Children's genetics and family dynamics:

An evocative effects analysis of the Norwegian Mother-Father-and-Child Study

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Abstract

Family demographic behavior is often studied as a response to structural conditions or parental characteristics, yet children may themselves shape key family trajectories, known as an evocative correlation. Using the Norwegian Mother, Father and Child Cohort Study (MoBa) and analyzing 40,000 genotyped mother–father–child trios, we test whether children's polygenic indices (PGIs) predict subsequent fertility, partnership dissolution, and transition to marriage. Accounting for parental PGIs, we found limited evidence for evocative effects across most traits. The exception was that children with a higher ADHD PGI predicted increased parental partnership dissolution (7% [CI=2.9-11.8%] per s.d.), especially in families with daughters (11% [CI=4.9-18%] per s.d.), higher socioeconomic status (15% [CI=7.7-23.2%] per s.d.), and older maternal age at first birth (10% [CI=3-17%] per s.d.). The main source of variation is the socio-economic dimension where child-driven effects are stronger in higher-SES families. The study provides evidence for evocative gene-environment correlations in family demography, demonstrating the importance of child-to-parent effects in shaping family trajectories.

Keywords

Evocative effects, family dynamics, child effects, population registers, MoBa, Norway

Introduction

Research on intergenerational family processes has overwhelmingly focused on how characteristics and behavior of parents are related to their children's outcomes (Amato 1996; Torche, Fletcher, and Brand 2024). Literature across multiple disciplines has examined outcomes such as the effects of parental divorce on children (Bernardi and Radl 2014; Amato 2000), the influence of parental socioeconomic status on children's life chances (Carvalho 2012; van Hootegem et al. 2025), and intergenerational transmission of demographic behaviors such as fertility (Anderton et al. 1987). Limited attention, however, has been paid to the possibility that the influence may operate in the opposite direction, or in other words, that children may impact parents. Children may themselves play an active role in shaping the environments in which they are raised. Children's traits, behaviors, and needs may elicit different responses from parents and other caregivers, setting in motion developmental and relational trajectories that ultimately affect the parents' family stability, future reproductive decisions, and relationship transitions.

The Scarr-McCartney Genotype- Environment Effects model proposed seminal theoretical work on how children shape the families they grow up in due to children's own (genetic) traits (Scarr and McCartney 1983). This theory outlines three distinct pathways through which an individual's genetic makeup (G) is correlated (r) with their environment (E), known as rGE, and how genetics influence the kinds of environments families experience. Passive rGE is where children inherit genetics from their parents while simultaneously being raised in an environment influenced by those parental genetics (e.g., parents with a high verbal ability pass on both genetics related to this trait and a home environment that stimulates verbal development). Active rGE is where individuals seek or create environments that match their genetic tendencies (e.g., a musically inclined child starts a band). Finally, evocative rGE, which we focus on in this study, is where children's genetically influenced traits elicit a particular response from others (e.g., a sociable child elicits more social interaction). Unlike passive or active rGE, evocative rGE captures how the child's traits themselves may prompt reactions from parents, caregivers and others in the family. This reframes the family not merely as a structure acting upon the child, but as a dynamic bidirectional system in which the child is also an actor eliciting responses that, ultimately, can influence parents' decisions around continued childbearing, relationship commitment, or partnership dissolution.

There is some evidence from observational studies of the relationship of children's traits on parental outcomes. The presence of childhood chronic illness or disability, for instance, can lead to parents forgoing or delaying subsequent childbearing or increase the likelihood of union dissolution (Corman and Kaestner 1992; Macinnes 2008; Loft 2022). MacInnes (2008), for instance, shows that mothers of children with disabilities are less likely to have additional children, interpreting this as a strategic response to the perceived demands of intensive parenting. Similarly, Corman and Kaestner (1992) report that having an unhealthy child significantly decreases the mother's likelihood of being married. Some childhood health conditions (e.g. congenital heart disease, cerebral palsy) have been found to be associated with a higher risk of divorce (Joesch and Smith 1997), while cancer in children has not (Syse, Loge,

and Lyngstad 2010). Loft (2014) shows a higher likelihood of parental relationship dissolution when a child is diagnosed with a disability or chronic illness. The literature also goes beyond health, demonstrating that child anger is positively associated with interparental conflict, and child pleasure is positively associated with interparental warmth (Ramos et al. 2022).These studies support the premise that child characteristics may act as stressors or inflection points in family dynamics.

Although some research exists, literature is limited. Most studies rely on inconsistent health measures or clinical diagnoses that often emerge only after major family decisions have been made. Moreover, children's behaviors and health are frequently reported by parents, making them vulnerable to measurement bias. While these studies suggest associations, they often cannot address questions of causality or timing with sufficient clarity. In this context, genetic data offers a promising path forward. Because genetic variation is randomly assigned at conception and remains stable across the life course, it provides a powerful tool for identifying how early emerging traits may evoke differential responses from the social environment without the confounding that is typically present in observational studies. Polygenic indexes (PGIs) thus allow us to measure a child's genetic relative risk for a given outcome. PGIs can help identify evocative pathways by isolating the child's own influence on the environment, independent of parental characteristics.

Recent work has begun to apply this framework to parenting behavior, showing, for example, that parents invest more in children with a higher PGI for educational attainment (Breinholt and Conley 2023). Crucially, they demonstrate that these effects are socially stratified: non-college-educated parents increase how often they read to children with high PGIs, while college-educated parents appear less responsive. Complementing this, recent findings from Norway show that children's PGIs for educational attainment are significantly associated with increased maternal literacy-focused parenting, and that this literacy-focused parenting mediates the effects of polygenic indices on their later educational performance, further evidencing the presence of evocative effects in parenting styles (Austerberry et al. 2025). This emerging line of inquiry offers a compelling extension of evocative rGE theory, suggesting that genetic information can uncover patterns of differential treatment that might otherwise remain hidden. At the same time, it highlights how social contexts, including socio-economic status and environment, continue to shape how biology can be linked to social outcomes.

The current study examines whether children's polygenic propensities influence family dynamics. Specifically, we examine whether a child's PGI for a range of behavioral and health outcomes predicts three key family dynamics aspects: the likelihood of consequent childbearing, the risk of partnership dissolution, and the transition into marriage. We identify evocative rGE by leveraging genetic trio data (i.e., genomic information from both a child and both parents), and test whether these evocative effects vary by socioeconomic and demographic characteristics.

Genetic trios offer a powerful framework to isolate and understand the causal effects of child traits on family dynamics. This design allows us to isolate the child's PGIs contributions from those of the parents, enabling a clearer disentanglement of direct genetic effects from

environmental influences. Analyzing both transmitted and non-transmitted alleles provides a mechanism to distinguish between the child's inherent dispositions and the genetically informed parental behaviors (often referred to as genetic nurture) where parents' genes shape the environment they provide, regardless of whether those genes are passed on to the child (Kong et al. 2018). Such differentiation is crucial for attributing specific family dynamics directly to the child's PGI rather than to confounding familial factors. Moreover, information on genetic trios facilitates rigorous variance decomposition, enabling researchers to partition the contributions of direct genetic influences versus those stemming from the family environment. When parental polygenic indices are accounted for in the analysis, any residual association between the child's genetic profile and observed family behaviors is more confidently interpreted as a causal influence. This method is especially valuable in studies examining evocative rGE, where a child's genetically influenced traits trigger specific reactions from caregivers or siblings. By using trio designs, researchers can therefore provide robust evidence that families are not just shaping children, but that children's characteristics and traits also play a role in the family environment, highlighting a bidirectional interplay between nature and nurture.

We draw on the Norwegian Mother, Father and Child Cohort Study (MoBa), a longitudinal, population-based birth cohort with genetic data from mothers, fathers, and children (Magnus et al. 2016; Brandlistuen et al. 2025), linked to administrative records with information available on family structures and reproductive behavior . Norway is a particularly informative setting for this work: as it is a universal welfare state with high levels of gender equality, and strong support systems for families (Lappegård and Noack 2015; Syltevik 2018).

Our goal is both empirical and conceptual. Empirically, we seek to identify whether children's genetic predispositions evoke differential family responses in domains beyond direct parenting behavior. Conceptually, we contribute to the integration of psychological and sociological models of family life with genetic theories of development and gene-environment interplay (e.g., Wedow et al, 2018). By extending the concept of evocative rGE to the study of fertility and partnership dynamics, and by situating genetic influences within a socially stratified and policy-rich context, we advance a more interactive and reciprocal view of family life - one in which children are not only shaped by their families but also shape them in return.

Results

We examined whether children's PGIs for a range of behavioral and health-related traits (Supplementary Table S1) were associated with three family dynamic outcomes: subsequent childbearing, partnership dissolution, and transition into marriage. Using, at first, data from all parent-child trios in our sample, we estimated the effects of children's PGIs while controlling for parental PGIs.

Child's PGI and Sex

As shown in Figure 1, most associations were small and statistically non-significant, indicating that children's PGIs were generally unrelated to these family outcomes. The one notable exception was the PGI for ADHD, which significantly predicted partnership dissolution. A unit increase in the s.d. of the child's ADHD PGI was associated with an approximate 7% increase in the odds of parental dissolution (OR = 1.073, 95% CI: 1.029, 1.118). This association remained statistically significant after applying a Bonferroni correction for multiple testing.

Analyses stratified by sex of the child revealed further association between the ADHD PGI and dissolution, revealing it was driven primarily by families with daughters (Figure 2). Amongst girls, a 1 s.d. increase in the ADHD PGI was associated with an 11% increase in the odds of parental dissolution (OR = 1.11, 95% CI: 1.049, 1.180), whereas the corresponding increase for boys was less than 4% (OR = 1.036, 95% CI: 0.978, 1.098). Figure 2 also shows that no other sex-specific associations were observed for any of the other PGIs or family outcomes.

Child's PGI and socioeconomic status

When stratifying families by socioeconomic status, defined by parental education, we found that the association between the child's ADHD PGI and partnership dissolution was significant only amongst families with an above-median SES (Figure 3). In these relatively advantaged families, a 1 s.d. increase in the child's ADHD PGI was associated with a 15% increase in the odds of partnership dissolution (OR = 1.152, 95% CI: 1.077, 1.232). By contrast, in lower-SES families (at or below the median SES), the effect was not distinguishable from zero (OR = 1.023, 95% CI: 0.971, 1.079). Notably, we did not observe significant effects for any other PGIs, underscoring the unique role of the child's genetic liability for ADHD in shaping union dissolution dynamics. Additionally, results from the multiverse analysis (Supplementary Figures 2 and 3) show that the primary source of variation in estimates (beyond the specific PGI and outcome examined) is socioeconomic status where evocative effects appear stronger among higher-SES families compared to lower-SES families.

Child's PGI and maternal age at first birth

Maternal age at first birth also moderated the evocative effects observed for the ADHD polygenic index. In families where the mother was older than the median age at first birth, 1 s.d. increase in child's ADHD PGI was associated with a 10% increase in the odds for parental partnership dissolution (OR = 1.099, 95% CI: 1.03, 1.170). In contrast, no statistically significant association was observed amongst families with younger mothers. Additionally, in the older maternal-age trios, we detected a significant relationship between the child's anxiety disorder PGI and parental transition into marriage: 1 s.d. increase in child's anxiety disorder PGI was associated with a 10% increase in odds of the parents marrying after the child's birth (OR = 1.096, 95% CI: 1.035, 1.160).

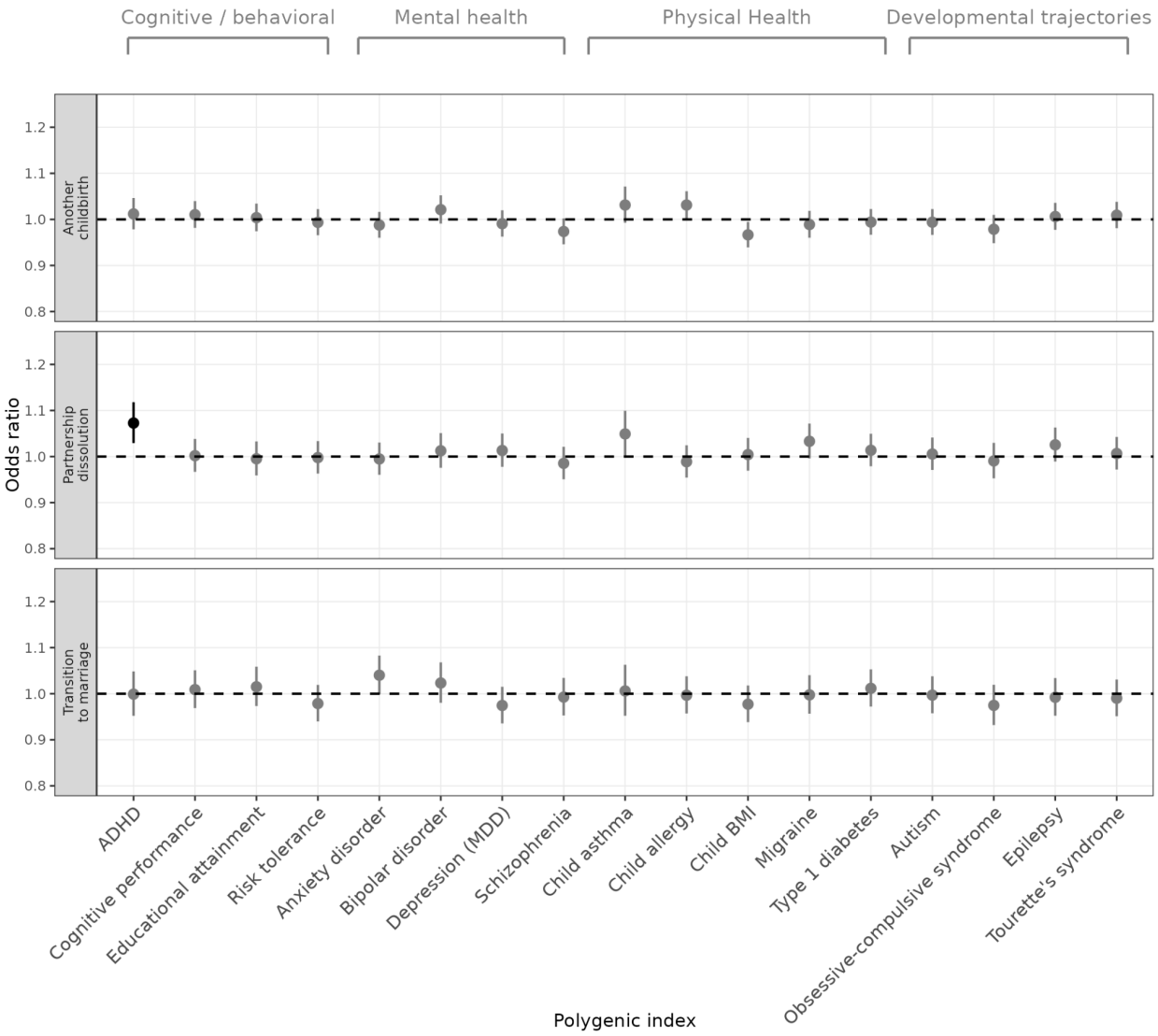

FIGURE 1. Odds ratios of family demographic events by child's polygenic index (PGI) for all families. Estimates and associated 95% confidence intervals for child's PGI from logit models where parents' PGIs are controlled. Dark color indicates the estimate is statistically significant after Bonferroni correction for the number of PGIs. N = 39,056 (fertility) / 39,037 (dissolution) & 19,079 (marriage).

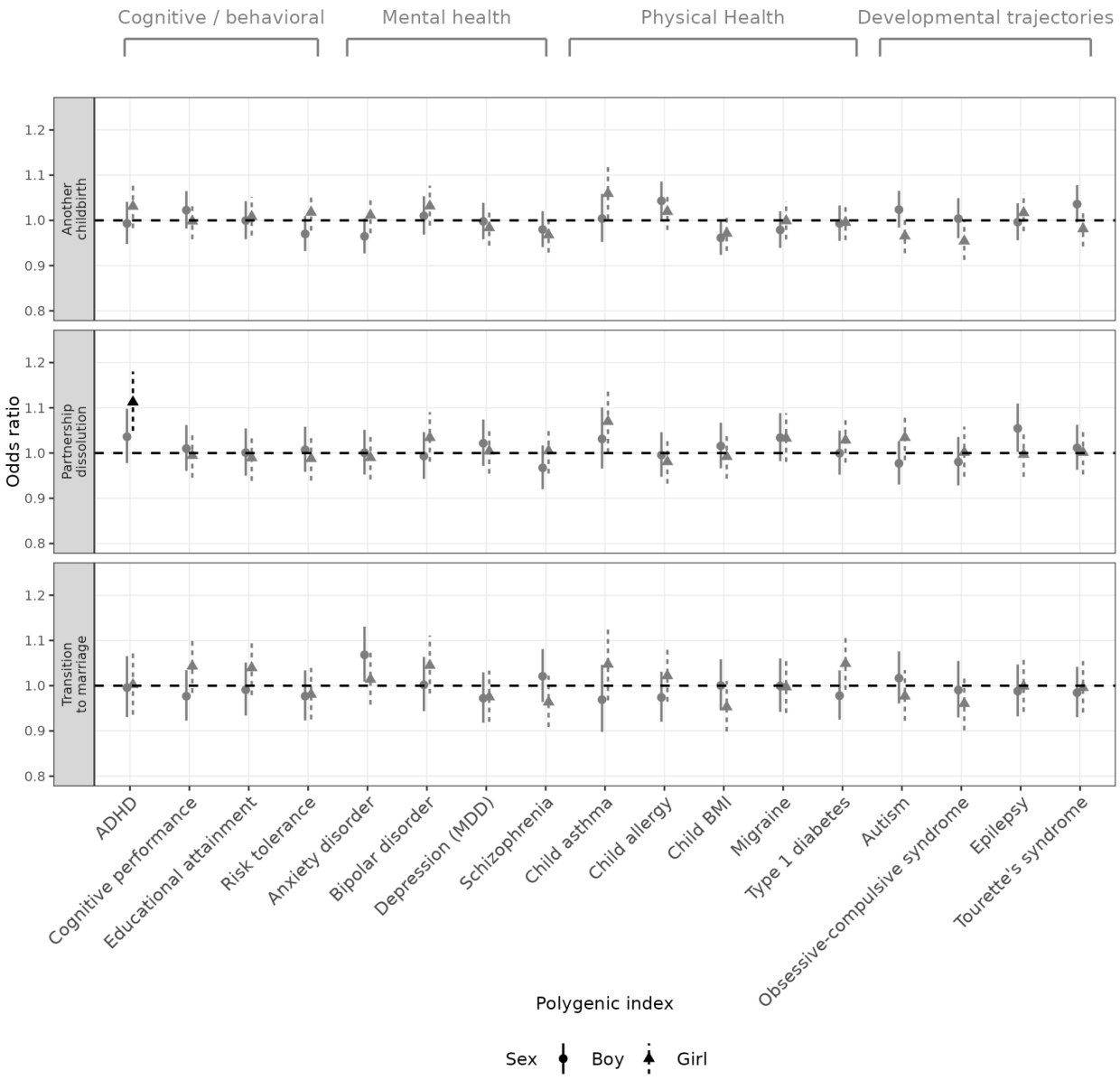

FIGURE 2. Odds ratios of family demographic events by child's polygenic index (PGI) with sample split by sex of the focal child. Estimates and associated 95% confidence intervals for child's PGI from logit models where parents' PGIs are controlled. Dark color indicates the estimate is statistically significant after Bonferroni correction for the number of PGIs.

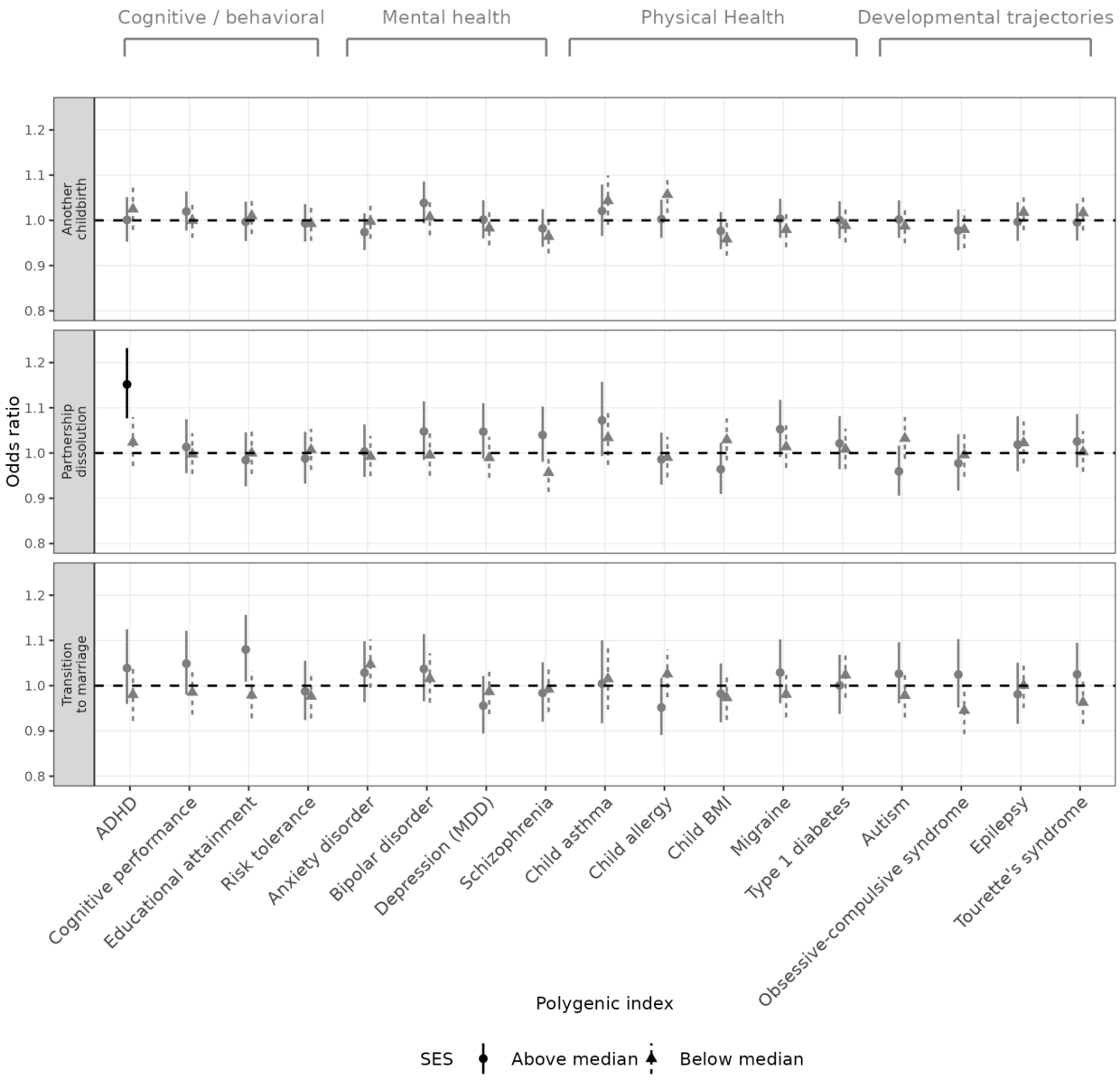

FIGURE 3. Odds ratios of family demographic events by child's polygenic index (PGI) with sample split by the SES of the parents. Estimates and associated 95% confidence intervals for child's PGI from logit models where parents' PGIs are controlled. Dark color indicates the estimate is statistically significant after Bonferroni correction for the number of PGIs.

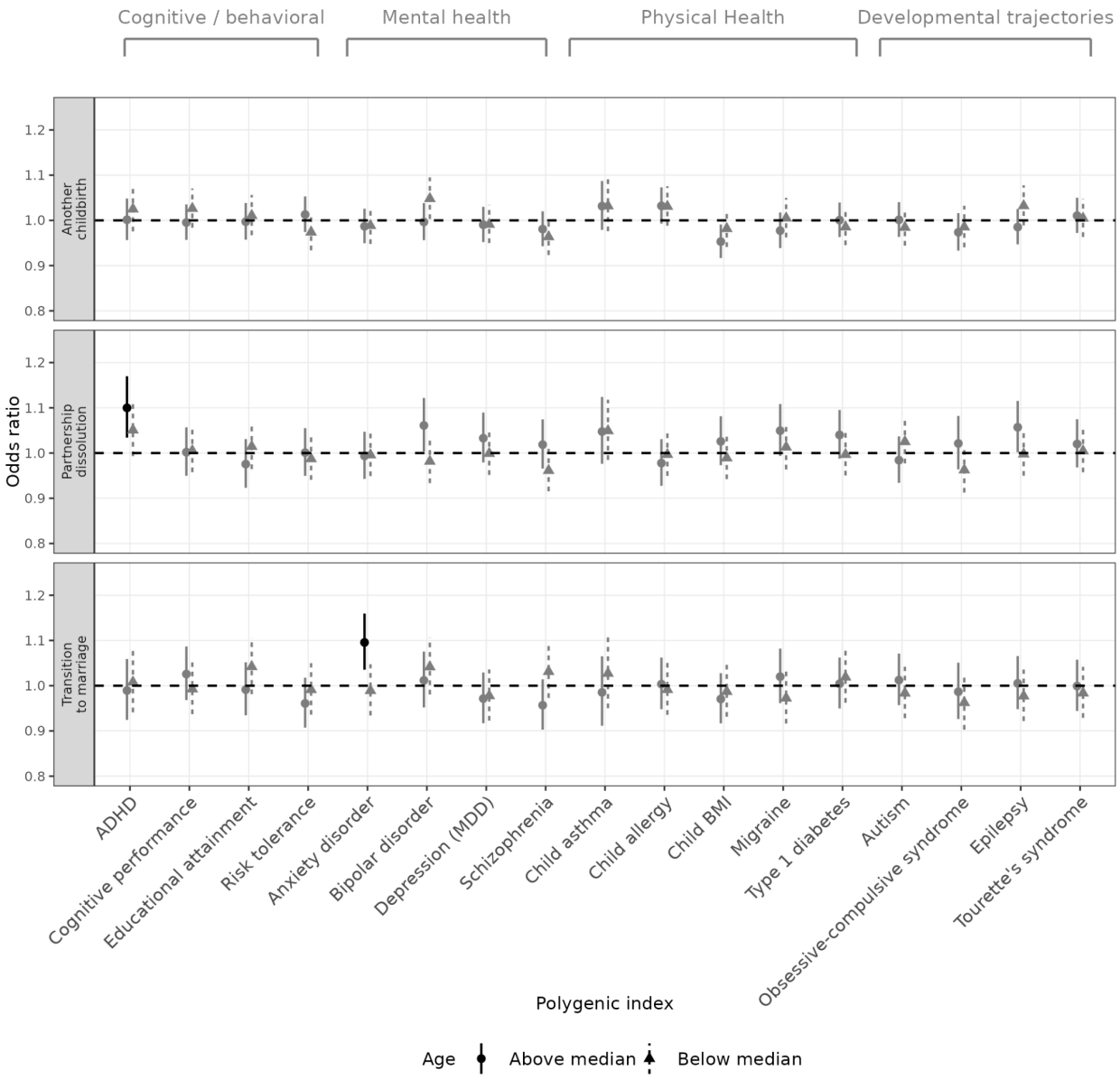

FIGURE 4. Odds ratios of family demographic events by child's polygenic index (PGI) with sample split by the age at first birth of the mother. Estimates and associated 95% confidence intervals for child's PGI from logit models where parents' PGIs are controlled. Dark color indicates the estimate is statistically significant after Bonferroni correction for the number of PGIs.

Discussion

In this study, we aimed to advance our understanding of family dynamics by examining child-driven evocative effects. Specifically, we investigated whether children's PGIs for behavioral and mental and physical health traits are associated with parental responses in the form of further childbearing, partnership dissolution, and transition to marriage. Using a genetically informed trio design, we estimated the direct influence of children's polygenic indices on family demographic outcomes, independent of parental genotype. While most polygenic indices were not significantly associated with these outcomes, we found consistent evidence that higher ADHD PGIs in children predicted increased risk of parental partnership dissolution. These findings offer novel evidence for evocative gene–environment correlations in the domain of family formation and stability, highlighting the role of child characteristics in shaping parental life trajectories.

Our findings on ADHD resonate with longstanding clinical observations: families of children with ADHD experience higher rates of marital conflict and divorce than families of children without ADHD (Schermerhorn et al. 2012). In other words, children's genetically influenced behavior can "evoke" stress in the family system. Moreover, our finding that the effect on dissolution was primarily driven by families with daughters further points to the highly sex-specific complexity of ADHD, where girls are often underdiagnosed and likely experience unaddressed behavioral challenges from both parents and social environments, potentially placing a strain on family relationships (Walters 2023). Importantly, sex differences in ADHD include girls showing less overt hyperactivity and more issues with attention and emotion regulation (De Ronda et al. 2024) which may contribute to under-recognition and differential responses from caregivers and parents. Additionally, gender norms and societal expectations may shape how parents perceive and respond to these behaviors, potentially compounding stress within the family system. Importantly, our result aligns with recent evidence that polygenic indices for risk-prone or psychopathological traits increase divorce risk. For example, partners' polygenic indices for internalizing symptoms and risk behavior increase the probability of partnership dissolution (Jørgensen et al. 2025), a pattern that is echoed here from the child's side. Together, these patterns underscore that intergenerational influence is bidirectional: children do not passively inherit their family environments but can actively contribute to the life-course trajectories of their parents.

Our findings also have significant conceptual implications for evocative rGE. Classical rGE theory holds that individuals' genetic propensities can elicit responses from their environment (Scarr and McCartney 1983). We extend this theory to family demography by showing that offspring genotype can influence parental family-level events. In our case, a child's genetic liability for ADHD appears to evoke changes in parental partnership behavior. We also highlight the importance of the genetic correlation-by-environment interaction framework (rGExE; (Wedow et al. 2018)) in understanding family dynamics, by showing that evocative effects depend on familial socio-economic status and maternal age. Consistent with our finding that children's anxiety PGI in older maternal-age trios was linked to a greater likelihood of parents marrying post-birth, prior research suggests that older mothers may buffer risks in anxious children (Yu and Yan 2022) and that genetically influenced traits can evoke stabilizing parental

responses (Reiss et al. 2022). Previous studies have also found genetic links between older maternal (and paternal) and ADHD as well as other forms of externalising behavior (Mills et al. 2021). One possible explanation for the stronger effect in high-SES families is that behavioral difficulties such as ADHD may conflict with more rigid normative expectations, especially where orderly development and academic success are presumed (Torche, Fletcher, and Brand 2024; Owens 2020). While divorce has been shown to be particularly consequential for children in high-SES families (Bernardi and Radl 2014; Brand et al. 2019), our findings suggest that such families may also be more vulnerable to dissolution when facing child-level challenges.

Overall, our stratified analyses by child sex, family SES, and maternal age at first birth indicated that among the full set of PGIs examined, ADHD stood out as the only behavioral trait exhibiting a robust evocative effect on family structure captured through our genetically informed trio design. While other traits may exhibit significant correlational patterns with family dynamics, these associations tend to attenuate under this research design, suggesting that they may be confounded by shared familial factors or other types of gene-environment interplay such as passive and active gene-environment correlations that are rather operating through parents' own characteristics.

However, the pattern of null results for other PGIs is also informative. We tested a range of indices and found no consistent evocative effects aside from ADHD. The absence of effects for other traits in our analyses underlines that child-evoked influences on partnership and fertility are likely to be trait-specific. Moreover, other types of gene-environment interplays (GxE), such as passive GxE selection and GxE interaction, might play a key role, and parental characteristics will be of greater importance in such instances. For instance, recent family-based analyses have emphasized passive rGE, where parents tend to structure children's learning environments in accordance with their own genotypes, rather than in response to each child's PGI (Zhou et al. 2025).

Our study has several strengths. A core strength is the mother-father-child trio design, which substantially controls for key family-level confounders. For example, it eliminates passive gene-environment correlation where parents' genes influence the home environment shared with the child (Davies et al. 2024). In addition, our approach adjusts for indirect parental genetic effects and any stable family-wide environmental factors that are shared within the family. By including genotype data from both parents and offspring, we differentiate alleles that the child inherits from those within the parents' genomes. This approach directly addresses genetic nurture - the phenomenon whereby non-transmitted parental alleles influence children's outcomes via the environment (Kong et al. 2018). In our design, those non-transmitted alleles (and any effects of parental polygenic indices) are held constant so that associations with the child's transmitted PGI reflect a direct effect of the child's genotype. In practice, this means our ADHD PGI association cannot be attributed to passive rGE or shared family background. This within-family framework is analogous to a sibling fixed-effects model, which generally yields more conservative but more credible genetic estimates than population-based analyses (Davies et al. 2024).

These strengths notwithstanding, the study also comes with several limitations. First, the analysis is based on Norwegian MoBa data. Norway's social policies, healthcare system, and

family norms are distinctive (Lappegård and Noack 2015; Syltevik 2018), and our findings may therefore not widely generalize to other social or cultural contexts. The combination of institutional support and normative openness, however, also makes Norway a valuable context in which to examine the subtle ways that child characteristics may influence family trajectories. If we assume that evocative effects generally are small, and smaller in magnitude than other social effects on family processes, the Norwegian context is ideal for observing them. However, to fully capture the complexity of intergenerational family dynamics, future research should extend this framework to more diverse populations and institutional settings.

Second, the MoBa study is subject to participation bias. Enrollment was voluntary, resulting in a sample skewed towards families with higher socioeconomic status whereas families experiencing multiple adversities are likely underrepresented. While previous work (Nilsen et al. 2009) suggests that such selection bias may not significantly distort exposure–outcome associations, it can affect prevalence estimates. Third, our polygenic indices were derived from GWAS of European ancestry populations. Because polygenic scores have reduced predictive accuracy in non-European groups (Duncan et al. 2019), our findings are not directly applicable to more diverse ancestral backgrounds. Fourth, despite growing GWAS sample sizes, current PGIs explain only a limited proportion of variance in the underlying traits, in part due to the exclusion of rare variants (Crouch and Bodmer 2020). For instance, the ADHD PGI explains approximately 6% of trait variance (Plomin and Von Stumm 2018), with a SNP heritability of 14% (i.e., proportion of trait variation explained by common genetic variants (SNPs)) (Demontis et al. 2023), implying that our estimates are likely conservative. As GWAS methods advance, more predictive PGIs may allow detection of additional child-driven effects. Finally, as with any non-experimental study, unmeasured confounding remains a possibility. Nonetheless, by leveraging within-family genetic variation and controlling for parental genotypes, our design substantially reduces confounding and strengthens the direction towards causal interpretation.

Conclusion

In a genetically informed trio design, we tested whether children's PGIs for behavioral and health traits predict parental subsequent childbearing, partnership dissolution, and marriage. This approach quantifies children's contributions to family dynamics, contrasting prior research focusing on parent-to-child transmission. Most PGIs showed no significant associations; however, children's higher ADHD PGI robustly predicted an increased risk of parental dissolution. These findings provide novel evidence of an evocative gene–environment correlation in family dynamics, underscoring how child-driven effects shape parents' life trajectories.

Methods

Data sources

The data used in analyses reported here are drawn from two sources, the Norwegian Mother-Father-and-Child Cohort study (MoBa) and Norwegian population-wide administrative registers. A system of personal ID numbers facilitates linking of data between these two sources.

Norwegian Mother, Father and Child Cohort Study

The Norwegian Mother, Father and Child Cohort Study (MoBa) is a population-based pregnancy cohort study conducted by the Norwegian Institute of Public Health (Magnus et al. 2016). Participants were recruited from across Norway during 1999–2008. Mothers consented to participation in 41% of the pregnancies. The cohort includes approximately 114,500 children, 95,200 mothers and 75,200 fathers. Blood samples were obtained from both parents during pregnancy and from mothers and children (umbilical cord) at birth (Paltiel et al. 2014). The Medical Birth Registry (MBRN) is a national health registry containing information about all births in Norway, from 1967 onwards, and was used to obtain children's year of birth and sex registered at birth (Irgens 2000). The establishment of MoBa and initial data collection was based on a license from the Norwegian Data Protection Agency and approval from The Regional Committees for Medical and Health Research Ethics (REK). MoBa is regulated by the Norwegian Health Registry Act.

Genotyping and quality control of genotype data

Genotyping was performed using the Illumina Global Screening Array, Illumina HumanCoreExome, Illumina HumanOmniExpress, and Illumina InfiniumOmniExpress Arrays. The genetic data have gone through quality control and imputation procedures according to the MoBaPsychGen pipeline (Corfield et al. 2022). The sample was restricted to families for whom genetic data were available for complete trios (i.e., mother, father, and child).

Administrative register data

Demographic outcome data on family dynamics were taken from population-wide administrative registers (Røed and Raaum 2003). These registers cover the whole Norwegian population. The demographic data has from little to no attrition, and are based on registered vital events, yielding objective measurements of changes in households and the marital and childbearing status of parents. The administrative data covers the period up to the end of 2020, which marks the end of the follow-up period; by that time, the children in the sample were between 11 and 21 years old.

Measures

Outcome measures

For all outcomes, we register both whether the event ever occurred and when it takes place, a duration variable. This allows us to use different outcome definitions with specific time frames. In subsets of models, we experiment with shorter follow-up periods (5 years, 10 years). Only events that took place within the specific follow-up window are counted.

Having another child is measured by whether the mother has had a child after the focal child, within the relevant follow-up window.

Partnership dissolution is measured as the transition from being married or cohabiting with a common child to having separated or divorced as marital status or not any longer cohabiting with the co-parent of a common child. The 'common child' in this sense will include the focal child, but could also include other common children, but not children from prior relationships.

The transition to marriage is measured by observing changes in marital status for cohabiting couples. Couples who were already married at the birth of the focal child are set to missing, and excluded from the analysis.

Covariates

Sex of child and year of birth were obtained from the administrative register data, specifically the population register. Family SES was obtained from the National Educational Database, and was measured as the mean of both parents' educational attainment (in years of education) measured the year before the child's birth. Mother's age at first birth and birth order is calculated from complete birth history data from the population register data. Both family SES and mother's age at first birth is split into subsamples at the median value in the sample. Birth order is split between families where the focal child is the firstborn and families where they are not.

Polygenic indices (PGIs) construction

PGIs were calculated based on genome-wide association studies listed in Supplementary Table S1. Following quality-control guidelines (Choi, Mak, and O'Reilly 2020; Burt 2024), we retained variants on autosomal chromosomes with minor allele frequency MAF ≥ 0.01 and imputation quality INFO scores ≥ 0.95 when available, removing ambiguous and duplicated SNPs. Polygenic scores for children, mothers, and fathers were computed for each trait using PRSice software (Choi and O'Reilly 2019). Variants from GWAS summary statistics were matched with SNPs available in MoBa, irrespective of their nominal association significance with the outcome. Clumping was performed by selecting the SNP with the smallest p-value in each linkage disequilibrium block, excluding correlated SNPs ($r^2 \geq 0.1$ within 250-kb windows).

Analytical strategy and statistical methods

Identification of evocative effects of child genetics on family dynamics

To isolate the influence of children's genetic predispositions on family dynamics, we employed a genetically informed trio design using genotype data from mother, father, and child. This approach leverages the random allocation of alleles from parents to offspring under Mendelian inheritance, enabling the estimation of child-specific genetic effects independent of parental genotype. By simultaneously modeling PGIs for the child and both parents, we account for shared genetic variants in the PGIs and obtain unbiased estimates of the effect of child PGI on the outcome.

Model specification

Our main results are parameter estimates for a model where we include the three family members (child, mother and father) as well as each parent's first five genomic principal components and genotyping batch effects. Principal components for the child would be superfluous as the variation in child PGI is random conditional on parents' PGIs. The model equation was:

$$(1)\ y \sim \alpha + \beta_1 PGI_{child} + \beta_2 PGI_{mother} + \beta_3 PGI_{father} + \sum_{m=1}^{5} \gamma_m PCm_{mother} + \sum_{m=1}^{5} \eta_m PCm_{father}$$

The parameters for this model are then estimated using one of the *k* polygenic indices, leading to a set of *k* estimates from the main model specification, one for each PGI.

Multiverse analysis

To assess the robustness of results, we created a number of outcome definitions, model specifications and subsamples, and estimated models for all relevant permutations of these definitions. This let us assess the robustness in results as well as any variations in results by sex of child and family SES.

Outcome definitions are the three outcomes (subsequent fertility, partnership dissolution and transition to marriage) combined with the length of the follow-up period: 5 years, 10 years, and until the observation window was closed. This gives us 9 outcome definitions.

The model specifications were: a) a single child's PGI model with genomic principal components, b) a trio model with child's PGI, father's PGI and mother's PGI, and genomic principal components for both parents, a trio model like c) with controls for sex (for mixed-sex samples) and birth cohort, a trio model like d) with no controls for parents' genomic principal components.

Sample definitions used were: a) All families (i.e., no restriction imposed), b) only families where the focal child is a boy, c) only families where the focal child is a girl, d) only families with above-median SES, e) only families with below-median SES, f) only families where the mother's age at first birth is below the median; and, g) only families where the mother's age at first birth is above the median.

Complete results from the multiverse analysis are shown in the Supplementary Materials. In short, they show that the main source of variation in estimates (beyond which PGI and outcome is studied) is the SES-dimension. Evocative effects are somewhat stronger in higher-SES families than in lower SES families. The choice of statistical model (logit versus OLS) also produces slightly different estimates, but not to the degree where it has any substantive implications.

Data availability statement

Data from the Norwegian Mother, Father and Child Cohort Study is managed by the Norwegian Institute of Public Health. Access requires approval from the Regional Committees for Medical and Health Research Ethics (REK), compliance with GDPR, and data owner approval. Participant consent does not allow individual-level data storage in repositories or journals. Researchers seeking access for replication must apply via www.helsedata.no. Access to administrative register data is available from Statistics Norway after an application procedure.

Code availability statement

All code used to produce the results presented in this paper, as well as the statistical results themselves are available in the Supplementary Materials and will be made available at: https://github.com/torkildl/evocative .

Contributions

THL and ETA designed the study. ETA created the polygenic indices. THL performed data merging and statistical analysis. ETA and THL wrote the paper. AF, AH, ECC, REJ and MCM commented on analysis and paper drafts.

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Competing interests

M.C.M. is a Trustee of the UK Biobank; on the Ethics Advisory Boards of the UK Biobank and Our Future Health, Scientific Advisory Board of Our Future Health, Netherlands Lifelines Biobank, US Health and Retirement Survey and UK CLS Cohort Studies.

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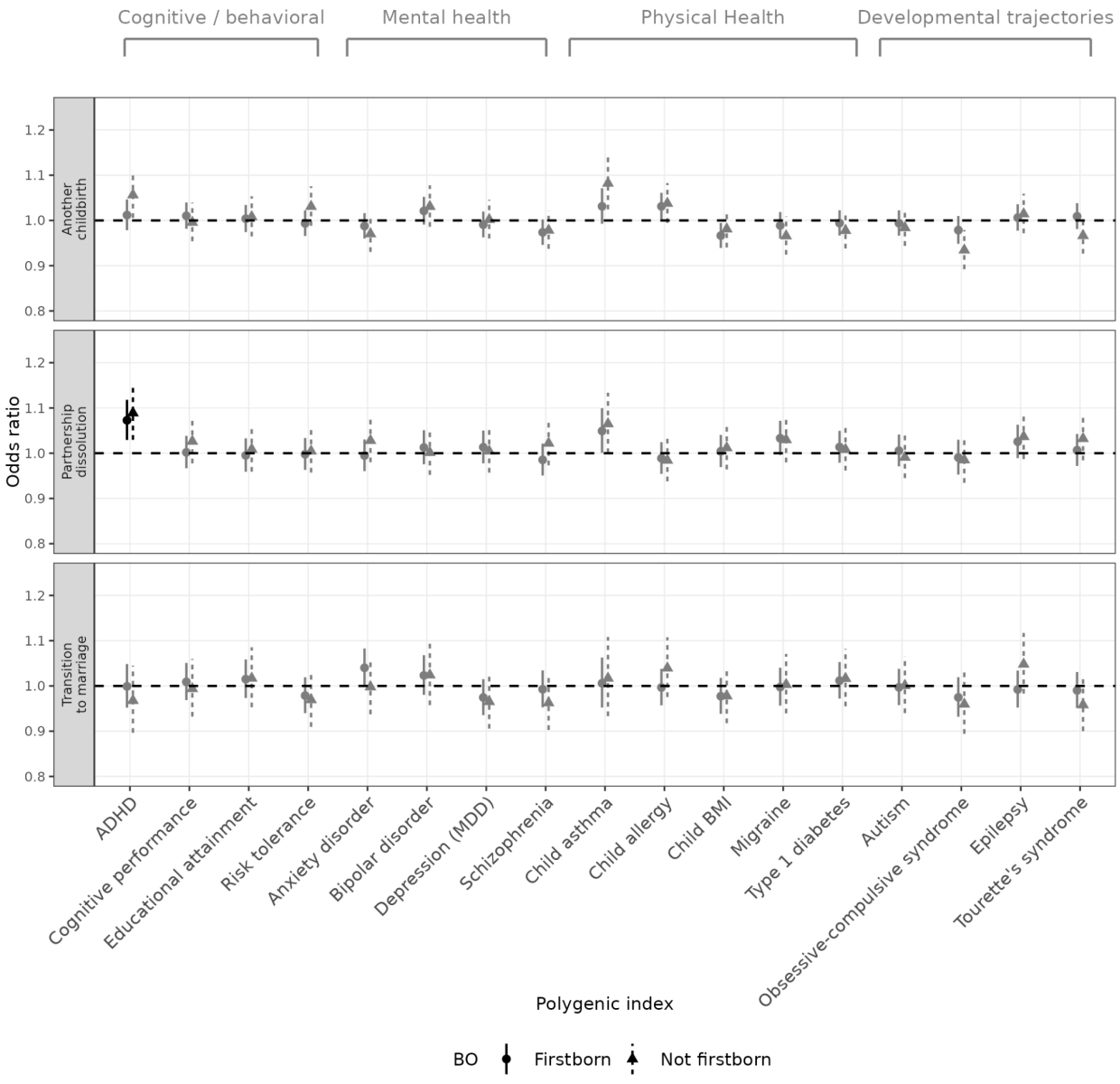
Supplementary Materials

This document accompanies the manuscript Akimova et al. Children's genetics and parental family dynamics: An evocative effects analysis of the Norwegian Mother-Father-and-Child Study.

Table of Contents

1. Results for birth order sample splits

Supplementary Figure 1 shows our coefficient plot with the sample split by the mother's parity (i.e. ,the child's birth order). The results indicate no difference by birth order.



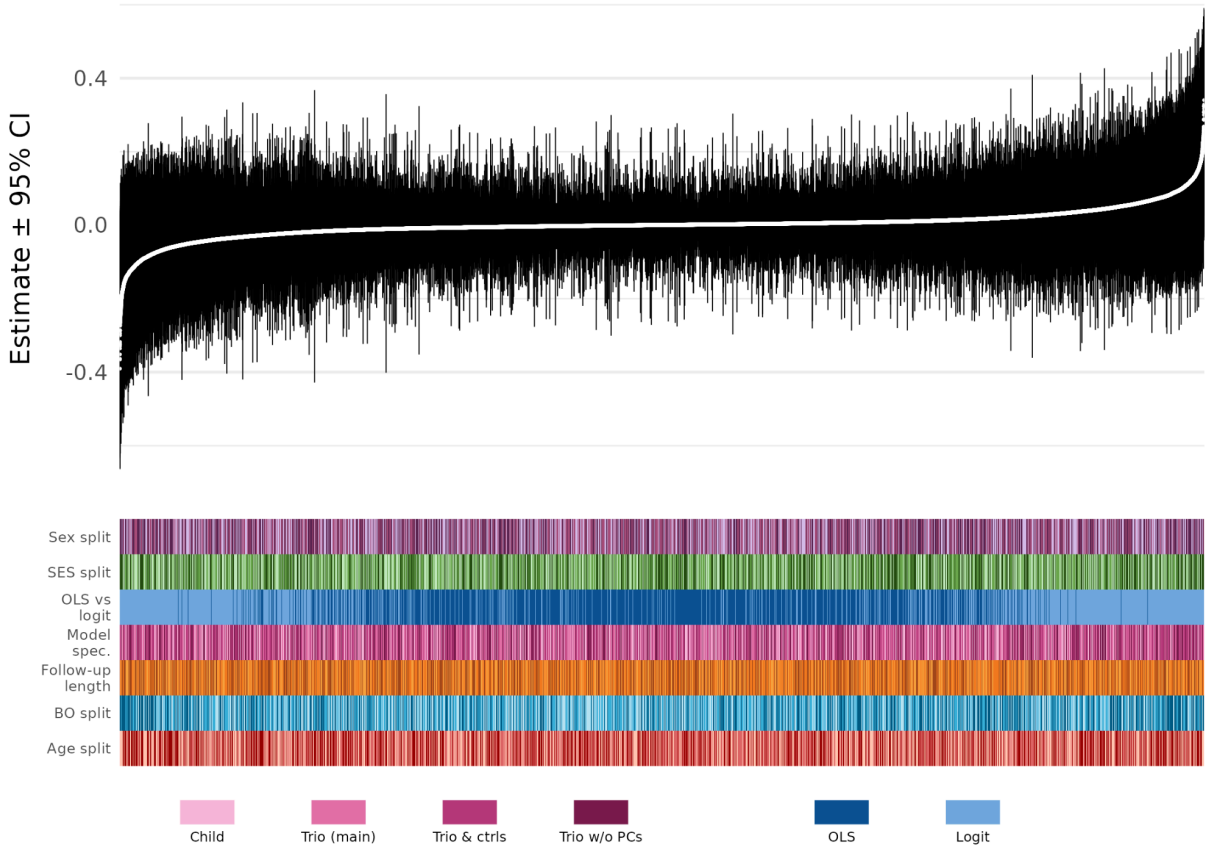
SUPPLEMENTARY FIGURE 1. Odds ratios of family demographic events by child's polygenic index (PGI) with sample split by the parity of the mother (the birth order of the focal child). Estimates and associated 95% confidence intervals for child's PGI from logit models where parents' PGIs are controlled. The dark color indicates the estimate is statistically significant after Bonferroni correction for the number of PGIs.

2. Multiverse analysis

To avoid letting arbitrary modelling choices affect conclusions too much, we performed a so-called multiverse analysis. In practice this means that instead of choosing a preferred model specification and proceeding with the preferred model, we estimated all variations of models across a set of choices. The set of choices are all permutations of 17 different PGIs, four different model definitions, four sample splits with three possibilities each, two link functions, three processes, and 3 follow-up lengths. In total, this amounts to 99,144 models. Results from all models are then presented and analyzed together.

2.1 Results across all model specifications

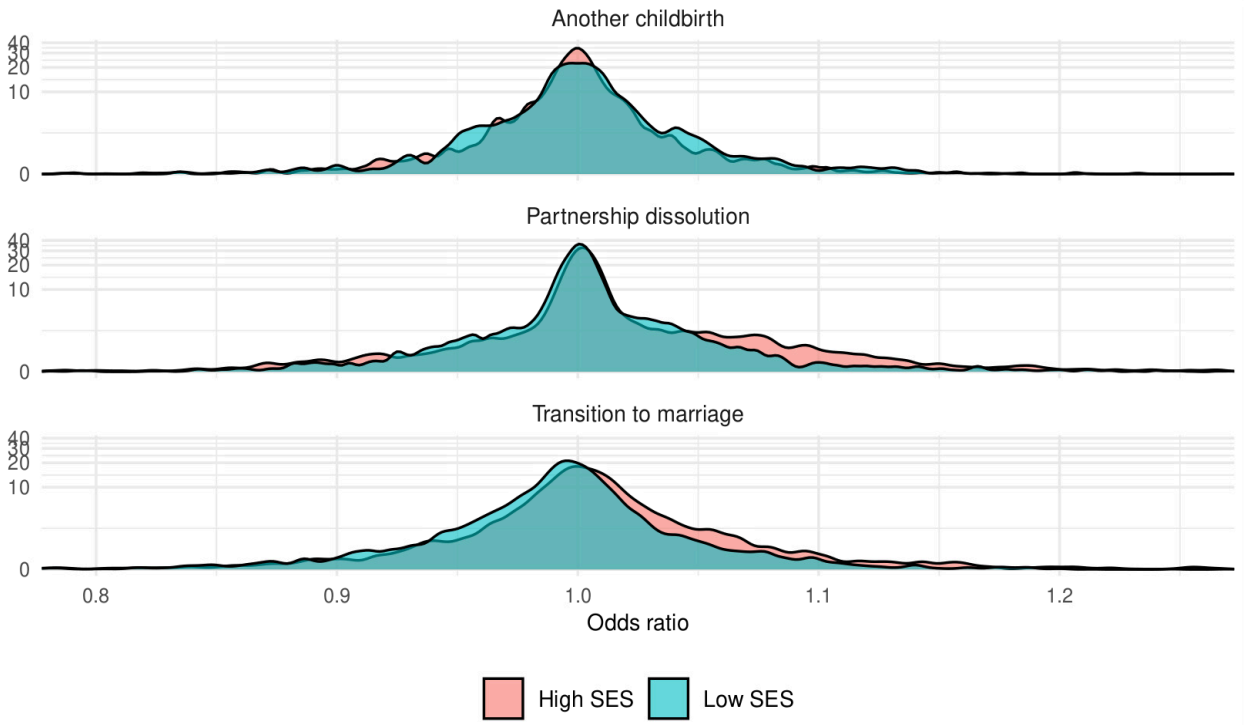
In Supplementary Figure 2, we show the results from this exercise. In the top panel, we display a specification curve of estimates for the evocative child PGI coefficient from all models. In the bottom panel, we show the color coding of each estimate. Using a meta-analytic model, we could estimate which factors contributed to higher or lower estimates. As reported in the manuscript, high SES families report higher evocative effects generally than lower SES families. Apart from this striking finding, there were few patterns in the estimates that warranted any further meta-analytic study. One exception is that the strongest estimates are found using logit models, which is not surprising.



SUPPLEMENTARY FIGURE 2. Results from multiverse analysis. Top panel: specification curve for child PGI coefficient. Models are sorted by the magnitude of the child PGI coefficient. Bottom panel: color coding of model choices.

2.2 Evocative effects in high SES vs. low SES families

Using the results data generated by the multiverse procedure, we can meta-analyse results across PGIs and subsamples. By regressing coefficient size on model choices, one important pattern stood out: High SES family samples generally yield higher estimates for the child PGI. This means that evocative effects are generally stronger among such families than among low SES families. Supplementary Figure 3 shows the distributions of coefficients for models estimated using the high SES sample or the low SES sample. It is evident from the figure that, although the distributions mostly overlap, there are more higher-value estimates found for the high SES families than for the low SES families.



SUPPLEMENTARY FIGURE 3. Evocative effects in high SES vs. low SES families. The densities of effect sizes for each of the three processes (another childbirth, partnership dissolution, transition to marriage). High SES families (light red) and low SES families (mint green).

3. Sample descriptive statistics

Variable	Valid N	M	SD	Min	Max
Birth order	39056	1.72	0.82	1	13
Birth year (child)	39056	2005.43	1.92	2001	2009
Birth year (father)	39065	1972.38	5.41	1942	1990
Birth year (mother)	40276	1974.74	4.72	1956	1991
Sex	39056	1.49	0.50	1	2
Age at first birth	40275	27.92	4.33	15	46
Partnership dissolution (t0)	39037	0.20	0.40	0	1
Partnership dissolution (t5)	39037	0.08	0.27	0	1
Partnership dissolution (t10)	39037	0.14	0.35	0	1
Subsequent childbearing (t0)	39056	0.56	0.50	0	1
Subsequent childbearing (t5)	39056	0.54	0.50	0	1
Subsequent childbearing (t10)	39056	0.56	0.50	0	1
Transition to marriage (t0)	19079	0.52	0.50	0	1
Transition to marriage (t5)	19079	0.34	0.47	0	1
Transition to marriage (t10)	19079	0.47	0.50	0	1
Educational attainment	40251	14.77	2.06	0	21

SUPPLEMENTARY TABLE S1: Sample descriptive statistics. The varying number of valid observations is due to a small number of missing values. For marriage outcomes, it is due to selecting families where the parents are not yet married.

4. Tables with numerical results

For reference, the numerical results plotted in Figures 1-4 in the main document are included here, as Supplementary Table 2. Tables for Figure 2 through 4 have two instances of each PGI, with the lowest sample split value first and the highest value last.

PGI	Another childbirth			Partnership dissolution			Transition to marriage		
	Beta	SE	P	Beta	SE	P	Beta	SE	P
FIGURE 1: No split									
ADHD	0.012	0.017	0.492	0.07	0.021	0.001	-0.001	0.025	0.969
Cognitive performance	0.01	0.015	0.487	0.002	0.018	0.916	0.009	0.021	0.667
Educational attainment	0.004	0.015	0.802	-0.005	0.019	0.8	0.015	0.021	0.486
Risk tolerance	-0.006	0.015	0.655	-0.002	0.018	0.9	-0.022	0.021	0.297
Anxiety disorder	-0.012	0.014	0.395	-0.005	0.018	0.772	0.039	0.02	0.055
Bipolar disorder	0.021	0.015	0.172	0.012	0.019	0.51	0.023	0.022	0.293
Depression (MDD)	-0.009	0.015	0.527	0.013	0.018	0.471	-0.026	0.021	0.214
Schizophrenia	-0.027	0.015	0.071	-0.015	0.018	0.417	-0.007	0.021	0.726
Child asthma	0.031	0.019	0.114	0.048	0.024	0.045	0.006	0.028	0.832
Child allergy	0.031	0.015	0.035	-0.011	0.018	0.532	-0.003	0.021	0.871
Child BMI	-0.034	0.015	0.019	0.004	0.018	0.815	-0.023	0.021	0.268
Migraine	-0.011	0.015	0.458	0.033	0.019	0.081	-0.002	0.021	0.908
Type 1 diabetes	-0.006	0.014	0.682	0.014	0.018	0.446	0.012	0.02	0.569

PGI	Another childbirth			Partnership dissolution			Transition to marriage		
	Beta	SE	P	Beta	SE	P	Beta	SE	P
Autism	-0.006	0.014	0.672	0.005	0.018	0.759	-0.003	0.021	0.88
Obsessive-compulsive syndrome	-0.022	0.016	0.176	-0.01	0.02	0.63	-0.026	0.023	0.263
Epilepsy	0.006	0.015	0.678	0.025	0.018	0.171	-0.008	0.021	0.711
Tourette's syndrome	0.009	0.014	0.529	0.007	0.018	0.715	-0.01	0.02	0.632
FIGURE 2: Boys									
ADHD	-0.007	0.024	0.775	0.035	0.03	0.231	-0.004	0.034	0.901
Cognitive performance	0.022	0.021	0.28	0.01	0.026	0.7	-0.023	0.029	0.421
Educational attainment	0	0.021	0.987	0.001	0.027	0.977	-0.009	0.03	0.765
Risk tolerance	-0.03	0.02	0.137	0.007	0.025	0.78	-0.023	0.029	0.419
Anxiety disorder	-0.036	0.02	0.077	0.001	0.025	0.98	0.066	0.029	0.023
Bipolar disorder	0.01	0.021	0.643	-0.007	0.027	0.797	0.002	0.031	0.951
Depression (MDD)	-0.002	0.021	0.914	0.021	0.026	0.402	-0.028	0.029	0.337
Schizophrenia	-0.02	0.021	0.324	-0.033	0.026	0.193	0.021	0.029	0.478
Child asthma	0.004	0.027	0.882	0.031	0.033	0.357	-0.031	0.039	0.421
Child allergy	0.042	0.02	0.038	-0.004	0.025	0.86	-0.026	0.029	0.365
Child BMI	-0.039	0.02	0.054	0.015	0.025	0.542	0	0.029	0.988
Migraine	-0.021	0.021	0.312	0.033	0.026	0.201	-0.001	0.03	0.984

PGI	Another childbirth			Partnership dissolution			Transition to marriage		
	Beta	SE	P	Beta	SE	P	Beta	SE	P
Type 1 diabetes	-0.007	0.02	0.734	0	0.025	0.992	-0.022	0.028	0.432
Autism	0.024	0.02	0.244	-0.023	0.025	0.357	0.017	0.029	0.566
Obsessive-compulsive syndrome	0.004	0.023	0.865	-0.02	0.028	0.48	-0.01	0.032	0.763
Epilepsy	-0.004	0.021	0.854	0.053	0.026	0.04	-0.012	0.03	0.683
Tourette's syndrome	0.035	0.02	0.081	0.011	0.025	0.648	-0.016	0.029	0.59
FIGURE 2: Girls									
ADHD	0.031	0.025	0.213	0.107	0.03	0	0.002	0.035	0.945
Cognitive performance	-0.002	0.021	0.936	-0.006	0.026	0.832	0.042	0.03	0.155
Educational attainment	0.008	0.022	0.696	-0.011	0.027	0.686	0.039	0.031	0.204
Risk tolerance	0.018	0.021	0.394	-0.012	0.026	0.629	-0.019	0.03	0.512
Anxiety disorder	0.011	0.021	0.582	-0.01	0.026	0.689	0.014	0.029	0.628
Bipolar disorder	0.031	0.022	0.155	0.034	0.027	0.215	0.044	0.031	0.161
Depression (MDD)	-0.016	0.021	0.433	0.004	0.026	0.869	-0.025	0.03	0.397
Schizophrenia	-0.033	0.021	0.119	0.005	0.026	0.854	-0.037	0.03	0.22
Child asthma	0.058	0.028	0.039	0.067	0.034	0.049	0.046	0.04	0.252
Child allergy	0.019	0.021	0.353	-0.019	0.026	0.452	0.022	0.03	0.461
Child BMI	-0.029	0.021	0.163	-0.008	0.026	0.76	-0.048	0.03	0.108

PGI	Another childbirth			Partnership dissolution			Transition to marriage		
	Beta	SE	P	Beta	SE	P	Beta	SE	P
Migraine	-0.001	0.021	0.964	0.032	0.027	0.225	-0.003	0.03	0.927
Type 1 diabetes	-0.005	0.02	0.818	0.028	0.025	0.267	0.048	0.029	0.102
Autism	-0.035	0.021	0.086	0.034	0.025	0.184	-0.023	0.029	0.425
Obsessive-compulsive syndrome	-0.047	0.023	0.04	0.002	0.028	0.957	-0.04	0.033	0.218
Epilepsy	0.017	0.021	0.425	-0.003	0.026	0.905	-0.001	0.03	0.967
Tourette's syndrome	-0.019	0.021	0.36	0.001	0.026	0.958	-0.004	0.029	0.886
FIGURE 3: Low SES									
ADHD	0.001	0.025	0.966	0.141	0.034	0	0.038	0.04	0.345
Cognitive performance	0.019	0.022	0.369	0.013	0.03	0.661	0.048	0.034	0.163
Educational attainment	-0.003	0.022	0.885	-0.016	0.031	0.612	0.077	0.035	0.027
Risk tolerance	-0.006	0.021	0.763	-0.012	0.03	0.682	-0.013	0.034	0.709
Anxiety disorder	-0.026	0.021	0.217	0.003	0.03	0.907	0.028	0.033	0.397
Bipolar disorder	0.038	0.023	0.095	0.047	0.031	0.139	0.037	0.036	0.316
Depression (MDD)	0.002	0.022	0.943	0.046	0.03	0.123	-0.045	0.034	0.184
Schizophrenia	-0.018	0.022	0.405	0.039	0.03	0.192	-0.016	0.034	0.635
Child asthma	0.021	0.028	0.47	0.07	0.039	0.074	0.004	0.046	0.924
Child allergy	0.003	0.021	0.899	-0.014	0.03	0.635	-0.049	0.034	0.143

PGI	Another childbirth			Partnership dissolution			Transition to marriage		
	Beta	SE	P	Beta	SE	P	Beta	SE	P
Child BMI	-0.024	0.021	0.268	-0.036	0.03	0.221	-0.018	0.034	0.593
Migraine	0.004	0.022	0.871	0.051	0.031	0.094	0.029	0.035	0.413
Type 1 diabetes	0.00	0.021	0.992	0.021	0.029	0.471	0.001	0.033	0.98
Autism	0.002	0.021	0.923	-0.041	0.029	0.159	0.026	0.034	0.438
Obsessive-compulsive syndrome	-0.022	0.023	0.34	-0.023	0.033	0.483	0.024	0.038	0.518
Epilepsy	-0.003	0.022	0.88	0.019	0.031	0.543	-0.019	0.035	0.586
Tourette's syndrome	-0.004	0.021	0.835	0.025	0.029	0.388	0.025	0.034	0.458
FIGURE 3: High SES									
ADHD	0.025	0.024	0.296	0.024	0.027	0.383	-0.02	0.031	0.524
Cognitive performance	0	0.02	0.989	-0.002	0.023	0.916	-0.015	0.026	0.576
Educational attainment	0.01	0.021	0.626	0	0.024	0.991	-0.021	0.027	0.439
Risk tolerance	-0.007	0.02	0.72	0.007	0.023	0.761	-0.024	0.026	0.365
Anxiety disorder	-0.002	0.02	0.902	-0.008	0.023	0.74	0.046	0.026	0.076
Bipolar disorder	0.007	0.021	0.728	-0.005	0.024	0.848	0.016	0.027	0.569
Depression (MDD)	-0.017	0.02	0.394	-0.01	0.023	0.652	-0.013	0.026	0.614
Schizophrenia	-0.036	0.02	0.074	-0.044	0.023	0.059	-0.008	0.027	0.776
Child asthma	0.042	0.027	0.112	0.033	0.031	0.278	0.015	0.035	0.67

PGI	Another childbirth			Partnership dissolution			Transition to marriage		
	Beta	SE	P	Beta	SE	P	Beta	SE	P
Child allergy	0.055	0.02	0.006	-0.01	0.023	0.665	0.026	0.026	0.329
Child BMI	-0.042	0.02	0.034	0.029	0.023	0.212	-0.027	0.026	0.307
Migraine	-0.021	0.021	0.32	0.014	0.024	0.57	-0.019	0.027	0.476
Type 1 diabetes	-0.012	0.02	0.552	0.008	0.023	0.712	0.023	0.026	0.368
Autism	-0.013	0.02	0.512	0.032	0.023	0.163	-0.022	0.026	0.404
Obsessive-compulsive syndrome	-0.02	0.022	0.354	-0.004	0.025	0.872	-0.056	0.029	0.052
Epilepsy	0.018	0.02	0.386	0.022	0.023	0.338	0.001	0.026	0.981
Tourette's syndrome	0.017	0.02	0.408	0.002	0.023	0.915	-0.038	0.026	0.147
FIGURE 4: Low age									
ADHD	0.025	0.025	0.326	0.05	0.029	0.085	0.008	0.035	0.82
Cognitive performance	0.026	0.022	0.229	0.005	0.025	0.835	-0.007	0.029	0.803
Educational attainment	0.011	0.022	0.635	0.014	0.026	0.577	0.041	0.031	0.18
Risk tolerance	-0.026	0.021	0.218	-0.013	0.025	0.602	-0.009	0.029	0.764
Anxiety disorder	-0.012	0.021	0.588	-0.004	0.025	0.865	-0.011	0.029	0.708
Bipolar disorder	0.047	0.022	0.036	-0.018	0.026	0.473	0.041	0.031	0.186
Depression (MDD)	-0.009	0.022	0.678	-0.001	0.025	0.969	-0.023	0.03	0.445
Schizophrenia	-0.037	0.022	0.089	-0.039	0.025	0.114	0.031	0.03	0.3

PGI	Another childbirth			Partnership dissolution			Transition to marriage		
	Beta	SE	P	Beta	SE	P	Beta	SE	P
Child asthma	0.031	0.028	0.269	0.048	0.032	0.138	0.027	0.04	0.499
Child allergy	0.03	0.022	0.159	-0.003	0.025	0.895	-0.009	0.03	0.769
Child BMI	-0.018	0.022	0.393	-0.011	0.025	0.656	-0.013	0.03	0.662
Migraine	0.005	0.022	0.819	0.013	0.025	0.612	-0.028	0.03	0.362
Type 1 diabetes	-0.015	0.021	0.485	-0.003	0.024	0.892	0.019	0.029	0.521
Autism	-0.016	0.021	0.463	0.025	0.024	0.311	-0.017	0.029	0.571
Obsessive-compulsive syndrome	-0.015	0.024	0.534	-0.038	0.027	0.162	-0.038	0.033	0.242
Epilepsy	0.032	0.022	0.147	-0.002	0.025	0.926	-0.023	0.03	0.436
Tourette's syndrome	0.005	0.021	0.828	0.005	0.025	0.849	-0.016	0.029	0.574
FIGURE 4: High age									
ADHD	0.001	0.023	0.955	0.095	0.031	0.002	-0.011	0.035	0.759
Cognitive performance	-0.005	0.02	0.816	0.002	0.027	0.945	0.025	0.029	0.388
Educational attainment	-0.003	0.021	0.886	-0.025	0.028	0.379	-0.009	0.03	0.774
Risk tolerance	0.013	0.02	0.519	0.001	0.027	0.973	-0.04	0.029	0.173
Anxiety disorder	-0.013	0.02	0.502	-0.006	0.027	0.81	0.091	0.029	0.002

PGI	Another childbirth			Partnership dissolution			Transition to marriage		
	Beta	SE	P	Beta	SE	P	Beta	SE	P
Bipolar disorder	-0.004	0.021	0.863	0.059	0.028	0.037	0.012	0.031	0.706
Depression (MDD)	-0.01	0.02	0.626	0.032	0.027	0.236	-0.029	0.029	0.326
Schizophrenia	-0.019	0.02	0.331	0.018	0.027	0.498	-0.044	0.03	0.136
Child asthma	0.031	0.027	0.244	0.046	0.036	0.197	-0.015	0.04	0.709
Child allergy	0.032	0.02	0.111	-0.022	0.027	0.404	0.004	0.029	0.904
Child BMI	-0.048	0.02	0.015	0.025	0.027	0.344	-0.03	0.029	0.307
Migraine	-0.023	0.021	0.263	0.048	0.028	0.083	0.02	0.03	0.511
Type 1 diabetes	0.001	0.019	0.977	0.039	0.026	0.137	0.004	0.029	0.879
Autism	0.001	0.02	0.951	-0.016	0.027	0.548	0.012	0.029	0.668
Obsessive-compulsive syndrome	-0.027	0.022	0.219	0.021	0.029	0.475	-0.013	0.032	0.676
Epilepsy	-0.015	0.02	0.461	0.055	0.027	0.043	0.005	0.03	0.863
Tourette's syndrome	0.01	0.02	0.599	0.02	0.027	0.456	-0.001	0.029	0.978

5. Overview of genome-wide association studies

SUPPLEMENTARY TABLE S3. Summary of genome-wide association studies (GWAS) used for constructing polygenic indices.

Outcome	GWAS reference
Cognitive/behavioral	
ADHD	Demontis, Ditte, et al. “Genome-wide analyses of ADHD identify 27 risk loci, refine the genetic architecture and implicate several cognitive domains.” *Nature Genetics* 55.2 (2023): 198-208.
Cognitive performance	Lee, James J., Robbee Wedow, Aysu Okbay, Edward Kong, Omeed Maghzian, Meghan Zacher, Tuan Anh Nguyen-Viet et al. "Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals." *Nature Genetics* 50, no. 8 (2018): 1112-1121.
Educational attainment	Okbay, Aysu, Yeda Wu, Nancy Wang, Hariharan Jayashankar, Michael Bennett, Seyed Moeen Nehzati, Julia Sidorenko et al. "Polygenic prediction of educational attainment within and between families from genome-wide association analyses in 3 million individuals." *Nature Genetics* 54, no. 4 (2022): 437-449.
Risk tolerance	Karlsson Linnér, Richard, et al. “Genome-wide association analyses of risk tolerance and risky behaviors in over 1 million individuals identify hundreds of loci and shared genetic influences.” *Nature Genetics* 51.2 (2019): 245-257.
Mental health	
Anxiety disorder	Forstner, Andreas J., et al. “Genome-wide association study of panic disorder reveals genetic overlap with neuroticism and depression.” *Molecular Psychiatry* 26.8 (2021): 4179-4190.
Bipolar disorder	O’Connell, K.S., Koromina, M., van der Veen, T. et al. Genomics yields biological and phenotypic insights into bipolar disorder. *Nature* 639, 968–975 (2025).
Major depressive disorder (MDD)	Adams MJ, Streit F, Meng X, Awasthi S, et al. (2025) Trans-ancestry genome-wide study of depression identifies 697 associations implicating cell types and pharmacotherapies. *Cell*. 6;188(3):640-652
Schizophrenia	Trubetskoy, Vassily, et al. “Mapping genomic loci implicates genes and synaptic biology in schizophrenia.” *Nature* 604.7906 (2022): 502-508.
Physical health	
Childhood-onset asthma	Ferreira, Manuel AR, Riddhima Mathur, Judith M. Vonk, Agnieszka Szwajda, Ben Brumpton, Raquel Granell, Bronwyn K. Brew et al. "Genetic architectures of childhood-and adult-onset asthma are partly distinct." *The American Journal of Human Genetics* 104, no. 4 (2019): 665-684.

Childhood allergy	We used GWAS summary statistics from the FinnGen project (R12 release, November 2024), which includes data from over 500,000 individuals linked to national health registry information. The file for endpoint F5_CHILDHOOD_ALLERGY (behavioural and emotional disorders with onset usually occurring in childhood and adolescence) was downloaded via Google Cloud Storage following public access approval.
Childhood BMI	Vogelezang, Suzanne, Jonathan P. Bradfield, Tarunveer S. Ahluwalia, John A. Curtin, Timo A. Lakka, Niels Grarup, Markus Scholz et al. "Novel loci for childhood body mass index and shared heritability with adult cardiometabolic traits." *PLoS Genetics* 16, no. 10 (2020): e1008718.
Migraine	Hautakangas, H., Winsvold, B.S., Ruotsalainen, S.E. et al. Genome-wide analysis of 102,084 migraine cases identifies 123 risk loci and subtype-specific risk alleles. *Nature Genetics* 54, 152–160 (2022).
Type 1 diabetes	Chiou, Joshua, Ryan J. Geusz, Mei-Lin Okino, Jee Yun Han, Michael Miller, Rebecca Melton, Elisha Beebe et al. "Interpreting type 1 diabetes risk with genetics and single-cell epigenomics." *Nature* 594, no. 7863 (2021): 398-402.
Developmental trajectories	
Autism spectrum disorder (ASD)	Grove, Jakob, et al. “Identification of common genetic risk variants for autism spectrum disorder.” *Nature Genetics* 51.3 (2019): 431-444
Obsessive-compulsive syndrome (OCD)	Strom, N.I., Burton, C.L., Iyegbe, C. et al. Genome-Wide Association Study of Obsessive-Compulsive Symptoms including 33,943 individuals from the general population. *Mol Psychiatry* (2024). 29(9):2714-2723
Genetic generalised epilepsy	GWAS meta-analysis of over 29,000 people with epilepsy identifies 26 risk loci and subtype-specific genetic architecture. *Nature Genetics* 55, no. 9 (2023): 1471-1482.
Tourette’s syndrome	Yu, Dongmei, et al. “Interrogating the genetic determinants of Tourette’s syndrome and other tic disorders through genome-wide association studies.” *American Journal of Psychiatry* 176.3 (2019): 217-227.