Parenting and polygenic scores in disruptive child behavior:

A test of gene-environment correlations

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We thank the families participating in this research. The data necessary to reproduce the analyses presented here are not publicly accessible. Analytic code and materials necessary to reproduce the analyses presented in this paper are not publicly accessible. All data, code and materials are available from the first author. The analyses presented here were not preregistered.

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Abstract

This study examined gene-environment (parenting) correlations as underlying mechanisms for disruptive child behavior. Polygenic scores for disruptive and externalizing behavior (PGS-DB and PGS-EXT) and parent-reported harsh and warm-supportive parenting were measured in 288 Dutch families with children (M_{age} = 6.26, *SD* = 1.31, 48% girls) with above-average parent-reported disruptive behavior. Harsh, but not warm-supportive, parenting and children's PGS-DB were associated with disruptive child behavior (β = 0.22), but no evidence emerged for gene-environment correlations or genetic nurture. However, harsh parenting was found to partially mediate the link between parental PGS-EXT and disruptive child behavior (β = 0.04). These findings suggest that when using developmentally appropriate PGS, genetic nurture can be identified as a relevant mechanism underlying disruptive child behavior.

Keywords: genetic nurture, externalizing behavior, harsh parenting, gene-environment correlation, supportive parenting, polygenic score

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The onset of disruptive behavior in childhood increases the risk of behavior problems later in life, including antisocial and criminal behavior (Campbell et al., 2000; Costello et al., 2006). In childhood, disruptive behavior is marked by defiance to authority, angry or irritable moods, disobedience, and verbal or physical aggression towards others (DSM-V; American Psychiatric Association, 2013). Untreated disruptive behavior often intensifies over time and has effects that last into adulthood, creating a financial and personal burden for individuals, their families, and society (Mesman et al., 2001; Scott et al., 2001; Rissanen et al., 2021; Rivenbark et al. 2017). Parents play an important role in the development of children's disruptive behavior (for a meta-analysis see Pinquart, 2017). For instance, parental warmth and harshness were found to be related to lower and higher levels of disruptive behavior in children, respectively (Pinquart, 2017). Importantly, higher levels of disruptive behavior may further elicit harsher parenting, leading to a cycle to coercive parent-child interactions that further increase disruptive behavior (Patterson,1982, 2002; Pettit & Arsiwalla, 2008).

Traditionally, developmental psychologists viewed parenting as a purely environmental construct (e.g., Kiff et al., 2011). However, behavioral genetic research, which burgeoned from the '80s onwards, suggests otherwise. Studies with genetically sensitive designs showed that individual differences in parenting are at least partially a function of genetic differences between persons (Wertz et al., 2019; Wertz et al., 2020; Runze et al., 2023; Klahr & Burt, 2014). Moreover, parental genes and their children's genes might be directly associated with the environment, a phenomenon known as *gene-environment correlation* (Plomin et al., 1977; Scarr & McCartney, 1983), and, by extension, explain elevated risk for disruptive child behavior.

One type of gene-environment correlation is *passive gene-environment correlation* (e.g., Scarr & McCartney, 1983). Parents may inherit genes that contribute to their own disruptive behavior, which may in turn lead them to create a harsh rearing environment for their children. Another type of gene-environment correlation is *evocative gene-environment correlation*. This occurs when an individual's genetically influenced behavior elicits a specific reaction from others in the environment (e.g., Burt, 2008). For instance, parents may use harsher discipline when their child has a higher genetic predisposition for disruptive behavior. When parental genes influence child outcomes via their parenting behavior over and above the effect of the child's genes, this is called *genetic nurture* (e.g., Kong et al., 2018). For example, parents with a higher genetic propensity for disruptive behavior may be more likely to manifest harsh and less supportive and warm parenting, leading to increases in disruptive behavior in their children (Jaffee et al., 2006).

In the last years, a number of first studies have investigated both parental and child genetics in the association between parenting and externalizing child behavior—a broader construct encompassing disruptive child behavior along with attention deficits. In childhood and adolescence, children's polygenic scores (PGS) of externalizing behaviors (and, in one study, aggression) predicted externalizing behavior (Kretschmer et al., 2022; Kuo et al., 2022; Teeuw et al., 2023; Luo et al., 2021) which is an indication of direct genetic effects on externalizing behavior. In a prospective-longitudinal study on more than 2,000 Dutch adolescents, followed between the ages of 11 and 29, Kretschmer et al. (2022) did not find a significant association between parental PGS of externalizing behavior and family dysfunction. Moreover, in terms of genetic nurture, Kretschmer et al. (2022) found that parents' PGS were not associated with adolescents' externalizing behaviors, while controlling for child PGS. However, they did find that adolescents' PGS were associated with more family dysfunction via adolescents' externalizing behavior—evidence for an evocative gene-

environment correlation. Kuo et al. (2022) studied an at-risk sample of around 1,000 US families with adolescents between 12 and 17 years old and found a significant association between parental PGS of externalizing behavior and lower parent-child closeness, but not parental involvement. Also, their study showed that parents' PGS *did* predict adolescent externalizing behavior. Notably, both the Kuo et al. (2022) and Kretschmer et al. (2022) studies focused on adolescents only; there is a lack of knowledge about the role of gene-environment correlations in families with younger children.

The Present Study

In the present study, we investigated the association between (1) genetic variants associated with disruptive behavior in parents and the parenting they provide (*passive* GxE correlation) and (2) genetic variants associated with disruptive behavior in children and the parenting they experience (*evocative* GxE correlation). Also, we investigated *genetic nurture,* in which parental genotype predicts children's disruptive behavior over and above genetic transmission, via parenting behavior that in turn predicts children's disruptive behavior.

Our polygenic score of disruptive behavior (PGS-DB) was based on a recent GWAS of 13 externalizing phenotypes (Baselmans et al., 2022). We selected the meta-analyzed phenotype GWAS data for disruptive behavior including summary statistics of six phenotypes; aggression, angry outbursts, extreme irritability, irritability, irritable for two days, and attention deficit/hyperactivity disorder (ADHD), to achieve a close match between polygenic score and phenotype. Genetic data of disruptive behavior from this GWAS consists of an effective sample size of 523,150 participants of several ages, ranging from early to middle childhood to middle and old age. To better compare our results to previous research, as supplementary analysis, we also computed a polygenic score of externalizing behaviors based on a GWAS that covered the following seven phenotypes: attention-deficit/hyperactivity disorder, problematic alcohol use, lifetime cannabis use, reverse-coded age at first sexual intercourse, number of sexual partners, general risk tolerance and lifetime

smoking initiation (Karlsson Linnér et al., 2021). This polygenic score has been used in previous research (e.g., Kretschmer et al., 2022) but encompasses broader phenotypes which are not yet seen in young children.

We expected to find (1) a correlation between parenting behavior (i.e., harsh and warm-supportive) and disruptive child behavior, (2) gene-environment correlations indicated by significant association between parents' PGS-DB and the parenting they provide (passive) and from children's PGS-DB to the parenting they receive (evocative), (3) genetic nurture, indicated by significant direct association between parents' PGS-DB and disruptive child behavior—while controlling for genetic transmission from parent to child (3a), or the link between parents' PGS-DB and their children's disruptive behavior environmentally mediated by parenting (3b).

Methods

Participants

This study used baseline data of the larger (*masked for review*) project (*masked for review*), a randomized controlled trial (RCT) of the Incredible Years parenting program. Others have described the intervention and its effects in full (Chhangur & Weeland et al., 2012; Overbeek, et al., 2021; Weeland & Chhangur et al., 2017). The original RCT was preregistered with the Netherlands Trial Register (*masked for review*) and was approved by a relevant medical-ethical review board (*masked for review*). Families were recruited in two cohorts for logistical reasons (September-October 2012 and 2013) based on a screening for above average disruptive behavior (>75th percentile on the Eyberg Child Behavior Inventory, ECBI, Eyberg & Pincus, 1999; Weeland et al., 2018).). Exclusion criteria were intellectual disability of the parent and/or child (IQ ≤ 70) and not mastering the Dutch language. Children with a diagnosis of conduct-related or other psychological disorder were not included. Half of the parents were randomly assigned to receive the intervention, however intervention effects

did not bias the current analysis which was based on pretest data. Out of the 387 families in the original sample, 296 (77%) parents agreed to provide saliva samples at 2.5 years follow-up for DNA extraction, and a total of 288 samples of parents and children passed quality controls (see Figure S1). Parents (92% mothers) were between 27.07 and 49.27 years old (M = 38.10, SD = 4.84). Most fathers (91%) and mothers (93%) were born in the Netherlands. Children (48% girls) were between 3.61 and 8.61 years old (M = 6.26, SD = 1.31) and the majority was born in the Netherlands (97%). Families included in the current study did not significantly differ from families that were not included in terms of, sex of the child, sex of the parent, age of the child, age of the parent, harsh parenting, warm-supportive parenting, and disruptive child behavior (see Table S1).

Measures

Eyberg Child Behavior Inventory

The intensity of disruptive child behavior was reported by parents with a Dutch translation of the Intensity subscale of the Eyberg Child Behavior Inventory (ECBI; Eyberg & Pincus, 1999; Weeland et al., 2018). Parents reported the frequency of disruptive behavior by using a 7-point Likert scale (1 = *never*, 7 = *always*) on a total of 36 items (e.g., "Does not obey house rules", "Has temper tantrums", and "Whines"). The ECBI has sufficient psychometric properties, is widely used internationally to assess the effectiveness of caregiver-training programs, and accurately discriminates between children with and without conduct-related disorders (Abrahamse et al., 2015**;** Leijten et al., 2017; Rich & Eyberg, 2001). Internal consistency was $\alpha = .85$.

Parenting Practices

Parenting practices were measured using the Parent Practice Inventory (PPI; Webster-Stratton et al., 2001). This self-report consists of several sections, each including multiple items (7-point Likert scale: 1 = *not likely at all/never*, 7 = *likely/always*) related to parents'

frequency of responses to parenting situations . To assess warm-supportive parenting we used the praise and incentive dimensions scale (11 items, e.g., “Giving a hug or compliment”) combined with the positive verbal scale (9 items, e.g., “In an average week, how often do you praise or reward your child for doing a good job at home or school?”). To assess harsh parenting, we used the harsh and inconsistent discipline scale (15 items, e.g., “Threatening but not punishing”) combined with the punishment scale (6 items, e.g., “Slapping or hitting when misbehavior occurs”). The internal consistency of both dimensions, i.e., harsh parenting and warm-supportive parenting, was $\alpha = .75$ and $\alpha = .72$, respectively.

Genotyping and Computation

Saliva was collected via passive drool with the Oragene-DNA OG-600 container (DNA Genotek, Canada) following manufacturer’s instructions and stored at room temperature. Genomic DNA was extracted from saliva and bisulfite converted with the ZYmo EZ DNA methylation kit (Zymo research, Irvine, CA, USA). Genotyping was performed on saliva samples using the Infinium iSelect GSA array (Illumina, San Diego CA, USA). Genotypes were called using Illumina’s GenomeStudio software and quality control was performed using PLINK v1.90b6.17 (Purcell et al., 2007). Participants were excluded if genetic quality controls failed i.e., (1) the sample call rate for this person was low (< 95%) ($n_{child} = 2$ and $n_{parent} = 0$), (2) the parent-child genetic relatedness did not match the expected familial relation (proportion of IBD in PLINK > 0.2; $n_{child} = 0$ and $n_{parent} = 0$), (3) there was a phenotype-genotype gender mismatch ($n_{child} = 2$ and $n_{parent} = 5$), or (4) a person was identified as an ethnic outlier based on a principal component analysis to account for population stratification ($n_{child} = 11$ and $n_{parent} = 10$). There were 10 children and 9 parents for whom the heterozygosity value was ± 3 SD from the mean. We decided not to remove them because the values were strongly shifted to the mean (i.e., high median) in such a way that it was easy to fail this criterium. Besides, none of the heterozygosity rates were above .04, indicating no sign of low sample quality.

Before imputation, single nucleotide polymorphisms (SNPs) were removed when (1) they had a low call rate (i.e., genotype missingness was < 95%) or, in other words, SNPs that are missing in a large proportion of individuals (n_{child} = 10623 SNPs and n_{parent} = 6220 SNPs), (2) minor allele frequency (MAF) was low (< 5%) (n_{child} = 407136 and n_{parent} = 409292 SNPs), or (3) if they were not in Hardy-Weinberg equilibrium (p < 1e-6, n_{child} = 421 SNPs and n_{parent} = 84). To impute the genotypes, we made use of the Michigan Imputation Server (Das et al., 2016) and imputed the genotypes using the HapMap2, build GRCh37/hg19 as reference panel. Monomorphic SNPs (with MAF < 0.1%) and SNPs with low imputation quality (R^2 < 0.3) were excluded, resulting in 2,377,699 SNPs for the children and 1,720,570 for the parents.

Polygenic Score Computation

We computed two polygenic scores. First, a polygenic score for disruptive behavior (PGS-DB) was based on the most recent meta-analytic genome-wide association study (GWAS) summary statistics based on six disruptive behavior phenotypes in 523,150 individuals (Baselmans et al., 2021). Phenotypes from the original studies were aggression, angry outbursts, extreme irritability, irritability, irritable for two days, and ADHD. Participants ranged from early childhood to old age. Second, for the supplementary analysis, we used a polygenic score of externalizing behaviors (PGS-EXT) based on multivariate genomic analyses of the following seven phenotypes: attention-deficit/hyperactivity disorder, problematic alcohol use, lifetime cannabis use, reverse-coded age at first sexual intercourse, number of sexual partners, general risk tolerance and lifetime smoking initiation (Karlsson Linnér et al., 2021). We used LDpred2 to compute the polygenic scores. This method utilizes GWAS summary statistics and LD information from an external LD reference sample (Hapmap3 with independent LD blocks) to calculate the posterior mean effect size of each

SNP (Privé et al., 2021). We performed standard quality control procedures on the summary statistics following the tutorial of Choi et al. (2020).

Statistical Analyses

We tested our hypotheses with SEM in R (R Core Team, 2022) using the lavaan package (Rosseel, 2012). We included a covariance between harsh parenting and warm-supportive parenting because those constructs were expected to be highly correlated. Because the model was saturated—the number of data points equaled the number of estimated parameters—evaluation of model fit was uninformative. Before analyzing, the data were screened to verify the statistical assumptions of structural equation modeling (SEM). Skewness and kurtosis of the variable residuals were examined and showed univariate and multivariate normality (Bentler, 2006; Bryne, 2010). Furthermore, we controlled for 10 genetic principal components (PC). Because of the relatively small sample size and relatively large model, we regressed out the effects of the PCs and used the residuals in subsequent analyses. We used full information maximum likelihood (FIML) estimation to handle missing data because data were missing completely at random (MCAR, $\chi 2$ (18) = 12.16, p = .839). Path coefficients were assumed to be significantly different from zero if the confidence intervals (CIs) around them did not include 0. CIs were calculated using the standard error (SE) of the parameter estimate.

Results

Table 1 displays all univariate means and standard deviations and bivariate correlations for each variable. Bivariate correlations indicated significant, albeit small, positive associations between (1) disruptive child behavior and children's own PGS-DB (r = .13) and (2) disruptive child behavior and harsh parenting (r = .21), as well as (3) a small negative association between warm-supportive parenting and harsh parenting (r = -.15), and

(4) a strong positive association between children's polygenic score for disruptive behavior and parents' polygenic score for disruptive behavior (r = .50).

Harsh Parenting

Path coefficients are shown in Table 2 and Figure 1. The model was just identified and explained 6.6% of the variance in disruptive child behavior. Harsh parenting was, as we expected, positively associated with disruptive child behavior, indicating that harsh parents were more likely to have children with higher levels of disruptive behavior (β = 0.22, 95% CI [0.13, 0.32], p < .001). Also as expected, children's PGS-DB was positively associated with their disruptive behavior (β = 0.15, 95% CI [0.01, 0.28], p = .031), and parents' PGS-DB was directly correlated with children's PGS-DB (β = 0.50, 95% CI [0.42, 0.59], p < .001).

We did not find evidence for passive or evocative gene-environment correlations, as the paths from parents' PGS-DB to harsh parenting (β = 0.06, 95% CI [-0.09, 0.20], p = .349) and from children's PGS-DB to harsh parenting (β = -0.06, 95% CI [-0.21, 0.08] , p = .393) were non-significant. To investigate the genetic nurture of disruptive child behavior, we assessed whether parents' PGS-DB predicted disruptive child behavior, controlling for children's PGS-DB, but the result was not significant (β = -0.04, 95% CI [-0.17, 0.10] , p = .600). Note that the sample size was small and confidence intervals were large preventing strong interpretation of these null results.

We also assessed the two indirect pathways from PGS-DB of parents to disruptive child behavior. Children's PGS-DB partially mediated the association between parents' PGS-DB and children's disruptive behavior (β = 0.07, 95% CI [0.01, 0.14], p = .035). However, we did not find evidence for an environmentally mediated effect of parents' PGS-DB on disruptive child behavior via harsh parenting (β = 0.01, 95% CI [-0.02, 0.05], p = .447).

Table 1

Descriptive Statistics of and Correlations Between the Polygenic Scores, Characteristics, Parenting Behaviors and Disruptive Child Behavior

Variable	*n*	*M*	*SD*	1	2	3	4	5	6	7	8	9
1. Parental PGS-DB	280	0.00	0.08	-	.03	.00	.02	.02	**.50**	.05	-.02	.05
2. Harsh parenting	286	4.64	1.05		-	**-.15**	-.04	.04	-.03	**.21**	.01	-.01
3. Warm-supportive parenting	287	9.69	1.15			-	.07	**-.10**	.03	.05	.00	**-.14**
4. Parental sex	288	0.92	0.27				-	**-.17**	.00	.04	-.02	.09
5. Parental age	285	38.22	4.63					-	.00	-.04	.02	**.23**
6. Child PGS-DB	281	0.00	0.09						-	**.13***	-.01	.01
7. Disruptive child behavior	287	132.53	19.27							-	**-.21**	.08
8. Child sex	288	0.48	0.50								-	-.01
9. Child age	285	6.26	1.31									-

Note. PGS-DB = polygenic score of disruptive behavior; bold estimates are significant ($p < .05$).

Table 2

Unstandardized and Standardized Direct and Indirect Effects Using a Polygenic Score of Disruptive Behaviors

Effect	*b*	*SE*	β	95% CI	
				LL	*UL*
PGS-DB child					
PGS-DB parent	**0.51**	**0.05**	**0.50**	**0.42**	**0.59**
Harsh parenting					
PGS-DB parent	0.74	0.95	0.06	-0.09	0.20
PGS-DB child	-0.81	0.95	-0.06	-0.21	0.08
Warm-supportive parenting					
PGS-DB parent	-0.28	0.93	-0.02	-0.16	0.12
PGS-DB child	0.44	0.93	0.03	-0.10	0.17
Harsh parenting	**-0.19**	**0.07**	**-0.15**	**-0.25**	**-0.05**
Disruptive child behavior					
PGS-DB parent	-8.18	15.58	-0.04	-0.17	0.10
PGS-DB child	**32.87**	**15.25**	**0.15**	**0.01**	**0.28**
Harsh parenting	**3.87**	**0.87**	**0.22**	**0.13**	**0.32**
Warm-supportive parenting	1.40	0.84	0.08	-0.02	0.18
Genetic transmission via PGS child	**16.60**	**7.89**	**0.07**	**0.01**	**0.14**
Environmental mediation via HP	2.86	3.76	0.01	-0.02	0.05
Environmental mediation via WSP	-0.39	1.32	-0.00	-0.01	0.01

Note. CI = confidence interval; LL = lower limit; UL = upper limit; PGS-DB = polygenic score of disruptive behaviors. HP = Harsh parenting; WSP = Warm-supportive parenting; Significant estimates ($p < .05$) are in bold.

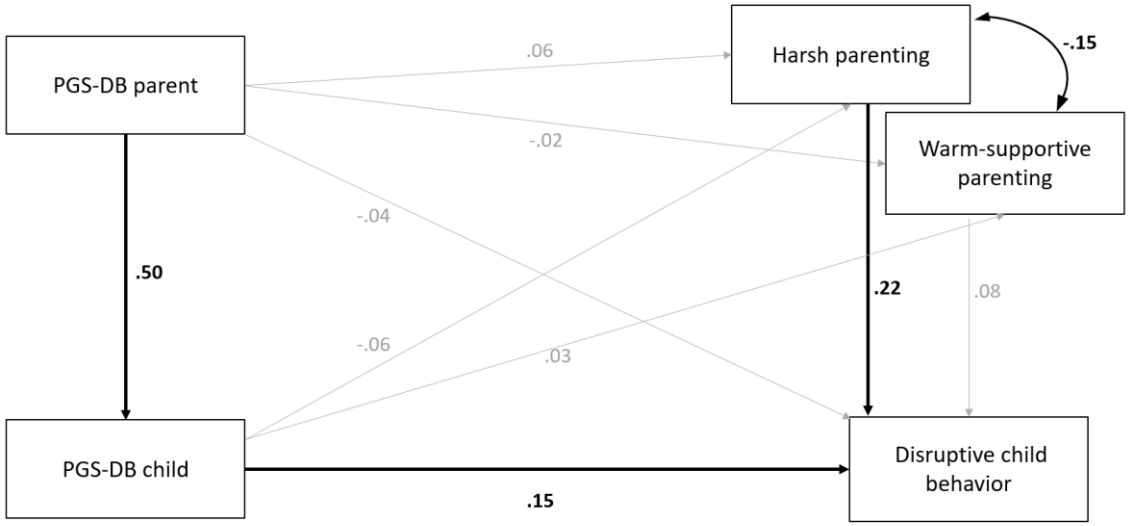
Warm-Supportive Parenting

We did not find a significant association between warm-supportive parenting and disruptive child behavior (β = 0.08, 95% CI [-0.02, 0.18], $p = .095$). Children did not evoke warmer or more supportive parenting based on their own PGS-DB (β = 0.03, 95% CI [-0.10, 0.17], $p = .639$), and parents did not engage in less warm or supportive parenting based on their PGS-DB (β = -0.02, 95% CI [-0.16, 0.12], $p = .764$). Finally, we found no evidence for

an environmentally mediated effect from parents' PGS-DB to disruptive child behavior via warm-supportive parenting (β = -0.00, 95% CI [-0.01, 0.01], *p* = .767).

Figure 1

Estimated Model of Gene-Environment Correlation, Genetic Confounding, and Genetic Nurture for Disruptive Child Behaviors



Note. Standardized regression coefficients are presented. Significant estimates are in bold and have bold arrows. PGS-DB = polygenic score of disruptive behaviors.

Sensitivity Analyses

We repeated our main analyses including a polygenic score of the broader construct externalizing behavior (i.e., PGS-EXT). Results were similar to the main analysis with a few exceptions (see Supplemental materials S1). Briefly, in this model children's PGS-EXT did not predict disruptive child behavior (β = 0.08, 95% CI [-0.06, 0.22], *p* = .252). We did find that higher PGS-EXT scores of parents were associated with more harsh parenting (β = 0.18, 95% CI [0.04, 0.32], *p* = .011). Moreover, children with lower PGS-EXT scores tended to have parents that were harsher (β = -0.14, 95% CI [-0.28, -0.00], *p* = .047). In this model, we

found a significant, albeit very small, genetic nurture —i.e., environmentally mediated — effect of parent's PGS-EXT on disruptive child behavior via harsh parenting (β = 0.04, 95% CI [0.00, 0.08], p = .028).

Discussion

In the present study, we aimed to advance our understanding of gene-environment interplay in disruptive child behavior. Using a polygenic score of disruptive behavior (PGS-DB), we found that parents that report harsher (but not warm-supportive) parenting behavior also report having a child with more disruptive behavior. Moreover, a higher child PGS-DB was associated with more disruptive behavior, a finding that corresponds with outcomes of previous research (Kretschmer et al., 2022; Kuo et al., 2022; Teeuw et al., 2023; Luo et al., 2021). However, using this PGS-DB no evidence emerged for gene-environment correlations: none of the associations between children's PGS-DB and parenting, parents' PGS-DB and parenting, and parents' PGS-DB and disruptive behavior were significant.

Interestingly, in a supplementary analysis, we investigated a polygenic score of broader externalizing behavior (PGS-EXT) and found no direct genetic effect (i.e., child genotype predicting phenotypic behavior while controlling for parent genotype), but *did* find a genetic nurture effect: parent's higher PGS-EXT predicted more harsh parenting which in turn predicted more disruptive child behavior. Moreover, a higher PGS-EXT score in the child was associated with parenting, potentially indicating an evocative type of gene-environment correlation, whereby a child's genetic predisposition to externalizing behaviors associates with parenting behavior.

An interesting finding of this study is that although harsh parenting was associated with disruptive child behavior, warm-supportive parenting was not. One potential explanation might be related to the fact that our sample contained a relatively high proportion of families reporting mild or sub-clinical, and not necessarily clinical levels of disruptive child behavior.

In this sample, many parents might occasionally lose their patience and engage in harsher parenting in challenging situations, but still be able to maintain an overall positive relationship. The relatively low correlation between harsh and warm-supportive parenting (r = -.15) indeed seems to suggest that these parenting dimensions do not act in tandem, and are largely independent constructs. Another possible explanation is that for children showing more disruptive behavior, parents may actively and deliberately try to maintain praise and positive reinforcement in response to their child's behavior (Fischer et al., 2020).

The genetic nurture effects revealed in the supplementary analysis is partly consistent with the wider literature. For example, Kuo et al. (2022) found a similar genetic nurture effect using parental externalizing behaviors instead of parenting behavior. In none of the previously discussed studies, however, a genetic nurture effect with parenting was found. This may be explained, in part, by different choices of measurement across studies. For instance, Kretschmer et al. (2022) used a broad measure of family dysfunction and Kuo et al. (2022) assessed constructs such as involvement that focused, for example, on whether the parent would help with schoolwork). These constructs might not fully capture the nature of harsh or warm-supportive parenting.

The discrepancy between results using the PGS-DB and the PGS-EXT may be due to the phenotypes and age groups in the GWAS of the summary statistics that the PGS were constructed from. While the PGS-DB quite closely matched the phenotype under study (i.e., disruptive child behavior) and included childhood samples in its associated GWAS, the PGS-EXT included a broader set of phenotypes encompassing problematic alcohol use, number of sexual partners and lifetime smoking initiation which are less relevant in a child sample. This may explain why in the PGS-EXT model, the link between parental genotype and parenting emerged—as the PGS-EXT was more developmentally appropriate for adults than the PGS-

DB. Similarly, this may explain why the link between child genotype and disruptive child behavior did emerge in the PGS-DB model, but not in the PGS-EXT model.

Several limitations of our study warrant mentioning. Overall, the ability to detect a genetic effect, let alone complex gene-environment interplay effects, was still relatively limited in the present study due to the small sample size and subsequent low statistical power. Future studies may increase power by either collecting larger samples or by pooling data from several, comparable, studies. Another limitation is that we collected genetic data from only one parent. This could bias our estimates of both direct genetic effects and genetic nurture effects (Tubbs et al., 2020). In particular, when only one parent's genotype is not included in the model the child's indirect genetic effect is biased in the direction of the unmodelled parent's effect, while the effect of the parent's genotype is biased in the opposite direction. Also, we had to rely on parent self-report measures which may be biased (Runze & van IJzendoorn, 2023), although harsh parenting practices might be difficult to observe. Nevertheless, our study provides valuable insight into the gene-environment interplay and adds to the scarce knowledge to date regarding the role of genetic predisposition in parenting and disruptive child behavior. Moreover, our sample was comprised of children with higher disruptive behavior problems compared to – often used – normative population samples. Future research should invest in testing the direct and indirect effects of genetic predisposition for other phenotypes on disruptive child behavior, and the potential mediating role of parenting, with models that contain genetic predisposition for both parents. Additionally, it should be established whether PGS that are instead derived from GWAS that already partition indirect and direct genetic effects may have better power to predict genetic nurture effects.

Conclusion

We found associations between children's early environment (i.e., parenting behavior) and their genetic predisposition on disruptive child behavior, as well as evocative gene-

environment correlations and genetic nurture effects. However, this depended on whether genetic predisposition was based on a more specific score for disruptive behaviors seen in children or a broader externalizing score. Our findings emphasize the need to include both genetic and environmental data to provide a complete understanding of pathways that shape complex traits such as disruptive child behavior and parenting behavior. It is therefore important to replicate this study with multi-informant, multi-method assessments, trio genetic data, and various PGS for various behavior-related phenotypes.

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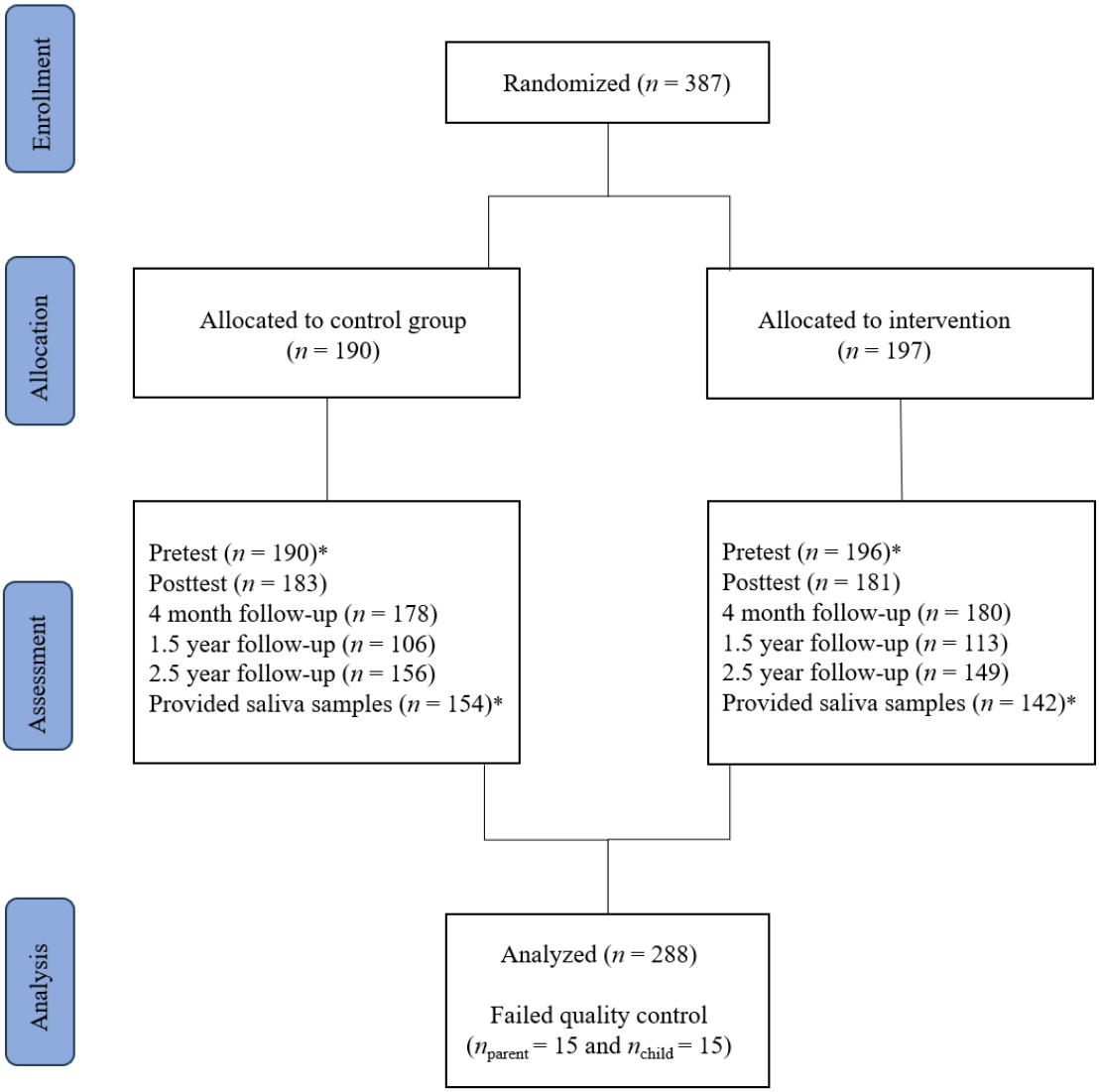
Supplemental Materials

Figure S1

Flow Chart Diagram of Participants in the Study



Note. * = assessments included in the current study

Table S1

Families With Both Polygenic Scores Versus Families Without Polygenic Scores

	Families with PGS *N* = 288		Families without PGS *N* = 98			
	N	%	*N*	%	χ^2	*p*
Dutch child	279	97.21	95	96.93	0.00	.999
Dutch father	261	90.63	77	78.57	8.68	.003
Dutch mother	269	90.63	78	79.59	13.87	< .001
Girls	138	47.92	36	38.71	2.05	.153
Mothers	265	92.01	90	91.84	0.00	.999
	M	*SD*	*M*	*SD*	*t*	*p*
Age child	6.26	1.31	6.47	1.37	1.36	.176
Age parent	38.22	4.63	37.73	5.42	-0.81	.419
Harsh parenting	4.64	1.05	4.82	1.28	1.22	.224
Warm-supportive parenting	9.69	1.15	9.75	1.14	0.43	.671
Disruptive behavior	132.53	19.27	135.56	19.14	1.35	.179

Note. PGS = polygenic score.

S2. Gene-environment Interplay in Externalizing Behavior

Harsh parenting

Path coefficients appear in Table S2 and are shown in Figure S2. The model was just identified and explained 5.4% of the variance in disruptive child behavior. Harsh parenting was, as expected, positively associated with disruptive child behavior, suggesting that harsh parents were more likely to have children with higher levels of disruptive behavior. This small association reflects that increase in harsh parenting would lead to an increase in disruptive child behavior, controlling for all other predictors in the model. As expected, parents PGS-EXT was also positively associated with harsh parenting (i.e., passive gene-environment correlation). We also found that parents' PGS-EXT was directly correlated with children's PGS-EXT which was, as we would expect based on genetic linkage, around $\beta = .50$. Children's PGS-EXT was also significantly associated with parents' harsh parenting (b_h; evocative gene-environment correlation). Notably, this association was in the opposite

direction of what we expected, showing that children with higher PGS-EXT scores tended to have parents that are less harsh.

To assess the genetic nurture of externalizing behavior we assessed whether parents' PGS-EXT predicted disruptive child behavior, controlling for children's PGS-EXT. We did not find evidence for this genetic nurture hypothesis. As indirect pathways could still be significant, even in the absence of direct correlations (i.e., complete mediation; Baron & Kenny, 1986), we also assessed the two indirect pathways from parents' PGS-EXT to disruptive child behavior. However, we also did not find evidence for genetic transmission; children's PGS-EXT did not mediate the association between parents' PGS-EXT and children's disruptive behavior. We did, however, find evidence for a statistical effect of parents PGS-EXT on children's PGS-EXT that was not transmitted via genetic predisposition, but partially mediated via harsh parenting (i.e., an environmentally mediated effect).

Warm-supportive Parenting

Path coefficients of direct and indirect effects of warm-supportive parenting are also depicted in Table S2 and Figure S2. Contrary to harsh parenting and expectations, we did not find a significant correlation between warm-supportive parenting and disruptive child behavior. We additionally did not find evidence for a passive gene-environment correlation, which would be indicated by a significant direct relationship between parents' PGS-EXT and warm-supportive parenting. There was also no evidence for an evocative gene-environment correlation, which would have been indicated by a significant direct relationship between children's PGS-EXT and warm-supportive parenting. Also warmer and more supportive parenting was no associated with parents' PGS-EXT. Lastly, no evidence was found for an environmentally mediated effect from parents' PGS-EXT to disruptive child behavior via warm-supportive parenting.

Table S2

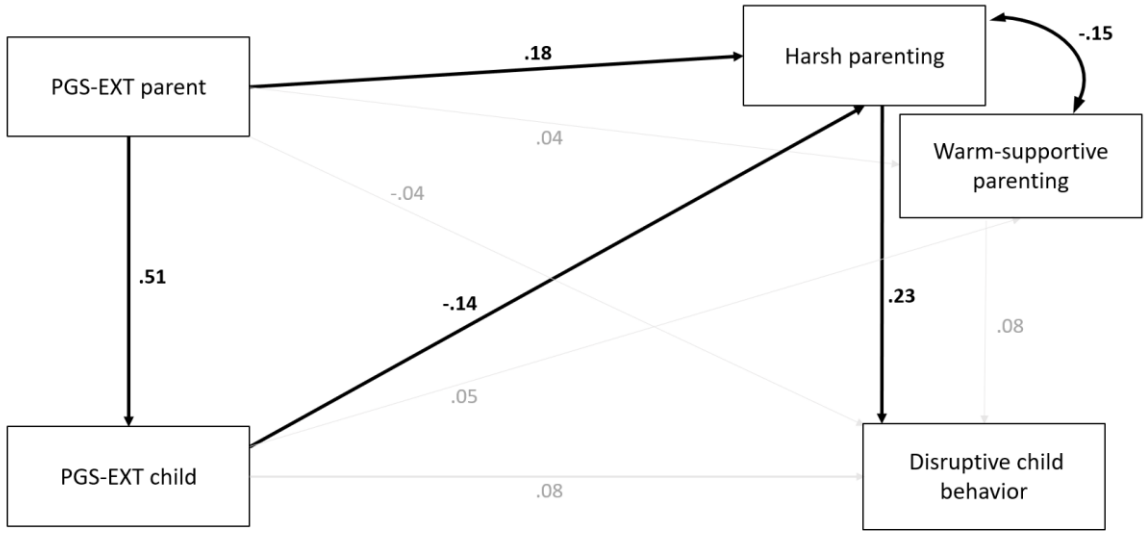
Unstandardized and Standardized Direct and Indirect Effects Using the Polygenic Score of Externalizing Behaviors

Effect	*b*	*SE*	β	95% CI	
				LL	*UL*
PGS-EXT child					
PGS-EXT parent	**0.51**	**0.05**	**0.51**	**0.42**	**0.60**
Harsh parenting					
PGS-EXT parent	**1.51**	**0.60**	**0.18**	**0.04**	**0.32**
PGS-EXT child	**-1.19**	**0.60**	**-0.14**	**-0.28**	**-0.00**
Warm-supportive parenting					
PGS-EXT parent	0.33	0.59	0.04	-0.10	0.18
PGS-EXT child	0.46	0.60	0.05	-0.08	0.19
Harsh parenting	**-0.19**	**0.07**	**-0.15**	**-0.25**	**-0.05**
Disruptive child behavior					
PGS-EXT parent	-5.18	10.11	-0.04	-0.17	0.10
PGS-EXT child	11.39	9.97	0.08	-0.06	0.22
Harsh parenting	**3.91**	**0.89**	**0.23**	**0.13**	**0.32**
Warm-supportive parenting	1.40	0.85	0.08	-0.02	0.18
Genetic transmission via PGS child	5.77	5.08	0.04	-0.03	0.11
Environmental mediation via HP	**5.92**	**2.71**	**0.04**	**0.00**	**0.08**
Environmental mediation via WSP	0.47	0.88	0.00	-0.01	0.02

Note. CI = confidence interval; LL = lower limit; UL = upper limit; PGS-DB = polygenic score of disruptive behaviors. HP = Harsh parenting; WSP = Warm-supportive parenting; Significant estimates are in bold.

Figure S2

The Estimated Model of Gene Environment Correlation, Genetic Confounding, and Genetic Nurture Using the Polygenic Score of Externalizing Behaviors



Note. Path model testing gene-environment correlation, genetic confounding, and genetic nurture. Each coefficient represents the standardized regression coefficient. Significant estimates are in bold and have bold arrows. PGS-EXT = polygenic score of externalizing behaviors.