenic risk factor to antisocial behaviors. Second, the criminogenic risk factor should be entrenched in criminological theories. Findings generated from research examining the causes of criminogenic risk factors allow for greater theoretical specificity regarding the complex arrangement of factors that are etiologically related to antisocial behavior. Third, the criminogenic risk factor should be the focus of some prevention and intervention programs. Knowing what causes variation in criminogenic risk factors is able to assist in the development of prevention and intervention programs. Instead of intervention programs focusing only on the direct causes or correlates to crime and delinquency, programs are able to target for change more distal risk factors. In other words, there are more opportunities to intervene and break the potential developmental pathways that ultimately culminate in antisocial behavior. One criminogenic risk factor that meets these criteria is parental socialization. To illustrate, measures of parental socialization have been found to correlate with measures of antisocial behaviors (Cullen et al. 2008; Simons et al. 2004), an overwhelming number of criminological theories identify parenting as a causal agent in the etiology of antisocial

behaviors (Simons et al. 2004), and a host of intervention and prevention programs have targeted parental socialization (Piquero et al. 2009).

Given the central importance of parental socialization to criminological theory and research and to prevention/intervention programs, it is noteworthy that very few criminological studies have examined what causes variation in parental socialization. Loeber et al. (2000:363) recognized this gap in the literature and responded to criticisms that it was outside the purview of research related to the development of antisocial behaviors. According to them:

The present study, unlike many other studies on child deviance, selected family interaction patterns as dependent variables. We justified this approach in two ways. First, there is abundant evidence that various negative family interaction patterns are linked to different forms of deviant child behavior, including delinquent offending. Second, the explanation of child deviance can be strengthened by an explanation of the best predictors of such deviance, including parent–child interactions. Thus, parent–child interactions themselves can be studied suitably as outcomes.

The current study follows the lead of Loeber et al. (2000) and examines the various contributors to *perceptions* of parental socialization from a behavioral genetic perspective. While perceptions of parental socialization may not be viewed as reliable and valid as objective measures of parental socialization, there are at least two reasons why focusing on perceptions is warranted. First, an overwhelming amount of criminological research employs measures of parenting that are based on perceptions. Understanding what accounts for variation in perceived parenting is critical to both criminological theory and research. Second, perceptions of parenting likely mediate any association between actual parenting and adolescent outcomes (Rowe 1983). Against this backdrop, focusing on perceptions of parenting should not be viewed as a limitation, but rather a key contribution of the current study.

## Genetic Influences on the Environment

Behavioral geneticists are interested in decomposing the variance in phenotypes (i.e., measurable characteristics) into three different components: a heritability component, a shared environmental component, and a nonshared environmental component. The heritability component captures the extent to which genetic variance explains individual differences in phenotypes. Shared environmental factors refer to environmental factors that are the same between siblings. Nonshared environmental effects, in contrast, are environmental factors that are different between siblings. Behavioral geneticists estimate the relative effects of genetic, shared environmental, and nonshared environmental factors on a phenotype by analyzing samples of kinship pairs, including twin pairs. By employing samples of twin pairs, behavioral geneticists are able to compare the phenotypic similarity of monozygotic (MZ) twin pairs to the phenotypic similarity of dizygotic (DZ) twin pairs. MZ twins share 100% of their genetic material, while DZ twins, on average, share 50% of their genetic material. As a result, if the assumptions of twin-based research are met, the only reason that MZ twins should be phenotypically more similar than DZ twins is because they share twice as much genetic material. Using this logic, it is possible to estimate the proportion of phenotypic variance that is explained by genetic factors, shared environmental factors, and nonshared environmental factors (Plomin et al. 2008).

Behavioral geneticists have used twin-based methodologies to decompose the genetic and environmental underpinnings to phenotypes. The precise amount of variance that is explained by genes and the environment in these measures waxes and wanes across studies depending on sample characteristics and the phenotype being studied. Nonetheless, there are some commonalities that tend to cut across all studies, such as genetic effects being detected on virtually all human phenotypes that have ever been studied, family environments having relatively small effects on phenotypes, and nonshared environments having relatively strong effects on phenotypes (Turkheimer 2000). These findings are known as the “laws of behavioral genetics” and are generally accepted among the vast majority of behavioral geneticists.

With the available evidence indicating significant genetic effects on human phenotypes, behavioral geneticists began to theorize that the effects genes have may also extend to environments (Plomin and Bergeman 1991; Plomin et al. 1977; Scarr 1992; Scarr and McCartney 1983). Dubbed the “nature of nurture,” this line of research has examined the heritability of environmental measures. To do so, the same twin-based methodologies that were employed to decompose the variance in human phenotypes are used. However, instead of using a behavioral phenotype as the outcome measure, an environmental measure is entered into the analysis as the dependent variable. The findings culled from these studies provide direct evidence of the extent to which genetic factors are implicated in explaining variance in measures of the family environment (Plomin et al. 1994).

There is a relatively rich pool of behavioral genetic research that has examined genetic influences on measures of environments. Kendler and Baker (2007) conducted a review of this research and the results of their study were quite revealing (see also Plomin and Bergeman 1991). They identified 19 studies that had estimated genetic influences on parenting behaviors. Their review of the literature indicated that genetic influences accounted for between 12 and 35% of the variance in parental socialization. Other measures of the family environment, such as conflict and organization, were also found to be heritable, with genetic factors explaining between 18 and 30% of the variance. Based on their review of all the studies Kendler and Baker (2007:615) concluded that “genetic influences on measures of the environment are pervasive in extent and modest to moderate in magnitude.” One of the looming questions—especially for criminologists—is how environments could be influenced by genetic factors. To address this question, it is essential to turn attention to the logic of gene-environment correlations.

## Gene-Environment Correlations

Gene-environment correlations provide the theoretical scaffolding needed to understand the underlying mechanisms that might lead from genotypic variance to environmental variance (Jaffee and Price 2007; Walsh 2002). There are two main types of gene-environment correlations, each of which captures a different way in which genes are intertwined with environments, that have direct application to the study of genetic influences on parenting (Plomin et al. 1977; Scarr and McCartney 1983). The first type of gene-environment correlation is known as a passive gene-environment correlation. Passive gene-environment correlations draw attention to the fact that parents pass along two entities to their children: genes and a rearing environment. Since both genes and the rearing environment are traced to the same source (i.e., parents), they are likely to be correlated (Rutter 2006).

For example, highly aggressive and violent parents are likely to pass along to their children the genetic predisposition to be aggressive and violent. Likewise, aggressive and violent parents are likely to rear their children in environments that are typified by abuse, maltreatment, and negativity (Farrington and Welsh 2007). The end result is that the child's genetic predispositions (e.g., the genetic predisposition to be aggressive and violent) are correlated with the environment (e.g., an abusive and negative rearing environment) into which they are born.

The second gene-environment correlation is known as an evocative gene-environment correlation. According to the logic of evocative gene-environment correlations, genes are involved in eliciting responses from the environment and these environmental reactions, in turn, are correlated with genotype. For instance, children who are characterized as being behaviorally disordered (i.e., a genetically influenced phenotype) are more likely to be disciplined by their parents than are children who are relatively well-behaved. In this case, the genetic predisposition to be behaviorally disordered is evoking negative responses from the environment (i.e., parental discipline). It is important to note that the genetic predisposition (e.g., the predisposition to be behaviorally disordered) and the environmental response (e.g., parental discipline) are highly correlated.

Evocative gene-environment correlations are in line with "child effects" explanations (Lytton 1990) except that the unit of analysis is the gene instead of the behavior. According to advocates of child effects explanations, the association between parental socialization is not necessarily the result of the parent socializing the child to act in a certain way, but rather this association is driven by the behavior of the child. There is empirical support for child effects in the genesis of parental socialization (Beaver and Wright 2007; Harris 1998; Hu et al. 2006; Lytton 1990), and there is some evidence indicating that child effects may be the result of evocative gene-environment correlations (Ge et al. 1996; O'Connor et al. 1998).

## The Current Study

The current study adds to the literature examining genetic influences on environmental measures by decomposing the variance in measures of perceived parental socialization into genetic, shared environmental, and nonshared environmental components. In this study, parental socialization is operationalized with individual dimensions of perceived parenting as well as with global parenting scales. The parenting techniques of the father and of the mother are examined separately to determine whether genetic factors differentially affect perceptions of paternal and maternal parental socialization. To address these issues, a sample of twin pairs drawn from the National Longitudinal Study of Adolescent Health was analyzed by using DeFries–Fulker analysis.

## Methods

### Data

Data for the current study come from the National Longitudinal Study of Adolescent Health (Add Health), which is a longitudinal study of a nationally representative sample of American youths enrolled in seventh through twelfth grade (Udry 2003). The first wave of data was collected in 1994–1995 when students enrolled in 132 middle and high schools

were asked to complete a self-report survey at school (i.e., the wave 1 in-school survey). More than 90,000 adolescents completed the survey that included questions asking about their families, their peers, and their behaviors. In order to gain more detailed information about sensitive topics, a subsample of youths, along with their primary caregivers, was reinterviewed in their homes (i.e., the wave 1 in-home survey). Questions were asked about the adolescents' social relationships, their involvement in risk behaviors, and their use of drugs and alcohol. In total, 20,745 youths and 17,700 of their primary caregivers participated in the wave 1 in-home component of the Add Health study (Harris et al. 2003).

The second wave of data was collected during 1996 when 14,738 of the original wave 1 respondents were reinterviewed. Since relatively little time lapsed between waves, most of the participants were still adolescents. Thus the questions asked at the previous wave were still applicable and, as a result, the survey instruments remained very similar. For instance, adolescents were asked about their family and peer relationships, their school experiences, and their involvement in delinquency. Approximately 5–6 years later, the third round of interviews was conducted. During wave 3 interviews, most of the respondents were young adults and thus the questions asked at the previous two waves were no longer age-appropriate. Consequently, the survey instruments were redesigned to include items that were germane to adults. Questions were asked, for instance, about child-rearing tactics, employment status, and lifetime contact with the criminal justice system. In total, 15,197 respondents were successfully reinterviewed at wave 3 (Harris et al. 2003).

One of the unique features of the Add Health study is that sibling and twin pairs were oversampled. During wave 1 interviews, youths were asked whether they currently lived with a co-twin, a half-sibling, a stepsibling, or a cousin. If they indicated that they did, and if their sibling was 11–20 years old, then they were also added to the sample. A probability sample of full siblings is also nested within the data (Jacobson and Rowe 1999). Importantly, analyses have been conducted to determine whether the sibling pairs differ from the nationally representative sample on a range of demographic and behavioral measures. The results of these studies did not reveal any significant differences between the sibling pairs sample and the larger sample of youths (Beaver 2008; Jacobson and Rowe 1998). For reasons to be discussed momentarily, the final analytical sample is confined to  $N = 537$  twin pairs ( $n = 289$  MZ twin pairs and  $n = 248$  same-sex DZ twin pairs).

## Measures

### Parenting Scales

#### *Maternal Attachment*

Maternal attachment is one of the most widely examined dimensions of parenting in the criminological literature (Hirschi 1969; Sampson and Laub 1993). As a result, a maternal attachment scale was included in the analysis. During wave 2 interviews, adolescents were asked to indicate how closely they feel to their mother and how much they think their mother cares about them. Responses to these questions were coded on a 5-point Likert scale. The two items were summed together to create the maternal attachment scale, where higher values indicated greater levels of maternal attachment ( $\alpha = .53$ ). Previous researchers analyzing the Add Health data have used this same maternal attachment scale (Beaver 2008; Haynie 2001; Schreck et al. 2004).

### *Maternal Involvement*

Theoretical and empirical research has revealed that maternal-child involvement is strongly related to a range of antisocial behaviors (Loeber and Stouthamer-Loeber 1986). The Add Health data include ten items that measured how involved the mother is with their child. During wave 2 interviews, adolescents were presented with a list of ten activities and were asked which, if any, they had done with their mother in the past 4 weeks. Respondents, for instance, were asked whether they and their mother had gone to a movie together, played a sport together, worked on a project for school together, and gone shopping together. These items were coded dichotomously, where 0 = no and 1 = yes. Responses to the ten questions were summed together to form the maternal involvement index ( $\alpha = .53$ ). A similar index has been used previously (Crosnoe and Elder 2004).

### *Maternal Disengagement*

Children raised by parents who are cold, withdrawn, and disengaged have been found to be at-risk for a range of maladaptive outcomes, including delinquent involvement (Loeber and Stouthamer-Loeber 1986). Consequently, a maternal disengagement scale was created that has been used previously (Beaver 2008). During wave 2 interviews, respondents were asked seven questions that measured maternal disengagement. For instance, adolescents were asked to indicate whether their mother is warm and loving, whether they are satisfied with the way their mother communicates with them, and whether they are satisfied with their relationship with their mother. Responses to these items were summed together to create the maternal disengagement scale, where higher values reflect greater levels of maternal disengagement ( $\alpha = .86$ ).

### *Maternal Negativity*

The three maternal parenting scales described above each measure a different dimension of maternal parenting. However, there is empirical research indicating that global measures of parenting are stronger predictors of adolescent delinquency than are separate parenting scales (Wright and Cullen 2001). To take this finding into account, I created a global maternal negativity scale that was a function of the three maternal parenting scales. In doing so, the maternal attachment scale and the maternal involvement index were reverse-coded such that higher values indicated less attachment and less involvement. Then the maternal attachment scale, the maternal involvement index, and the maternal disengagement scale were subjected to factor analysis. The results revealed that all three items loaded on the same construct and, as a result, a weighted factor score was created. Higher values on this scale indicated more maternal negativity.

### *Parental Supervision*

Parental supervision has emerged as one of the most consistent predictors of adolescent delinquency (Gottfredson and Hirschi 1990). All else being equal, adolescents who are not monitored by their parents are at-risk for becoming involved in antisocial behaviors. To explore the genetic and environmental correlates of parental monitoring, a parental supervision scale was created. During wave 2 interviews, adolescents were asked seven questions about the amount of supervision that they receive from their parents. For instance, respondents were asked to indicate whether their parents let them make their own

decisions about what time they must be at home on weekend nights, about the people they hang around with, and about which television programs they watch. These items were coded dichotomously (0 = no, 1 = yes). Responses to these questions were then summed together to create the parental supervision scale, where higher values indicate more parental supervision ( $\alpha = .70$ ).

### *Paternal Attachment*

Although paternal parenting has been linked to various outcomes in adolescent development, the effects that fathers have on their children's delinquency has not been studied as thoroughly as the effects that mothers have on adolescent delinquent involvement. Even so, there is reason to believe that paternal attachment may be related to antisocial behaviors (Glueck and Glueck 1950). To explore the factors related to producing variation in paternal attachment, a paternal attachment scale was created. During wave 2 interviews, respondents were asked how close they feel to their father and how much they think their father cares about them. Responses to these items were coded on a 5-point Likert scale. These two items were summed together to create the paternal attachment scale, where higher values indicate more paternal attachment ( $\alpha = .71$ ).

### *Paternal Involvement*

The Add Health data also include a paternal involvement index. Similar to the maternal involvement index, the paternal involvement index was created from ten different items. During wave 2 interviews, respondents were presented with a list of ten activities and asked which, if any, their father had done with them in the past 4 weeks. More specifically, adolescents were asked whether they had gone shopping with their father, whether they had played a sport with their father, and whether they had worked on a project for school with their father. Items were coded dichotomously (0 = no, 1 = yes). Responses to the ten items were summed together to create the paternal involvement index, where higher values indicate more paternal involvement ( $\alpha = .61$ ).

### *Paternal Negativity*

Following the logic of the maternal negativity scale, a paternal negativity scale was created that acted as a more global measure of the father's parenting skills. This scale was created by using the paternal attachment scale and the paternal involvement index. Importantly, these two scales were reverse-coded so that higher values indicated less paternal attachment and less paternal involvement. Both of these scales were then subjected to a factor analysis which revealed that they could be accounted for by a unitary construct. As such, the weighted factor scores were used to create the paternal negativity scale. Higher values on this scale reflect more paternal negativity.<sup>1</sup>

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<sup>1</sup> Bivariate correlations were calculated to ensure that the maternal and paternal measures were not tapping the same underlying construct. The results of these bivariate correlations revealed statistically significant associations between all of the maternal and paternal parenting measures. These correlations, however, ranged between  $r = .09$  and  $r = .48$ , indicating that the maternal and paternal scales were measuring parent-specific behaviors, not family-wide parenting practices.



## Nonshared Sources of Variance

### *Low Self-Control*

In order to take into account possible child-effects, where the child's behaviors are influencing parenting, a low self-control scale was included in the analyses. During wave 1 interviews, respondents and their parents were asked twenty-three questions designed to measure individual variation in levels of self-control. For instance, respondents were asked whether they have trouble paying attention in school, whether they have trouble keeping their mind focused, and whether they go with their "gut feeling" when making a decision. Mothers were asked to indicate whether their child has a bad temper, whether they can trust their child, and whether their child gets along well with other children. Responses to these items were summed together to create the low self-control scale, where higher values indicate lower levels of self-control ( $\alpha = .75$ ). This same low self-control scale has been used by previous Add Health researchers (Beaver et al. 2009). For a complete listing of items that are included in this scale see Appendix 1.

### *Delinquency*

There is some evidence indicating that parental socialization, especially among adolescents, is the result of the way in which the adolescent behaves (Lytton 1990). To account for this possibility, a delinquency scale was included in the analyses. During wave 1 interviews, respondents were asked to indicate how frequently in the past year they had engaged in eleven different delinquent acts. For instance, youths were asked how frequently they had sold marijuana or other drugs, how frequently they had stolen something worth more than \$50, and how often they had taken part in a group fight. Responses to the items were summed together to create the delinquency scale ( $\alpha = .81$ ). Similar scales have been used previously by researchers analyzing the Add Health data (Guo et al. 2007). Appendix 1 contains a listing of the individual items that are included in this scale.

## **Plan of Analysis**

The analysis for this paper will be carried out in a series of linked steps. First, twin correlations will be calculated as an initial step towards determining the genetic and environmental underpinnings of various measures of parenting. A twin correlation is a correlation between the variable of one twin with the same variable of their co-twin. To illustrate, a twin correlation for maternal attachment would be calculated by correlating the maternal attachment scale for one twin with the maternal attachment scale for their co-twin. For genetic effects to be influential, the twin correlations for MZ twins must be significantly greater than the twin correlations for DZ twins. Twin correlations will be calculated for all of the parenting scales. Genetic influences on parenting will be inferred to the extent that the MZ twin correlations are significantly greater than the DZ twin correlations.

Although twin correlations provide an important first step when examining the potential genetic influences on a measure, additional analyses can be conducted to provide specific estimates of the relative effects of genetic and environmental factors. One technique that has emerged in the behavioral genetic research is DeFries–Fulker (DF) analysis (DeFries and Fulker 1985). DF analysis is a regression-based statistic that can be used when

analyzing samples of kinship pairs. The results generated from DF analysis provide precise estimates of the proportion of variance in perceptions of parenting (or any other variable) accounted for by genetic factors and the proportion of variance in perceptions of parenting accounted for by environmental factors.

The DF equation has been modified since it was originally proposed (Rodgers and Kohler 2005; Rodgers et al. 1994). The DF equation that is now used among samples drawn from the general population 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 the parenting scale being analyzed,  $K_2$  is their co-twin's score on that same parenting scale,  $R$  measures genetic similarity ( $R = 1.0$  for MZ twins,  $R = .5$  for DZ twins), and  $R * K_2$  is an interaction term created by multiplying  $R$  and  $K_2$ . In this equation,  $b_0$  = the constant,  $b_1$  = the proportion of variance in the parenting scale that is explained by shared environmental influences,  $b_2$  is not usually interpreted in the DF model, and  $b_3$  = the proportion of variance in the parenting scale that is accounted for by genetic factors. The effects of the nonshared environment (plus error) on the parenting scale is captured by the error term,  $e$ .

Rodgers and Kohler (2005) recently proposed a slightly different DF equation that represents an improvement over the one presented in Eq. 1. The new DF equation takes the following form:

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

where  $K_1$  remains the score on the parenting scale for one twin,  $K_2$  remains the co-twin's score on that same parenting scale, and  $R$  remains a measure of genetic similarity. The most obvious difference between Eqs. 1 and 2 is that Eq. 2 includes a new term,  $K_m$ . In this DF equation,  $K_m$  = the mean of the parenting scale of interest (i.e., the mean for  $K_2$ ). Equation 2 also indicates that  $K_2$  is being mean centered, while the main effect of  $R$  is eliminated (although it is still retained in the interaction term). The substantive meaning of the coefficients does not change between Eqs. 1 and 2. For instance,  $b_1$  = shared environmental effects on parenting and  $b_2$  = genetic effects on parenting, while  $e$  = the proportion of variance in parenting that is accounted for by nonshared environmental effects and error.

The coefficients in the DF model represent latent factors because they only estimate the proportion of variance in parenting accounted for by genetic, shared environmental, and nonshared environmental factors; they do not reveal which particular genes or which specific environments are accounting for the variance. Equation 2, however, can be modified to include measured nonshared sources of variance (Rodgers et al. 1994). The DF equation that includes nonshared sources of variance takes the following form:

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

The equation presented above is an almost exact duplicate of Eq. 2 with the important exception that Eq. 3 includes a new term, ENVDF. ENVDF is a difference measure that is created by subtracting twins' scores on a variable. The resulting value measures the difference between twins on the variable. To illustrate, Twin 2's score on the delinquency scale could be subtracted from Twin 1's score on the delinquency scale. The end result would be a new variable (ENVDF) that measures the difference in delinquent involvement between twins. Unlike the other coefficients where the value of  $b$  represents the explained variance, the  $b$  associated with the ENVDF variables does not have the same meaning. In short, the interpretation of the ENVDF centers on statistical significance and on traditional

effect sizes (e.g., *t*-values). All of the other coefficients are interpreted the same way as discussed in reference to Eq. 2.

Two DF models will be estimated for each parenting measure. First, a baseline DF model will be employed by using the Eq. 2. This model will provide estimates of the proportion of variance in parenting accounted for by genetic factors, shared environmental factors, and nonshared environmental factors. The second DF model will introduce low self-control and delinquency into the equation as nonshared sources of variance (i.e., ENVDF terms). Equation 3 will be used when estimating these models. Following the lead of other researchers using DF analysis (Beaver et al. 2008), all of the models will be estimated by using the statistical software package, AMOS. AMOS uses a full-information maximum likelihood imputation algorithm to estimate values for missing data. As a consequence, all of the DF models are based on the full sample of MZ and same-sex DZ twins ( $N = 537$  twin pairs).

## Results

The analysis begins by estimating twin correlations for all of the perceived parenting measures for the full sample and separately for MZ and DZ twin pairs. As Table 1 shows, all of the twin correlations across all three groups are statistically significant. A close inspection of the pattern of twin correlations reveals that all of the MZ twin correlations are larger than the DZ twin correlations. These findings suggest that parenting is at least partially influenced by genetic factors.

Table 2 contains the results of the DF models for the three perceived maternal parenting scales: maternal attachment, maternal involvement, and maternal disengagement. Remember that for each of the parenting measures two models will be calculated: one is a baseline model and the other introduces measures of low self-control and delinquency as nonshared sources of variance. Model 1 shows that shared environmental factors explain 42% of the variance, while 17% of the variance in maternal attachment is due to genetic factors. The nonshared environment (plus error) accounts for the remaining 41% of the variance in maternal attachment. Model 2 portrays the results of the model with low self-control and delinquency entered as nonshared sources of variance. As can be seen, both of these variables have statistically significant effects on maternal attachment. The significant coefficient for low self-control can be interpreted to mean that the twin with lower levels of self-control also reported lower levels of maternal attachment. Interestingly, the opposite

**Table 1** Twin correlations for the parenting scales

	All twins	MZ twins	DZ twins
Maternal attachment	.540*	.626*	.452*
Maternal involvement	.327*	.411*	.234*
Maternal disengagement	.413*	.482*	.332*
Parental supervision	.414*	.542*	.256*
Paternal attachment	.340*	.484*	.195*
Paternal involvement	.429*	.543*	.301*
Maternal negativity	.522*	.610*	.433*
Paternal negativity	.491*	.611*	.345*

\*  $p < .05$ , two-tailed tests

**Table 2** DF analysis of the maternal parenting scales

	Maternal attachment				Maternal involvement				Maternal disengagement			
	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE
DF analysis components												
Shared environment	.42*	.03	.43*	.03	.10*	.03	.10*	.03	.19*	.03	.24*	.03
Heritability	.17*	.04	.16*	.04	.31*	.04	.31*	.04	.29*	.04	.24*	.04
Nonshared sources of variance												
Low self-control			-.02*	.00			-.02*	.01			.07*	.02
Delinquency			.02*	.01			.02	.02			.07	.04

\*  $p < .05$ , two-tailed tests

set of findings emerged for delinquency. In this case, there was a positive association, meaning that the twin who reported more delinquent involvement actually reported higher levels of maternal attachment.

Models 3 and 4 of Table 2 contain the results for the DF models using the maternal involvement index as the dependent variable. These models indicate that about 10% of the variance in maternal involvement is due to the shared environment, about 31% of the variance is attributable to genetic factors, and about 59% of the variance is the result of nonshared environmental factors. Similar to Model 2, low self-control maintains a statistically significant and negative association with maternal involvement, meaning that the twin (from each twin pair) who had lower levels of self-control reported less maternal involvement.

The last two models in Table 2 portray the findings generated from the DF models when the maternal disengagement scale was entered as the dependent variable. Similar to the previous models, a significant amount of variance (between 24 and 29%) was accounted for by genetic factors. The shared environment also accounted for between 19 and 24% of the variance in the maternal disengagement scale, with the nonshared environment explaining approximately 50% of the variance in maternal disengagement. Also of interest is that low self-control had a statistically significant effect on maternal disengagement, where the twin with lower levels of self-control reported greater levels of maternal disengagement. There was no association between delinquency and maternal disengagement.

The results generated thus far indicate that a moderate amount of variance in perceptions of maternal parenting is due to genetic factors. Next, DF models were estimated for perceived parental supervision and the two perceived paternal parenting scales. Models 1 and 2 display the results for parental supervision. Recall that parental supervision was not parent-specific; rather the questions pertained to both parents. As a result, the parental supervision scale should not be considered a maternal scale or a paternal scale. As can be seen, the shared environment explains none of the variance in parental supervision, genetic factors account for about 54% of the variance in parental supervision, and nonshared environmental factors account for the remaining 46% of the variance. When low self-control and delinquency were introduced as sources of nonshared variance (Model 2), low self-control maintained a negative and statistically significant association with parental supervision. In substantive terms, this association indicates that the twin who had lower levels of self-control reported less parental supervision. The effect of delinquency on parental supervision was nonsignificant.

**Table 3** DF analysis of the parental supervision and paternal parenting scales

	Parental supervision				Paternal attachment				Paternal involvement			
	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE
DF analysis components												
Shared environment	.00	–	.00	–	.00	–	.00	–	.00	–	.00	–
Heritability	.54*	.04	.54*	.04	.46*	.05	.46*	.05	.55*	.05	.56*	.05
Nonshared sources of variance												
Low self-control			–.02*	.01			–.02*	.01			–.02*	.01
Delinquency			–.02	.02			.02	.01			.01	.02

\* *p* < .05, two-tailed tests

Models 3 and 4 of Table 3 contain the results of the DF models where the paternal attachment scale was employed as the dependent variable. These models indicate that shared environmental factors explain none of the variance in the paternal attachment scale. About 46% of the variance in the paternal attachment scale is attributable to genetic factors and nonshared environmental factors account for about 54% of the variance. In line with all of the previous models, low self-control had a statistically significant effect on paternal attachment, while delinquency was unrelated to levels of paternal attachment. The last two models in Table 3 show the findings for the DF models where the paternal involvement index was included as the dependent variable. Strikingly similar to the previous model, genetic factors explain about 55% of the variance in paternal involvement and the nonshared environment accounts for about 45% of the variance. The shared environment once again had no effect on the paternal involvement scale. Model 6 reveals that the low self-control scale was inversely related to paternal involvement, meaning that the twin with lower levels of self-control reported less paternal involvement. Delinquency was not related to paternal involvement.

The subsequent set of models examines the genetic and environmental underpinnings to the global measure of perceived maternal negativity and the global measure of perceived paternal negativity. Models 1 and 2 of Table 4 show that about 25% of the variance in maternal negativity is attributable to genetic factors. The remaining 75% of variance is divided between the shared environment (~35%) and the nonshared environment (~40%). In addition, low self-control and maternal negativity were significantly related, where the twin with lower levels of self-control was subjected to more maternal negativity.

The results for the perceived paternal negativity scale are presented in Models 3 and 4 of Table 4. These DF models reveal two findings that are of particular interest. First, the shared environment explains none of the variance in paternal negativity, genetic factors account for about 62% of the variance in paternal negativity, and the nonshared environment accounts for about 38% of the variance in paternal negativity. Second, low self-control is positively associated with paternal negativity, which suggests that the twin with lower levels of self-control reports greater paternal negativity. There is no relationship between delinquency and paternal negativity.<sup>2</sup>

<sup>2</sup> I tested for harmful levels of multicollinearity by calculating variance inflation factors (VIF) and tolerance values. The results of these statistics indicated the presence of some multicollinearity (as a function of the heritability term and the shared environmental term) for the models in Table 2 and the maternal negativity

**Table 4** DF analysis of maternal negativity and paternal negativity scales

	Maternal negativity				Paternal negativity			
	Model 1		Model 2		Model 3		Model 4	
	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE
DF analysis components								
Shared environment	.31*	.03	.35*	.03	.00	–	.00	–
Heritability	.29*	.04	.25*	.04	.62*	.04	.63*	.04
Nonshared sources of variance								
Low self-control			.02*	.00			.01*	.00
Delinquency			–.00	.01			–.01	.01

\*  $p < .05$ , two-tailed tests

Because low self-control and to a lesser extent delinquency were important sources of nonshared variance in explaining variance in the parental socialization scales, it is important to explore the various factors that account for variance in these scales. As a result, the next set of models decomposes the variance in the low self-control and delinquency scales using DF analysis. Table 5 displays the results of these models. As can be seen, the shared environment did not explain any of the variance in low self-control, genetic factors explained 41% of the variance, and the nonshared environment accounted for the remaining 59% of variance. Additionally, 18% of the variance in delinquency was accounted for by the shared environment, while genetic factors accounted for 38% of the variance and the nonshared environment explained the remaining 44% of the variance.

## Discussion

The effect that parental socialization has on child and human development is at the heart of much criminological theory and research (Harris 1998). For the most part, criminologists assume that the ways in which parents treat their children will have long-term effects on their child's behaviors, personalities, and life outcomes. At the same time, very little criminological research is ever expended on examining what explains variation in parental socialization both within- and between-families. Behavioral genetic research, in contrast,

Footnote 2 continued

model in Table 4. This is not surprising because the DF model is essentially an interaction model and interaction models are known to be affected by multicollinearity (for the models with the paternal measures as the dependent variable, the shared environmental effect was dropped from the equation because it was non-significant and so collinearity was not a problem). Nonetheless, I recalculated the DF models using different variants of the DF model and the results were virtually identical. Similarly, I recalculated the DF models in three steps. First, I entered only the ENVDIF measures into the equation (this was the baseline model). I then sequentially introduced the heritability term and then the shared environmental term. The standard errors for all of the coefficients were then examined across all of the models. The results revealed that the magnitude of the standard errors remained very similar across all of the models. Additionally, the results of these models are in line with the extant literature examining the genetic basis to family environments. Last, I recalculated all of the models by removing all of the ENVDIF measures and the shared environmental component. These models had no issues with collinearity (because they were essentially bivariate models) and the heritability estimates were similar to the ones that were reported in the full model (i.e., the confidence intervals overlapped). As a result, it does not appear as though the results are a function of collinearity or multicollinearity.

**Table 5** DF analysis of the low self-control and delinquency scales

	Low self-control		Delinquency	
	<i>b</i>	SE	<i>b</i>	SE
DF analysis components				
Shared environment	.00	–	.18*	.03
Heritability	.41*	.04	.38*	.03

\*  $p < .05$ , two-tailed tests

has explored the potential factors that explain variation in all types of environments, including those that are found in the family. The results of these studies have revealed that virtually all environments that have been examined by behavioral geneticists are partially shaped by genetic factors (Kendler and Baker 2007). The current study used these findings as a springboard to examine the extent to which genetic factors explained variance in perceptions of maternal and paternal parental socialization.

Analysis of twin pairs from the National Longitudinal Study of Adolescent Health revealed four broad findings. First, genetic factors explained a statistically significant amount of variance in all of the measures of perceived maternal and paternal parenting. Although the precise amount of variance attributable to genetic factors varied across the various dimensions of parenting, genetic factors consistently explained at least 25% of the variance in parenting. The magnitude of these genetic effects is consonant with the genetic effects reported in other studies (Kendler and Baker 2007).

The precise interpretation of the heritability estimates for perceived parenting remains somewhat obfuscated. On the one hand, these heritability estimates may be capturing child-effects that are the result of genetic factors in the adolescent. For example, adolescents who have a bad temper (a genetically influenced trait) are likely to evoke negative reactions from their parents. On other hand, however, variation in parenting between twins could represent gene-environment interactions, where the parents' genetic predispositions interact with each of their child's unique suite of traits. The end result would be differential parental treatment between twins that is the result of complex interactions between genes and the environment. Still another interpretation is that the heritabilities of the perceived parenting measures could represent genetic influences on how the adolescents perceive and interpret parental treatment (Jaffee et al. 2004). Unfortunately, the research design employed in this study was unable to disentangle these different interpretations.

Regardless of the underlying mechanism that is producing the heritability of perceived parenting, the results of the current study clearly indicate that perceived parenting is a function of genetic factors. This finding has serious ramifications for criminological theory and research. First, from a theoretical standpoint, there are very few, if any, criminological perspectives that highlight the very real possibility that parental socialization is the result of genetic factors. Gottfredson and Hirschi's (1990) theory is a prime example. They argue that variation in levels of self-control is almost completely the result of variation in parental management techniques. Gottfredson and Hirschi are essentially arguing that variation in parenting produces variation in levels of self-control. This is likely a gross oversimplification because in all actuality, variation in parental management techniques is produced by genetic influences in both the parent and the child. And, there is not a single study published in a criminology journal examining the genetic influences on measures of parenting. Without directly examining the role of genetics, it is likely that criminological theories and research that examine parental socialization are misspecified (Harris 1998).

The second main finding to emerge from the current study is that a significant amount of variance in the maternal parenting measures was explained by shared environmental factors. At first glance, this finding seems to provide unequivocal evidence that parenting is a shared environment that makes siblings more similar to each other—that is, parental socialization has effects on human development that are in line with the traditional social science perspective. Such a conclusion, however, needs to be tempered by the fact that the shared environmental component also includes the effects of any passive gene-environment correlations, whereby genetic factors cause parents to treat their children similarly (Schulz-Heik et al. 2009). As a result, the statistically significant effects of the shared environmental component may simply be capturing passive gene-environment correlations, not purely environmentally mediated effects. Unfortunately, the research design employed in the current study was unable to explore whether the shared environmental component was a reflection of shared environmental effects, passive gene-environment correlations, or some combination of the two. Future research would benefit by exploring this issue in greater detail.

Third, for most of the parenting measures, the nonshared environmental component explained at least 50% of the variance in parental socialization. In substantive terms, this finding suggests that nongenetic child-specific characteristics and idiosyncratic parental behaviors are a driving force behind variation in parental socialization. While it was not possible to examine all of the various child-specific characteristics that would fall under the rubric of the nonshared environment, the effects of two specific nonshared environments were explored: low self-control and delinquent involvement. The results of the DF models revealed that the twin who had lower levels of self-control elicited more negative parental reactions. Stated differently, twin differences in levels of self-control represent a significant nonshared source of variance in parental socialization.

In contrast, adolescent delinquency failed to reach statistical significance in seven of the eight models. These null results stand in opposition to much criminological research showing a statistically significant association between measures of parenting and measures of adolescent delinquency. Recall, however, that DF models remove all of the variance that is attributable to genetic factors and shared environmental factors before estimating the effects of specific nonshared sources of variance. This methodology represents a significant departure from most social science research that confounds shared, nonshared, and genetic effects when examining associations between parenting and antisocial outcomes thereby producing upwardly biased estimates of the parenting-antisocial behavior nexus. Supplementary analyses were conducted to examine whether there were statistically significant associations between the parenting measures and the adolescent delinquency scale when using a simple bivariate test that does not account for genetic and shared environmental effects. Statistically significant associations were found in two of the four maternal parenting measures ( $p < .05$ ), with the two null results just barely failing to reach statistical significance ( $p < .11$ ). Similar results were detected for the three paternal parenting measures, where a statistically significant effect was detected in one of the three models ( $p < .05$ ) and a marginally significant effect was detected in another one ( $p < .06$ ). There was not a significant association between parental supervision and delinquency. These results indicate that studies that fail to control for genetic influences likely over-estimate the association between parental socialization and delinquency (Harris 1995; Rowe 1994; Wright and Beaver 2005).

The results of the DF analysis for the low self-control and delinquency scales tend to reinforce this perspective. For low self-control, none of the variance was accounted for by shared environmental factors (e.g., family-wide parenting practices), 41% was the result of



genetic factors, and the remaining 59% of variance was attributable to nonshared environmental factors. These findings strongly suggest that criminologists studying the development of self-control need to explore the complex ways in which genetic and nonshared environmental factors create variation in levels of self-control. A slightly different pattern of results was detected for the delinquency scale in that the shared environment explained 18% of the variance in delinquency. The overwhelming majority of variance, however, was still accounted for by genetic factors (38%) and nonshared environmental factors (44%). In order for criminologists to keep pace with these results, they need to begin to analyze genetically sensitive datasets which are capable of separating shared environmental effects from genetic and nonshared environmental effects. Existing criminological theories focusing on the development of antisocial behaviors, moreover, need to be modified in order to take into account the results generated from biosocial criminological research indicating the importance of genetic and nonshared environmental effects.

The fourth key finding to emerge from the analyses was the significant differences in heritability between the maternal parenting scales and the paternal parenting scales. Across all of the models the heritability estimates were much higher for the paternal parenting scales in comparison with the maternal parenting scales. For example, the heritabilities of the maternal parenting scales were relatively modest, with heritability estimates ranging between .16 and .31. The heritabilities of the paternal parenting scales, in contrast, were much stronger, with heritability estimates ranging between .46 and .63. *z*-scores for testing for the difference between coefficients were estimated as outlined by Paternoster et al. (1998) and the results confirmed that heritability estimates were significantly different for attachment ( $z = 4.69, p < .05$ ), involvement ( $z = 3.91, p < .05$ ), and negativity ( $z = 6.72, p < .05$ ). What accounts for the differences in heritability between maternal and paternal parenting is not known, but remains an important line of inquiry for future researchers to explore.

Although the results of this study add to research examining gene-environment correlations and the genetic basis to parenting behaviors, the results need to be interpreted with caution in light of at least two key limitations. First, the analysis was based on a sample of twin pairs, which necessarily raises questions about the generalizability of the results. Whether the findings would generalize to singletons remains a limitation of all analyses that employ sibling pairs; however, studies that analyze kinship pairs represent a significant improvement over studies that only include one sibling per household in the data because genetic effects can be estimated directly (Harris 1998; Rowe 1994; Wright and Beaver 2005). Second, the parenting measures were drawn from data collected during adolescence. It would be interesting to examine whether genetic factors explain an equal amount of variance in parental socialization during infancy and childhood.

Replication studies addressing these limitations need to be conducted before a great deal of stock can be placed in the findings generated in the current study. Nonetheless, the results presented here pose a significant challenge to criminological research that assumes that “environmental” measures reflect purely social processes and are not affected by genetic influences. Instead, a more accurate perspective is to view environments as partially an extension of genotype, where each person’s unique suite of genetically influenced phenotypes are manifested in the shaping and molding of their environment (Dawkins 1982). This perspective does not downplay the significance of the environment, but rather adds clarity to understanding why environments are not randomly distributed across people, something that purely social science explanations are not able to do. Integrating

genetic research into criminological research, in short, will help criminological theories to become more believable, more accessible, and most importantly, more accurate.

**Acknowledgments** This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (<http://www.cpc.unc.edu/addhealth>). No direct support was received from grant P01-HD31921 for this analysis.

## Appendix 1

See Table 6.

**Table 6** Individual items comprising the nonshared sources of variance measures

### *Low self-control scale*

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's usually because you worked hard for it.
7. You never get sad.
8. You never criticize other people.
9. You usually go out of your way to avoid having to deal with problems in your 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 problem, 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 your homework done?
23. Do you have trouble getting along with other students?

### *Delinquency scale*

In the past 12 months, how often did:

1. You hurt someone badly enough to need bandages or care from a doctor or nurse?

**Table 6** continued

2. Someone hurt you badly enough to need bandages or care from a doctor or nurse?
3. You use or threaten to use a weapon to get something from someone?
4. You take part in a fight where a group of your friends were against another group?
5. You deliberately damage property that did not belong to you?
6. You steal something worth more than \$50?
7. You steal something worth less than \$50?
8. You go into a house or building to steal something?
9. You sell marijuana or other drugs?
10. You shot or stabbed someone?
11. You pulled a knife or gun on someone?

\* A question asked to the parent. All other questions were asked directly to the respondent

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