

Physical Punishment and Childhood Aggression: The Role of Gender and Gene–Environment Interplay

Brian B. Boutwell^{1*}, Cortney A. Franklin¹, J.C. Barnes², and Kevin M. Beaver³

¹Sam Houston State University, College of Criminal Justice, Huntsville, Texas

²The University of Texas at Dallas, School of Economic, Political and Policy Sciences, Richardson, Texas

³Florida State University, College of Criminology and Criminal Justice, Tallahassee, Florida

.....

A large body of research has linked spanking with a range of adverse outcomes in children, including aggression, psychopathology, and criminal involvement. Despite evidence concerning the association of spanking with antisocial behavior, not all children who are spanked develop antisocial traits. Given the heterogeneous effects of spanking on behavior, it is possible that a third variable may condition the influence of corporal punishment on child development. We test this possibility using data drawn from a nationally representative dataset of twin siblings. Our findings suggest that genetic risk factors condition the effects of spanking on antisocial behavior. Moreover, our results provide evidence that the interaction between genetic risk factors and corporal punishment may be particularly salient for males. *Aggr Behav* 37:559–568, 2011. © 2011 Wiley Periodicals, Inc.

.....

Keywords: antisocial behavior; corporal punishment; gene–environment interaction

There is currently a wealth of research linking the use of corporal punishment with deleterious outcomes in children [Gershoff, 2002; Lansford et al., 2011; Taylor et al., 2010]. Despite consistent correlations between physical punishment and antisocial outcomes, however, at least one issue remains largely unresolved. That is, the majority of children who are exposed to corporal punishment do not develop aggressive tendencies. This heterogeneity in response to spanking suggests that additional variables may play a role in moderating the link between spanking and later-life behaviors [Rutter et al., 2006]. One possibility, stemming from research in the fields of molecular and behavioral genetics, is that genetic factors may condition the effects of punishment on behavior. A burgeoning line of research indicates that certain genetic variants moderate the influence of physical abuse on the development of violent and antisocial behavior [Kim-Cohen et al., 2006].

Although some researchers have correctly pointed out that most parents in the population do not physically abuse their offspring [Harris, 1998], the majority of parents will, at some point, rely on corporal punishment to discipline their children [Taylor et al., 2010]. Given the large number of

parents who spank, as well as the consistent evidence linking spanking to antisocial behavior, there remains a need to examine whether genetic influences might moderate the effects of corporal punishment on behavior. This study addresses this question by analyzing a sample of twin siblings drawn from a nationally representative data set. We begin first, however, by examining prior research concerning the link between the use of corporal punishment and aggressive behavior in children.

CORPORAL PUNISHMENT AND CHILDHOOD ANTISOCIAL BEHAVIOR

For the past several decades, researchers across multiple disciplines have devoted a significant amount of attention to testing the relationship

*Correspondence to: Brian B. Boutwell, Sam Houston State University, College of Criminal Justice, P.O. Box 2296, Huntsville, TX 77341-2296. E-mail: brian.boutwell@shsu.edu

Received 10 March 2011; Revised 23 May 2011; Accepted 18 June 2011

Published online 6 September 2011 in Wiley Online Library (wileyonlinelibrary.com). DOI: 10.1002/ab.20409

between corporal punishment and individual differences in personality and behaviors [Gershoff, 2002; Glueck and Glueck, 1950]. Gershoff [2002] recently conducted an extensive meta-analysis on the topic. The review covered more than 50 studies and examined the effects of spanking across 11 different outcomes, including mental health, parent-child relationships, aggression, and crime. The findings of the meta-analysis revealed a statistically significant association between corporal punishment and adverse phenotypes. Corporal punishment exerted an average effect size of .36 on measures of aggression collected during childhood. Delinquent and antisocial behaviors were also shown to be significantly related to spanking ($d = .42$). Similar results were observed for the relationship between spanking and mental health ($d = -.49$), being a victim of physical abuse ($d = .69$), abusing one's own child or spouse in adulthood ($d = .13$), and engaging in adult criminal and antisocial behaviors ($d = .42$). In short, the pattern of findings produced by Gershoff suggests that spanking is consistently related to an increased risk of adverse outcomes both in childhood and across the life course.

Although the findings described above offer support for a link between spanking and antisocial behavior, many of the studies included in Gershoff's [2002] meta-analysis were cross-sectional. As a result, the ability to definitively state the temporal ordering between spanking and the child outcomes remains impossible, and thus any conclusions about causality remain undecided. Recently, however, Taylor et al. [2010] addressed this limitation by analyzing data drawn from the Fragile Families and Child Wellbeing Study to examine the longitudinal association between corporal punishment and aggression in a sample of young children. After controlling for a host of potentially confounding factors, including the child's levels of antisocial behavior at prior waves, the results revealed that increased spanking at age 3 corresponded to higher levels of aggression once the child had reached the age of 5.

Lansford et al. [2011] reported similar findings from a longitudinal analysis of two independent samples of children in the United States. The first sample in the study included more than 500 males and females ranging in age from 6 to 9 years old. Analysis of this sample revealed that higher levels of physical discipline in a given year corresponded to increased levels of behavior problems in the following year. The second sample analyzed by Lansford et al. [2011] included more than 200 at-risk boys between the ages of 10 and 15. The results revealed

that the use of corporal punishment in 1 year predicted increased antisocial behavior in the following year. Interestingly, Lansford and her team also reported evidence of a reciprocal effect between spanking and aggression. In this case, although spanking was linked to behavioral problems, childhood behavioral problems were also found to elicit the use of more corporal punishment.

The findings presented by Lansford et al. [2011] suggest that a transactional relationship may exist between spanking and antisocial behavior. In other words, the spanking-behavior correlation may not necessarily reflect a unidirectional influence of spanking on behavioral problems. Other studies have suggested that difficult and taxing behavior in children, for example, can elicit the use of physical disciplinary tactics [Jaffee et al., 2004]. Jaffee et al. [2004] analyzed data from more than 1,000 twins drawn from the Environmental Risk (E-Risk) Longitudinal Twin Study. The results of the study demonstrated that the effect of spanking on antisocial behavior was, in large part, child driven. In this case, genetic factors in the child played a significant role in predicting the use of corporal punishment.

That there is an association between spanking and behavior seems evident from the body of research mentioned above. Whether the relationship represents a causal effect of spanking on behavior, a child-driven effect, or a reciprocal effect remains an unresolved empirical issue. None of these possibilities, however, can fully account for why most children who are spanked fail to develop behavioral problems. As a result, an alternative explanation is needed that might offer more insight into why certain children seem especially vulnerable to the influence of spanking. One possibility—assuming for a moment that spanking has a causal influence on the child's behavior—is that spanking effects become more or less pronounced depending on the presence of additional factors in the child. A body of research is beginning to suggest that genes might play a key role in moderating certain environmental risk factors. Known broadly as gene-environment interaction ($G \times E$), this line of inquiry is discussed in some detail below.

GENE-ENVIRONMENT INTERACTION AND CORPORAL PUNISHMENT

Research on the topic of $G \times E$ focuses on the interplay that takes place between genetic and environmental factors in the prediction of numerous

human outcomes, including various diseases and different forms of psychopathology [Moffitt, 2005; Moffitt et al., 2006]. Existing research on the topic of $G \times E$ has revealed that the influence of certain genes may be contingent on exposure to various environmental pathogens [Rutter et al., 2006]. Conversely, the effects of certain environmental risk factors may become most pronounced when paired with genetic vulnerabilities [Rutter et al., 2006].

Along this line of reasoning, it is possible that children who possess a genetic vulnerability for the development of antisocial behaviors may be disproportionately susceptible to adverse environmental experiences. Based on the logic of $G \times E$ research, the effects of physical or harsh discipline may influence the emergence of behavioral problems in some children by exacerbating a genetic predisposition for aggressive and antisocial behavior [Caspi et al., 2002; Jaffee et al., 2005; Kim-Cohen et al., 2006]. Variation in genetic factors, in other words, may help explain why not all children who are spanked also develop behavioral problems.

To date, there is relatively little evidence bearing directly on the issue of whether genetic risk conditions the influence of corporal punishment on antisocial behavior [Lynch et al., 2006]. Despite this paucity of research, indirect evidence of an interaction between spanking and genetic factors can be gleaned from studies examining the effects of child abuse and neglect. Jaffee et al. [2005], for example, examined whether genetic susceptibilities to conduct disorder were conditioned by the experience of physical maltreatment in children. These researchers analyzed a sample of twin siblings drawn from the E-Risk Study. The findings presented by Jaffee et al. suggested that exposure to maltreatment conditioned genetic influences on the emergence of conduct problems.

Caspi et al. [2002] were the first group of researchers to detect a statistical interaction between a measured gene and a measured environment in the prediction of antisocial behavior. Analyzing data drawn from the Dunedin Multidisciplinary Health and Development Study, these researchers were interested in whether variants of the monoamine oxidase A (MAOA) gene were conditioned by the experience of abuse or neglect in childhood. The study examined a range of behavioral phenotypes, including measures tapping violence, aggression, and overt criminality. The findings suggested that the low functioning variant of the MAOA gene conditioned the influence of child abuse on myriad antisocial outcomes over the life course.

Efforts to replicate the original $G \times E$ detected by Caspi et al. [2002] have provided further support for

the ability of environments to condition the effects of genes [for exceptions, see Haberstick et al., 2005]. Kim-Cohen et al. [2006] reviewed findings from a number of studies examining the MAOA-child abuse interaction. This group of researchers found a significant pooled interaction effect for the low functioning MAOA allele and exposure to maltreatment. A more recent meta-analysis yielded a very similar set of findings [Taylor and Kim-Cohen, 2007]. Taylor and Kim-Cohen [2007] reported that maltreatment consistently conditioned the influence of MAOA on antisocial outcomes both in childhood and across development.

Finally, Paaver et al. [2008] analyzed approximately 222 boys and 261 girls drawn from the European Youth Heart Study. Paaver et al. [2008] were interested in whether certain variants of the 5-HTTLPR serotonin transporter gene conditioned the influence of adverse family environments on levels of impulsivity. The results of the study provided evidence of a significant interaction between the short allele of the gene and family adversity in the prediction of impulsive behaviors. Interestingly, this significant $G \times E$ was constrained only to females in the sample. For males, the two variables did not produce a statistically significant interaction.

THIS STUDY

Despite growing interest by researchers on the issue of $G \times E$, there has been little attention devoted to exploring whether genetic risk factors condition the effects of spanking in the prediction of childhood conduct problems. The dearth of evidence in this area is surprising for at least two reasons: (1) spanking in childhood has been tied to a host of deleterious outcomes and (2) an overwhelming majority of parents in the population spank their children. In light of the gaps that exist in this line of inquiry, this study has two goals. First, we examine whether the use of corporal punishment during the first 5 years of life moderates a genetic predisposition for antisocial behavior. Second, we test whether spanking moderates genetic influences similarly for males as compared with females.

METHODS

Sample

This study was conducted using data drawn from the Early Childhood Longitudinal Study, Birth

Cohort (ECLS-B). The ECLS-B is a nationally representative sample of all children born in the United States in the year 2001. Children were sampled via birth certificates registered with the National Center for Health Statistics. This design allowed efficient coverage of the target population (i.e., all children born in 2001). The primary aim of the ECLS-B was to gather a range of information concerning various aspects of child development. The sampled children were followed for the first 4 years of life, allowing for observation of numerous developmental milestones and the factors that underlie their emergence. During each wave of the ECLS-B, researchers relied on multiple reporting sources, including the child's biological mother and father (even if the father was a nonresident father), as well as information reported by trained observers [Bethel et al., 2005]. To date, three waves of data have been collected and are available for analysis [Nord et al., 2004].

Beginning in the fall of 2001 and ending in the fall of 2002, data for Wave I was collected from the survey participants ($n = 10,700$ children; $n = 5,450$ males; $n = 5,250$ females).¹ At this time, the children were approximately 9 months old. Thus, the majority of data collection during Wave I occurred in the focal child's home. Information was collected concerning the child's developmental status, motor skill development, language abilities, and behavioral regulation. Questions were also included in order to assess the caregiver's occupational status, educational history, socioeconomic status, parenting practices, and overall health and wellbeing.

The second wave of data collection (Wave II) began in the fall of 2003, and included roughly 90% of parental respondents sampled during Wave I. At the time of the Wave II interviews, the focal children had reached the approximate age of 2 years. The survey procedures employed during Wave II closely mirrored those of Wave I. The child's primary caregiver once again completed a personal interview as well as a self-report questionnaire. Children were also assessed on a range of outcomes, including levels of parent-child attachment, cognitive development, physical health and wellbeing, and behavioral regulation.

Wave III data collection began in the fall of 2005 and ended in the spring of 2006. Similar to Wave II, approximately 90% (unweighted) of respondents were retained in the sample during the Wave III follow-up. During the third wave of the ECLS-B,

most of the focal children were approximately 4 years old. During Wave III, several elements of the study were necessarily modified in order to include more age-appropriate survey content. Parents, for example, were asked about specific behavioral indicators for their children, such as the child's level of aggressiveness, impulse control, and their capabilities of getting along with their peers in a social setting. Additionally, parents were also asked a number of questions related to the home environment, relationship status, and parenting practices employed with their child (e.g., disciplinary strategies).

Embedded within the ECLS-B data are a sample of twin children who lived in the same household ($n = 1,600$ twins). Roughly, 250 of the sampled twins were identified as being monozygotic (MZ) or identical twins. The remaining twins in the sample were identified as being dizygotic (DZ) or fraternal twins ($n = 1,300$).² Twin pairs that lacked a clear determination of zygosity were excluded from the current analysis. It is important to point out that the parents of twins reported information separately for each child.

Measures

Childhood antisocial behavior. For this study, childhood antisocial behavior was assessed using data drawn from the Wave III in-home parental interviews. Eight items, adapted from the Preschool and Kindergarten Behavior Scales-Second Edition [PKBS-2; Merrell, 2003], were analyzed in order to measure the presence of antisocial behavior in the focal children. The PKBS-2 is a standardized instrument designed to tap a range of aspects associated with both disruptive and overtly aggressive behaviors that emerge during the early years of development. Respondents, for example, were asked about the frequency with which their child threw temper tantrums and had problems concentrating. Additionally, parents were asked how often their child destroyed personal property. Responses to the individual items ranged in frequency from 1 (never) to 5 (very often).

In order to evaluate the psychometric properties of the eight observable indicators, we conducted both exploratory and confirmatory factor analyses. The findings suggested that each of the items tapped a unified construct assessing variation in antisocial behavior. In order to construct the childhood antisocial behavior scale, each of the eight items were summed and coded, such that higher values on the scale were indicative of increased antisocial

¹In compliance with the ECLS-B privacy procedures, all sample sizes and case counts have been rounded to the nearest 50.

²Approximately 100 twins were excluded because they either lacked zygosity information or data related to their cotwin.

TABLE I. Correlation Matrix and Descriptive Statistics

		X1	X2	X3	X4	X5	X6	X7	X8
Childhood antisocial behavior	X1	1.00							
Genetic risk	X2	0.23*	1.00						
Corporal punishment	X3	0.25*	0.07*	1.00					
Low self-control	X4	0.29*	0.16*	0.14*	1.00				
Maternal depression	X5	0.31*	0.21*	0.16*	0.17*	1.00			
Family adversity	X6	0.14*	0.05	0.01	0.03	0.16*	1.00		
Child's sex	X7	0.16*	0.07*	0.04	0.10*	-0.02	0.04	1.00	
Child's race	X8	-0.08*	-0.03	-0.07*	-0.16*	-0.14*	-0.05	0.02	1.00
Mean		19.23	1.02	0.84	8.95	17.09	17.93	0.50	0.61
SD		4.90	0.57	1.80	4.33	5.63	4.65	0.50	0.48

*Significant at the 0.05 level, two-tailed.

behavior in the child ($\alpha = .80$). Table I presents descriptive statistics and bivariate correlations for all the measures included in this study.

Genetic risk scale. Although the ECLS-B lacked genotypic information for the participants, we were able to construct a latent measure of genetic risk using the sample of twins [Beaver et al., 2009; Jaffee et al., 2005; Vaske et al., 2011]. In order to create a measure of genetic risk, the first step was to randomly select one child from within each twin pair to be designated as the *target twin*. The target twin's sibling was then assigned the designation of *cotwin*. Next, the cotwin's score on the antisocial behavior scale (described above) was transformed into a dichotomous variable. In order to create the dichotomous measure, the continuous scale was recoded so that individuals scoring at or above the 90th percentile were given a score of "1." All other individuals were assigned a score of "0."

After creating the dichotomous measure, it was then used to model levels of genetic risk as a function of twin relatedness (i.e., twin zygosity) [Vaske et al., 2011]. Given that MZ twins share 100% of their genetic material and DZ twins share approximately 50% of their distinguishing genes, behaviors that are under genetic influence should correlate more closely between MZ twins than between DZ twins.³ As a result, MZ twins with a cotwin scoring "1" on the dichotomous behavioral

problems scale are considered to possess the highest level of genetic risk for displaying antisocial behavior. DZ twins with a sibling scoring "1" on the dichotomous measure are considered to rank second in terms of genetic risk. DZ twins with a sibling scoring "0" on the dichotomous scale would be at the third highest level of risk, genetically speaking. MZ twins, finally, with a cotwin scoring "0" on the dichotomous behavioral scale are considered to have the lowest level of genetic risk.

The genetic risk scale included in this study was coded so that 0 = MZ target twins whose cotwin was assigned a score of 0 on the dichotomous externalizing behaviors variable; 1 = DZ twins whose cotwin was assigned a score of 0 on the dichotomous behavioral measure; 2 = DZ target twins whose cotwin was assigned a score of 1 on the dichotomous externalizing behaviors variable; and 3 = MZ target twins whose cotwin was assigned a score of 1 on the dichotomous externalizing behaviors variable. Higher scores on the genetic risk scale are intended to capture higher levels of genetic predisposition for the development of antisocial behavior.

Corporal punishment. The Wave III interviews with the focal child's mother included a series of items intended to assess the disciplinary strategies used to deal with a range of childhood misbehaviors. Included within these questions were items related to whether the mother spanked her child. If the subject responded affirmatively to spanking their child, they were then asked to indicate the number of times in the previous week that they had used corporal punishment in order to discipline both the focal child as well as their cotwin. In line with prior research, responses to this item were coded continuously such that higher scores corresponded to increased use of spanking by the parent [Gershoff, 2002].

Low self-control. Difficulties with self-control, impulse regulation, and attention problems early in

³A primary assumption underlying the construction of the genetic risk scale is that MZ twins correlate more closely for the measure of antisocial behavior (i.e., the scale used to construct the genetic risk index) than do DZ twins. We examined this issue directly, and the cross-twin correlations were as follows: MZ twins, $r = .67$; DZ twins, $r = .38$. Because both sex-discordant and same-sex DZ twins were included in the analysis, we also examined whether sex differences might influence the correlation observed for DZ twins. These results revealed that the cross-twin correlation for same-sex DZ twins was almost identical to that for sex discordant DZ twins ($r = .38$ and $.36$, respectively).

life represents a risk factor for a host of adverse phenotypes, including antisocial and aggressive behaviors [Arseneault et al., 2003; Pratt and Cullen, 2000; Tremblay et al., 2004]. In order to account for the relationship between low self-control and behavioral problems, we included a measure of childhood self-control taken during the Wave II interviews with the child's primary caregiver.

To assess variation in self-control, the ECLS-B included items drawn from the Infant/Toddler Symptom Checklist [DeGangi et al., 1995]. Parents of the focal children were asked a series of questions designed to capture the child's capability of paying attention and focusing during certain activities. Responses to the individual items were coded such that 0 = never, 1 = used to be, 2 = sometimes, and 3 = most times. The results of both exploratory factor analysis and reliability analysis indicated that each of the observable indicators loaded on a single construct ($\alpha = .69$). To create the scale, the individual items were summed and coded so that higher scores reflected lower levels of self-control.

Maternal depression. Prior research has suggested that exposure to maternal depression increases the risk of developing antisocial behavior in childhood [Kim-Cohen et al., 2005]. In order to take account of findings concerning the depression-antisocial behavior link, we included a modified version of the Center for Epidemiologic Studies Depression Scale (CES-D) collected during Wave III of the ECLS-B [Radloff, 1977]. The CES-D has been used by researchers to measure depressive symptoms and has shown to be a valid method of assessing depressive symptoms among the general population [Radloff and Teri, 1986].

During Wave III, mothers in this study responded to 12 self-administered items designed to tap depressive symptomology. The participants were asked to indicate how often they experienced feelings of depression, loneliness, and fearfulness. Additional items tapped the respondent's ability to focus their attention on various tasks and to achieve desired goals. For this analysis, we summed each of the individual items in order to create a measure of maternal depression. Higher scores on the Wave III depression scale corresponded to increased levels of depressive symptoms ($\alpha = .87$).

Family adversity. Exposure to adverse family environments is a risk factor for the development of chronic and severe antisocial behavior in children [Moffitt, 1993]. As a result, we included a measure of family adversity in this analysis. During the first wave of data collection, mothers in the study were asked a series of questions in order to measure how

often they argued with their spouse over a number of topics. Participants, for example, were asked to report on the frequency of disagreements concerning household finances and duties around the home. Responses to each item were coded such that 1 = never, 2 = hardly ever, 3 = sometimes, and 4 = often. Each of the items were coded such that higher scores reflected a more disruptive home environment and summed to create the measure of family adversity ($\alpha = .77$).

Child's sex. To explore potential gender differences in this study, as well as to avoid any potential problems with confounding owing to differences between the sexes, we controlled the gender of the child. The child's gender was measured using information gleaned from the birth certificate and was coded so that 0 = female and 1 = male.

Child's race. In order to limit the potential for confounded results, this study also included a measure of race in the analyses. The child's race was measured using information gleaned from the birth certificate and was coded so that 0 = non-white and 1 = white.

PLAN OF ANALYSIS

The analysis for this study proceeded in a series of interrelated steps. First, we began by using ordinary least squares regression to estimate the influence of both genetic risk and corporal punishment on childhood antisocial behavior.⁴ Second, we examined whether exposure to corporal punishment conditioned the influence of genetic factors on antisocial behavior. In order to test for the presence of $G \times Es$, we constructed a multiplicative interaction term using the genetic risk scale and the measure of corporal punishment. The third and final step was to examine gender differences for the emergence of antisocial behavior. For this study, gender differences were explored using a two-pronged approach. First, the sample was subdivided into male and female subsamples. Second, the first two steps described in the plan of analysis were repeated separately using the male sample and the female sample, respectively.

RESULTS

We begin by first examining the impact of corporal punishment and genetic risk on the development of

⁴It is important to mention that the twin pairs in this study were double entered into the dataset. As a result, every twin serves as both the *target* twin and the *co-twin*.

TABLE II. OLS Regression Models Predicting Childhood Antisocial Behavior

	Model 1			Model 2		
	B	β	SE	B	β	SE
Genetic risk	0.82	.10*	.25	0.40	.05	.30
Corporal punishment	0.56	.21*	.11	0.05	.02	.11
Low self-control	0.22	.20*	.03	0.22	.20*	.03
Maternal depression	0.19	.21*	.03	0.20	.21*	.03
Family adversity	0.10	.10*	.03	0.10	.10*	.03
Child's sex	1.48	.16*	.26	1.46	.15*	.30
Child's race	-0.05	-.00	.30	-0.00	-.00	.30
Genetic risk X corporal punishment	-	-	-	0.46	.21*	.21

*Significant at the 0.05-level, two-tailed; Huber/White standard errors are presented.

antisocial behavior. The findings from this portion of the analysis are presented in Model 1 of Table II. As seen, both corporal punishment and genetic risk exerted significant main effects on the behavioral problems scale. In this case, children exposed to increased use of corporal punishment exhibited increased behavioral problems, even after controlling for the influence of genetic factors. Not surprisingly, children exposed to higher levels of family adversity increased maternal depression and those with lower levels of self-control also tended to evince more behavioral problems.

Although Model 1 provides evidence that both genetic risk and corporal punishment influence behavioral problems, it does not offer insight into whether the use of corporal punishment conditions the effects of genes. As a result, the second step in the analytical process was to examine whether corporal punishment interacted with genetic factors to influence antisocial behavior in the child. The results contained in Model 2 of Table II suggest that the effects of genetic risk on behavioral problems were moderated by the use of corporal punishment. Stated another way, corporal punishment has a more pronounced effect for children with greater genetic risk.⁵

⁵We examined whether controlling for maternal and paternal antisocial behavior substantively altered the results. To do so, we included a measure of maternal antisocial behavior along with the original covariates and recalculated our models. The results were unchanged from the findings presented above. Moreover, we also tested whether controlling for paternal antisocial behavior affected the results. The standardized effect size (i.e., coefficient) for the interaction term, controlling both maternal and paternal antisocial behavior, remained substantively identical to the original models. Because the introduction of both maternal and paternal behavioral measures resulted in the removal of a number of cases or to listwise deletion, we opted to retain our original set of covariates.

The next phase of the analysis was to test whether the $G \times E$ presented above might be conditional upon the child's gender. Essentially, this portion of the analysis tests whether a three-way interaction exists between corporal punishment, genetic risk, and gender in the prediction of antisocial behavior. To address this point, the sample was divided by gender and the models presented in Table II were reestimated. The findings obtained using the gender-specific models are presented in Table III. As can be seen, Model 1 of Table III reveals a similar pattern of findings for males that was obtained using the full sample. Both the genetic risk scale as well as the corporal punishment measure exerted significant influences on antisocial behavior. Importantly, Model 2 in Table III indicates that the significant $G \times E$ detected in the full sample remained significant for males in the study.

The results for the female sample are presented in Model 3 of Table III. The pattern of findings that emerged for females indicated several items of note. First, the genetic risk scale failed to significantly predict female antisocial behavior. Corporal punishment, on the other hand, remained a statistically significant predictor of behavioral problems. Model 4 reveals the findings for the test of $G \times E$ in the female subsample; the interaction between genetic risk and corporal punishment failed to reach statistical significance.

In order to present a more interpretable image of the interaction between genetic risk and corporal punishment for males, we adopted a two-stage approach. First, we created a new spanking variable based on the continuous measure of corporal punishment. This new variable included three categories: 0 = no corporal punishment, 1 = low use of corporal punishment, and 2 = high use of corporal punishment. Second, we plotted the effects of the corporal punishment measure across varying levels of genetic risk for males. As seen in Figure 1, the effect of corporal punishment on antisocial behavior functioned based on the measure genetic risk. In other words, as levels of genetic risk increased, so too did the *effect* of corporal punishment on antisocial behavior. Thus, males with the highest levels of genetic risk *and* the greatest exposure to corporal punishment exhibited more antisocial behavior than male children with other combinations of these two factors. When considered with the findings presented earlier, this body of results suggests that genetic factors condition the influence of spanking differently for males than for females. We further discuss the implications of our findings in the section that follows.

TABLE III. OLS Regression Models Predicting Childhood Antisocial Behavior in Split Samples

	Model 1			Model 2			Model 3			Model 4		
	Males			Males			Females			Females		
	B	β	SE	B	β	SE	B	β	SE	B	β	SE
Genetic risk	1.11	.13*	.38	0.30	.03	.45	0.55	.07	.33	0.46	.06	.40
Corporal punishment	0.60	.25*	.15	-0.29	-.12	.30	0.50	.17*	.15	0.40	.02	.13
Low self-control	0.22	.20*	.05	0.22	.20*	.05	0.22	.21*	.05	0.22	.21*	.05
Maternal depression	0.18	.20*	.04	0.20	.20*	.04	0.21	.24*	.04	0.21	.24*	.04
Family adversity	0.11	.10*	.04	0.11	.11*	.03	0.08	.08*	.04	0.10	.10*	.03
Child's race	-0.06	-.01	.45	0.05	.00	.45	-0.04	-.00	.42	-0.03	-.00	.42
Genetic risk X corporal punishment		-		0.80	.41*	.23		-		.11	.05	.25

*Significant at the 0.05-level, two-tailed; Huber/White standard errors are presented.

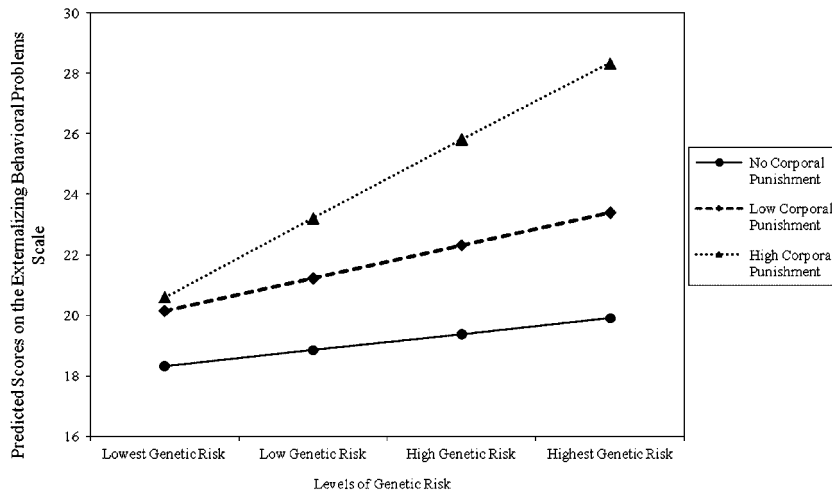


Fig. 1. Evidence of a gene–environment interaction for males.

DISCUSSION

Studies examining the biosocial underpinnings of behavior are continuing to provide evidence that both heritable influences and environmental risk factors may work closely together to condition the emergence of antisocial phenotypes [Kim-Cohen et al., 2006; Taylor and Kim-Cohen, 2007]. Exposure to risky environments, when coupled with a genetic vulnerability, may increase the likelihood that a child will develop conduct problems and persistent antisocial behavior. Although a number of environmental risk factors have been examined, to our knowledge there has been no effort to test whether corporal punishment conditions genetic effects on behavior. This study constructed a latent measure of genetic risk in order to directly test whether spanking interacted with genetic factors to influence the emergence of early childhood antisocial behavior. Our analysis of the ECLS-B produced two

notable findings regarding the interaction of genetic factors and corporal punishment.

First, spanking interacted with the measure of genetic risk in order to influence antisocial behavior. In this case, children scoring higher on the measure of genetic risk, and who were spanked more often, also scored higher on the measure of childhood antisocial behavior. This finding is important because it may offer insight into why some children seem especially vulnerable to the negative influence of spanking. Our findings necessarily suggest that children exposed to a double dose of risk, both genetic and environmental, may be those most at risk for developing antisocial traits [Moffitt, 2005].

The second noteworthy finding concerned the moderating role of gender. Specifically, the significant $G \times E$ observed in the full sample of participants seemed to be isolated to male subjects. When the sample was split by gender, the interaction between genetic risk and spanking failed to reach statistical

significance for females. This finding is not surprising, given the results produced by Paaver et al. [2008]. These researchers detected a significant interaction between genetic factors and family risk in the prediction of female impulsivity. Both the findings presented by Paaver et al. and our current findings suggest that the confluence of environmental and genetic risk factors may operate differently for males and females. Given the somewhat novel nature of this result, it is important to consider the possible interpretation of this finding in greater detail.

Previous evidence has suggested that males, on average, are more vulnerable to the influence of environmental pathogens than are females [Vahter et al., 2007]. Other studies have arrived at very similar results concerning the increased susceptibility of males to deleterious environmental effects [Vaske et al., 2011]. Vaske et al. [2011] produced evidence that the threshold of genetic risk for females, when predicting antisocial behavior, is significantly higher than is observed for males. Vaske et al.'s findings indicate that females may be shielded, to some extent, from the influence of genetic risk factors on antisocial behavior. In contrast, males may require comparatively lower levels of both genetic and environmental risk to reach a threshold for the emergence of antisocial behavior. Along this line of reasoning, it is possible that increased genetic and environmental vulnerability for males was responsible for the differences between samples in the finding of a significant $G \times E$.

Before concluding, it is important to note that this study was not without limitations. The first limitation concerns the presence of a gene-gene interaction ($G \times G$). In other words, it is possible that the interaction between genetic factors and corporal punishment does not represent a pure $G \times E$. Alternatively, the $G \times E$ detected in this analysis may reflect an interaction between unmeasured genetic factors tied to both spanking and antisocial behavior (i.e., a $G \times G$). Put differently, if genetic factors influenced both behavioral problems and corporal punishment (via child-driven effects), then it is possible that our findings reflect an interaction between the genetic risk scale and the underlying genetic factors that influence spanking [Harris, 1998]. We attempted to directly examine this possibility by regressing the corporal punishment scale on the measure of genetic risk and saving the residuals as a new variable. Next, the residualized scores for the corporal punishment variable were used to construct a multiplicative interaction term with the genetic risk scale. The results obtained from these sensitivity models were substantively identical

to those obtained in the original analytical approach. Even so, we cannot definitively rule out the possibility of a $G \times G$. Additional research aimed at replicating our findings will help to determine whether the results of this study are indeed robust.

Second, the findings presented in this study concern the development of childhood antisocial behavior (i.e., behavioral problems emerging before the age of 5). As a result, it remains unclear whether our findings have implications for the development of chronic and severe antisocial behavior (e.g., crime and delinquency) across later points in the life course. Some scholars have suggested, however, that behavioral problems emerging in the first few years of life represent a strong predictor of life course offending [Moffitt, 1993; Olweus, 1979; Robins, 1966]. Consequently, it is possible that our findings help to shed light on the developmental origins of adverse behaviors that will persist over time. At this point, however, any prediction along these lines would be premature.

A third limitation concerns the use of parental reports to measure both childhood antisocial behavior as well as corporal punishment. Because the primary caregiver reported on both the child's behavior and their exposure to corporal punishment, it necessarily raises the possibility that shared methods variance (i.e., correlated errors) may account for a portion of the correlation between the two measures. Future research is needed to explore this issue further. We should point out, however, that Arsenault et al. [2003] explored the issue of shared methods variance in some detail by examining the convergence of several reporting sources for behavioral outcomes in children (e.g., mothers, teachers, independent observers, and child self-reports). Concerning the results of their study, Arsenault et al. [2003; p 842] noted that "the information provided by all four informants (mothers, teachers, examiner-observers, and 5 year old children) is valuable for research uses because the four reports of children's antisocial behaviour included reliable and non-biased information that was agreed upon by all informants."

Ultimately, the pattern of findings produced in this study suggests that the use of corporal punishment may exacerbate genetic vulnerabilities in some children, thereby increasing the risk of antisocial behavior. Moreover, our results suggest that this process may apply most directly to males rather than females. Given the evidence of a "gendered" $G \times E$, the results presented here may call into question prior social scientific explanations posited to explain the origin of gender differences in antisocial behavior. Specifically,

scholars have suggested that females exhibit lower levels of antisocial and offending behaviors as compared with males in part, because of stereotypical gender roles and socialization effects [e.g., Adler, 1975; Chesney-Lind and Pasko, 2004; Hagan et al., 1987, 1979; Simon, 1975]. In contrast, our findings provide evidence that females may be less likely to develop antisocial behavior for a host of reasons, some of which are environmental and some of which are genetic [Vaske et al., 2011].

REFERENCES

- Adler F. 1975. *Sisters in Crime: The Rise of the New Female Criminal*. New York: McGraw Hill.
- Arseneault L, Moffitt TE, Caspi A, Taylor A, Fruhling S, Rijdsdijk V, Jaffee SR, Abow JC, Measelle JR. 2003. Strong genetic effects on cross-situational antisocial behaviour among 5-year-old children according to mothers, teachers, examiner-observers, and twins' self-reports. *J Child Psychol Psychiatry* 44:832–848.
- Beaver KM, DeLisi M, Wright JP, Vaughn MG. 2009. Gene–environment interplay and delinquent involvement: Evidence of direct, indirect, and interactive effects. *J Adolesc Res* 24:147–168.
- Bethel J, Green JL, Nord C, Kalton G, West J. 2005. *Early Childhood Longitudinal Study, Birth Cohort (ECLS-B), Sampling*. Volume 2 of the ECLS-B Methodology Report for the 9-Month Data Collection, 2001–02 (NCES 2005-147). U.S. Department of Education. Washington, DC: National Center for Education Statistics.
- Caspi A, McClay J, Moffitt TE, Mill J, Martin J, Craig IW, Taylor A, Poulton R. 2002. Role of genotype in the cycle of violence in maltreated children. *Science* 297:851–854.
- Chesney-Lind M, Pasko L. 2004. *The Female Offender: Girls, Women and Crime* (2nd ed.). Thousand Oaks: Sage.
- DeGangi GA, Poisson S, Sickel RZ, Wiener AS. 1995. *Infant/Toddler Symptom Checklist*. San Antonio, TX: The Psychological Corporation.
- Gershoff ET. 2002. Corporal punishment by parents and associated child behaviors and experiences: A meta-analytic and theoretical review. *Psychol Bull* 128:539–579.
- Glueck S, Glueck E. 1950. *Unraveling Juvenile Delinquency*. Cambridge, MA: Harvard University Press.
- Haberstick BC, Lessem JM, Hopfer CJ, Smolen A, Ehringer MA, Timberlake D, Hewitt JK. 2005. Monoamine oxidase A (MAOA) and antisocial behaviors in the presence of childhood and adolescent maltreatment. *Am J Med Genet* 135:59–64.
- Hagan J, Simpson J, Gillis AR. 1979. The sexual stratification of social control: A gender-based perspective on crime and delinquency. *Br J Sociol* 30:25–38.
- Hagan J, Simpson J, Gillis AR. 1987. Class in the household: A power-control theory of gender and delinquency. *Am J Sociol* 92:788–816.
- Harris JR. 1998. *The Nurture Assumption: Why Children Turn Out the Way They Do*. New York, NY: The Free Press.
- Jaffee SR, Caspi A, Moffitt TE, Polo-Tomas M, Price TS, Taylor A. 2004. The limits of child effects: Evidence for genetically mediated child effects on corporal punishment but not on physical maltreatment. *Dev Psychol* 40:1047–1058.
- Jaffee SR, Caspi A, Moffitt TE, Dodge KA, Rutter M, Taylor A, Tully L. 2005. Nature x nurture: Genetic vulnerabilities interact with physical maltreatment to promote conduct problems. *Dev Psychopathol* 17:67–84.
- Kim-Cohen J, Moffitt TE, Taylor A, Pawlby S, Caspi A. 2005. Maternal depression and children's antisocial behavior. *Arch Gen Psychiatry* 62:73–181.
- Kim-Cohen J, Caspi A, Taylor A, Williams B, Newcombe R, Craig IW, Moffitt TE. 2006. MAOA, maltreatment, and gene-environment interaction predicting children's mental health: New evidence and a meta-analysis. *Mol Psychiatry* 11:903–913.
- Lansford JE, Criss MM, Laird RD, Shaw DS, Pettit GS, Bates JE, Dodge KA. 2011. Reciprocal relations between parents' physical discipline and children's externalizing behavior during middle childhood and adolescence. *Dev Psychopathol* 23:225–238.
- Lynch SK, Turkheimer E, Slutske WS, D'Onofrio BM, Mendle J, Emery RE. 2006. A genetically informed study of the association between harsh punishment and offspring behavioral problems. *J Fam Psychol* 20:90–198.
- Merrell KW. 2003. *Preschool and Kindergarten Behavior Scales* (2nd ed.). Austin, TX: PRO-ED.
- Moffitt TE. 1993. Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychol Rev* 100:674–701.
- Moffitt TE. 2005. The new look of behavioral genetics in developmental psychopathology: Gene–environment interplay in antisocial behaviors. *Psychol Bull* 131:533–554.
- Moffitt TE, Caspi A, Rutter M. 2006. Measured gene–environment interactions in psychopathology. *Perspect Psychol Sci* 1:5–27.
- Nord C, Edwards B, Hilpert R, Branden L, Andreassen C, Elmore A, Sesay D, Fletcher P, Green J, Saunders R, Dulaney R, Reaney L, Flanagan KD, West J. 2004. *Early Childhood Longitudinal Study, Birth Cohort (ECLS-B), User's Manual for the ECLS-B Nine-Month Restricted-Use Data File and Electronic Code Book (NCES 2004-092)*. U.S. Department of Education. Washington, DC: National Center for Education Statistics.
- Olweus D. 1979. Stability of aggressive reaction patterns in males: A review. *Psychol Bull* 86:852–875.
- Paaver M, Kurrikoff T, Nordquist N, Oreland L, Harro J. 2008. The effect of 5-HTT gene promoter polymorphism on impulsivity depends on family relations in girls. *Prog Neuro-Psychopharmacol Biol Psychiatry* 32:1263–1268.
- Pratt TC, Cullen FT. 2000. The empirical status of Gottfredson and Hirschi's general theory of crime. *Criminology* 38:931–964.
- Radloff LS. 1977. The CES-D: A self-report depression scale for research in the general population. *Appl Psychol Meas* 1:385–401.
- Radloff LS, Teri L. 1986. Use of the CES-D with older adults. *Clin Gerontol* 5:119–136.
- Robins LN. 1966. *Deviant Children Grown Up*. Baltimore, MD: Williams and Wilkins.
- Rutter M, Moffitt TE, Caspi A. 2006. Gene-environment interplay and psychopathology: Multiple varieties but real effects. *J Child Psychol Psychiatry* 47:226–261.
- Simon RJ. 1975. *Women and Crime*. Lexington, MA: Lexington Books.
- Taylor A, Kim-Cohen J. 2007. Meta-analysis of gene–environment interactions in developmental psychopathology. *Dev Psychopathol* 19:1029–1037.
- Taylor CA, Manganello JA, Lee SJ, Rice JC. 2010. Mothers' spanking of 3-year-old children and subsequent risk of children's aggressive behavior. *Pediatrics* 125:1057–1065.
- Tremblay RE, Nagin D, Seguin J, Zoccolillo M, Zelazo P, Bolvin M, Perusse D, Japel C. 2004. Physical aggression during early childhood: Trajectories and predictors. *Pediatrics* 114:43–50.
- Vahter M, Akesson A, Liden C, Ceccatelli S, Berglund M. 2007. Gender differences in the disposition and toxicity of metals. *Environ Res* 104:85–95.
- Vaske J, Wright JP, Boisvert D, Beaver KM. 2011. Gender, genetic risk, and criminal behavior. *Psychiatry Res* 185:376–381.