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Re-examining externalizing polygenic risks and psychosocial environments: Are gene-environment correlations really that pervasive?

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Abstract

Background. Gene-environment correlations (*r*GEs) are often observed for psychiatric traits, including externalizing problems. Some researchers believe *r*GEs reflect the effects of offspring genotype on psychosocial environments. Others consider *r*GEs to be a nuisance, reflecting effects of confounders, such as population stratification, assortative mating, or parental genotypes. Our study aims to examine whether *r*GEs persist when confounding effects are controlled for in a family-based design.

Methods. We examined *r*GEs between a polygenic score (PGS) for externalizing problems and adolescent psychosocial environments (parent- and child-reported parental relationships, delinquent peer affiliation, and school connectedness) in the National Longitudinal Study of Adolescent to Adult Health. We estimated *r*GEs within 777 families (1,589 twins/full-siblings) and also at the population-level using 7,635 unrelated individuals.

Results. *r*GEs were observed with parental relationships, delinquent peer affiliation, and school connectedness in the population sample. In family-based analyses, *r*GEs with parental relationships persisted at the within-family level. However, *r*GE with delinquent peer affiliation was attenuated and non-significant at the within-family level, suggesting the presence of confounding. Analyses regarding school connectedness were inconclusive.

Conclusions. While PGSs are often associated with psychosocial environments, some of these associations may be subject to substantial confounding. Our findings highlight the importance of testing *r*GE while controlling for potential confounds in large population-based studies.

Introduction

Externalizing (EXT) problems, such as aggression, rule-breaking behaviors, and harmful substance use, are characterized by impulsivity and behavioral undercontrol (Krueger et al., 2002). EXT problems in adolescence are prospectively associated with negative life outcomes, such as financial and legal difficulties (Gornik, Clark, Durbin, & Zucker, 2022) and emotional problems (Hamdi & Iacono, 2014). While genes are known to play a prominent role (Barr & Dick, 2019), EXT problems are also influenced by environmental factors (Fortuin, van Geel, & Vedder, 2015; Pinquart, 2017; Samek & Hicks, 2014). Furthermore, genetic and environmental factors for EXT problems are closely intertwined, as genetic risks for EXT are often correlated with psychosocial environments (i.e. gene-environment correlation, *r*GE) (Elam, Lemery-Chalfant, & Chassin, 2023; Kuo et al., 2021). Emerging studies have begun to show that genetic effects on EXT problems may be mediated by environments (Dong, Sasia, & Li, 2026; Li, 2019), highlighting the role of psychosocial influences in what are putatively believed to be genetic effects.

That said, the very notion of environmentally mediated genetic effects is complicated, in part because it may also reflect *r*GEs driven by confounders, such as population stratification, assortative mating, or parental genotypes. While these particular confounds are notoriously challenging to control for in large population-based designs (Pingault et al., 2022), a family-based design provides one robust way to account for these confounds. For example, researchers can estimate *r*GE effects within families by comparing twins and full-siblings, who are born to the same household, have the same ancestral backgrounds, and are influenced by similar family environments. They also randomly inherit genotypes from the same parents during meiosis, such that PGS variances within a family are independent of environmental confounders. Therefore, family-level confounding effects are mostly accounted for when estimating *r*GE within families (see Figure 1) (McAdams, Rijsdijk, Zavos, & Pingault, 2021; Selzam et al., 2019; Tanksley et al., 2025; Zhou et al., 2025). *r*GEs that persist within families may offer stronger support for a conceptualization where social environments mediate genetic effects on EXT development, whereas *r*GEs that substantially attenuate when estimated within families may indicate the presence of confounding. As such,

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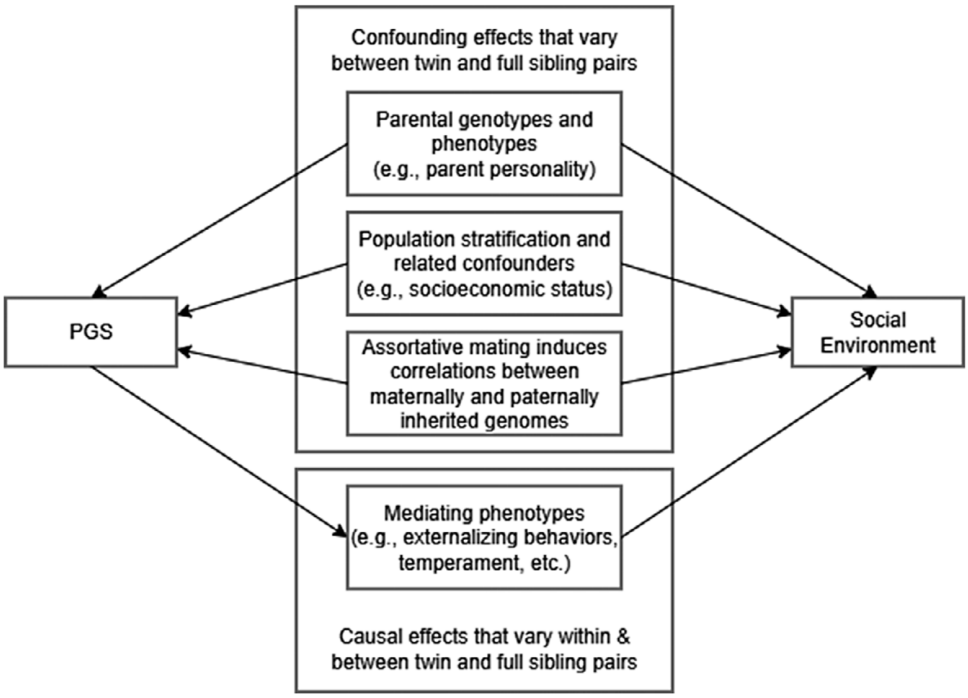


Figure 1. Explanations of rGE between PGS and social environments.

our study aims to test whether *r*GEs for EXT problems are potentially robust to family-level confounds by leveraging a family-based design.

Gene-environment correlations in the EXT literature

Twin studies have shown that 50%–80% of the variation in EXT problems can be attributable to genetic factors within a given population (Hicks et al., 2004; Krueger et al., 2002). More recently, the field has moved toward identifying loci associated with EXT problems at the individual level through genome-wide association studies (GWASs). A GWAS performed by Karlsson Linnér et al. (2021) examined the association of single nucleotide polymorphisms (SNPs) with EXT in a pooled sample of 1.5 million individuals, in which a latent genetic factor for EXT was estimated using GWAS summary statistics for problematic alcohol use, lifetime smoking initiation, lifetime cannabis use, general risk tolerance, attention-deficit/hyperactivity disorder, age at first sex, and number of sexual partners. The effects of all SNPs were then aggregated into a polygenic score (PGS), which is calculated as a weighted sum of the dosages of effect alleles an individual carries across their genomes (Purcell et al., 2007). A PGS for EXT computed from the Karlsson Linnér study was found to outperform PGSs based on other studies in capturing genetic risks for a spectrum of EXT problems (Deng et al., 2025), explaining up to 10% of the variance in an EXT factor and 1%–7% of the variance in specific EXT traits across two held-out samples.

While a GWAS is meant to identify primarily genetic variances that are associated with the trait in question (e.g. EXT problems), EXT PGS computed from GWAS signals consistently show a high degree of correlation with environments (Dong et al., 2026; Kretschmer et al., 2022; Kuo et al., 2021), suggesting that GWAS signals may reflect both genetic and environmental variances. *r*GE may reflect active and evocative effects, where a higher PGS may lead individuals to select into or evoke certain environmental responses. For example, adolescents with higher genetic risks for EXT may affiliate with more substance-using peers (Elam et al., 2017) or elicit harsher parenting responses (Yan, Ansari, & Peng, 2021). Meta-analyses of twin studies have indeed shown that many psychosocial environments share a substantial proportion of heritable variance with EXT problems (Hicks et al., 2009; Kendler & Baker, 2007; Klahr et al., 2013; Marceau et al., 2013). Further, these 'heritable' environments may in turn impact development down-the-stream, thereby mediating genetic effects on future EXT problems (Elam et al., 2023; Kretschmer et al., 2022). In summary, the presence of *r*GE may suggest that PGS for EXT not only reflects direct genetic effects, but also mediated effects via heritable environmental exposures (Kendler & Baker, 2007; Li, 2019; Pingault et al., 2022).

On the other hand, *r*GE may reflect passive, or confounding, effects. For example, population stratification arises when genetic associations are confounded by ancestry-related differences in the genotype (i.e. differences in allele frequencies). This could inflate correlations among otherwise independent alleles in the genome, even after controlling for genetic principal components (Elhaik, 2022; Grinde et al., 2024). Assortative mating occurs when individuals select their partners based on similarity or dissimilarity regarding a phenotype. Parental genotypes may also represent a family-level confound in *r*GE because individuals inherit half of their genotype from each parent, such that apparent genetic associations may inadvertently capture the effect of the parents' genotype as well. Collectively, these passive/confounding effects are often present in population-based designs and may inflate associations between genes and environmental exposures (Kong et al., 2018; Price, Zaitlen, Reich, & Patterson, 2010).

Adolescent psychosocial environments

We focus on measured environments during adolescence because it is a developmental stage characterized by heightened neural plasticity and increasing personal autonomy, making it especially

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salient for studying *r*GE in the context of EXT problems (Baker, Galván, & Fuligni, 2025; Sebastian, Viding, Williams, & Blakemore, 2010). Genetic factors have been previously shown to influence how adolescents select, evoke, and respond to their environments in the context of EXT problems (Andersen, Steinberg, & Belsky, 2021; Dick, Adkins, & Kuo, 2016). The role of parenting has been extensively studied with respect to the development of EXT, with studies showing that higher genetic risk for EXT is associated with less supportive parenting (Pinquart, 2017; Samek et al., 2015). Peer effects are another dimension theorized and studied in relation to EXT. For example, delinquent peer affiliation has been found to be positively associated with genetic risks for EXT (Samek et al., 2016). Researchers have also linked broader social contexts, such as school environments, to EXT genetic risks. For instance, Li (2019) found that PGS for EXT problems predicted trajectories of substance use and antisocial behaviors both directly and indirectly through perceived school safety and belonging. Crucially, these environmental factors are themselves risk factors for maladaptive outcomes, including the development of EXT problems (Carroll, Klump, & Burt, 2023; Monahan, Rhew, Hawkins, & Brown, 2014; Pinquart, 2017).

Current study

The current study investigated *r*GEs between PGS for EXT problems and previously well-studied adolescent psychosocial environments in relation to EXT problems (i.e. parental relationships, school connectedness, and delinquent peer affiliation) using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health). We first examined *r*GE effects in a population sample comprised of genetically unrelated individuals. Next, we estimated the same *r*GE effects at the within- and between-family levels using a family-based sample of twins and full-siblings. The within-family effects were estimated by comparing twins/siblings within the same families and therefore reflect *r*GEs when controlling for family-level confounds. The between-family effects were derived from comparisons of family means and therefore resemble estimates of *r*GE in the general population.

We hypothesized that: (1) in the population sample (i.e. unrelated individuals), higher PGS will be correlated with poorer parental relationships and school connectedness and greater delinquent peer affiliation, (2) at the between-family level (i.e. comparisons between sets of twins/siblings), twins/siblings from a family with higher average PGS will on average experience poorer parental relationships and school connectedness and greater delinquent peer affiliation, (3) at the within-family level (i.e. comparisons of twins/siblings within the same family), twins/siblings with higher PGS will experience poorer parental relationships and school connectedness and greater delinquent peer affiliation than their co-twin/sibling, and (4) within-family *r*GE effects, where confounding effects are controlled for, will be smaller in magnitude than between-family estimates.

Methods

Participants

Add Health is a longitudinal study of a large sample of youths from the United States who were in grades 7–12 during the 1994–1995 school year. Over 90,000 adolescents were initially sampled from the population through a stratified set of qualifying high schools. 20,745 adolescents were then randomly selected from the previous school sample and administered the in-home interview at Wave I, including oversamples from underrepresented populations. Participants were followed from adolescence to adulthood for five waves of data collection to date: Wave I (1994–1995, grades 7–12, $N = 20{,}745$), Wave II (1995–1996, grades 8–12, $N = 14{,}738$), Wave III (2001–2002, ages 18–26, $N = 15{,}197$), Wave IV (2007–2008, ages 24–32, $N = 15{,}701$), and Wave V (2016-2018, aged 33–43, $N = 12{,}300$). Information on attrition across waves was documented by Harris et al. (2019). The full Add Health sample at Wave I was comprised of 50.5% females and 61.45% who self-identified racial-ethnically as 'White', 23.17% 'Black or African American', 7.64% 'Asian or Pacific Islander', 3.57% 'Native American', 16.99% 'Hispanic', and 9.64% as 'Other'.[1] Add Health also included more than 3,000 pairs of individuals of varying genetic relatedness living in the same household, including 289 monozygotic (MZ) twin pairs, 452 dizygotic (DZ) twin pairs, 1,251 full-sibling pairs, 442 half-sibling pairs, 201 cousin pairs, 408 unrelated pairs, and 96 other related pairs (i.e. aunt/uncle, niece/nephew, group homes, spouses, and in-laws).

Our study leverages two subsamples of Add Health participants. First, we formed our admixed population subsample from 7,635 genetically unrelated individuals. Then we formed a family-based subsample of 1,589 individuals from 777 families. These individuals are MZ/DZ twins and full-siblings who were born to and grew up in the same household (Harris, Halpern, Haberstick, & Smolen, 2013).

Genotyping

Saliva samples were collected at Wave IV and genotyped using the Illumina Human Omni-1 Quad or the Omni-2.5 Quad BeadChip. Quality control procedures were conducted at the participant level (i.e. removing participants with call rates <90%, poor duplicate concordance, unclear sex chromosomes, identifier abnormalities) and the marker level (i.e. removing variants with call rate <90%, alignment uncertainty, tri-allelic status, duplicate discordance, ancestry-specific Hardy-Weinberg Equilibrium $p < 5 \times 10^{-5}$, and minor allele frequency <0.5%). Genotypes were then merged between arrays and imputed using the 1000 Genomes Phase 3 panel from the GLGC/GIANT protocol. Imputed data containing 9,664,514 markers were available for 9,974 participants, including 2,014 genetically related individuals from 975 families. Participants were partitioned into four genetic similarity groups based on genetic similarity to European, African, East Asian, and Hispanic ancestries, which is defined by distance from the mean of the first two genetic principal components (PCs), as described in Braudt and Harris (2020).

Measures

Polygenic scores for EXT (PGS_{EXT})

PGS_{EXT} was calculated based on a GWAS of EXT behaviors (Karlsson Linnér et al., 2021) that has shown superior predictive performance relative to other GWASs (Deng et al., 2025). PGS_{EXT} was calculated using PRS-CS (Ge et al., 2019), which accounts for linkage disequilibrium and optimizes prediction by weighting individual SNP effects in GWAS summary statistics based on external reference panels using Bayesian continuous shrinkage priors. PRS-CS has been shown to have better predictive performance and generalizability across genetic ancestries (Ahern, Thompson, Fan, & Loughnan, 2023). To further control for population stratification, PGS_{EXT} was residualized on the first 20 genetic PCs that were estimated using GCTA (Yang, Lee, Goddard, & Visscher, 2011)

[1]Proportions of self-identified racial-ethnic identities sum larger than 100% because participants are asked to indicate all appropriate identities that apply.

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and standardized within each genetic similarity group (Price et al., 2006). To maximize sample sizes, we ran analyses with individuals from all genetic similarity groups while including genetic similarity groups as a dummy-coded covariate.

Parental relationships

Parent report. At Wave I, the same parent reported on their relationship with each adolescent in the household. Six items assessed the closeness, collaboration, trust, antagonism, and overall satisfaction of the relationship (Supplementary Table S1). Parent report of the parental relationship has satisfactory reliability with Cronbach's $\alpha = 0.71$.

Child report. At Wave I, participants self-reported on how they were parented as adolescents: five items measured aspects of supportive parenting by each resident parent, including perceived care, closeness, warmth, communication, and overall satisfaction of the relationship (Supplementary Table S2). The maternal and paternal scales both showed high reliability with Cronbach's αs = 0.85 and 0.88, respectively.

Delinquent peer affiliation

At Wave I, three items assessed how many of the participant's three closest friends used cigarettes, marijuana, and alcohol. Another item assessed how often the participant took part in a fight where their group of friends fought against another group of people in the past year. These four items (Supplementary Table S3) showed satisfactory reliability ($\alpha = 0.71$).

School connectedness. At Wave I, six items assessed the participants' feelings of belongingness, interpersonal connectedness, teacher support, and safety at school (Supplementary Table S4). These items were rated on the same five-point scale and showed high reliability ($\alpha = 0.77$).

Covariates. Adolescent age and biological sex were cluster-mean centered and covaried out within-family. Family means of age and sex, parental education, family income, and dummy-coded genetic similarity groups were covaried out at the between-family level.

Analysis

Confirmatory factor analysis (CFA)

Psychosocial environments were measured as factor scores because they generally have better psychometric properties than composite scores (McNeish, 2023), are normally distributed, and better suit our analyses. A CFA model was estimated for each environmental variable in the entire genotyped sample using the WLSMV estimator. Item-level missing data were handled by pairwise deletion. Standardized factor scores were computed using the maximum a posteriori method.

Primary analysis

We used Bayesian Structural Equation Modeling (SEM) in Mplus (Muthén & Muthén, 1998) with the Gibbs sampler and 30,000 iterations of Markov Chain Monte Carlo draws, which allows for probabilistic interpretations of the posterior distributions. Missing data were handled with Bayesian data augmentation in Mplus (Muthén & Asparouhov, 2012). We elicited non-informative priors for all parameters because of a lack of empirical precedent. Model convergence was evaluated by visual examination of trace and autocorrelation plots for fixed effect parameters and posterior predictive checking of the chi-square statistic. Because Bayesian models yield posterior probability distributions, we report our results in terms of point estimates (i.e. median of the posterior) and their 95% credible intervals.

Psychosocial environments were modeled as correlated outcomes in the same Bayesian SEM. To test the first hypothesis, psychosocial environments were regressed on PGS_{EXT} and covariates in the population sample (i.e. 7,635 unrelated individuals of Add Health), using multiple regression. The population-level effect therefore reflects the expected difference in psychosocial environments given a one-unit increase in PGS_{EXT} among unrelated individuals in the general population.

To test the second and third hypotheses, a multilevel SEM was estimated with random intercepts for families using the family-based sample (1,589 twins/full-siblings from 777 families), where psychosocial environments were regressed on PGS_{EXT} and covariates at both within- and between-family levels. At the between-family level, psychosocial environments were regressed on family means of PGS. The between-family effect therefore reflects the extent to which twins/siblings from a family with higher average PGS_{EXT} experienced more/less of the environment. At the within-family level, PGS was centered around the family means to reflect a twin/sibling's deviation from their family mean. The within-family effect therefore reflects the extent to which the twin/sibling with higher PGS_{EXT} in a family experienced more/less of the environment relative to their co-twin/sibling.

Finally, to test the fourth hypothesis, the difference of the between-family effect from the within-family effect for each psychosocial environment was estimated as $\Delta\beta = \beta_{between}\text{-}\beta_{within}$. This was evaluated at each Markov Chain Monte Carlo draw and aggregated into a posterior distribution, which allowed us to test whether within- and between-family effects were statistically different in the same multilevel model.

We used a Bayesian analytic framework with non-informative priors for all parameters, thus circumventing the need for multiple-testing correction (Sjölander & Vansteelandt, 2019). We report all tested effects, including those considered null in the frequentist sense.

Results

Descriptive statistics

Table 1 shows descriptive statistics for the population sample and the family-based sample. Supplementary Tables 5 and 6 show descriptive statistics by pair type and genetic similarity grouping. Supplementary Table 7 shows missing data patterns for primary analysis (i.e. SEM).

CFA models

CFA models achieved satisfactory fit for parent-reported (N = 8,736; CFI = 0.991; TLI = 0.986; RMSEA = 0.044) and child-reported parental relationship (N = 9,796; CFI = 0.988; TLI = 0.981; RMSEA = 0.082), delinquent peer affiliation (N = 9,934; CFI = 0.999; TLI = 0.997; RMSEA = 0.029), and school connectedness (N = 9,942; CFI = 0.984; TLI = 0.966; RMSEA = 0.086). The factor structures were consistent with our expectation. For child-reported parental relationships, two highly correlated factors emerged for maternal and paternal relationships, and the prorated mean of maternal and paternal factor scores was therefore used to measure the average quality of parental relationships in the household. Details regarding CFA models can be found in Supplementary Tables S1–S4.

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Table 1. Sample descriptive statistics

Variable	Twins and full-siblings		Unrelated individuals	Subsample comparison
	Statistics	ICC	Statistics	*p*-value
Number of individuals	1,589		7,635	
Number of families	777			
Age at Wave 1	16.10 (1.66)	36.3%	16.08 (1.72)	0.815
Female sex	815 (51.3%)	26.5%	4,041 (52.9%)	0.238
Genetic similarity group		100%[a]		
European	986 (62.1%)		4,741 (62.1%)	0.118
African	282 (17.7%)		1,695 (22.2%)	
East Asian	66 (4.2%)		371 (4.9%)	
Hispanic	163 (10.3%)		828 (10.8%)	
Self-identified race/ethnicity[b]				
White	1,102 (69.4%)		5,190 (68.0%)	0.392
Black/African	296 (18.6%)		1,680 (22.0%)	0.012
Native	48 (3.0%)		193 (2.5%)	0.243
Asian/Pacific Islander	77 (4.8%)		405 (5.3%)	0.532
Other	130 (8.2%)		426 (5.6%)	0.001
Hispanic	194 (12.2%)		817 (10.7%)	0.151
Annual household income	$47,741 ($59,568)	100%[a]	$46,179 ($44,273)	0.721
Parental education[c]		100%[a]		
Never went to school	2 (<0.1%)		5 (<0.1%)	0.988
8th grade or less	48 (3.0%)		213 (2.7%)	
Less than high school	111 (7.0%)		569 (7.5%)	
High school graduate	475 (29.9%)		2,256 (29.5%)	
Less than college	348 (21.9%)		1,582 (20.7%)	
College graduate	368 (23.2%)		1,729 (22.6%)	
More than college	173 (10.9%)		863 (11.3%)	
PGS	−0.03 (0.99)	57.0%	0.00 (0.99)	0.210
Parental relationship				
Parent-reported	−0.02 (1.01)	50.0%	−0.01 (0.99)	0.979
Child-reported	0.04 (0.97)	27.6%	−0.01 (1.01)	0.300
Delinquent peer affiliation	−0.04 (0.98)	38.0%	0.00 (1.00)	0.151
School connectedness	0.04 (1.00)	28.8%	0.00 (1.00)	0.043

Note: Means are estimated for continuous variables with standard deviations in parentheses, while counts and percentages are shown for categorical variables. ICC, intra-class correlation (i.e. proportion of between-family variance over total variance of the variable). ICC is estimated for dichotomous variables by dummy coding. Parental relationships, delinquent peer affiliation, and school connectedness were measured as standardized factor scores estimated in the entire Add Health genotyped sample (i.e. population + related sample combined). Subsample comparison was performed by first randomly selecting one twin/sibling from each family and comparing these adolescents to the unrelated adolescents in the population sample. Continuous outcomes were compared using linear regression, binary outcomes using logistic regression, and categorical outcomes using chi-square test.
[a]ICCs = 100% because these variables are identical across twins/siblings within the same family.
[b]Sum of percentages larger than 100% because individuals were allowed to endorse multiple racial-ethnic identities.
[c]Parental education was measured as the higher of maternal and paternal education levels. In our analysis, ordinal categories of parental education were modeled as a continuous variable.

Primary analysis

Figure 2 shows the association between PGS_{EXT} and environmental variables estimated in the population sample and at the within- and between-family levels in the family-based sample.

Population-level effects

Higher PGS_{EXT} was associated with less supportive parental relationship (parent-reported: $\beta = -0.093$, 95% Credible Interval [CI] = −0.116 ~ −0.070; child-reported: $\beta = -0.049$, 95% CI = −0.071 ~ −0.027), higher levels of delinquent peer affiliation ($\beta = 0.113$, 95% CI = 0.092 ~ 0.134), and lower school connectedness ($\beta = -0.070$, 95% CI = −0.093 ~ −0.048) in the population sample.

Between-family effects

The between-family effects of PGS_{EXT} in the family-based sample are largely consistent with the estimates in the population sample in

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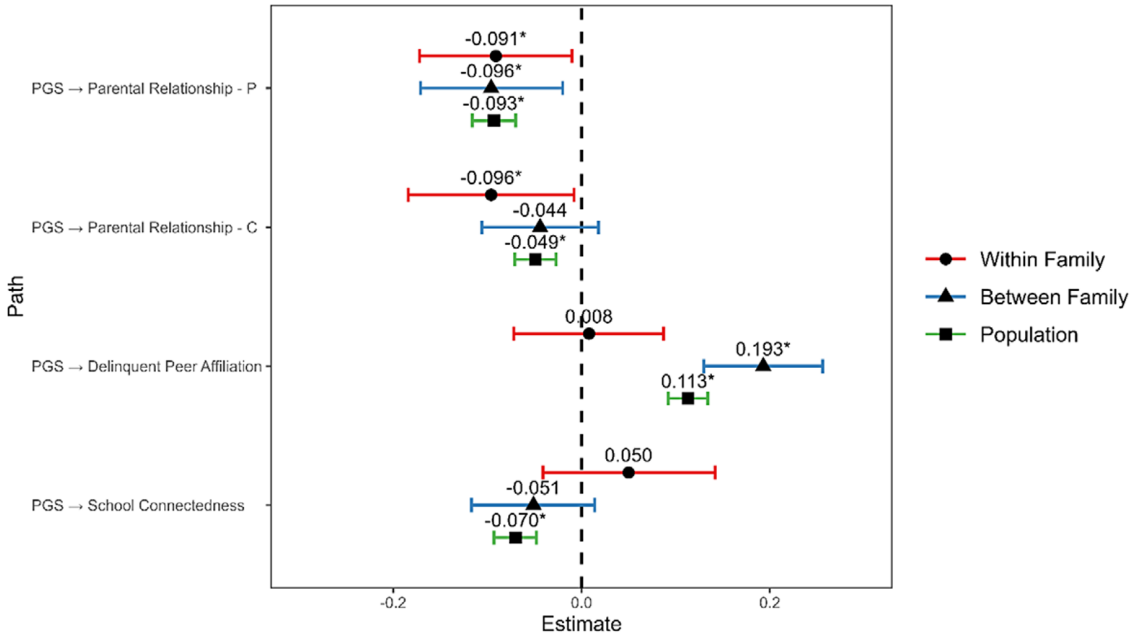


Figure 2. Estimates of PGS effects on social environments. *Note*: Point estimates and 95% credible intervals at the within-family level, between-family level, and in the population (i.e. unrelated) sample were shown. P, parent-reported. C, child-reported. *****, 95% credible interval does not contain the null, or is statistically significant in the frequentist sense.

terms of effect sizes. Higher PGS_{EXT} is significantly associated with a less supportive parental relationship by parent report ($\beta = -0.096$, 95% CI = −0.171 ~ −0.020), and higher delinquent peer affiliation (β = 0.193, 95% CI = 0.130 ~ 0.256). The association of PGS_{EXT} with child-reported parental relationship ($\beta = -0.044$, 95% CI = −0.106 ~ 0.018) and school connectedness ($\beta = -0.051$, 95% CI = −0.117 ~ 0.014) did not reach statistical significance, although the estimates were in the expected direction. The Bayesian posterior probability was also quite high for a negative association of PGS_{EXT} with child-reported parental relationship and school connectedness (i.e. 91.5% and 93.8%, respectively).

Within-family effects

A robust within-family effect of PGS_{EXT} on parental relationships was observed across informants: parents report less supportive parental relationships with the twin/sibling with higher PGS_{EXT} ($\beta = -0.091$, 95% CI = −0.172 ~ −0.010) in the family; and the twin/sibling with higher PGS_{EXT} was more likely to report less supportive parental relationships with their parents ($\beta = -0.096$, 95% CI = −0.184 ~ −0.008) relative to their co-twin/sibling. However, PGS_{EXT} was not significantly associated with delinquent peer affiliation ($\beta = 0.008$, 95% CI = −0.072 ~ 0.087) or school connectedness ($\beta = 0.050$, 95% CI = −0.041 ~ 0.142).

Between-to-within differences

Between- and within-family effects were not significantly different for parent-reported ($\Delta\beta = -0.005$, 95% CI = −0.115 ~ 0.106) and child-reported parental relationship ($\Delta\beta = 0.052$, 95% CI = −0.056 ~ 0.160) and for school connectedness ($\Delta\beta = -0.101$, 95% CI = −0.214 ~ 0.011). However, there was a significant attenuation in *r*GE from between- to within-family with respect to delinquent peer affiliation ($\Delta\beta = 0.185$, 95% CI = 0.084 ~ 0.286).

Discussion

By comparing a family-based design alongside a population sample, the current study explored whether *r*GEs observed between PGS_{EXT} and measured adolescent psychosocial environments are robust to family-level confounds, such as population stratification, assortative mating, and parental genotypes. At the population level, we found robust correlations between PGS_{EXT} and all measured environmental variables (i.e. parent- and child-reported parental relationships, delinquent peer affiliation, and school connectedness). When we estimated *r*GE at the between-family level, *r*GEs were generally consistent with those found in the population sample. When examined at the within-family level, however, *r*GEs were only observed with parental relationships but no other environments. We also found significant attenuation in *r*GE for delinquent peer affiliation, such that its within-family effect was much smaller than between-family effect. Our results provide new insights into the role confounders play in often-observed but still partially understood *r*GEs in the psychiatric and behavioral genetics literature.

First, and as expected, we found that *r*GEs between PGS_{EXT} and psychosocial environments are quite ubiquitous when examined at the population level and mostly present when examined at the between-family level. This is also consistent with previous studies that identified *r*GE using similar PGS and psychosocial environmental variables (de la Paz et al., 2023; Kuo et al., 2021; Li, 2019). We suspect that the slight differences between our findings at the population level and those at the between-family level may be due to demographic differences and smaller sample sizes in the family-based subsample (see Table 1).

When *r*GEs were examined at the within-family level, we only found *r*GEs with respect to parental relationships: the twin/sibling with a higher PGS_{EXT} was more likely to have a less supportive parental relationship than their co-twin/sibling. This finding was replicated by both self- and parent-reported data, suggesting that this result is robust to reporter bias. There was also no evidence of confounding, as the between- and within-family effects were not significantly different for parental relationships. One explanation is that PGS_{EXT} may contribute to the development of certain traits and characteristics (e.g. behavior problems, temperament) in adolescents, which in turn evokes less supportive parental relationships (de la Paz et al., 2023; Elam et al., 2017; Klahr et al., 2013;

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Kretschmer et al., 2022; Kuo et al., 2022; Marceau et al., 2013; Wang et al., 2017). To illustrate, adolescents with higher PGS_{EXT} may develop behavior problems that are perceived negatively by their parents, or perhaps make parenting more difficult (Akcinar & Baydar, 2016; Patterson, 2002). Prior work has also shown that difficult temperaments may contribute to negative parent-offspring dynamic (Ayoub et al., 2019). Genetic susceptibility to EXT may also explain some of the variability in response to parenting-based psychosocial interventions (Reiss & Leve, 2007).

Unlike what was observed in the population-based sample, the family-based analyses uncovered no *r*GE effects between PGS_{EXT} and delinquent peer affiliation. Instead, we found significant attenuation of *r*GE with delinquent peer affiliation from the between- to within-family level, suggesting that family-level confounders may contaminate *r*GEs observed at the population-level (Bornovalova et al., 2014; Klahr & Burt, 2014; Kuo et al., 2022). One source of confounding could involve broader, system-level influences that are associated with population differences, such as socioeconomic status and regional and neighborhood factors. For example, PGS_{EXT} may be associated with higher delinquent peer affiliation through the effect of poverty, urbanicity, or other social vulnerabilities, which are reflected in PGS_{EXT} due to population stratification and/or assortative mating (Foster et al., 2017). Additionally, PGS_{EXT} may reflect heritable traits and characteristics of the parents such as parental monitoring, which has been found to negatively associate with delinquent peer affiliation of their children (Elam et al., 2017; Li, Chen, Li, & Deater-Deckard, 2015). Finally, family-level psychosocial factors, such as parental psychopathology, are not differentially experienced by twins/siblings in a family. These factors may contribute to the association between genetic risks and delinquent peer affiliation at the population level, but go undetected in our family-based design.

Lastly, with respect to school connectedness, there was no evidence for *r*GE after confounding effects were controlled for. However, the within-family estimate did not significantly differ from the between-family estimate, such that there was no clear evidence of confounding either. This set of results may reflect imprecise *r*GE estimates due to the relatively smaller sample size for twins and full-siblings and correspondingly insufficient statistical power to draw robust conclusions.

In sum, our findings suggest that PGS-related *r*GE effects, though commonly observed in population-based studies, may offer different interpretations. Some *r*GEs (e.g. with parental relationship) may reflect the effect of offspring genotype on the environment. This possibility is consistent with the idea that genetic effects may be mediated by psychosocial environments (Elam et al., 2023). Other *r*GEs (e.g. with delinquent peer affiliation), however, may reflect a passive/confounding pathway, where offspring PGS and their environments are spuriously associated due to family/population-level confounders. Such confounding effects are challenging to control for in large population-based designs. As such, we advocate for the use of rigorous, family-based designs that account for population- and family-level confounders in conjunction with polygenic methods. For instance, our approach, which compares population, between-family, and within-family PGS-environment associations, could be extended to investigating other heritable psychiatric traits and to other family-based, genotyped cohorts. Because parental genotypes were not measured in this sample, our within-family design is limited to the offspring and controls for the combined influence of confounders (i.e. parental genotype, assortative mating, and population stratification). Parsing out specific sources of confounding may be an important future direction. As an example, extended family data with parent and offspring genotypes can be leveraged to isolate the specific contribution of parental genotype.

We acknowledge several other limitations to our study. First, we operationalized parental, peer, and school environments as unidimensional constructs. Not only are these constructs not necessarily unidimensional, but there are also other environments across development that could have been examined. Next, we acknowledge that our within-family design may be subject to some residual confounding effects – such as due to bias in reporting processes. Twins and siblings in the same family may also imitate, contrast, or otherwise influence each other through family dynamics, leading to under-/over-estimation of within-family effects (Carey, 1986; Jensen & McHale, 2017). Additionally, our family-based design limited us to a relatively small within-family subsample, contributing to a greater degree of uncertainty in estimating *r*GE effects relative to our larger population subsample of unrelated individuals. This is further complicated by our use of PGS, which generally has modest effects. Our design therefore cannot rule out the possibility of true *r*GE effects that operate through aspects of genetic liability not captured by the current PGS. Finally, our family-based model also controls for factors that are shared by twins/siblings and may fail to capture true *r*GE effects that act on shared family environments. Future research should aim to replicate these findings using other family-based designs, such as with genotyped parent-offspring trios.

Supplementary material. The supplementary material for this article can be found at http://doi.org/10.1017/S0033291726106242.

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Ethical standards. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008. This study was approved by the University of Wisconsin-Madison Institutional Review Board (UW-Madison IRB# 2015-1189). Consent to participate was obtained by the Add Health study.

Transparency and openness. Our analyses were pre-registered at https://osf.io/3s5tx. Restricted-access data from the Add Health study are available at: https://addhealth.cpc.unc.edu/data/#restricted-use. Analysis codes are available at https://github.com/lichendong/2024_within_fam_rge. We report how we determined all data exclusions, all manipulations, and all measures in the current study.

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