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**The Effects of Spanking on Psychosocial Outcomes:
Revisiting Genetic and Environmental Covariation**

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Abstract

The current research analyzed data from the Children of the National Longitudinal Survey of Youth (Study 1) and conducted simulation models using parameters from the literature (Study 2) to provide a summative evaluation of the psychosocial effects of spanking with regard to genetic and nonshared environmental covariation. Results of Study 1 replicated previous work showing that associations between spanking and outcomes of delinquency, depression, and alcohol use were explained by moderate-to-large degrees of genetic covariation, and small-to-moderate degrees of nonshared environmental covariation. Simulation estimates from Study 2 suggest that genetic covariation could account for a substantial amount of the phenotypic effect between spanking and psychosocial outcomes ($\approx 60\%$ - 80%), with the remainder likely attributable to nonshared environmental covariation ($\approx 0\%$ - 40%). The results of the current research indicate that continued work on the effects of spanking is best served by behavior genetic research on a broader range of outcomes than what is currently available.

Keywords: spanking; genetic covariation; environmental covariation; simulation modeling; behavioral genetics.

The Effects of Spanking on Psychosocial Outcomes: Revisiting Genetic and Environmental Covariation

The extent to which a child's experiences with a parent or primary caregiver have a lasting impact across the life course has historically been a focal topic for developmental researchers (Durrant & Ensom, 2012). Parental use of spanking, or "hitting a child on their buttocks or extremities using an open-hand" (Gershoff & Grogan-Kaylor, 2016, p. 453) with the intention of modifying undesirable child behavior, in particular, has received substantial empirical attention within the developmental sciences (Gershoff, 2002; Gershoff & Grogan-Kaylor, 2016; Larzelere & Kuhn, 2005; Paolucci & Violato, 2004). ("Spanking," "corporal punishment," and "mild physical punishment" are viewed largely as synonymous. Here, we maintain the use of the term "spanking" for consistency throughout, as defined above.) The use of spanking has been posited to have direct links with internalizing and externalizing problems in childhood and adolescence (e.g., Lansford, Wager, Bates, Pettit, & Dodge, 2012), mental health problems in adulthood (e.g., Afifi et al., 2017), and a range of other adverse psychosocial outcomes across the life course (see Gershoff & Grogan-Kaylor, 2016).

Although substantial empirical effort has been devoted to elucidating the enduring outcomes of spanking children, meta-analyses of this body of work have reached varying conclusions. For example, results differ regarding whether spanking is effective at reducing problem behaviors (e.g., Gershoff, 2002; Larzelere & Kuhn, 2005), and the degree to which spanking is linked with negative psychosocial outcomes (e.g., Ferguson, 2013; Paolucci & Violato, 2004). Gershoff and Grogan-Kaylor (2016) provide the most recent meta-analytic addition to the spanking literature in an effort to address remaining concerns and unclear findings. One primary concern addressed in the 2016 meta-analysis is ensuring that spanking (as

defined above) is not confounded with harsher forms of physical punishment perpetrated against children (e.g., hitting children with objects; abusive behaviors including choking and beating – “physical abuse”). This distinction is important: whereas there is a clear consensus that child *physical abuse* can be detrimental for child development (see Ferguson, 2013; Jaffee et al., 2004; Larzelere & Kuhn, 2005), it is less clear whether controlled spanking is: (1) an effective form of positive punishment to reduce problem behavior (Grogan-Kaylor et al., 2018), and (2) associated with negative psychosocial outcomes throughout development and into adulthood (Cecil, Barker, Jaffee, & Viding, 2012; Lynch et al., 2006).

The results of the Gershoff and Grogan-Kaylor (2016) meta-analysis suggest that spanking is, indeed, associated with wide-ranging negative psychological and behavioral outcomes across development. Psychosocial outcomes resulting from spanking vs. non-spanking, importantly, did not differ as a function of study design (e.g., retrospective, longitudinal), measurement (e.g., observational, child-report, parent-report), or age of children at the time of spanking (ranging from toddlerhood to adolescence). In summary, the results of Gershoff and Grogan-Kaylor align with the general consensus that spanking is consistently associated with poor developmental outcomes, appears to be largely ineffective at reducing problem behavior, and has not been shown to have any positive effects on child development (Durrant & Ensom, 2012; Gershoff, 2013; Grogan-Kaylor et al., 2018).

Despite associations between spanking and adverse psychosocial outcomes (Gershoff & Grogan-Kaylor, 2016), methodological limitations of prior work preclude making firm *causal* inferences. For ethical and practical reasons, research on spanking is largely correlational (retrospective reporting, cross-sectional, longitudinal; more than 90% of studies included in Gershoff & Grogan-Kaylor, 2016) rather than experimental. Like much social science research,

scholars in this area must primarily rely on survey datasets (e.g., retrospective, longitudinal) and statistical techniques (e.g., cross-lagged panel analyses) to parse selection effects from the purported causal effects of spanking (see Gershoff et al., 2018). The standard model of causation in the social sciences (Holland, 1986) is therefore often applied to ethically constrained research topics like spanking. The association between spanking and adverse psychosocial outcomes is generally inferred to be *consistent with* a causal interpretation given that (1) correlational research documents significant, reliable associations between parental use of spanking and adverse psychosocial outcomes across development, (2) spanking precedes (rather than follows) adverse psychosocial outcomes, and (3) alternative, potentially confounding variables are ruled out via statistical controls (see Grogan-Kaylor et al., 2018). Because empirical work has provided support for each of these criteria (see Gershoff et al., 2018), the consensus is that spanking is, at the least, *consistent with* causal interpretations, such that spanking has a direct, negative association on psychosocial outcomes – although an indisputable causal conclusion is nearly impossible to defend (see Gershoff & Grogan-Kaylor, 2016; Lynch et al., 2006).

A substantive criticism of the claim of a direct association between spanking and adverse psychosocial outcomes primarily focuses on criterion 3, in that it is not clear that all relevant confounds have been definitively ruled out. Such criticisms fall under the umbrella of selection effects and omitted variables bias. Although a large proportion of Americans, for example, report using spanking (Gershoff & Grogan-Kaylor, 2016), there remains variation in who spans, how frequently they spank, when spanking is used, and how aggressively spanking is inflicted (see Benjet & Kazdin, 2003). Personality traits, demographic variables, and a range of psychosocial factors influence parenting strategies (including those associated with spanking; e.g., Gershoff & Grogan-Kaylor, 2016). Children also vary on factors that influence parenting strategies and

spanking (Cecil et al., 2012; Larzelere, Kuhn, & Johnson, 2004) – known as evocative child effects (Scarr & McCartney, 1993). Finally, parents provide rearing environments for their children (in addition to their genes), which impact the child's behavior – known as passive child influences (Scarr & McCartney, 1993). These factors can therefore introduce confounds that increase the probability of alternative explanations for the association between spanking and psychosocial outcomes.

When assessing parental effects on child outcomes, in particular, gene-environment correlations can potentially explain seemingly robust associations between parental behavior and child outcomes. Gene-environment correlation refers to the association between heritable phenotypic characteristics and nonrandom exposure to environmental experiences that are associated with that same phenotypic characteristic (Plomin et al., 1977). Put differently, genotypes are non-randomly associated with the environment via manifested behavior and psychological traits (Kandler & Zapko-Willmes, 2017) such that the environment is essentially an extension of an individual's phenotype, which is heavily influenced by its corresponding genotype. When applied to developmental associations within families, the problem of genetic covariation can emerge. Genetic covariation refers to the problem in which two variables that covary at the phenotypic level – such as spanking and psychosocial outcomes – are each heritable and, therefore, potentially covary at the genetic level. The implications being that an association between two variables at the phenotypic level may be a result of a third variable – shared genetic covariation – underpinning the phenotypic traits.

The term “heritable” refers to traits for which a proportion of phenotypic variation is explained by genetic variation (Polderman et al., 2015). Nearly 50 years of behavioral genetics research have provided robust “laws” concerning the interplay of genes and environment in the

development of complex phenotypes (Chabris et al., 2015; Plomin et al., 2016; Polderman et al., 2015; Turkheimer, 2000): (1) That essentially all complex phenotypic traits – including spanking (the receipt and use), mental health problems, self-control, etc. – are to varying degrees heritable; (2) That similarities between biological relatives are largely the result of shared genetic variation; and (3) That complex phenotypic traits are polygenic and pleiotropic, meaning that each complex trait is influenced by many genes, and that each gene is associated with multiple complex traits. An implication of these laws is that, to the extent to which two heritable traits covary at the genetic level, genetic covariation explaining the observed phenotypic association is a plausible (alternative) explanation to purported direct environmental effects of spanking, which must be addressed empirically (Barnes et al., 2014a). Just as researchers include statistical control variables in regression-based analyses to identify unique associations between variables, if known genetic variation underpinning traits of interest is unaccounted for then it is possible that a significant phenotypic association appears despite the fact that the phenotypic association may largely be explained by genetic covariation.

Data produced via standard social science research designs, such as longitudinal designs or retrospective reports, often include assessments from only one child per family and, therefore, cannot account for potential genetic covariation underpinning the traits of interest (Barnes et al., 2014a; 2014b). The only direct way to account for genetic covariation in developmental research is to use a genetically-informative research design, which typically means analyzing data secured from twins or siblings (Plomin et al., 2013). With regard to spanking and consequent psychosocial outcomes, the variables accord with the behavioral genetic laws, such that each are heritable (see, Barnes et al., 2013; Burt, 2009; Polderman et al., 2015), and the traits covary genetically (Button et al., 2008; Jaffee et al., 2004); therefore, genetic covariation is not only

plausible (Barnes et al., 2013), but could explain a large proportion of the observed phenotypic association between spanking and psychosocial outcomes (see, Button et al., 2008; Jaffee et al., 2004).

Genetically-informative research designs capable of identifying shared genetic covariation underpinning spanking and relevant psychosocial outcomes have supported this position. Jaffee and colleagues (2004) used a sample of twins to investigate whether the association between spanking (referred to as “corporal punishment” in Jaffee et al., 2004) and children’s antisocial behavior was explained by genetic covariation between the traits. Jaffee et al. found that genetic covariation between spanking and antisocial behavior accounted for 86% of the observed phenotypic association, whereas the remainder was primarily explained by nonshared environmental covariation. Another twin study conducted by Button and colleagues (2008) found that genetic covariation between parental punitive punishment and children’s externalizing behaviors explained 61% (for maternal punitive punishment) and 98% (for paternal punitive punishment) of the phenotypic association. Together these studies are consistent with the notion that children’s experiences of spanking and behavioral outcomes are largely explained by genetic covariation.

The twin studies by Jaffee et al. (2004) and Button et al. (2008) show that genetic covariation can account for a large proportion of the observed phenotypic effect between spanking-like behaviors and negative child outcomes. There is, however, an important distinction between spanking and harsher forms of parental punishment (see Gershoff & Grogan-Kaylor, 2016). In addition to spanking, Jaffee et al. examined genetic and environmental covariation of the association between harsh parenting (i.e., physical abuse) and children’s antisocial behavior. In contrast to significant genetic covariation found for spanking and antisocial behavior, Jaffee et

al. found minimal genetic influences on children's experiences of physical abuse, but significant and substantial effects of shared environmental variation on children's experiences of physical abuse. Similarly, a study by Lynch and colleagues (2006) using the children-of-twins design investigated effects of mild physical punishment (i.e., spanking) and harsh physical punishment (i.e., physical abuse) on internalizing and externalizing problems, and substance use. The results reported by Lynch et al. show that, after controlling for genetic relatedness, spanking had only slight associations with negative outcomes; in contrast, however, harsh physical punishment did have significant negative effects on outcomes after genetic relatedness was accounted for. Together the results of Jaffee et al. and Lynch et al. show that harsh physical punishment (i.e., physical abuse) does appear to have a direct, environmentally-mediated link with negative outcomes, whereas spanking appears to have primarily a genetically-mediated link with negative outcomes. These results also accord with the conclusion of Gershoff and Gorgon-Kaylor (2016) that proper operationalization of spanking (vs. physical abuse) is necessary to understand the precise effects of spanking on child development.

A longitudinal twin study conducted by Cecil et al. (2012) demonstrates evocative child effects of spanking, which are suggested by the results of the above genetically-informed studies. Using cross-lagged panel analyses, Cecil et al. examined whether harsh punishment (i.e., smacking and shouting) was associated with self-control difficulties from early childhood to adolescence. Results indicated bi-directional effects of harsh punishment and self-control such that both influenced and were influenced by one another; but, interestingly, between seven and 12 years, only evocative effects were found such that self-control difficulties predicted later harsh punishment, but harsh punishment did not predict later self-control difficulties. Cecil et al. did find long-term effects of harsh punishment on early adolescent conduct problems, but for

only boys. These results are consistent with the general notion that children's individual behavior can evoke or exacerbate parental punishment such as spanking, with the unique contribution that the effects of harsh punishment may be particularly relevant in early childhood, as opposed to later childhood and adolescence (Cecil et al., 2012).

Finally, adoption studies provide additional support for the evocative child effects model, such that genetically at-risk children prompt harsher parenting responses – which could subsequently influence negative outcomes (see Cecil et al., 2012). Research by O'Connor and colleagues (1998) provided general support for this evocative model such that genetically at-risk adoptees (i.e., based on biological mother's antisocial behavior scores prior to the birth of their children) were more likely to receive negative parenting from adoptive parents as compared to children at low genetic risk. Subsequent adoption studies accord with these initial findings in adoptive children, and also show that poor environments (Hao & Matsueda, 2006) may exacerbate evocative effects, and that genetically at-risk children appear to react more negatively to harsh parenting than do children at low genetic risk (Riggins-Caspers et al., 2003). Together these studies demonstrate that harsh parenting, such as spanking, can be evoked by problem behaviors in children, and suggest that children at genetic risk for problem behaviors may also react more negatively to parental punishment, possibly exacerbating the effects of negative parenting over development.

Collectively, the above studies are consistent with the conclusion that at least a proportion of the direct links between spanking (but not physical abuse) and negative psychosocial outcomes are due to genetic covariation (Button et al., 2008; Jaffee et al., 2004) via an evocative process whereby children's genetic propensities toward problem behavior prompt parental punishment (O'Connor et al., 1998), which appear to have negative effects particularly

in early and middle childhood (Cecil, et al., 2012). The remaining proportion of the phenotypic association appears to be explained by nonshared environmental covariation (Button et al., 2008; Jaffee et al., 2004), consistent with the notion that spanking may have small negative effects on psychosocial outcomes in children (Gershoff & Grogan-Kaylor, 2016; Lynch et al., 2006), but the enduring, or long-term effects of spanking are less clear (see Cecil et al., 2012). If the magnitude of the enduring effects of spanking are to be accurately estimated, then known genetic covariation needs to be accounted for (see Briley et al., 2019).

The purpose of the current research is provide an initial evaluation of the current state of the spanking literature with regard to genetic and (nonshared) environmental covariation by making use of the most up-to-date meta-analysis by Gershoff and Grogan-Kaylor (2016) assessing the phenotypic associations between spanking and a broad range of psychosocial outcomes across the life course. The available behavior genetic research on spanking includes a limited range of outcomes, such as internalizing and externalizing problems, and antisocial or conduct behavioral problems. In contrast, the broader developmental spanking literature has assessed a far wider range of psychosocial outcomes as a function of spanking: 17 specific psychosocial outcomes and the “overall effect” of spanking in the recent meta-analysis (Gershoff & Grogan-Kaylor, 2016). In an effort to provide an evaluative summary of the literature and groundwork for future behavior genetic research, we provide probable ranges of estimates of the degree to which genetic and nonshared environmental covariation can explain the reported phenotypic effects in Gershoff and Grogan-Kaylor. Toward these aims, we use parameters available from the published literature, in addition to parameters generated from new analyses on data from the Children of the National Longitudinal Survey of Youth (CNLSY). By obtaining heritability estimates and environmentality estimates on spanking and the significant

psychosocial outcomes in Gershoff and Grogan-Kaylor, combined with genetic and nonshared environmental correlations provided by Button et al. (2008), Jaffee et al. (2004), and original analyses reported here from the CNLSY, we are able to provide estimates of genetic and nonshared environmental covariation for all significant phenotypic effects.

Study 1

Study 1 reports the results of new analyses on data from the Children of the National Longitudinal Survey of Youth (CNLSY). The study assesses relative genetic and environmental contributions to the association between maternal reports of spanking and the outcomes of delinquency, depression, and alcohol use, in addition to providing genetic and nonshared environmental correlations between spanking and the above mentioned psychosocial outcomes. The results provide an original contribution to the spanking literature with regard to genetic and nonshared environmental covariation underpinning the associations between spanking and psychosocial outcomes, and these results serve as input parameters for simulation analyses in Study 2.

Method

Sample. Data for the present study are drawn from the Children of the National Longitudinal Survey of Youth (CNLSY) sample. The CNLSY is a sample of youth born to a nationally representative sample of women from the National Longitudinal Survey of Youth 1979 (NLSY79). Children of mothers from the NLSY79 were assessed every two years from 1986 to 2014. Multiple children born to the same mother are included in the CNLSY. As such, many participants are biologically related to one another. Over the past decade, researchers have created a reliable and well-validated linking algorithm capable of identifying biologically related sibling pairs nested within the CNLSY. This algorithm has been used to assign levels of additive

genetic relatedness between siblings in the CNLSY (Rodgers et al., 2016). The present study utilizes this algorithm to identify sibling pairs within the CNLSY in order to conduct a genetically-informative analysis to assess the magnitude of additive genetic and environmental overlap between variation in spanking during childhood and delinquency, depression, and alcohol use in adolescence. The analytic sample consisted of 2,868 respondents. Nested within these 2,868 respondents are 934 full-sibling pairs and 500 half-sibling pairs. See Table SOM1 for descriptive statistics.

Measures. *Spanking:* Mothers in the CNLSY were asked during each assessment wave to report how often their one to 14-year old children were spanked in the past week, focusing on age intervals 4-5, 6-7, and 8-9 because this yielded the most valid data on spanking for children in the CNLSY. Responses ranged from 0 to 18 for ages 4-9 with over 92% of responses ranging from 0 to 10. Values of 11-18 were recoded to have a value of 10 to create consistency in response categories across ages. Frequency of spanking during ages 4-5 was significantly and positively correlated with frequency of spanking at ages 6-7 ($r = .37, p < .001$) and frequency of spanking at ages 6-7 was significantly and positively correlated with frequency of spanking at ages 8-9 ($r = .29, p < .001$). Values across all age intervals were summed to create a cumulative measure of the frequency of spanking for children between ages 4-9. A Blom transformation was used to reduce nonnormality in the measure before behavior genetic analyses was conducted. Descriptive statistics for this measure before transformation are presented in Table SOM1.

Delinquency: From ages 14 to 17, participants were administered a self-report survey which asked them to report on their involvement in seven different types of delinquent behaviors during the past 12 months: 1) hurting someone on purpose; 2) lying to parents; 3) shoplifting; 4)

destroying property at school; 5) skipping school; 6) staying out all night without permission, and 7) having a parent come to school because of their behavior. Response categories were no (0) or yes (1). Items were summed together to create variety indexes of delinquency at ages 14-15 and 16-17. Across ages 14-17 years, the median alpha for the index was .65. Both measures were summed to capture the frequency of self-reported delinquent behavior from ages 14-17. This measure has been used extensively by researchers (Connolly et al., 2015; Harden et al., 2012; Van Hulle et al., 2007) and validated by a previously published report showing that it significantly predicts criminal convictions among youth in the CNLSY sample (Lahey et al., 2006). Similar to the measure of spanking, the created measure for delinquency from ages 14-17 was Blom-transformed to reduce nonnormality before behavior genetic analyses.

Depression: Depression was assessed at ages 14-15 and 16-17 by a 7-item version of the Center for Epidemiological Studies – Depression (CES-D) scale that asked participants how often in the past week they felt the following ways: 1) did not feel like eating; 2) had trouble keeping mind on tasks; 3) felt depressed; 4) felt everything was an effort; 5) sleep was restless; 6) felt sad, and 7) could not “get going”. Response categories for each item were 0 = *rarely, none of the time, 1 day*, 1 = *some, a little of the time, 1-2 days*, 2 = *occasionally, moderate amount of time, 3-4 days*, and 3 = *most, all of the time, 5-7 days*. Values were summed to assess frequency of depressive symptoms at each age interval. The median alpha across ages 14-17 was .88. Scores for respondents at ages 14-15 and 16-17 were summed to create a measure of depressive symptomology from ages 14-17.

Alcohol Use: Alcohol use was measured by asking participants to report, on average, how often in the past 12 months they consumed an alcoholic beverage including beer, wine, or liquor. Response categories were 0 = *did not drink alcohol in the past 12 months*, 1 = *1 to 2 days*, 2 = 3

to 5 days, 3 = every other month or so (6 to 11 days a year), 4 = 1 to 2 times a month (12 to 24 days a year), 5 = several times a month (25 to 51 days a year), 6 = 1 or 2 days a week, 7 = 3 to 6 days a week, and 8 = daily. The average of alcohol use reported during ages 14-15 was 2.68 (3 to 5 days in the past month) and 3.41 (every month or so [6 to 11 days a year]) during ages 16-17. Values were summed to create a measure of average alcohol use from ages 14-17.

Demographic Covariates: To control for confounding effects of family income, race, sex, and sibling-pair age difference, each key variable was regressed on family income (total household income reported by mothers at age 30), race (0 = Black or Hispanic, 1 = Non-Black/Non-Hispanic), sibling-pair age difference (sibling 2 age [in years] – Sibling 1 age [in years]), and sex (0 = female, 1 = male). The standardized residuals from this regression-based analysis were used in subsequent univariate and bivariate quantitative behavioral genetic analyses. The racial composition of the sample was approximately 40% non-Black, non-Hispanic, 38% Black, and 21% Hispanic, and the sex composition was approximately 50% male and 50% female (see Table SOM1).

Analytic Plan. The analysis for the present study was conducted in a series of three interconnected steps. First, phenotypic and within-sibling correlations for spanking, delinquency, depression, and alcohol use were examined. Phenotypic correlations were estimated with standard errors adjusted for non-independence since the data were secured on siblings from the same household (Asparouhov & Muthén, 2006). After inspecting bivariate associations between key variables, within-sibling pair correlations were estimated. Within-sibling pair correlations assess the strength of association between siblings within a pair on a measurable phenotype. If within-sibling pair concordance on a phenotype is stronger for full siblings (who share, on average, 50% of their additive genetic material) than for half-siblings (who share, on average,

25% of their additive genetic material), this can be interpreted as preliminary evidence that the phenotype under examination is under some degree of genetic influence.

Based on results from the phenotypic correlation and within-sibling pair analysis, univariate ACE models were then fitted to the data to partition the observed variation in spanking, delinquency, depression, and alcohol use into latent additive genetic, shared environment, and nonshared environment variance components. The latent additive genetic component (A) represents shared additive genetic variation between siblings, the shared environment component (C) represents family-level environmental experiences that make siblings similar to one another (e.g., culture, household family dynamics, socioeconomic status), and the nonshared environment component (E) represents individual-level environmental experiences unique to each sibling that create differences between siblings (e.g., different parent-child relationships, different peers, different school experiences). The nonshared environment variance component also includes the effects of systematic measurement error. To estimate the magnitude of additive genetic influences, the correlation between the A components was fixed based on the amount of additive genetic material shared between siblings. The correlation between A components for full siblings was fixed to .50 and the correlation for half-siblings was fixed to .25. Because the shared environment reflects all shared experiences between siblings, the correlation between C components was fixed to 1.0; and the correlation between the E components was fixed to 0 because the nonshared environment assumes that siblings share 0% of their unique environment and these experiences are unique to each sibling. The magnitude of additive genetic, shared environment, and nonshared environment effects on an examined phenotype is calculated by comparing the observed cross-sibling correlations to the predicted

cross-sibling correlations generated by the model. Model fit statistics are used to compare alternative biometric structures (AE and CE) to baseline ACE models.

The last step in the analysis involved fitting a series of bivariate Cholesky decomposition models to partition the covariance between spanking, delinquency, depression, and alcohol use into latent additive genetic, shared environment, and nonshared environment components. Figure SOM1 depicts a path diagram for the bivariate Cholesky decomposition model. Bivariate ACE models can provide information on the extent to which common and unique additive genetic and environmental variation explains the total amount of covariance between two phenotypes. The proportion of total variance in spanking due to genetic influences shared with delinquency, depression, and alcohol use was estimated by fitting a series of bivariate Cholesky decomposition models to the data.

All univariate and bivariate models were estimated using *Mplus* 8.1 (Muthén & Muthén, 1998-2018) with full information maximum likelihood estimation and using the WLSMV estimator. Baseline models were compared alongside nested models using values from an adjusted χ^2 difference test (Santorra, 2000), Akaike Information Criteria (AIC), and Bayesian information criteria (BIC). A non-significant change in χ^2 indicates that the nested model with fewer parameters fits the data equally well and is the best-fitting model based on parsimony.

Results

Table SOM2 presents the phenotypic and intra-class correlations for full- and half-sibling pairs. Spanking during childhood was positively and moderately associated with delinquency ($r = .27$, 95% CI: .20-.34), depression ($r = .17$, 95% CI: .11-.28), and alcohol use ($r = .20$, 95% CI: .13-.30) during adolescence. Full-siblings demonstrated stronger concordance on maternal reports of spanking ($r = .21$, 95% CI: .12-.31), adolescent delinquency ($r = .33$, 95% CI: .26-.42),

adolescent depressive symptomology ($r = .30$, 95% CI: .25-.37), and adolescent alcohol use ($r = .27$, 95% CI: .21-.38) compared to half-siblings. Comparisons of same-sex and opposite-sex pairs for full- and half-siblings revealed overlapping confidence intervals suggesting non-significant differences in spanking, delinquency, depression, and alcohol use across same-sex and opposite-sex pairs. Results from univariate sex-limitation ACE models also revealed no evidence of significant sex differences in the magnitude of genetic influence on spanking, delinquency, depression, or alcohol use (results available upon request).

The next step of the analysis focused on decomposing the variance in all key measures into latent additive genetic and environmental components. Table 1 presents the results from all estimated univariate ACE models. The best-fitting model for spanking was a full ACE model, which suggested that 36% of the variation in spanking was due to latent additive genetic factors, 26% of the variation was due to shared environment, and 38% of the variance was due to nonshared environment (including error). Model fit statistics indicated that an AE model provided the best fit to the data for delinquency and suggested that 46% of the variation was due to additive genetic factors, while 54% of the variation was due to nonshared environment. An AE model also provided the best fit for depression suggesting that 40% of the variation in depressive symptomology was attributable to latent additive genetic factors, whereas 60% of the variation was attributable to nonshared environment factors. Lastly, variation in alcohol use was accounted for by genetic and nonshared environmental sources, such that 26% of the variation was attributable to latent additive genetic influences and 74% of the variation was attributable to nonshared environment.

Insert Table 1 About Here

The last step in the analysis focused on identifying genetic and environmental influences on spanking shared with delinquency, depression, and alcohol use. Standardized parameter estimates from all best-fitting bivariate models are presented in Table SOM3 and are used to calculate the genetic correlation (r_g), shared environment correlation (r_c), and nonshared environment correlation (r_e) as well as the proportion of covariance attributable to genes, shared environment, and nonshared environment. The r_g for spanking and delinquency was .54 (95% CI: .40-.68), while the calculated r_e was .07 (95% CI: .03-.14). There was no r_c between spanking and delinquency since constraining the c12 parameter to “0” improved model fit, whereby the AE model was the best fit to the data. As presented in Figure 1, additive genetic covariation accounted for 66% of the phenotypic correlation between spanking and delinquency, and nonshared environment covariation accounted for the remaining 34% of the phenotypic association.

Insert Figure 1 About Here

A r_g of .74 (95% CI: .59-.90) and a r_e of .21 (95% CI: .10-.41) was calculated for spanking and depression. Additive genetic covariation accounted for 48% of the phenotypic correlation between spanking and depression, and nonshared environment covariation accounted for 52% of the phenotypic correlation. With respect to spanking and alcohol use, the r_g and r_e estimates were .81 (95% CI: .62-.92) and .05 (95% CI: .02-.16), respectively. Additive genetic covariation accounted for 61% of the phenotypic correlation, and nonshared environment covariation accounted for 39% of the phenotypic correlation

Study 2

Results of Study 1 indicate that the associations between spanking and psychosocial outcomes are substantially accounted for by both genetic and nonshared environmental

covariation, consistent with previous studies using similar variables on different data sets (Button et al., 2008; Jaffee et al., 2004). A limitation of the genetically-informed analyses on spanking and psychosocial outcomes is the limited range of outcomes assessed. The Gershoff and Grogan-Kaylor (2016) meta-analysis makes clear, however, that the general spanking literature has investigated a broad range of outcomes showing direct associations with spanking. In an attempt to bridge the gap between the broader spanking literature and the behavior genetic spanking literature, Study 2 uses simulation modeling to contribute informed estimations of genetic and nonshared environmental covariation of the 18 phenotypic effects of spanking reported in the 2016 meta-analysis.

Study 2 uses a simulation tool and parameters available in the literature (i.e., heritability estimates, environmentality estimates, genetic correlations, nonshared environmental correlations, and phenotypic correlations) to estimate the degree to which the 18 phenotypic effects of spanking reported by Gershoff and Grogan-Kaylor (2016) may be accounted for by genetic and nonshared environmental covariation. The output of the simulations contribute an evaluative summary of the current state of the literature on developmental outcomes of spanking from a genetically-informed perspective, in complement to the recent, informative meta-analysis by Gershoff and Grogan-Kaylor. The primary contribution of this approach are summative estimates of genetic and nonshared environmental covariation on a much wider range of outcomes (see Gershoff & Grogan-Kaylor, 2016) than has been available previously to researchers (Button et al., 2008; Jaffee et al., 2004; Study 1), which will hopefully motivate continued research on genetic and nonshared environmental covariation on previously unexamined psychosocial outcomes.

Method

The *R* simulation tool developed by Barnes et al. (2017) allows calculations of the degree to which genetic covariation underpinning two traits is likely to explain an observed phenotypic effect. Such genetic covariation calculations have been used previously within the spanking literature (Button et al., 2008; Jaffee et al., 2004), and there are other similar tools available for these types of calculations as well (e.g., Purcell, 2016).

The present analysis uses the Barnes et al. (2017) simulation tool to estimate the degree to which the phenotypic association between spanking and the psychosocial outcomes reported in Gershoff and Grogan-Kaylor (2016) can be accounted for by (1) shared genetic covariation, and (2) nonshared environmental covariation. We do not model the impact of shared environmental covariation given that previous analyses demonstrate very small proportions, if any, of the phenotypic associations are accounted for by shared environmental covariation, with the overwhelming majority of the effect accounted for by genetic covariation and nonshared environmental covariation (Button et al., 2008; Jaffee et al., 2004).

The output of the simulations are (1) the estimated degree to which genetic covariation can account for the observed phenotypic effect, h^2_{cov} , and (2) the estimated degree to which nonshared environmental covariation can account for the observed phenotypic effect, e^2_{cov} . These outputs are the result of solving the equations below, with equation 1 assessing genetic covariation and equation 2 assessing nonshared environmental covariation. Each of these equations requires four pieces of information: (1) the phenotypic effect size (correlation) between the two traits, r_p , (2) the heritability estimate (equation 1) or the nonshared environmental estimate (equation 2) of the predictor variable, h^2_x or e^2_x , respectively, (3) the heritability estimate (equation 1) or the nonshared environmental estimate (equation 2) of the

outcome variable, h^2_y or e^2_y , respectively, and (4) the genetic correlation (equation 1) or the nonshared environmental correlation (equation 2) between the two traits, r_g or r_e , respectively.

$$(1) \quad h^2_{cov} = \frac{\sqrt{h^2_x} * r_g * \sqrt{h^2_y}}{r_p} \qquad (2) \quad e^2_{cov} = \frac{\sqrt{e^2_x} * r_e * \sqrt{e^2_y}}{r_p}$$

The input parameters can be obtained, to varying degrees, from the published literature. First, we had to specify the observed phenotypic effects for the associations between spanking and psychosocial outcomes. For the present analysis, we relied on the meta-analytic effects reported in Gershoff and Grogan-Kaylor's (2016). This meta-analysis provides standardized effect sizes for 17 specific psychosocial outcomes across the lifespan, and also provides an "overall effect" of spanking on psychosocial outcomes, more generally. Table 2 lists all 18 phenotypic effects reported by the Gershoff and Grogan-Kaylor meta-analysis. Of the phenotypic effects provided, 13 were demonstrated to be significant, meaning that exposure to spanking had a statistically significant adverse effect on psychosocial outcomes in childhood or adulthood. Phenotypic effect sizes (d) from Gershoff and Grogan-Kaylor are displayed in Table 2, with significant effects in bold.

We obtained heritability and nonshared environmental estimates for spanking behavior (the predictor variable), and for each of the 18 psychosocial outcome variables assessed by Gershoff and Grogan-Kaylor (2016). For many psychosocial outcomes, we consulted the database of meta-analyzed heritability and environmentality estimates provided by Polderman et al. (2015). Of the 18 psychosocial variables examined in the present research, nine (50%) were included in the Polderman et al. database, and these estimates are provided in Table 2. Meta-

analyzed heritability and environmentality estimates of six (33%) psychosocial outcomes were obtained from Burt (2009). The remaining heritability and environmentality estimates were obtained from individual published studies using academic search engines (e.g., Google Scholar, PsychInfo) to identify behavior genetic analyses of each psychosocial outcome. For the majority of the psychosocial outcomes examined, we identified multiple estimates from the published literature in addition to the estimates presented in meta-analyses. For the predictor variable of spanking, we obtained seven heritability and environmentality estimates from four measures across three separate studies (Barnes et al. 2013; Jaffee et al., 2004; Wade & Kendler, 2000). The specific measurement of each variable from each source is noted in the “Measure” column of Table 2.

Insert Table 2 About Here

Next, we converted the effect sizes from d , as reported in Gershoff and Grogan-Kaylor (2016), to effect sizes of r using a standard d -to- r conversion equation (below), with the results listed in the r column of Table 2.

$$r = \sqrt{\frac{d^2}{4 + d^2}}$$

The final input parameter needed for the simulation tool is the genetic correlation, r_g , or nonshared environmental correlation, r_e , between each variable pair (i.e., spanking and a specific psychosocial outcome). Estimation of these correlations is limited in the literature. Because of

this, Barnes et al. (2017) recommend using a range of plausible, yet conservative values. For the present research we implemented a range of $r_g = 0.30-0.60$ and $r_e = 0.00-0.30$ to be used across each psychosocial outcome based on results of Button et al. (2008), Jaffee et al. (2004), and estimates from Study 1.

Because the input parameters used for these calculations are never “fixed” given that heritability and environmental estimates, and phenotypic effects, can fluctuate across time, space, and sample, the simulation tool (Barnes et al., 2017) allows for constructing beta-distributions for the input values of r_p , h^2_x , h^2_y , e^2_x , and e^2_y . (Barnes et al. provide formal mathematical details for building the beta-distributions to use the simulation tool.) The beta-distributions allow for natural range and unspecified error (including measurement error, sampling bias) to be taken into account when calculating h^2_{cov} and e^2_{cov} . The beta-distributions for r_p s are approximately centered on the meta-analyzed effect size reported by Gershoff and Grogan-Kaylor (2016). The beta-distributions for the heritability and environmentality inputs, which account for variation of estimates around the simple mean, are approximately centered on the simple arithmetic means for the heritability and environmentality estimates of each variable (provided in the $M h^2$ and $M e^2$ column of Table 2). The beta-distributions for the overall effect of spanking are shown in Figure SOM2 and SOM3, and beta-distributions for the 17 remaining specific outcomes are provided and in Figure SOM4a-m and Figure SOM5a-m.

When solving for h^2_{cov} or e^2_{cov} at a specified value of r_g or r_e , respectively, 10,000 calculations are computed. Each calculation draws a random value of r_p , h^2_x , and h^2_y , from the respective beta-distributions for the genetic covariation simulation, and a random value of r_p , e^2_x , and e^2_y , from the respective beta-distributions for the nonshared environmental covariation simulation. Put differently, 10,000 calculations are computed at each value of r_g at intervals of

0.01 beginning with $r_g = 0.30$ through $r_g = 0.60$, and at each value of r_e at intervals of 0.01 beginning with $r_e = 0.00$ through $r_e = 0.30$. Therefore, in the present analysis, 310 thousand calculations are computed for each phenotypic effect, resulting in more than 11 million calculations in total with this tool. The output, described below, is the mean value and 95% credible interval of the 10,000 h^2_{cov} and e^2_{cov} estimates at each value of r_g or r_e , respectively.

Results

Shared genetic covariation. The simulation output collectively suggests that the phenotypic effects of spanking on psychosocial outcomes are likely to be explained substantially by shared genetic covariation underpinning both traits (see Figure SOM6a-k for figures corresponding to each outcome variable). For all psychosocial outcomes except one (discussed below), average estimates of genetic covariation were greater than 50%, given the specified phenotypic effect (see Table 2 across the range of genetic correlations indicated by previous research and the results of Study 1, $r_g = .30 - .60$.) Examining the model output for the Overall Effect of spanking, it can be observed that at the given effect size ($d = .33$, $r_p = .16$), nearly all of the phenotypic covariation will likely be accounted for by shared genetic covariation between spanking and the trait of interest once genetic correlations become large in size, $r_g > .50$ (see Figure 2). There were traits for which the simulation output suggested that genetic covariation could potentially explain the entire effect – meaning that the mean h^2_{cov} and associated 95% credible interval were all equal to 1. These traits, Impaired Cognitive Ability and Self-Esteem, had very small phenotypic effect sizes, $r_p < .1$.

The one exception to the overall trend of substantial genetic covariation explaining the phenotypic effect was the outcome Victim of Physical Abuse. This outcome showed, overall, low explanation by genetic covariation, unlike the rest of the variables assessed, such that even at

the high end of the genetic correlation range ($r_g = .60$), genetic covariation was estimated at only approximately 20%.

Insert Figure 2 About Here

Non-shared environmental covariation. The simulation output collectively suggests that the phenotypic effects of spanking on psychosocial outcomes are likely to be explained by small-to-moderate degrees of nonshared environmental covariation underpinning both traits of interest (see Figure SOM7a-m for figures corresponding to each outcome variable). For most significant psychosocial outcomes except one (discussed below), average estimates of nonshared environmental covariation ranged from 0%-40%, with some outcomes showing nonshared environmental covariation of 80% or greater, given the specified phenotypic effect (see Table 2) across the range of nonshared environmental correlations indicated by previous research and the results of Study 1, $r_e = .00-.30$. Examining the model output for the Overall Effect of spanking, in particular, it can be observed that at the given effect size ($d = .33$, $r_p = .16$), nonshared environmental covariation may explain small-to-moderate proportions of the phenotypic effect between spanking and psychosocial outcomes. The simulation output yielded very high estimates of nonshared environmental covariation for Self-Esteem, which had a very small phenotypic effect size in the Gershoff and Grogan-Kaylor (2016) meta-analysis – meaning that the mean e^2_{cov} and associated upper-bound 95% credible interval were equal to 1 at the upper end of the r_e range assessed here (approaching $r_e = .30$; see Figure 2).

The one exception to the overall trend of moderate nonshared environmental covariation explaining the phenotypic effect was, again, the outcome Victim of Physical Abuse. This

outcome showed, overall, low explanation by nonshared environmental covariation, unlike the rest of the variables assessed, such that even at the high end of the nonshared environmental correlation range ($r_e = .30$), nonshared environmental covariation was estimated at only approximately 10%.

General Discussion

The current research analyzed sibling data from the Children of the National Longitudinal Survey of Youth (CNLSY; Study 1) and conducted simulation models using input parameters from the existing literature (Study 2) to provide a summative evaluation of genetic and nonshared environmental covariation explaining the associations between spanking and psychosocial outcomes. Study 1 found moderate effects of spanking on outcomes of adolescent delinquency, depression, and alcohol use, with results suggesting a moderate-to-large degree of genetic covariation accounting for the associations, and a small-to-moderate degree of nonshared environmental covariation accounting for the associations. Study 2 estimated plausible ranges of genetic and nonshared environmental covariation on a broad range of psychosocial outcomes of spanking reported in a meta-analysis by Gershoff and Grogan-Kaylor (2016). Broadly, the simulations suggest that the effects of spanking on psychosocial outcomes are likely to be explained largely by genetic covariation, with the remainder attributable to nonshared environmental covariation.

The results of the analyses on the CNLSY data and the simulations are consistent with results by Jaffee et al. (2004) and Button et al. We find moderate effects of spanking on delinquent behavior, such that the majority of the phenotypic effect was attributable to genetic covariation, and the remainder attributable to nonshared environmental covariation. The simulation models using the most up-to-date effect sizes of the effects of spanking on a wide

range of outcomes provides a more comprehensive understanding of the negative effects of spanking. In the context of the broader behavior genetic literature on spanking, it can reasonably be concluded that genetic covariation explains a non-trivial proportion of the covariance between spanking and psychosocial outcomes, with the remaining proportion accounted for by nonshared environmental covariation.

This pattern of results is largely due to larger observed genetic correlations between spanking and psychosocial outcomes as compared to the smaller observed nonshared environmental correlations between the traits (see Study 1; see also, Button et al., 2008; Jaffee et al., 2004). The overall findings produce two key points. First, it is plausible to suggest that spanking, in some cases, may exert a negative impact, and certainly not a positive impact, on psychosocial development across the life course. Second, continued investigation of the developmental outcomes of spanking must increase its use of behavior genetic research designs to provide the accurate effect sizes of spanking on psychosocial outcomes.

The one exception to this general conclusion is the association between spanking and physical abuse. Behavioral genetic research has clearly demonstrated that (1) spanking and physical abuse show non-trivial shared environmental effects (Jaffee et al., 2004; Study 1), (2) physical abuse has clear, negative psychosocial outcomes (Lynch et al., 2006), and (3) the association between spanking and physical abuse is primarily explained by *shared* environmental covariation (Jaffee et al., 2004). Our simulation output yields very small estimates of genetic ($\approx 20\%$) and nonshared environmental covariation ($\approx 10\%$), suggesting that *shared* environmental covariation largely explains the phenotypic effect, consistent with previous work (Jaffee et al., 2004; Lynch et al., 2006).

Research Implications and Future Directions

The evidence of genetic covariation explaining a substantial proportion of the associations between spanking and psychosocial outcomes (with the exception of physical abuse), has implications for the magnitude of the reported effects of spanking on development. Overall, the pattern of results suggesting moderate-to-large degrees of genetic covariation imply that once genetic variation is considered, the environmentally-mediated effects of spanking on psychosocial outcomes will be smaller in magnitude than what non-genetically-informed studies suggest (see also, Lynch et al. 2006). The meta-analyzed effect sizes of spanking on psychosocial outcomes using behavior genetic designs will likely be smaller in magnitude than those reported in Gershoff and Grogan-Kaylor (2016).

That the true causal effects of spanking are likely smaller in magnitude than what the reported literature suggests is not at all to say that the negative effects of spanking on child development are nil. The extant research and the data reported here strongly suggests that non-shared environmental covariation explains a non-trivial proportion of the phenotypic effect – a finding that is, in fact, consistent with *causal* interpretations of the negative effects of spanking (see Turkheimer & Harden, 2013).

The simulation models highlight a general disconnect in the breadth and scope of the general spanking literature and the behavior genetic spanking literature. A limited range of psychosocial outcomes has been assessed using behavior genetic data (Button et al., 2008; Cecil et al., 2012; Jaffee et al., 2004; Lynch et al., 2006; O'Connor et al., 1998; Riggins-Casperson et al., 2003) in comparison to the wide range of outcomes assessed in spanking meta-analyses using standard social science approaches (Barnes et al., 2014a; Gershoff & Grogan-Kaylor, 2016). For example, psychosocial outcomes such as moral internalization, negative parent-child relationship, cognitive impairment, self-esteem, and adult support for physical punishment have

thus far not been subject to behavior genetic analyses capable of controlling for shared genetic variation. The output of our simulations for these unstudied variables are consistent with the pattern of findings on the commonly studied outcomes of spanking such as externalizing behaviors and conduct problems. Given the consistency in the pattern of results, it is reasonable to predict that genetic covariation will also account for a substantial proportion of the phenotypic effect on outcomes such as moral internalization, negative parent-child relationship, cognitive impairment, self-esteem, and adult support for physical punishment. A primary contribution of the simulation models is to demonstrate the plausibility and estimated magnitude of genetic covariation in a broader range of psychosocial outcomes.

As the genetically-informed literature examining spanking and various psychosocial outcomes continues to grow, the logical next step is for a meta-analysis of effect sizes to be conducted in which genetic covariation is included as a moderator. The non-genetically informative literature does, in fact, show wide ranging negative effects of spanking on psychosocial outcomes (Gershoff & Grogan-Kaylor, 2016), yet the behavior genetic literature is not yet mature enough to support a meta-analysis. Here, we have contributed additional behavior genetic data (see Study 1) in an explicit effort to contribute to the dearth of currently available studies. Our goal with the inclusion of the simulation analyses in Study 2 is to demonstrate the need for continued behavior genetic work on spanking and child development. Continued contribution to the literature will allow new meta-analyses to be conducted to more accurately discern the magnitude of the causal effect of spanking on psychosocial outcomes. We hope researchers will answer this call for research.

The spanking literature would also benefit from more rigorous longitudinal designs capable of investigating evocative child effects (e.g., Cecil et al., 2012). The spanking literature

is largely built on cross-sectional and retrospective studies (Gershoff & Grogan-Kaylor, 2016). Available longitudinal work (Cecil et al., 2012) and adoption studies (Hao & Matsueda, 2006; O'Connor et al., 1998; Riggins-Casperson et al., 2003) clearly indicate the bi-directional and evocative nature of spanking and psychosocial outcomes. Although the age at which spanking is received does not appear to impact the magnitude of associations between spanking and psychosocial outcomes (Gershoff & Grogan-Kaylor, 2016), evidence from Cecil et al. (2012) seems to indicate that enduring effects of spanking may be smaller relative to effects in childhood – however, more longitudinal studies are needed to clarify the role of age and enduring effects. Longitudinal behavior genetic studies assessing a wide-range of outcomes is an appropriate path forward for understanding the nature of spanking and its consequent effects (see also, Gershoff et al., 2018).

Limitations

The primary limitation of the current research is the boundary conditions within which the simulation output must be interpreted. As with all simulations, the output relies on the accuracy of the inputs. One way in which we worked to be as inclusive as possible with regard to variation of input parameters was to build and sample from beta-distributions (see Figures 3 and 4, for example), rather than using only a single input value for each trait. The simulation output is calculated from thousands of random values drawn from each distribution, which allow for natural variation in each value due to error, time, space, and sample (Barnes et al., 2017). The beta-distributions themselves were also largely built from values obtained from meta-analyses (Burt, 2009; Polderman et al., 2015), thus providing the most accurate values currently available in the literature.

The most important note with regard to interpreting the simulation output is that the output is most informative within the range of the phenotypic effect (r_p = association between spanking and outcome) specified in the beta-distribution, which is centered on the meta-analyzed effect sizes from Gershoff and Grogan-Kaylor (2016). Studies yielding larger phenotypic effect sizes will yield smaller genetic covariation at the same r_g values used here. Larger phenotypic effect sizes require correspondingly larger r_g to explain similar proportions of the phenotypic effect sizes (Barnes et al., 2014a).

Our interpretations of the extent to which the phenotypic effects may be explained by shared genetic covariation assume that the true genetic correlation falls within the range specified in our models, which is .30-.60. This range was chosen based on the only three available studies for which genetic correlations have been calculated between spanking and some of the psychosocial outcomes assessed by Gershoff and Grogan-Kaylor (including Study 1, here). If the true genetic correlation between any two phenotypes (e.g., spanking and moral internalization) is smaller than what we have specified, then the extent to which genetic covariation can explain the phenotypic effect will be reduced. The extent to which the phenotypic effects reported in Gershoff and Grogan-Kaylor would be explained by genetic covariation if the genetic correlation were assumed to be smaller, $r_g = .00-.25$, are provided in Table SOM3a-f)

With regard to Study 1, a limitation is the use of full siblings and half siblings, rather than a more complete quantitative genetic design employing MZ and DZ twins. On one hand, we were limited by the nature of the dataset, as only a handful of twin pairs with available data on the variables of interest were included in the sample. Nonetheless, this characteristic of the data introduces the possibility of bias in the calculation of our genetic correlations. The two primary

sources of bias in quantitative genetic research involves failure to satisfy the equal environments assumption (EEA; Bhattacharjee & Sarkar, 2017) and the assumption of random mating (for the phenotypes being examined) (Barnes et al., 2014). For humans, an assumption of random mating seems warranted rarely, if ever, and thus results in the possibility of obtaining downwardly biased heritability coefficients. The EEA, when violated, functions to artificially inflate heritability coefficients. As a recent in-depth analysis suggested, however, even intentional violations of *both* the EEA and random mating assumptions resulted in relatively minimal bias in the parameter estimates obtained using twin designs (Barnes et al., 2014). Even *if* the nature of analyzing full siblings and half siblings resulted in a higher likelihood of violating one, or both, assumptions, the results would remain substantively unchanged.

Conclusions

A significant body of work documents associations of spanking with various negative psychosocial outcomes (Gershoff & Grogan-Kaylor, 2016). A comparatively limited subset of this work, however, uses genetically-informative designs that allow for examination of genetic and environmental covariation underpinning known phenotypic effects (Button et al., 2008; Cecil et al., 2012; Jaffee et al., 2004; Lynch et al., 2006; O'Connor et al., 1998; Riggins-Caspers et al., 2003). Results of Study 1 replicate previous work (e.g., Jaffee et al., 2004) documenting associations between spanking and delinquency, depression, and alcohol use, and finds that these associations are explained by moderate-to-large degrees of genetic covariation and small-to-moderate degrees of nonshared environmental covariation. Study 2 used a simulation modeling procedure (Barnes et al., 2017) to provide estimates of genetic and nonshared environmental covariation underpinning recent meta-analyzed phenotypic effects of spanking across a plausible range of genetic and nonshared environmental correlations. The simulation output estimates that,

in most cases, genetic covariation will likely account for the majority of the observed phenotypic effect between spanking and the psychosocial outcome of interest (plausibly, in most cases, between 60%-80%), with the remainder attributable to nonshared environmental covariation (plausibly, in most cases, upwards of 40%). The findings of the current research indicate that continued investigation of the developmental consequences of spankings would be best served by focusing on behavior genetic research designs assessing a wide range of psychosocial outcomes to comprehensively understand the underlying nature of spanking on human development.

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Table 1. Standardized Portions of Variance from Univariate ACE Models

Model	A	C	E	AIC	BIC	$\Delta\chi^2$	Δdf	<i>p</i>
Spanking								
ACE	.36 (.08)	.26 (.07)	.38 (.03)	2377.241	2397.641	-	-	-
AE	.49 (.10)	.00 (.00)	.51 (.03)	2379.846	2403.721	5.662	1	.026
CE	.00 (.00)	.32 (.12)	.68 (.03)	2378.241	2403.325	14.503	1	<.001
Delinquency								
ACE	.29 (.08)	.15 (.09)	.56 (.03)	20618.11	20720.79	-	-	-
AE	.46 (.07)	.00 (.00)	.54 (.02)	20618.09	20707.88	5.893	1	.17
CE	.00 (.00)	.10 (.08)	.90 (.02)	20619.03	20725.62	12.375	1	<.001
Depression								
ACE	.38 (.06)	.02 (.02)	.60 (.03)	22437.57	22540.65	-	-	-
AE	.40 (.05)	.00 (.00)	.60 (.03)	22434.86	22525.05	5.608	1	.13
CE	.00 (.00)	.05 (.04)	.95 (.04)	22436.10	22534.17	10.484	1	<.001
Alcohol Use								
ACE	.21 (.07)	.08 (.06)	.71 (.02)	20638.71	20741.31	-	-	-
AE	.26 (.05)	.00 (.00)	.74 (.02)	20635.71	20738.31	8.243	1	.25
CE	.00 (.00)	.12 (.08)	.88 (.03)	20637.37	20739.15	10.675	1	<.001

Notes: Model estimator = maximum likelihood with robust standard errors. AIC = Akaike information criteria. BIC = Bayesian information criteria. *Df* = degrees of freedom. $\Delta\chi^2$ = Santorra-Bentler scaled change in model chi-squared. Standardized parameter estimates presented. Standard errors in parentheses. Results control for race and sex. Best fitting model bolded.

Figure 1. Latent Additive Genetic and Nonshared Environment Overlap between Spanking and Phenotypes

Notes: Proportions of covariance are calculated from coefficient estimates reported in Table SOM3. Estimates control for race and sex.

Table 2. Summary of Data Used for Simulation-Modeling Input

Outcome Variable	<i>d</i>	<i>r</i>	<i>h</i> ²	<i>M h</i> ²	<i>e</i> ²	<i>M e</i> ²	Measure	Source
Low moral internalization	.38	.19	.31	.47	.42	.32	Meta-analysis—Social values	Polderman et al. (2015)
			.37, .26, .30, .47, .51, .51, .52, .60, .62, .72,		.20, .30, .40, .31, .39, .28, .24, .32, .31, .38		Revised Rutter Parent Scale for Preschool Children—Prosocial Behavior (ages 2, 3, 4, 7)	Knafo et al. (2006)
Child aggression	.37	.18	.47, .48, .44, .40	.49	.25, .28, .27, .25	.26	Devereux Child Behavior rating scale (Aggressive behavior subscale)	van Beijsterveldt et al. (2004)
Child antisocial behavior	.39	.19	.73	.66	.27	.28	Achenback family of instruments & DSM-V	Jaffee et al. (2004)
			.58		.28		Meta-analysis—Conduct problems	Burt (2009)
Child externalizing behavior problems	.41	.20	.70, .78	.63	.30, .22	.22	Preschool & Kindergarten Behavior Scale (2ed; 7-itmes)—Parent reports	Barnes et al. (2013)
			.42, .82		.27, .18		Preschool & Kindergarten Behavior Scale (2ed; 6-itmes)—Teacher reports	
			.52, .56		.16, .14		The Child Behavior Checklist (external problem items only)	van der Valk et al. (2002)
			.59		.26		Meta-analysis—Externalizing	Burt (2009)
Child internalizing behavior problems	.24	.12	.35, .38	.41	.30, .32	.32	The Child Behavior Checklist (internal problem items only)	van der Valk et al. (2002)
			.51		.33		Meta-analysis—Internalizing	Burt (2009)
Child mental health problems	.53	.26	.20, .23	.36	.53, .57	.45	Center for Epidemiological Studies Depression Scale & Zung Anxiety Scale (10-12 years)	Zheng et al. (2016)
			.44		.42		Meta-analysis—Depression	Burt (2009)
			.48		.40		Meta-analysis—Anxiety	Burt (2009)
			.44		.36		Meta-analysis—Emotional disorders with childhood onset	Polderman et al. (2015)
	.51	.25	.19	.25	.62	.50	Meta-analysis—Family relationships	

Negative parent-child relationship			.30		.37		Meta-analysis—Problems related to upbringing	Polderman et al. (2015)
Impaired cognitive ability	.17	.08	.51	.51	.25	.25	Meta-analysis—Higher-order mental functions	Polderman et al. (2015)
Low self-esteem	.15	.07	.36, .40	.37	.60, .64	.62	Rosenberg Self-Esteem Inventory	Roy et al. (1995)
			.29, .32		.66, .71			Kendler et al. (1998)
			.47		.51			Neiss et al. (2006)
Victim of physical abuse	.64	.30	.07	.07	.05	.05	Parent interview	Jaffee et al. (2004)
Adult antisocial behavior	.36	.18	.47	.48	.36	.35	Meta-analysis—Mental & behavioral disorders	Polderman et al. (2015)
			.49		.33		Meta-analysis—Conduct disorders	
Adult mental health problems	.24	.12	.47	.49	.36	.41	Meta-analysis—Mental & behavioral disorders	Polderman et al. (2015)
			.61		.31		Meta-analysis—Mood disorders	
			.38		.55		Meta-analysis—Dissocial personality disorder	
Adult support for physical punishment	.38	.19	.31	.31	.50	.50	Meta-analysis—Attitudes	Polderman et al. (2015)
Overall effect size	.33	.16	.45	.45	.40	.40	Meta-Analysis—Mental functions (Big-5 Personality and IQ)	Polderman et al. (2015)
Predictor Variable			h^2	$M h^2$	e^2	$M e^2$	Measure	Source
Spanking			.25	.31	.10	.19	Parent interview	Jaffee et al. (2004)
			.37		.39		Child reports of parent spanking	Wade et al. (2000)
			.16		.21		Parent reports of spanking	
			.27, .50		.07, .18		Parent report of spanking frequency	Barnes et al. (2013)

Figure 2. Top: Mean h^2_{cov} (black, solid line) with 95% Credibility Intervals (gray, dashed lines) for Overall Effect of Spanking on Child and Adult Outcomes at Different Values of r_g Assuming Effect Size of $d = .33$, $r_p = .16$. Bottom: Mean e^2_{cov} (black, solid line) with 95% Credibility Intervals (gray, dashed lines) for Overall Effect of Spanking on Child and Adult Outcomes at Different values of r_e Assuming Effect Size of $d = .33$, $r_p = .16$.

SUPPLEMENTAL MATERIAL

[Supplemental Online Material – For Review](#)

[Supplemental Online Material – Unreviewed](#)