The Journal of Child Psychology and Psychiatry

Journal of Child Psychology and Psychiatry **:* (2024), pp **–** doi:10.1111/jcpp.13956

Genetic influences on sibling bullying and mental health difficulties

Umar Toseeb,[1] John Vincent,[2] and Kathryn Asbury[1]

[1]Department of Education, University of York, York, UK; [2]Institute of Psychiatry Psychology and Neuroscience, Social Genetic and Developmental Psychiatry Centre, King's College London, London, UK

Background: Sibling bullying is associated with mental health difficulties; both in the short and long term. It is commonly assumed that sibling bullying leads to mental health difficulties but additional explanations for the relationship between the two are seldom investigated. **Methods:** To address this gap in knowledge, we used a genetically sensitive design with data from the Avon Longitudinal Study of Parents and Children (maximum $N = 3{,}959$, 53% female). At ages 11–13 years, individuals self-reported their involvement in sibling bullying, as a victim and perpetrator, and parents reported on their child's mental health difficulties. Polygenic scores, indices of genetic risk for psychiatric disorders (major depressive disorder and attention deficit hyperactivity disorder) were computed using children's genetic data. Regression and structural equation models were fitted to the data. **Results:** Sibling bullying, victimisation and perpetration, and polygenic scores both predicted mental health difficulties in an additive manner but there was no interaction between them. Polygenic scores for mental health difficulties were also associated with sibling bullying. **Conclusions:** These findings suggest that sibling bullying, victimisation and perpetration, is associated with mental health difficulties, even after accounting for some genetic effects. Additionally, the relationship between sibling bullying and mental health difficulties may be, at least partly, due to shared genetic aetiology. One possibility is that genetic risk for mental health difficulties influences the onset of mental health difficulties which in turn make children more susceptible to sibling bullying. **Keywords:** Sibling bullying; mental health; ALSPAC; childhood; genetics.

Introduction

Sibling bullying and its prevalence

Sibling bullying is ubiquitous in families throughout the world (Bowes, Wolke, Joinson, Lereya, & Lewis, 2014; Deniz et al., 2023; Liu et al., 2020; Lopes, Relva, & Fernandes, 2019; Sabah, Aljaberi, & Lin, 2022; Toseeb, Deniz, & Noret, 2023; Tucker, Finkelhor, Shattuck, & Turner, 2013). It is defined as 'any unwanted aggressive behaviour(s) by a sibling that involves an observed or perceived power imbalance and is repeated multiple times or is highly likely to be repeated; bullying may inflict harm or distress on the targeted sibling, including physical, psychological, or social harm' (Wolke, Tippett, & Dantchev, 2015, p. 918). In the United Kingdom, up to half of children report being involved in sibling bullying as a victim, perpetrator, or both (Toseeb, McChesney, & Wolke, 2018). This decreases to around a third as children grow older and enter adolescence (Toseeb, McChesney, Oldfield, & Wolke, 2020). Therefore, it is necessary to investigate the aetiological influences on sibling bullying and the reasons why it might be associated with negative outcomes in an effort to reduce its incidence and potential consequences.

Correlates of sibling bullying

Evidence of the circumstances and personal characteristics that make some children vulnerable to sibling bullying is starting to emerge, specifically in the UK and USA. Some of these are common across victimisation and perpetration. For example, the number of siblings in the household is associated with both victimisation and perpetration (Toseeb, McChesney, Dantchev, & Wolke, 2020). Similarly, having a disability is associated with increased victimisation and perpetration (Toseeb et al., 2018; Tucker, Finkelhor, & Turner, 2017). Other factors are associated with *either* victimisation *or* perpetration. Males are more likely to be perpetrators and females are more likely to be victims, older siblings are more likely to be perpetrators and younger siblings are more likely to be victims, perpetrators tend to have high levels of emotional dysregulation, and children who are victimised by peers are at increased risk of being victimised by siblings (Dantchev & Wolke, 2019; Toseeb, McChesney, Dantchev, & Wolke, 2020; Toseeb, McChesney, Oldfield, & Wolke, 2020). Additionally, victims of sibling bullying are more likely to be victims of peer bullying (Tucker, Finkelhor, & Turner, 2019); indeed both sibling and peer bullying victimisation have some common correlates (Tucker, Finkelhor, & Turner, 2020). Cross-nationally, a number of correlates of sibling bullying victimisation have been identified. Whilst the exact correlates differ depending on country, they broadly fall into individual- (e.g.

Conflict of interest statement: No conflicts declared.

sex), family- (e.g. resources, functioning, etc.), peer- (e.g. friendships), neighbourhood- (e.g. feeling safe, belonging, etc.), and school-level (e.g. bullying, positive school environments, etc.) correlates (Toseeb, Deniz, & Noret, 2023). A key limitation of previous work is that it only considers behavioural correlates of sibling bullying without consideration of genetic confounding. This is problematic because all behaviours are at least partly heritable and result from a complex interplay between genetic and environmental effects (Turkheimer, 2000). Therefore, to fully understand the aetiology of sibling bullying, genetic influences need to be considered.

Sibling bullying and mental health

A number of investigations have focussed on the correlates and prospective outcomes associated with sibling bullying; the most common being mental health. Sibling bullying is associated with a number of adverse mental health outcomes both concurrently and in the longer term. Cross-sectional studies demonstrate that sibling bullying is associated with symptoms of depression, anxiety, and behavioural disorders (Liu et al., 2020; Lopes et al., 2019; Toseeb et al., 2018; Tucker et al., 2013). There is also evidence for prospective longitudinal relationships. Children who are involved in sibling bullying during childhood are more likely to subsequently experience symptoms of psychosis, depression, anxiety, and suicidal ideation (Bowes et al., 2014; Dantchev, Zammit, & Wolke, 2018), to have low levels of well-being and self-esteem (Toseeb & Wolke, 2022), and to engage in high-risk behaviours in late adolescence (Dantchev & Wolke, 2018), compared with those who are not involved in sibling bullying. The mechanisms through which these potentially negative effects might manifest are becoming apparent. For example, in some children, sibling bullying appears to be followed by a reduction in self-esteem, which is associated with subsequent mental health difficulties (Deniz & Toseeb, 2023). This might explain why some children who are involved in sibling bullying go onto experience poor mental health whereas others do not. Such investigations of the relationship between sibling bullying and mental health are limited as they do not consider genetic effects. This is problematic because mental health difficulties are influenced by the interplay between genetic and environmental factors (Allegrini et al., 2020).

Behavioural genetics

Behavioural genomic methods allow for the investigation of genetic influences on a given behavioural trait or disorder. Genetic risk for a given trait can be calculated by aggregating the effects of common genetic variants, such as single-nucleotide polymorphisms, to a polygenic score (PGS). Samples of hundreds of thousands of individuals have been used to develop PGSs for common mental health difficulties, such as major depressive disorder, anxiety, and attention deficit hyperactivity disorder (Demontis et al., 2019; Howard et al., 2019; Purves et al., 2020). A key finding from behavioural genomics research is that genetic influences tend to be general rather than specific. That is, PGSs for one type of mental health difficulty (e.g. emotional problems) are likely to also be associated with another type of mental health difficulty (e.g. behavioural problems), given that common mental health difficulties share genetic aetiology (Brikell et al., 2020).

Such general genetic effects have also provided insight into the aetiology of some of the correlates of mental health difficulties and potentially point towards genetic mediation effects. For example, PGSs for common mental health difficulties are associated with *peer* bullying (Schoeler et al., 2019). This suggests that *peer* bullying and mental health difficulties might share genetic aetiology. This is important because it provides additional insights into the possible mechanisms through which *sibling* bullying and mental health difficulties might be related with at least three possibilities. First, that sibling bullying causes mental health difficulties. Second, that experiencing mental health difficulties make children more susceptible to being bullied or bullying their siblings. Third, that both bullying and mental health difficulties are influenced by a common, usually unmeasured, factor, in this case, genetics. The most likely possibility is that it is a combination of the three.

The investigation of such gene–environment interplay has yielded mixed findings. Theoretically, environmental stressors (e.g. sibling bullying) may trigger pre-existing genetic vulnerabilities for mental health difficulties (Diathesis stress model: Zuckerman, 1999). Alternatively, genetically influenced phenotypic manifestation of mental health difficulties may lead to sibling bullying (i.e. evocative gene–environment interplay). In empirical work, however, the evidence is mixed. For example, in adults, stressful life events and childhood trauma interact with polygenic scores to predict phenotypic expression of depression (Colodro-Conde et al., 2018; Peyrot et al., 2014). But in some recent work with adolescents, there was no interaction between polygenic scores and *peer* bullying (Armitage, Wang, Davis, & Haworth, 2022), suggesting that those with high genetic risk for depression do not fair worse when involved in bullying compared with those with low genetic risk for depression. The mixed findings from empirical studies necessitate the investigation of gene–environment interplay in relation to sibling bullying and mental health difficulties.

The current study

Our investigation addressed these gaps in the literature. To the best of our knowledge, there has

not been an investigation of whether there are common genetic influences on both sibling bullying and mental health difficulties. Doing this will allow for more nuanced understanding of the aetiology of sibling bullying, why it is correlated with mental health difficulties, and why some children experience mental health difficulties when involved in sibling bullying. It is necessary to investigate sibling bullying separate to peer bullying for two reasons. First, the precursors and correlates of sibling bullying are somewhat unique. For example, the number of siblings and birth order are associated with sibling but not peer bullying (Dantchev & Wolke, 2019; Toseeb, McChesney, Dantchev, & Wolke, 2020). Second, sibling and peer bullying are independently associated with mental health difficulties (Dantchev, Hickman, Heron, Zammit, & Wolke, 2019), suggesting that their aetiologies might also be unique. We addressed this gap in knowledge by investigating associations between genetic vulnerability for mental health difficulties, sibling bullying, and the phenotypic manifestation of mental health difficulties in early adolescence.

We answered two research questions. First, does genetic risk for mental health difficulties moderate the relationship between sibling bullying and mental health difficulties? (research question 1). In line with the diathesis stress model (Zuckerman, 1999), we expected that individuals with a high genetic propensity for mental health difficulties, who are also involved in sibling bullying, will experience more mental health difficulties compared with those with a high genetic propensity for mental health difficulties who are not involved in sibling bullying. Second, we were interested in whether genetic vulnerability for mental health difficulties is associated with both sibling bullying and mental health difficulties? (research question 2). Given that genetic effects tend to be general, rather than specific, and the expectation that there may be some genetic mediation at play, we expected genetic vulnerabilities for mental health difficulties to be associated with both sibling bullying and mental health difficulties.

Method

Ethical approvals

The study was a secondary analysis of existing data from the Avon Longitudinal Study of Parents and Children (ALSPAC). Ethical approval for data collection was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committees (Health Authority). Informed consent for the use of data collected via questionnaires and clinics was obtained from participants following the recommendations of the ALSPAC Ethics and Law Committee at the time. Consent for biological samples has been collected in accordance with the Human Tissue Act (2004). Full details of ethics processes can be accessed on the ALSPAC webpage (http://www.bristol.ac.uk/alspac/researchers/research-ethics/). No further ethical approval was sought for the secondary analysis of existing data from the ALSPAC cohort; this is in line with the recommendations of Education Ethics Committee at the University of York.

Sample

Pregnant women resident in Avon, UK with expected dates of delivery between 1st April 1991 and 31st December 1992 were invited to take part in the study. The initial number of pregnancies enrolled was 14,541, of which 13,988 children were alive at 1 year of age. Parents and children provided biological samples, questionnaire data and took part in direct assessments. Full details of the cohort are reported elsewhere (Boyd et al., 2013; Fraser et al., 2013). The study website contains details of all the data available and provides a fully searchable data dictionary and variable search tool (http://www.bristol.ac.uk/alspac/researchers/our-data/).

We applied the following inclusion criteria; child responded to questionnaire about sibling bullying at age 12 years; had at least one sibling; and provided genetic data. Therefore, the maximum sample size was 3,959 (53% female) but the achieved sample size varied depending on the statistical model. In terms of siblings, 1,440 (36%) had at least one older sibling, 1,510 (38%) had at least one younger sibling, and 487 (12%) were the first born child. In terms of family background, 727 (19%) of mothers and 417 (11%) of fathers had a university-level education, and 3,232 (86%) of families owned their home. The analysis sample included a higher proportion of females, white children, university education parents, and higher levels of home ownership than the sample excluded from the analysis (see Table S1).

Genotyping and quality control

All children in the ALSPAC sample were genotyped using the Illumina Human Hap 550-quad genotyping array at the Wellcome Trust Sanger Institute, Cambridge, UK and the Laboratory Corporation of America, Burlington, NC, US. The resulting genome-wide data were then subjected to standard quality control. This included excluding individuals due to sex mismatches, minimal or excessive heterozygosity, missingness of >3% and identity by descent of <0.8. Population stratification was assessed by multidimensional scaling analysis and all individuals with non-European ancestry were removed, as genetic effects on any given trait vary across ancestral groups due to differences in linkage disequilibrium (LD) structure. SNPs with a minor allele frequency (MAF) of <1% and a call rate of <95% were removed, as were those that violated Hardy–Weinberg equilibrium ($p < 5 \times 10^{-7}$). Cryptic relatedness was measured as proportion of identity by descent (IBD >0.1). Related individuals that passed all other quality control thresholds were retained during subsequent phasing and imputation. Imputation was then performed using Impute v3 software and the HRC (HRC Version 1.1) imputation reference panel. Further quality control was then conducted with exclusion criteria applied for SNPs with MAF <0.01, imputation INFO scores <0.8 and call rates of <95%. Any violation from Hardy–Weinberg equilibrium ($p < 5 \times 10^{-7}$) was also assessed. Initially, there were 38,898,739 SNPs. Following quality control processes, a total of 6,774,469 genotyped and imputed SNPs remained for analysis.

Measures

Parents and children were mailed questionnaires in a postal campaign and were asked to answer questions on sibling bullying and mental health difficulties as part of a wider battery of measures.

Sibling bullying. When participants were 12 years old, they were asked to report their involvement in sibling bullying

using an adapted version of the Olweus Bullying Questionnaire (Olweus, 2007). Participants were told that sibling bullying is:

> when a brother or sister tries to upset you by saying nasty and hurtful things, or completely ignores you from their group of friends, hits, kicks, pushes or shoves you around, tells lies or makes up false rumours about you.

They were then asked six questions about how often they had been bullied by a sibling: hit, kicked, pushed or shoved (a); belongings taken or damaged (b); called names (c); made fun of (d); ignored or left out (e); and sibling told lies or spread rumours (f). They responded on a five-point scale (0 = *never*, 1 = *only ever once or twice*, 2 = *two or three times a month*, 3 = *about once a week*, 4 = *several times a week*). These questions were then repeated for sibling bullying perpetration. Established cut-offs were used (i.e. about once a week or more) to create binary bullying variables for each of the types of bullying (see Bowes et al., 2014; Toseeb et al., 2018). These variables were used to generate an additional two variables: sibling bullying victimisation and sibling bullying perpetration. For example, anyone who responded 'about once a week' to one question was given a score of 1, those who responded 'about once a week' to two questions were given a score of 2, and so on. This meant that each participant was given two scores of between 0 and 6 (a score for victimisation and another for perpetration) capturing the frequency and mode of bullying victimisation and perpetration; higher scores indicated more frequent bullying and using more modes of bullying.

Mental health difficulties. The parent-reported Strengths and Difficulties questionnaire (SDQ: Goodman, 1997) was used to screen for mental health difficulties when the child was 11 and 13 years old. Three subscales of the SDQ were used: emotional problems (e.g. 'often unhappy, downhearted'); conduct problems (e.g. 'often has temper tantrums or hot tempers'); and hyperactivity (e.g. 'constantly fidgeting or squirming'). In line with the psychopathology factor, we conceptualised both emotional and behavioural difficulties as different outward manifestations of underlying distress (Patalay et al., 2018). Parents responded on a three-point scale (0 = *not true*, 1 = *somewhat true*, 2 = *certainly true*). Each subscale consisted of five questions so sum scores ranged from 0 to 10, with higher scores representing more difficulties. The SDQ has good reliability in the ALSPAC sample (Speyer, Auyeung, & Murray, 2022). Given that SDQ data were not available at age 12 years to align with the sibling bullying data, we generated mean scores of SDQ subscales at ages 11 and 13 years. Then, we summed the scores on all three subscales to create a 'total problems' variables, which ranged from 0 to 15; higher scores indicated more mental health difficulties.

Polygenic scores (PGSs). Initially, two sets of PGSs were generated: major depressive disorder (MDD, Howard et al., 2019) and attention deficit hyperactivity disorder (ADHD, Demontis et al., 2019). Full details of how these were generated are reported elsewhere (Toseeb, Vincent, Oginni, Asbury, & Newbury, 2023). In short, summary statistics were accessed and subject to standard quality control procedures, before generating PGSs for each individual within the current sample. Next, associations with the SDQ subscales at a range of *p*-value thresholds ranging from .001 to 1 were tested. The *p*-value threshold of .3 was consistently significant across both PGSs (MDD and ADHD) in predicting SDQ scores at a range of ages between 7 and 16 years and increasing the threshold did not explain more variance. Principal components were included as covariates when creating the PGSs to control for population stratification. Therefore, PGSs were created at the .3 threshold and taken forward. For use in the analysis, we generated a 'combined PGS' by summing the MDD PGS and ADHD PGS; such an additive approach is in line with previous work (Schoeler et al., 2019).

Statistical analysis

The analysis was conducted in STATA/MP 17.0 (StataCorp, 2021) using complete cases. To address research question 1, two sets of regression models were fitted using a robust estimator. In the first set of models (models 1–4), the predictors were sibling bullying victimisation, combined PGS, sibling bullying victimisation by combined PGS interaction, and sex as a covariate. The outcome variable differed depending on the model: emotional problems (model 1), conduct problems (model 2), hyperactivity (model 3), and total problems (model 4). Given that PGSs best predict outcomes at the extremes, we generated tertiles of the combined PGS and focussed only on the upper and lower tertiles. This resulted in a binary variable (0 = *lowest one third combined PGS*, 1 = *highest one third combined PGS*). Given that much of the previous work demonstrates an effect at further extremes (i.e. the lowest 10% vs. the highest 10%), our decision to use tertiles was pragmatic as we were underpowered to run such a centile analysis; it would mean a combined sample size of ~750. Models 5–8 were a repeat of models 1–4 except that the continuous PGS was replaced with a binary PGS variable. The second set of models (models 9–16) were a repeat of models 1–8 except that sibling bullying victimisation was replaced with sibling bullying perpetration. To address research question 2, two structural equation models (*SEM*) were fitted using maximum likelihood estimation (*SEM* 1 for sibling bullying victimisation and *SEM* 2 for sibling bullying perpetration). Structural and measurement models were fitted concurrently. The measurement models consisted of two latent factors: sibling bullying (victimisation for *SEM* 1 and perpetration for *SEM* 2) and mental health difficulties (emotional problems, conduct problems, and hyperactivity). The structural model estimated the shared genetic influences on mental health difficulties and sibling bullying. The analysis was not preregistered.

Results

Descriptive statistics for the prevalence of sibling bullying and mental health difficulties are shown in Table 1. Approximately, a quarter of children reported being bullied by their siblings (once a week or more). Slightly less than a fifth (17%) reported bullying their siblings (once per week or more). The most common type of bullying was name calling (13%–19%), followed by making fun of/being made fun of (8%–15%) and physical aggression (10%–14%). Of all the children involved in sibling bullying (N = 1,173), just under half (N = 506) were both victims and perpetrators, whilst the remainder were either victims (N = 482) or perpetrators (N = 185).

Genetic risk as a moderator

To determine whether genetic risk for common psychiatric disorders (i.e. the combined PGS) moderates the relationship between sibling bullying and mental health, a series of regression models were fitted (see continuous PGS models in Tables 2 and 3). There was a consistent pattern of findings across all models. Sibling bullying (victimisation and

Table 1 Descriptive statistics for sibling bullying and mental health difficulties

	At least once per week *N* (%)
Sibling bullying victimisation	
Total sibling bullying victimisation	988 (25%)
Hit, kicked, pushed, or shoved by sibling	554 (14%)
Things damaged or taken by sibling	107 (3%)
Called names by sibling	735 (19%)
Made fun of by sibling	575 (15%)
Ignored or left out of sibling's games	189 (5%)
Sibling told lies or spread rumours about child	148 (4%)
Sibling bullying perpetration	
Total sibling bullying perpetration	691 (17%)
Hit, kicked, pushed, or shoved sibling	410 (10%)
Took money or other things from sibling	33 (1%)
Called sibling nasty and hateful names	514 (13%)
Made fun of sibling	307 (8%)
Ignored or left sibling out of games	124 (3%)
Spread rumours or tried to make others dislike sibling	30 (1%)

Sibling bullying roles	Prevalence
Uninvolved	2,786 (70%)
Victim-only	482 (12%)
Bully-only	185 (5%)
Bully-victim	506 (13%)

Parent-Report Mental Health Difficulties	Mean (*SD*)
Emotional problems	1.38 (1.50)
Conduct problems	1.17 (1.26)
Hyperactivity	2.66 (2.00)

Parent-report mental health difficulties represent the mean of age 11 and 13 years. *N*, number of children; *SD*, standard deviation.

perpetration) was associated with emotional problems, conduct problems, hyperactivity, and total problems (main effect of bullying). The combined PGS was also associated with a range of mental health difficulties (main effect of PGS). The relationship between sibling bullying and mental health difficulties did not vary as a function of the combined PGS (lack of interaction effects). As expected, females fared worse on emotional problems and males fared worse on conduct problems and hyperactivity. This pattern of results did not change when the combined PGS was converted into a binary variable (see binary PGS models in Tables 2 and 3). That is, both sibling bullying, victimisation and perpetration, and genetic risk for mental health difficulties are associated with mental health difficulties but sibling bullying does not exacerbate genetic risk for mental health difficulties.

Shared genetic influences on sibling bullying and mental health

To test whether both sibling bullying and mental health difficulties are associated due to shared genetic influences, two *SEM*s were fitted (Figure 1 victimisation and Figure 2 perpetration). The combined PGS was associated with both sibling bullying, victimisation and perpetration, and mental health difficulties, albeit to a lesser extent with the former. Even after accounting for some shared genetic influence, sibling bullying and mental health difficulties were still associated. That is, sibling bullying, victimisation and perpetration, and mental health difficulties co-occur only *partly* due to common genetic influences; the possibility of causal influences, in either direction, remains open.

Sensitivity analysis

We carried out a number of sensitivity analyses for both research questions. For these analyses, we replaced parent-report SDQ with either teacher-report SDQ (age 11) or self-report depression symptoms (Costello & Angold, 1988). These are reported in the supporting information (see Models 17–36 in Tables S2 and S3 and Figures S1–S3). For teacher-report SDQ, the general pattern of findings held for conduct problems and hyperactivity but not for emotional problems (likely due to poor factor loading of the emotional problems subscale). The findings held more consistently for self-report depression symptoms.

Discussion

Summary of key findings

In the current study, we used a genetically sensitive design to investigate the relationship between sibling bullying, victimisation and perpetration, and mental

Table 2 Predicting sibling bullying victimisation using polygenic scores for common psychiatric disorders

Predictor PGS	Main effect bullying b [95% CI]	Main effect PGS b [95% CI]	Bullying × PGS interaction b [95% CI]	Covariate sex b [95% CI]
Continuous PGS models				
Model 1: Emotional problems	0.11 [0.06, 0.15]***	0.06 [0.03, 0.10]***	0.00 [−0.03, 0.04]	0.33 [0.23, 0.42]***
Model 2: Conduct problems	0.15 [0.11, 0.20]***	0.08 [0.05, 0.11]***	0.00 [−0.03, 0.03]	−0.17 [−0.25, −0.09]***
Model 3: Hyperactivity	0.16 [0.11, 0.22]***	0.14 [0.09, 0.18]***	0.02 [−0.02, 0.06]	−0.88 [−1.00, −0.76]***
Model 4: Total problems	0.43 [0.31, 0.54]***	0.28 [0.20, 0.36]***	0.02 [−0.06, 0.10]	−0.72 [−0.95, −0.49]***
Binary PGS models				
Model 5: Emotional problems	0.14 [0.06, 0.23]**	0.22 [0.09, 0.34]**	−0.03 [−0.14, 0.09]	0.38 [0.26, 0.50]***
Model 6: Conduct problems	0.17 [0.09, 0.26]***	0.24 [0.14, 0.34]***	−0.02 [−0.12, 0.09]	−0.14 [−0.25, −0.05]**
Model 7: Hyperactivity	0.17 [0.06, 0.27]**	0.40 [0.23, 0.56]***	0.02 [−0.13, 0.16]	−0.88 [−1.03, −0.72]***
Model 8: Total problems	0.49 [0.28, 0.69]***	0.86 [0.56, 10.17]***	−0.03 [−0.32, −0.26]	−0.64 [−0.93, −0.35]***

*$p < .05$, **$p < .01$, ***$p < .001$. Values are unstandardised beta [95% confidence intervals]. PGS, combined polygenic score.

Table 3 Predicting sibling bullying perpetration using polygenic scores for common psychiatric disorders

Predictor PGS	Main effect bullying b [95% CI]	Main effect PGS b [95% CI]	Bullying × PGS interaction b [95% CI]	Covariate sex b [95% CI]
Continuous PGS models				
Model 9: Emotional problems	0.12 [0.06, 0.19]***	0.06 [0.02, 0.09]**	0.02 [−0.02, 0.06]	0.35 [0.25, 0.44]***
Model 10: Conduct problems	0.26 [0.20, 0.32]***	0.08 [0.05, 0.10]***	0.01 [−0.02, 0.04]	−0.14 [−0.21, −0.06]***
Model 11: Hyperactivity	0.21 [0.14, 0.30]***	0.14 [0.10, 0.19]***	0.01 [−0.04, 0.06]	−0.85 [−0.98, −0.73]***
Model 12: Total problems	0.60 [0.44, 0.76]***	0.28 [0.19, 0.36]***	0.05 [−0.05, 0.15]	−0.64 [−0.86, −0.41]***
Binary PGS models				
Model 13: Emotional problems	0.11 [0.02, 0.22]*	0.16 [0.04, 0.29]**	0.11 [−0.04, 0.27]	0.41 [0.29, 0.53]***
Model 14: Conduct problems	0.27 [0.16, 0.37]***	0.21 [0.11, 0.31]***	0.04 [−0.11, 0.19]	−0.10 [−0.20, −0.00]*
Model 15: Hyperactivity	0.25 [0.11, 0.39]**	0.39 [0.23, 0.56]***	0.03 [−0.17, 0.23]	−0.84 [−0.99, −0.68]***
Model 16: Total problems	0.62 [0.36, 0.88]***	0.77 [0.48, 10.07]***	0.19 [−0.21, −0.58]	−0.52 [−0.81, −0.24]***

*$p < .05$, **$p < .01$, ***$p < .001$. Values are unstandardised beta [95% Confidence Intervals]. PGS, combined polygenic score.

health difficulties in early adolescence. We found that (a) *both* sibling bullying *and* genetic risk for mental health difficulties are additively associated with mental health difficulties, (b) genetic risk for mental health difficulties does not moderate the relationship between sibling bullying and mental health difficulties, and (c) sibling bullying and mental health difficulties co-occur, in part, due to shared genetic influences. Our findings make a unique contribution to the literature as we provide, for the first time using a genetically sensitive design, evidence for an additional explanation for why sibling bullying and mental health difficulties are related. We demonstrate that whilst sibling bullying might lead to mental health difficulties (or vice versa), shared genetic influences also contribute to the relationship between sibling bullying and mental health difficulties. In the subsequent sections, we discuss these findings with reference to previous work.

Sibling bullying and genetics are associated with mental health difficulties

In line with expectations, we found that sibling bullying is associated with mental health difficulties. This remains the case even after accounting for some genetic risk for mental health difficulties. This suggests that both sibling bullying, irrespective of whether it is victimisation or perpetration, and

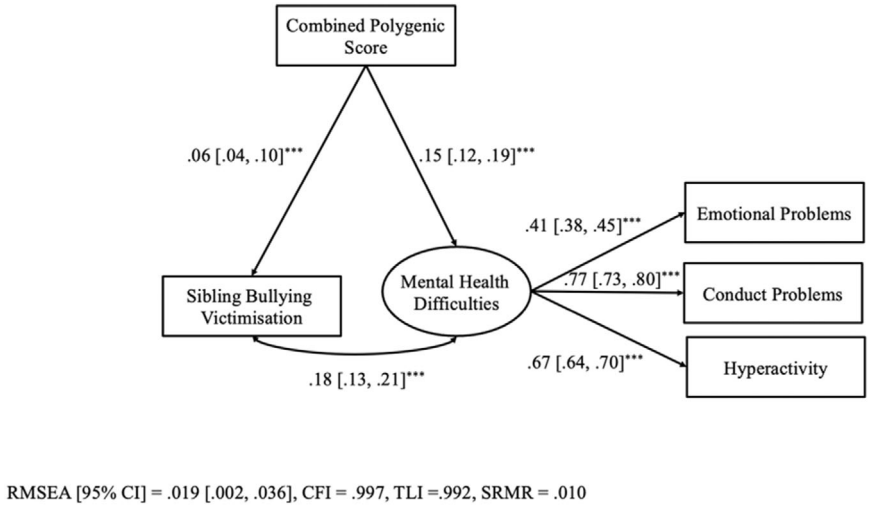

Figure 1 Common genetic influences on sibling bullying victimisation and mental health difficulties

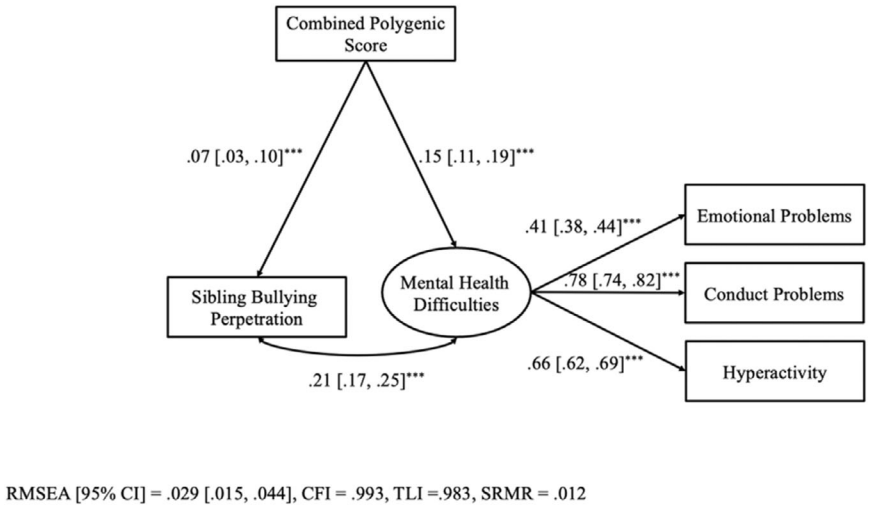

Figure 2 Common genetic influences on sibling bullying perpetration and mental health difficulties

genetic risk for mental health difficulties explain unique variance in mental health difficulties during early adolescence. This is important as it adds weight to the well-established literature suggesting that both genetic and environmental factors influence mental health difficulties (Allegrini et al., 2020). These relationships appear to be similar for sibling bullying victimisation and perpetration, suggesting that similar influences may be at play for both.

The lack of gene–environment interplay in the relationship between sibling bullying and mental health difficulties does not support our hypothesis. We predicted that mental health difficulties associated with sibling bullying would vary as a function of genetic risk for mental health difficulties. If such an effect had been observed, it would have been in line with gene–environment interplay theoretical frameworks. That is, genetic propensities may affect how individuals respond to environmental stressors or they may help to shape environments (evocative gene–environment interplay). We propose three explanations for the lack of observed effect. First, PGSs best predict outcomes at extreme ends. We attempted to model this by creating tertiles but much of the previous work demonstrates an effect at further extremes (i.e. the lowest 10% vs. the highest 10%); we were underpowered to run such a centile analysis; it would mean a combined sample size of ~750. We suspect that with a larger sample size, we would be better powered to better detect moderation effects. The second possibility is that the measure of mental health difficulties was not taken at the same time as the sibling bullying measure (it was an aggregate of a year before and a year after). It may be that genetic risk moderates immediate psychological distress, which we were not able to capture. These remain open possibilities that should be tested in future research. Finally, other recent attempts at investigating gene–environment interplay using PGSs have similarly not found effects (Armitage et al., 2022; Klingenberg et al., 2023). This may be indicative of the limitations of PGSs for investigating

the effects of gene–environment interplay given they only represent a proportion of genetic variation. Alternatively, the effects may simply be cumulative. That is, genetic and environmental influences may not interact but instead may simply have an additive effect on phenotypic outcomes.

Shared genetic influences

A particularly novel aspect of our work was the investigation of whether common genetic effects influence both sibling bullying and mental health difficulties; we found this to be true for both victimisation and perpetration. We speculate that these findings may be due to an evocative gene–environment correlation. For example, children with high genetic propensities for depression may develop symptoms of depression and thus become vulnerable to victimisation. Furthermore, children with high genetic propensity for conduct problems might be more likely to be hot tempered or fight with their siblings leading to bullying perpetration. This challenges common assumptions around the causal direction of the relationship between sibling bullying and mental health difficulties. Our findings do not exclude the possibility that sibling bullying leads to mental health difficulties, instead, we provide evidence for the possibility that sibling bullying and mental health difficulties are partly related because they are both influenced by a common set of genetic influences. Future work should test the direction of these effects (e.g. mediated pleiotropy) and consider whether a common set of environmental factors (and their interplay with genetic risk) influence both sibling bullying and mental health difficulties. This could lead to the possibility of a single set of interventions improving mental health difficulties and reducing the incidence of sibling bullying.

Strengths, limitations, and directions for future research

Our study was conducted with a large community sample. A particular strength of our work was the combination of genetic data with behavioural data. Our findings should, however, be viewed with a number of limitations in mind. First, we did not account for the multiple other factors that might influence the association between sibling bullying and mental health difficulties (e.g. peer bullying, socioeconomic status etc.). Second, we used data from a largely affluent northern European sample, which is problematic because the prevalence and correlates of sibling bullying vary across the world (Toseeb, Deniz, & Noret, 2023) and also because genetic effects can depend upon environmental influences. Future work should investigate these effects with a range of environmental confounders across different cultural contexts. Third, we did not distinguish biological siblings from non-biological siblings and thus the genetic correlation might be inflated; although this is likely to be minimal as we expect that most of the sample were reporting on biological siblings. Fourth, our index of genetic propensity, the combined PGS, is limited because it does not capture all genetic variation when compared with twin studies (i.e. missing heritability), and like all PGSs, some variation it explains may be indirect effects. Therefore, it does not provide any indication of the upper or lower bounds of genetic influences. Finally, the *SEM*s that we fitted do not include the multiple other influences on sibling bullying, mental health difficulties, and the relationship between the two. These are all correlated with individual (e.g. sex, disabilities, birth order, pre-existing mental health difficulties, well-being), family (e.g. single-parent families, socioeconomic status) and wider environmental factors (e.g. neighbourhood deprivation). Future work should model these relationships more comprehensively, for example, using network analysis (see Bjørndal, Ebrahimi, Lan, Nes, and Røysamb (2023)).

Conclusions

We report a genetically sensitive investigation of the relationship between sibling bullying and mental health difficulties. Our findings indicate that both sibling bullying and genetic propensities for mental health difficulties are associated with mental health difficulties in early adolescence. We also demonstrate that in addition to sibling bullying and mental health difficulties being directly associated, they are at least partly related due to shared genetic influences.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Table S1 Comparison of Analysis Sample with Remainder of ALSPAC Sample.

Table S2 Predicting Sibling Bullying Victimisation Using Polygenic Scores for Common Psychiatric Disorders.

Table S3 Predicting Sibling Bullying Perpetration Using Polygenic Scores for Common Psychiatric Disorders.

Figure S1. Common Genetic Influences on Sibling Bullying Victimisation and Mental Health Difficulties (Teacher-Report).

Figure S2. Common Genetic Influences on Sibling Bullying Perpetration and Mental Health Difficulties (Teacher-Report).

Figure S3. Common Genetic Influences on Sibling Bullying Victimisation and Perpetration and Depression Symptoms (Self-Report).

Acknowledgements

The UK Medical Research Council and Wellcome (Grant ref: 217065/Z/19/Z) and the University of Bristol

provide core support for ALSPAC. This publication is the work of the authors and they will serve as guarantors for the contents of this paper. GWAS data were generated by Sample Logistics and Genotyping Facilities at Wellcome Sanger Institute and LabCorp (Laboratory Corporation of America) using support from 23andMe. A comprehensive list of grants funding is available on the ALSPAC website (http://www.bristol.ac.uk/alspac/external/documents/grant-acknowledgements.pdf). The authors are extremely grateful to all the families who took part in this study, the midwives for their help in recruiting them, and the whole ALSPAC team, which includes interviewers, computer and laboratory technicians, clerical workers, research scientists, volunteers, managers, receptionists and nurses. The authors have declared that they have no competing or potential conflicts of interest.

Data availability statement

Information on how to access the ALSPAC data can be found here: http://www.bristol.ac.uk/alspac/researchers/access/.

Correspondence

Umar Toseeb, Department of Education, University of York, York YO10 5DD, UK; Email: umar.toseeb@york.ac.uk

Key points

- Previous work shows that sibling bullying is associated with poor mental health but genetic confounds have not been considered.
- We found that sibling bullying is associated with parent-report mental health difficulties, even after controlling for some genetic effects.
- Additionally, we found that sibling bullying and parent-report mental health difficulties are influenced by common genetic factors.
- Future work should consider whether a common set of environmental factors (and their interplay with genetic risk) influence both sibling bullying and mental health difficulties. This could lead to the possibility of a single set of interventions improving mental health difficulties and reducing the incidence of sibling bullying.

References

Allegrini, A.G., Cheesman, R., Rimfeld, K., Selzam, S., Pingault, J.B., Eley, T.C., & Plomin, R. (2020). The p factor: Genetic analyses support a general dimension of psychopathology in childhood and adolescence. *Journal of Child Psychology and Psychiatry*, *61*, 30–39.

Armitage, J.M., Wang, R.A.H., Davis, O.S.P., & Haworth, C.M.A. (2022). A polygenic approach to understanding resilience to peer victimisation. *Behaviour Genetics*, *52*, 1–12.

Bjørndal, L.D., Ebrahimi, O.V., Lan, X., Nes, R.B., & Røysamb, E. (2023). Mental health and environmental factors in adults: A population-based network analysis. *American Psychologist*. https://doi.org/10.1037/amp0001208. Epub ahead of print.

Bowes, L., Wolke, D., Joinson, C., Lereya, S.T., & Lewis, G. (2014). Sibling bullying and risk of depression, anxiety, and self-harm: A prospective cohort study. *Pediatrics*, *134*, E1032–E1039.

Boyd, A., Golding, J., Macleod, J., Lawlor, D.A., Fraser, A., Henderson, J., … Davey Smith, G. (2013). Cohort profile: The 'Children of the 90s'-the index offspring of the Avon longitudinal study of parents and children. *International Journal of Epidemiology*, *42*, 111–127.

Brikell, I., Larsson, H., Lu, Y., Pettersson, E., Chen, Q., Kuja-Halkola, R., … Martin, J. (2020). The contribution of common genetic risk variants for ADHD to a general factor of childhood psychopathology. *Molecular Psychiatry*, *25*, 1809–1821.

Colodro-Conde, L., Couvy-Duchesne, B., Zhu, G., Coventry, W.L., Byrne, E.M., Gordon, S., … Martin, N.G. (2018). A direct test of the diathesis-stress model for depression. *Molecular Psychiatry*, *23*, 1590–1596.

Costello, E.J., & Angold, A. (1988). Scales to assess child and adolescent depression: Checklists, screens, and nets. *Journal of the American Academy of Child & Adolescent Psychiatry*, *27*, 726–737.

Dantchev, S., Hickman, M., Heron, J., Zammit, S., & Wolke, D. (2019). The independent and cumulative effects of sibling and peer bullying in childhood on depression, anxiety, suicidal ideation, and self-harm in adulthood. *Frontiers in Psychiatry*, *10*, 651.

Dantchev, S., & Wolke, D. (2018). Sibling bullying at 12 years and high-risk behavior in early adulthood: A prospective cohort study. *Aggressive Behavior*, *45*, 18–32.

Dantchev, S., & Wolke, D. (2019). Trouble in the nest: Antecedents of sibling bullying victimization and perpetration. *Developmental Psychology*, *55*, 1059–1071.

Dantchev, S., Zammit, S., & Wolke, D. (2018). Sibling bullying in middle childhood and psychotic disorder at 18 years: A prospective cohort study. *Psychological Medicine*, *48*, 2321–2328.

Demontis, D., Walters, R.K., Martin, J., Mattheisen, M., Als, T.D., Agerbo, E., … Hollegaard, M.V. (2019). Discovery of the first genome-wide significant risk loci for attention deficit/hyperactivity disorder. *Nature Genetics*, *51*, 63–75.

Deniz, E., Derinalp, P., Gulkanat, I., Kaz, C., Ozhan, N., & Toseeb, U. (2023). Sibling bullying in Turkish adolescents: Translation and cross-cultural validation of the sibling bullying questionnaire. *Journal of Family Violence*, *38*, 379–392.

Deniz, E., & Toseeb, U. (2023). A longitudinal study of sibling bullying and mental health in autistic adolescents: The role of self-esteem. *Autism Research*, *16*, 1533–1549.

Fraser, A., Macdonald-Wallis, C., Tilling, K., Boyd, A., Golding, J., Davey Smith, G., … Lawlor, D.A. (2013). Cohort profile: The Avon longitudinal study of parents and children: ALSPAC mothers cohort. *International Journal of Epidemiology*, *42*, 97–110.

Goodman, R. (1997). The strengths and difficulties questionnaire: A research note. *Journal of Child Psychology and Psychiatry*, *38*, 581–586.

Howard, D.M., Adams, M.J., Clarke, T.-K., Hafferty, J.D., Gibson, J., Shirali, M., … McIntosh, A.M. (2019). Genome-wide meta-analysis of depression identifies 102 independent variants and highlights the importance of the prefrontal brain regions. *Nature Neuroscience*, *22*, 343–352.

Klingenberg, B., Guloksuz, S., Pries, L.K., Cinar, O., Menne-Lothmann, C., Decoster, J., … Rutten, B.P.F. (2023). Gene–environment interaction study on the polygenic risk score for neuroticism, childhood adversity, and parental bonding. *Personality Neuroscience*, *6*, Article e5.

Liu, X., Peng, C., Yu, Y., Yang, M., Qing, Z., Qiu, X., & Yang, X. (2020). Association between sub-types of sibling bullying and mental health distress among Chinese children and adolescents. *Frontiers in Psychiatry*, *11*, 368.

Lopes, P.P., Relva, I.C., & Fernandes, O.M. (2019). Psychopathology and sibling violence in a sample of Portuguese adolescents. *Journal of Child and Adolescent Trauma*, *12*, 11–21.

Olweus, D. (2007). *The Olweus bullying questionnaire*. Center City, MN: Hazelden.

Patalay, P., Fonagy, P., Deighton, J., Belsky, J., Vostanis, P., & Wolpert, M. (2018). A general psychopathology factor in early adolescence. *British Journal of Psychiatry*, *207*, 15–22.

Peyrot, W.J., Milaneschi, Y., Abdellaoui, A., Sullivan, P.F., Hottenga, J.J., Boomsma, D.I., & Penninx, B.W. (2014). Effect of polygenic risk scores on depression in childhood trauma. *British Journal of Psychiatry*, *205*, 113–119.

Purves, K.L., Coleman, J.R.I., Meier, S.M., Rayner, C., Davis, K.A.S., Cheesman, R., … Eley, T.C. (2020). A major role for common genetic variation in anxiety disorders. *Molecular Psychiatry*, *25*, 3292–3303.

Sabah, A., Aljaberi, M. A., Lin, C., Chen, H.-P. (2022). The associations between sibling victimization, sibling bullying, parental acceptance-rejection, and school bullying. *International Journal of Environmental Research and Public Health*, *19*, 16346.

Schoeler, T., Choi, S.W., Dudbridge, F., Baldwin, J., Duncan, L., Cecil, C.M., … Pingault, J.B. (2019). Multi-polygenic score approach to identifying individual vulnerabilities associated with the risk of exposure to bullying. *JAMA Psychiatry*, *76*, 730–738.

Speyer, L.G., Auyeung, B., & Murray, A.L. (2022). Longitudinal invariance of the strengths and difficulties questionnaire across ages 4 to 16 in the ALSPAC sample. *Assessment*, *30*, 1884–1894.

StataCorp. (2021). Stata Statistical Software: Release 17.

Toseeb, U., Deniz, E., & Noret, N. (2023). The prevalence and correlates of sibling bullying victimisation: A socio-ecological investigation of over 40,000 children in 20 countries. *PsyArXiv*. https://doi.org/10.31234/osf.io/gw56f

Toseeb, U., McChesney, G., Dantchev, S., & Wolke, D. (2020). Precursors of sibling bullying in middle childhood: Evidence from a UK-based longitudinal cohort study. *Child Abuse and Neglect*, *108*, 104633.

Toseeb, U., McChesney, G., Oldfield, J., & Wolke, D. (2020). Sibling bullying in middle childhood is associated with psychosocial difficulties in early adolescence: The case of individuals with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, *50*, 1457–1469.

Toseeb, U., McChesney, G., & Wolke, D. (2018). The prevalence and psychopathological correlates of sibling bullying in children with and without autism spectrum disorder. *Journal of Autism and Developmental Disorders*, *48*, 2308–2318.

Toseeb, U., Vincent, J., Oginni, O., Asbury, K., & Newbury, D.F. (2023). The development of mental health difficulties in young people with and without developmental language disorder: A gene-environment interplay study using polygenic scores. *Journal of Speech, Language, and Hearing Research*, *66*, 1639–1657.

Toseeb, U., & Wolke, D. (2022). Sibling bullying: A prospective longitudinal study of associations with positive and negative mental health during adolescence. *Journal of Youth and Adolescence*, *51*, 940–955.

Tucker, C.J., Finkelhor, D., Shattuck, A.M., & Turner, H. (2013). Prevalence and correlates of sibling victimization types. *Child Abuse and Neglect*, *37*, 213–223.

Tucker, C.J., Finkelhor, D., & Turner, H. (2017). Victimization by siblings in children with disability or weight problems. *Journal of Developmental and Behavioral Pediatrics*, *38*, 378–384.

Tucker, C. J., Finkelhor, D., & Turner, H. (2019). Patterns of sibling victimization as predictors of peer victimization in childhood and adolescence. *Journal of Family Violence*, *34*, 745–755.

Tucker, C.J., Finkelhor, D., & Turner, H. (2020). Family predictors of sibling versus peer victimization. *Journal of Family Psychology*, *34*, 186–195.

Turkheimer, E. (2000). Three laws of behavior genetics and what they mean. *Current Directions in Psychological Science*, *9*, 160–164.

Wolke, D., Tippett, N., & Dantchev, S. (2015). Bullying in the family: Sibling bullying. *Lancet Psychiatry*, *2*, 917–929.

Zuckerman, M. (1999). *Vulnerability to psychopathology: A biosocial model*. American Psychological Association. https://doi.org/10.1037/10316-000

Accepted for publication: 20 December 2023