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The Intergenerational Transmission of Mental Health*

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Abstract

We use population-wide Danish administrative registers to construct an objective, comparable measure of mental health for parents and children, drawing on psychiatric contacts, diagnoses, psychotherapy, and psychotropic medication. Using first-borns from the 1996–2006 cohorts, we estimate substantial intergenerational transmission: parental mental health events occurring from five years before birth through the child's third birthday are associated with a 9.1 percentage point increase in the child's probability of experiencing a mental health event by age 15. To disentangle biological from environmental channels, we implement an adoptee design. About half of the parent-child association reflects the family environment. Environmental transmission is concentrated in maternal pathways, accounting for roughly three quarters of the mother-child transmission, while the father-child association is driven primarily by biology. We also find pronounced heterogeneity by child gender: transmission to daughters appears predominantly biological, whereas transmission to sons operates mainly through the family environment and is driven by mothers.

Keywords: Mental health, intergenerational transmission, nature, nurture.

JEL Classifications: TBD

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1 Introduction

Mental health disorders are common in childhood and adolescence. Globally, an estimated 7% of children aged 5–9 and 14% of adolescents aged 10–19 have a mental disorder (World Health Organization, 2025). These early-life conditions often persist: roughly half of all lifetime mental health cases are diagnosed by age 14 (Kessler et al., 2007). Because mental health is an important input into long-run human capital and socioeconomic success (Bhalotra et al., 2025; Biasi et al., 2025; Currie, 2025), persistence across generations has the potential to amplify inequality in both health and economic outcomes over the life course. Poor mental health can distort economic preferences, beliefs, and marginal utility (Ridley et al., 2020; Quidt and Haushofer, 2016). As a result, intergenerational transmission of mental health problems may perpetuate disadvantage across multiple dimensions.

In this paper, we use rich administrative data from Denmark to examine the intergenerational transmission of mental health and the roles of genetic and environmental factors in driving these associations. Our mental health measure combines information on contacts with health care providers, diagnostic records, use of psychotherapy, and psychotropic medications, and thus provides an objective register-based measure of mental health that is consistently defined for parents and children. We measure parental mental health from five years before birth until the child turns three, and we measure child mental health from birth through age 15. We find that parental mental health is strongly associated with child mental health. In the biological-child sample, a child whose parent experiences a mental health event is about 9 percentage points (83%) more likely to experience a mental health event by age 15. The associations are economically large and statistically significant for both parents and larger for mothers.

This strong association could reflect genetic transmission, shared environmental factors, or both. Distinguishing these channels is not merely an academic exercise; it has direct implications for policy. If transmission operates primarily through genetics, interventions may need to focus on early screening and treatment of at-risk children. If transmission reflects the family environment, then supporting parental mental health and parenting capacity could break the cycle. Standard observational designs cannot separate these channels because biological parents provide both genes and environment to their children.

We therefore implement an adoptee design to assess the extent to which intergenerational associations reflect biology versus the family environment. Following Sacerdote (2007), we focus on foreign adoptees who are genetically unrelated to their adoptive parents. Under the assumption that adoption assignments are as-good-as-random conditional on observables, any observed correlations between adoptive parent-child pairs identify environmental effects. We validate this assumption by showing that pre-adoption child characteristics (child gender and age at arrival) are uncorrelated with adoptive parent characteristics, consistent with prior work (Fagereng et al., 2021; Kleven et al.,

2021; Sacerdote, 2007). To address the positive selection of adoptive parents on observable characteristics, we implement inverse-probability-weighted regressions that reweight the biological-child sample to match the adoptee sample. We restrict attention to children adopted before age two to limit exposure to the pre-adoption environment and to reduce concerns that adoptees differ from biological children due to selection into adoption or pre-adoption conditions.

We implement this approach in order to separate the role of environment from that of "biology-related channels." We use this term rather than "genetic transmission" because associations between biological parents and their children can reflect multiple mechanisms: direct genetic inheritance of risk alleles, gene-environment interactions where genetic predispositions are expressed under certain environmental conditions, epigenetic effects transmitted through prenatal exposures, and assortative mating on heritable traits. While we cannot separately identify these mechanisms, distinguishing biology-related from purely environmental pathways remains valuable for policy design, as environmental channels are more readily amenable to intervention.

We document four main findings from the adoptee design. First, about half of the observed intergenerational persistence in mental health is attributable to biology-related pathways, with the remainder reflecting the family environment. Second, the relative importance of biology and environment differs by parent gender. Around 75% of the mother-child association reflects environmental transmission, while the father-child association is driven primarily by biology-related pathways. Third, there is important heterogeneity by child gender. Overall transmission is larger for sons than for daughters. The mother-son association exceeds the mother-daughter association, while the father-daughter association exceeds the father-son association. Finally, transmission to daughters appears to be driven mainly by biology-related pathways, whereas transmission to sons operates primarily through the family environment and is concentrated in maternal channels.

Our paper contributes to several strands of literature. First, we add to the small but growing body of research on the intergenerational transmission of mental health. The majority of this literature relies on survey data. Using the British Cohort Study, Johnston et al. (2013) report a mother-child association in mental health on the order of 0.19. Based on the UK Household Longitudinal Study, Bencsik et al. (2023) obtain closely related estimates, with persistence of roughly 0.21 when mental health is measured in levels and about 0.19 when measured in ranks. Using Australian HILDA data, Vera-Toscano and Brown (2022) similarly estimate intergenerational persistence of approximately 0.21. A notable exception to the survey-based literature is Bütikofer et al. (2024), who use Norwegian administrative data and document intergenerational persistence in mental health, measuring parental mental health using sickness absence records and child mental health using primary care and emergency contacts. They find that the probability of a child having a mental health event is 9.3 percentage points higher if a parent has a mental health problem. This estimate is very close to our baseline association of about 9 percentage points. We add to these studies by constructing an objec-

tive, comprehensive measure of mental health that captures not only diagnoses but also treatment through psychotherapy and psychotropic medication, and by applying this measure consistently to both parents and children. In addition, we provide evidence on the relative importance of biology and environment in intergenerational mental health transmission.

Second, our paper relates to the literature using adoptee designs to decompose intergenerational associations into genetic and environmental components. Sacerdote (2007) pioneered the use of Korean adoptees to study intergenerational mobility in education and income. Björklund et al. (2006) and Plug (2004) use Swedish and U.S. adoptee samples to study educational attainment. More recently, Black et al. (2020) and Fagereng et al. (2021) study wealth transmission using Swedish and Norwegian adoption data, respectively. While adoptee designs have been used to study educational and economic outcomes, and in the medical literature to address nature–nurture questions for specific conditions (e.g., Amstadter et al., 2024; Kendler et al., 2023, 2022, 2020; McAdams et al., 2015), we are the first to do so for overall mental health.[1]

Our findings have particular salience given substantial treatment gaps in mental health care. Even in high-income countries, treatment gaps remain substantial, with an estimated 40% of people with mental health needs not receiving treatment (Ndetei et al., 2023). These gaps imply that intergenerational persistence in mental health can meaningfully amplify the long-run burden of mental illness by transmitting risk across generations. Against this background, our results point to the potential value of policies that reduce the downstream impact of parental mental health on children, including early identification of at-risk families, timely access to evidence-based psychotherapy, and family-based interventions that support parenting and caregiving capacity during early childhood. The heterogeneity in our findings further suggests that targeting support to households in which mothers experience mental health problems, and focusing on sons in particular, may yield especially high preventive returns.

2 Institutional Background

2.1 Mental Health Care in Denmark

Denmark has a tax-financed universal health insurance system in which general practitioners (GPs) typically serve as the first point of contact and act as gatekeepers to specialist care for both adults and children.

Mental health conditions are commonly identified in primary care through clinical symptom assessment and, when appropriate, standardized psychometric instruments. The process leading

[1] One exception is Marceau et al. (2025), who specifically study heterotypic (cross-domain) transmission in an adoptee design, but with a focus on child externalizing and internalizing behavior.

to diagnosis and treatment differs between adults and children. Adults are typically diagnosed in primary care and treatment can remain in primary care for milder conditions. Referral decisions depend on severity: clinical guidance emphasizes psychotherapy for mild cases, a combination of psychotherapy and pharmacological treatment for moderate cases (with GPs able to prescribe psychotropic medications), and referral to psychiatrists based in either hospital psychiatric departments or private practice for more severe conditions.[2]

For children and adolescents, referral thresholds are lower and official guidelines recommend diagnosis and treatment by specialists (child psychiatrists). In addition to GPs, teachers in kindergartens and in schools may raise concerns about children and relay them to parents or municipal services. The latter can also initiate evaluation and, when warranted, referral to specialized child and adolescent mental health care, with the majority of specialist treatment occurring in hospital child and adolescent psychiatric departments. Treatment guidelines in pediatric care indicate that treatment begins with non-pharmacological interventions (psychotherapy), and that when pharmaceutical treatment is indicated it is prescribed and managed by child psychiatrists. Given these differences in referral and treatment pathways between children and adults, it is important to use a mental health measure that captures both health care contacts and treatment, rather than relying solely on any single component such as diagnoses or prescriptions.

Financial barriers to health care are likely to be limited in Denmark's universal system. Consultations with general practitioners and hospital-based services are fully covered under the National Health Insurance Scheme, as is specialist psychiatric care when accessed through the referral system. Adult psychotherapy delivered by office-based psychologists requires out-of-pocket payments but is partially reimbursed when patients meet eligibility criteria and have a referral.[3] Prescription drugs are also not fully covered but subsidized. Prescription drug subsidies have shifted over time from fixed coinsurance (pre-2000) to a non-linear reimbursement schedule in which the coinsurance rate declines with cumulative prescription spending over the coverage year.[4]

2.2 Foreign Adoption in Denmark[5]

Danish law distinguishes between kin and non-kin adoption. Kin adoption includes stepchild adoption (adopting the child of a spouse or of a cohabiting partner) and family adoption (adopting a closely related child, or where there is a special attachment between the adopter and the child or

[2] Acute psychiatric emergency contacts do not require referral.

[3] Approximately 60% of a 12-week psychotherapy treatment are covered for eligible patients.

[4] Citalopram, a widely prescribed antidepressant, had a 25% coinsurance rate before Denmark adopted the non-linear subsidy scheme (Simonsen et al., 2021). The current full price of a 3-month supply of citalopram is below 75 DKK, or 10 EUR.

[5] This section draws on the description of the adoption landscape in Denmark provided in Henze-Pedersen and Olsen (2017).

the child's parents). In both cases, at least one of the adoptive parents is biologically related to the adopted child.

Non-kin adoption, on the other hand, refers to cases in which both of the adoptive parents are biologically unrelated to the adopted child, and it includes both national and international placements. Statistics Denmark figures over our sample period (1996–2006) indicate that non-kin adoptions of Danish children averaged about 52 per year, while non-kin adoptions from abroad were roughly ten times larger, at around 570 per year. This implies that, among non-kin adoptions, international placements were by far the dominant route for Danish adoptive parents during this period.

During this period, international non-kin adoption followed a clearly defined approval process in four stages. Prospective parents first underwent a municipal assessment of eligibility and suitability (phase 1). They then completed a mandatory adoption-preparatory course (phase 2). Phase 3 required them to submit a formal application to adopt and to receive the final approval decision. Approved applicants were subsequently placed on a waiting list through the relevant authority (the Danish International Adoption agency). Finally, adoptive parents received mandatory counseling immediately before and after the arrival of the adopted child in order to support the transition into adoptive family life (phase 4). This approval process, together with the eligibility criteria it enforces (including requirements related to health, housing, and economic circumstances), implies that adoptive parents are likely to be positively selected relative to biological parents, at least in terms of socioeconomic status.

3 Empirical strategy

Our objective is to estimate the intergenerational transmission of mental health and to disentangle the roles of genetic factors (*nature*) and environmental factors (*nurture*). We begin with a reduced-form specification:

$$y_i = \beta_0 + \beta_1 y_i^f + \beta_2 y_i^m + c_i + c_i^f + c_i^m + \nu_i, \tag{1}$$

where y_i, y_i^f, and y_i^m denote mental health measures for the child, father, and mother, respectively. Our preferred specification includes both parents to account for assortative mating and cohort fixed effects for all individuals (c_i, c_i^f, c_i^m) to control for differences in prevalence, diagnosis, and prescription behavior over time.

When estimated on a sample of biological families, β_1 and β_2 capture the combined influence of genetic and environmental transmission. To separate these channels, we estimate child-parent correlations for internationally adopted children, who are genetically unrelated to their adoptive parents. Hence, under the assumption that adoption assignments are as-good-as-random, any observed correlations between child-parent pairs in adoptive families identify environmental effects. We validate

this assumption by showing that pre-adoption child characteristics (child gender and age at arrival) are uncorrelated with adoptive parent characteristics, consistent with prior work (Fagereng et al., 2021; Kleven et al., 2021; Sacerdote, 2007).[6]

Even under the assumption of as-good-as-random assignment of adoptive children, child-parent correlations may differ between adoptive and biological families for reasons other than the absence of genetic links. As shown below, adoptive parents are on average older and better educated when they receive their first child; if the intergenerational transmission of mental health varies along these dimensions, such differences in parental characteristics will generate differences in correlations between the two samples.

To address this selection into adoption, we reweight the biological sample to match the sample of adoptive parents on observables.[7] We construct weights by first estimating a logistic regression of an indicator for being adoptive parents on child cohort, parental age at birth, education, and cohabitation status. We then compute inverse-odds weights as

$$w_i = \frac{\hat{p}_i}{1 - \hat{p}_i}, \tag{2}$$

where $\hat{p}_i$ is the predicted probability of being adoptive parents. Applying these weights to the biological sample ensures that differences in estimated correlations between biological and adoptive families are not driven by observable parental selection into adoption.

Finally, under the assumption that the intergenerational transmission of mental health is a linear combination of genetic transmission (or more precisely, biologically related transmission) and environmental transmission, we can estimate the role of biological transmission as the difference between the child-parent correlations estimated in the sample of biological children and those in the sample of adoptive children. This then allows us to estimate the relative share of each channel in the total intergenerational transmission of mental health.

4 Data

We use several population-level administrative data sets from Denmark. These data include individual-level records with unique personal identifiers that allow us to follow individuals over time and to link family members to one another.

[6] Another group of adoption studies use domestic adoptions with information on both adopting and biological parents to control for non-random allocation of adoptive children to parents (Black et al., 2020; Björklund et al., 2006; Plug, 2004). Lundborg et al. (2025), who study child–parent correlations for children conceived with donor egg and/or sperm ("in-uterus adoption"), also fall into this group.

[7] By only reweighting the biological sample, we preserve statistical power in the much smaller sample of adoptive parents.

Measurement of Mental Health. Our register-based mental health indicators provide an objective measure of one's health by capturing both mental health diagnosis and treatments. We combine information from three different registers with coverage beginning in the early to mid 1990s. For consistency, we use data starting in 1995, the year when the last of these three registers starts. First, we use the *Psychiatric Central Research Register*, which records all inpatient admissions, outpatient visits, and emergency department visits to psychiatric units in public and private hospitals covered by the national health insurance system. Second, we use the *National Health Insurance Register*, which contains reimbursements to private practices, including office based specialists, for all services covered by national health insurance. Third, we use the *Danish Prescription Register*, which covers all prescription drugs dispensed at Danish pharmacies and reports each medication's Anatomical Therapeutic Chemical (ATC) code. Using these sources, we define indicator measures for whether an individual has a mental health condition based on any qualifying hospital diagnosis, contacts with psychiatrists and psychologists (capturing psychotherapy), or dispensing of relevant psychotropic medications.[8]

Because register based measures of mental health care utilization begin in 1995, we cannot observe parents and children over identical age windows. We therefore measure parental mental health in an eight-year window spanning five years before childbirth and three years after childbirth. Including the post-birth years increases statistical power and reduces attenuation from transitory measurement error that would arise from relying on a single year, while capping the window at three years after childbirth limits concerns about reverse causality.[9] We measure child mental health from birth through age 15, a window that spans critical developmental periods and captures the onset of a large share of lifetime mental disorders, with roughly half of all lifetime cases diagnosed by age 14 (Kessler et al., 2007).

Additional Variables. We link in several additional registers to measure parental and child characteristics used in robustness checks and heterogeneity analyses. From the *Population Register*, we obtain year of birth, gender, and parents' marital or cohabitation status. The *Birth Register* provides information on the child's birth weight. The *Education Register* is used to measure the highest level of schooling of the parents. Finally, we use the *Income Statistics Register* to construct parents' decile rank in the national income distribution within their birth cohort. Unless otherwise noted, time-varying parental characteristics are measured two years prior to the child's birth.

[8] Appendix Table 1 lists the ICD-10 diagnosis groups, ATC codes, and provider and service codes used to construct the measures. The hospital diagnosis groupings follow Kessing et al. (2023, eAppendix1) and Momen et al. (2020, TableS1), and the prescription based list follows Kessing et al. (2023, eAppendix 2).

[9] Appendix Figure 1 shows the cumulative incidence of the mental health indicator by age for biological and adopted children: around 3% of children are classified as having a mental health condition by age 3 in both samples. In Denmark, age 3 also corresponds to entry into kindergarten, where early developmental and behavioral concerns are more likely to be detected.

Identification of Foreign Adoptees. The adoption records at Statistics Denmark are incomplete. Following Kleven et al. (2021), we therefore identify foreign adoptees as children meeting the following criteria: (i) the child's country of birth is a non-Western country; (ii) the child is first observed in the Danish Civil Registration System with an immigration date after their birth; (iii) both parents were born in Western countries; and (iv) both parents were legal residents of Denmark at the child's birth and have no emigration record at that date. We restrict the sample to children placed with adoptive parents before age 2 to limit exposure to the pre-adoption environment and to maximize time spent in the adoptive home.

Kleven et al. (2021) assess the accuracy of this procedure by comparing the inferred adoptee sample to the subset of years in which official adoption records exist. The match is close to one-to-one: very few biological children are incorrectly classified as adoptees (Type I errors), and very few adoptees fail to be identified (Type II errors). This implies that misclassification is limited and unlikely to meaningfully affect our estimates.

Analysis Sample. Our sample consists of first-born children from the 1996–2006 birth cohorts, which allows us to measure parental mental health for at least one year before childbirth and to follow children from birth through age 15 in the administrative registers. We focus on children whose parents are both born in Denmark, to reduce heterogeneity in attitudes toward mental health conditions and their treatment (Alonso et al., 2008). We further restrict attention to children who are observed at each age from 6 through 15, ensuring a common window in which we measure child mental health outcomes during middle childhood and adolescence, when clinically detected conditions are most prevalent. Finally, we exclude children whose parents cannot be observed in the Danish registers for at least 10 years during the child's life by age 16. This restriction ensures that the child has substantial exposure to each parent.[10] These sample restrictions leave us with 253,446 children, of which 251,538 are biological children and 1,908 are foreign adoptees.

Descriptive Statistics. Columns 1 and 2 of Table 1 report summary statistics for children and parents in the adoptee and biological-child samples, respectively. Because sample sizes differ sharply across groups, Column 3 assesses balance using standardized (normalized) differences rather than t-tests (McKenzie, 2017; Imbens and Rubin, 2015).[11] The statistics indicate that the adoptee sample differs from the biological-child sample along several dimensions. Standardized differences are large for child gender and for parental demographic and socioeconomic characteristics, indicating that

[10] This restriction is based on parental presence in the Danish registers (that is, being alive and resident in Denmark), not on parents' marital or cohabitation status.

[11] The standardized difference is defined as $\frac{\overline{X}_A - \overline{X}_B}{\sqrt{\frac{1}{2}\left(s_A^2 + s_B^2\right)}}$, where $\overline{X}_A$ and $\overline{X}_B$ are group means and s_A^2 and s_B^2 are variances. This provides a scale-invariant measure of the magnitude of imbalance.

the adoptee sample is positively selected along observed family stability and socioeconomic status. While the raw means also suggest that adoptive parents have lower prevalence of our mental health indicator, the corresponding standardized differences are comparatively small, indicating only modest imbalance in parental mental health across the two samples.

As described in Section 3, we address these imbalances by implementing inverse probability weighted regressions that reweight the full sample to match adoptees, where the weights are constructed using the following characteristics: parental marital or cohabitation status, parental age, parental education, child gender, and the birth year of the child. Column 4 reports summary statistics for the reweighted sample of biological children, with the corresponding standardized differences shown in Column 5. The inverse-probability weights substantially improve balance between the reweighted full sample and the adoptee sample, with standardized differences below 0.1 for all covariates except age at first diagnosis and fathers' income decile, for which the standardized differences remain slightly above 0.1. Importantly, even though we do not use parental mental health when constructing the weights, weighting reduces the standardized differences for both mothers' and fathers' mental health. This suggests that the sample of adoptive parents is not (positively) selected on mental health conditional on the observable characteristics used to construct the weights.

5 Results

5.1 Intergenerational Transmission of Mental Health

Table 3 reports estimates of intergenerational associations in mental health between parents and children based on Equation (1). Column 1 uses the sample of biological children, Column 2 uses the reweighted biological-child sample, and Column 3 uses the sample of adoptees. Panel A considers transmission from either parent, while Panel B estimates associations for mothers and fathers separately. The outcome means reported at the bottom of the table correspond to the share of children with at least one mental health event in the registers among those whose parents are not observed with any mental health event. This baseline prevalence is 11% among biological children and 15% among adoptees.

In the unweighted biological-child sample (Column 1), we find a strong association between parental and child mental health. Children whose parents have a mental health event in the window from five years before birth through three years after birth are 9.1 percentage points more likely to experience a mental health event between birth and age 16. Relative to the baseline prevalence, this corresponds to an 83% increase. In Panel B, we find evidence of substantial associations for both parents, but the association is larger for maternal mental health (9 percentage points) than for paternal mental health (6.6 percentage points).

It is difficult to compare our estimates of intergenerational persistence with prior studies because many papers rely on continuous, standardized survey indices and report correlations or rank-rank slopes rather than level effects on a binary outcome as in our setting (Davis et al., 2025). One notable exception is Bütikofer et al. (2024), who study intergenerational persistence in mental health using Norwegian administrative data, measuring parental mental health through sickness absence and adolescent mental health through GP and emergency contacts. Our baseline estimate of 9.1 percentage points is very close in magnitude to the 9.3 percentage point estimate reported in their study.

5.2 Role of Biology and Environment

Columns 2 and 3 of Table 3 report estimates of intergenerational associations in mental health in the reweighted sample of biological children and in the sample of adoptees. Relative to the unweighted biological child estimates in Column 1, the estimated associations in the reweighted biological child sample are somewhat smaller. This pattern is consistent with meaningful differences in observed family characteristics between biological and adoptive families, and with these characteristics being correlated with both parental and child mental health outcomes. Importantly, although the coefficients decline after reweighting, they remain sizable and precisely estimated, indicating that strong parent-child associations persist even after accounting for these observable compositional differences.

Turning to adoptees, Panel A of Column 3 indicates that adopted children whose adoptive parent experiences a mental health event are 4.1 percentage points more likely to experience a mental health event themselves. Relative to the baseline prevalence among adoptees whose parents are not observed with any mental health event, this corresponds to a 27% increase. The adoptee coefficient is about 55% of the reweighted biological child coefficient. Under the standard interpretation of the adoptee design, this ratio provides a descriptive decomposition in which roughly half of the intergenerational association reflects environmental transmission, with the remainder attributable to channels specific to biological links, including genetic transmission and other biology-linked mechanisms.

Panel B points to potentially important heterogeneity by parent. In the reweighted biological child sample, both maternal and paternal associations are sizeable. Among the adoptee sample, the maternal association remains economically meaningful, whereas the paternal association is close to zero and statistically insignificant. Our results suggest that approximately three quarters of the transmission from mothers is related to the environment. In contrast, the stronger father-child association observed among biological families is more tightly linked to biology-specific channels. Comparing to the literature on child-parent correlations in education, our results mirror those of Lundborg

et al. (2025) who also find that fathers seem to matter only throught their biological link to the child.

Internal validity and robustness of the adoptee design. As emphasized in prior work using the adoptee design (e.g., Holmlund et al., 2011), two issues are central for the comparison to biological families. First, adoptive parents may differ systematically from other parents, both due to self-selection into adoption and because adoption typically requires meeting eligibility criteria. In our data, adoptive parents are positively selected along several observable dimensions, including higher family stability at birth and higher socioeconomic status. A key feature of our analysis is that we address these compositional differences directly by reweighting the sample of biological families to match the adoptee sample on a parsimonious set of observables.

Second, adoptees themselves may differ from other children, either because of selection into adoption (including country-of-origin selection) or because pre-adoption conditions may affect later mental health. To mitigate exposure to the pre-adoption environment, we restrict the adoptee sample to children placed with adoptive parents before age 2. Table 4 further sheds light on the comparability of adoptees (and their parents) to biological children (and their parents) by examining how sensitive the estimated intergenerational associations in mental health are to the inclusion of additional control variables. Within each sample, we first add controls for child characteristics (gender and, for adoptees, age at adoption in days) and then add controls for family characteristics (parental education and income decile). The estimates are largely unchanged when these additional controls are included, indicating that the difference in intergenerational associations between biological children and adoptees is not materially driven by residual differences in these observed characteristics.

Heterogeneity by child gender. In Table 5, we examine heterogeneity in intergenerational mental health associations by child gender. In the reweighted biological child sample, the parent-child association is larger for sons than for daughters. Decomposing by parent, the maternal association exceeds the paternal association overall, and the mother-son association is stronger than the mother-daughter association. In contrast, the father-daughter association is larger than the father-son association. These gender-specific patterns are consistent with prior evidence documenting heterogeneous parent-child gradients by both the parent's and the child's gender (Mazumder, 2026).

The adoptee results highlight important differences in the relative roles of biology and environment by child gender. Among daughters, we find no detectable association in the adoptee sample, either in the "any parent" specification or when separating adoptive mothers and fathers. In contrast, we find a positive association among adopted sons, and this association is driven by adoptive mothers, while the estimate for adoptive fathers is small and statistically insignificant. Taken together, these patterns suggest that transmission to girls is more tightly linked to biology-specific pathways, whereas transmission to boys appears to operate primarily through the family environment and, in

particular, through maternal channels.

6 Conclusion

We document substantial intergenerational persistence in mental health using population-wide Danish administrative data and an objective measure that combines health care contacts, diagnoses, and treatment. We then implement an adoptee design to disentangle the roles of biology and the family environment and find that roughly half of the parent–child correlation in mental health is attributable to biology-linked transmission, with the remainder reflecting the family environment.

Several patterns in our results suggest that some interventions may be effective at reducing the transmission of poor mental health from parents to children. First, the sizable environmental component implies that intergenerational persistence is not purely mechanical. Interventions that improve parental mental health and the early-life home environment, including reducing barriers to early treatment, could shift the mental health trajectories of children. Second, environmental transmission is concentrated in maternal pathways, accounting for about three quarters of the mother–child association, while the father–child association is primarily driven by biology. Strengthening maternal mental health and caregiving capacity from pregnancy through early childhood (e.g., through improved screening and quick access to effective mental health care) could thus lead to improvements in child health. Third, we find pronounced heterogeneity by child gender: for girls, parent–child associations are largely biology-related, whereas for boys they operate mainly through the family environment and are driven by mothers. This pattern suggests that family-based prevention and early intervention in families experiencing maternal mental health problems would be expected to yield particularly large gains for sons.

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Table 1: Descriptive statistics

	Adopted children	Biological children (raw)	Std. diff.	Biological children (weighted)	Std. diff.
	(1)	(2)	(3)	(4)	(5)
A. Child characteristics					
Any mental health diagnosis/treatment	0.16	0.14	0.056	0.13	0.099
Age at first diagnosis/treatment	10.59	10.92	−0.086	11.20	−0.158
Girl	0.59	0.49	0.200	0.58	0.008
Parents cohabiting at birth	0.98	0.66	0.905	0.98	0.000
Mother's characteristics					
Any mental health diagnosis/treatment	0.20	0.25	−0.103	0.23	−0.053
Age at child birth	34.56	27.83	1.638	34.55	0.000
Years of education	14.81	13.78	0.471	14.81	0.003
Income decile	5.80	5.68	0.046	5.92	−0.047
Father's characteristics					
Any mental health diagnosis/treatment	0.11	0.16	−0.126	0.14	−0.089
Age at child birth	35.76	30.30	1.191	35.79	−0.006
Years of education	14.82	13.84	0.427	14.83	−0.003
Income decile	7.49	6.81	0.245	7.22	0.096
Observations	1,908	251,538		251,538	

Notes: Sample of first-born children of native parents or children adopted into previously childless native families, born between 1996–2006. Mental health diagnosis/treatment refers to any hospital visit to a psychiatric department with ICD-10 diagnosis codes F00–F99, any visit to a privately-practicing psychiatrist or psychologist, or the use of psychotropic medications (antipsychotics, antidepressants, lithium, anxiolytics, medication for ADHD, or medication for alcohol and opioid dependence). See Appendix Table 1. Child mental health is measured from birth to through age 15, while parental mental health is measured from 5 years before birth to until the child turns 3. Age at onset refers to the age of the child when the first mental health diagnosis or treatment is observed. Parental education and income are measured two year prior to birth. Income deciles are determined within the birth cohort of each parent. Standardized differences (Std. diff.) are calculated as the difference in means between the adopted children sample and the biological children sample, divided by the pooled standard deviation.

Table 2: Correlation between parental characteristics and predetermined characteristics of adopted children

	Girl		Age at immigration (days)	
	Bivariate regressions (1)	Multivariate regression (2)	Bivariate regressions (3)	Multivariate regression (4)
A. Mother's characteristics				
Years of education	−0.002	−0.000	−2.007	−1.749
	(0.005)	(0.006)	(1.812)	(1.972)
Income (million 2015 DKK)	0.000	0.000	−0.041	−0.060
	(0.000)	(0.000)	(0.030)	(0.042)
Mental health	0.005	0.006	0.015	0.093
	(0.028)	(0.028)	(9.683)	(9.824)
B. Dad characteristics				
Years of education	−0.005	−0.005	−0.939	−0.329
	(0.005)	(0.005)	(1.696)	(1.837)
Income (million 2015 DKK)	−0.000	−0.000	−0.011	0.026
	(0.000)	(0.000)	(0.026)	(0.036)
Mental health	−0.005	−0.007	−4.114	−4.343
	(0.036)	(0.036)	(12.325)	(12.467)
Mean dependent variable	0.586	0.586	326.650	326.650
Joint significance F-stat		0.343		0.587
p-value		0.914		0.741

Notes: Sample of 1,908 children adopted into previously childless native families, born between 1996–2006. MH is an indicator for any hospital visit to a psychiatric department with ICD-10 diagnosis codes F00–F99, any visit to a privately-practicing psychiatrist or psychologist, or the use of psychotropic medications (antipsychotics, antidepressants, lithium, anxiolytics, medication for ADHD, or medication for alcohol and opioid dependence). See Appendix Table 1. Child mental health is measured from birth to through age 15, while parental mental health is measured from 5 years before birth to until the child turns 3. Years of education and income (in million 2015 Danish Kroner) are measured two years before the birth of the child. Each coefficient in columns 1 and 3 comes from a separate regression of the dependent variable in the column on the independent variable in the row. Robust standard errors are shown in parentheses. Significance levels: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 3: Intergenerational correlation in mental health

	Biological children (raw) (1)	Biological children (weighted) (2)	Adopted children (3)
Any parent MH	0.091***	0.074***	0.041**
	(0.002)	(0.005)	(0.020)
Mother MH	0.090***	0.072***	0.053**
	(0.002)	(0.006)	(0.023)
Father MH	0.066***	0.050***	−0.009
	(0.002)	(0.007)	(0.028)
Mean child MH	0.109	0.104	0.153
Observations	251,538	251,538	1,908

Notes: Sample of first-born children of native parents or children adopted into previously childless native families, born between 1996–2006. MH is an indicator for any hospital visit to a psychiatric department with ICD-10 diagnosis codes F00–F99, any visit to a privately-practicing psychiatrist or psychologist, or the use of psychotropic medications (antipsychotics, antidepressants, lithium, anxiolytics, medication for ADHD, or medication for alcohol and opioid dependence). See Appendix Table 1. Child mental health is measured from birth to through age 15, while parental mental health is measured from 5 years before birth to until the child turns 3. The sample in column 2 is reweighted using inverse probability weights calculated based on the following set of characteristics: child gender, child birth cohort, parental cohabitation at birth, parental age at birth, and parental years of schooling. All specifications include indicators for the birth cohort of the child and each parent. Robust standard errors are shown in parentheses. Significance levels: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 4: Robustness of intergenerational correlation in mental health

	Biological children			Adopted children		
	(1)	(2)	(3)	(4)	(5)	(6)
Any parent MH	0.074***	0.074***	0.069***	0.041**	0.039*	0.039*
	(0.005)	(0.005)	(0.005)	(0.020)	(0.020)	(0.020)
Mother MH	0.072***	0.072***	0.069***	0.053**	0.052**	0.052**
	(0.006)	(0.006)	(0.006)	(0.023)	(0.023)	(0.023)
Father MH	0.050***	0.050***	0.044***	−0.009	−0.012	−0.012
	(0.007)	(0.007)	(0.007)	(0.028)	(0.028)	(0.028)
Child characteristics		X	X		X	X
Parent characteristics			X			X
Mean child MH	0.104	0.104	0.104	0.153	0.153	0.153
Observations	251,538	251,538	251,515	1,908	1,908	1,908

Notes: Sample of first-born children of native parents or children adopted into previously childless native families, born between 1996–2006. MH is an indicator for any hospital visit to a psychiatric department with ICD-10 diagnosis codes F00–F99, any visit to a privately-practicing psychiatrist or psychologist, or the use of psychotropic medications (antipsychotics, antidepressants, lithium, anxiolytics, medication for ADHD, or medication for alcohol and opioid dependence). See Appendix Table 1. Child mental health is measured from birth to through age 15, while parental mental health is measured from 5 years before birth to until the child turns 3. The sample of biological children (Columns 1–3) is reweighted using inverse probability weights calculated based on the following set of characteristics: child gender, child birth cohort, parental cohabitation at birth, parental age at birth, and parental years of schooling. All specifications include indicators for the birth cohort of the child and each parent. Robust standard errors are shown in parentheses. Significance levels: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 5: Intergenerational transmission of mental health by child gender

	Girls		Boys	
	Biological children (1)	Adopted children (2)	Biological children (3)	Adopted children (4)
Any parent MH	0.068***	0.025	0.082***	0.059*
	(0.007)	(0.023)	(0.007)	(0.036)
Mother MH	0.061***	0.024	0.087***	0.100**
	(0.008)	(0.026)	(0.009)	(0.041)
Father MH	0.055***	0.003	0.046***	−0.039
	(0.009)	(0.033)	(0.010)	(0.049)
Mean child MH	0.098	0.110	0.113	0.214
Observations	122,366	1,118	129,172	790

Notes: Sample of first-born children of native parents or children adopted into previously childless native families, born between 1996–2006. MH is an indicator for any hospital visit to a psychiatric department with ICD-10 diagnosis codes F00–F99, any visit to a privately-practicing psychiatrist or psychologist, or the use of psychotropic medications (antipsychotics, antidepressants, lithium, anxiolytics, medication for ADHD, or medication for alcohol and opioid dependence). See Appendix Table 1. Child mental health is measured from birth to through age 15, while parental mental health is measured from 5 years before birth to until the child turns 3. The sample of biological children (columns 1 and 3) is reweighted using inverse probability weights calculated based on the following set of characteristics: child gender, child birth cohort, parental cohabitation at birth, parental age at birth, and parental years of schooling. All specifications include indicators for the birth cohort of the child and each parent. Robust standard errors are shown in parentheses. Significance levels: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

The Intergenerational Transmission of Mental Health

Online Appendix

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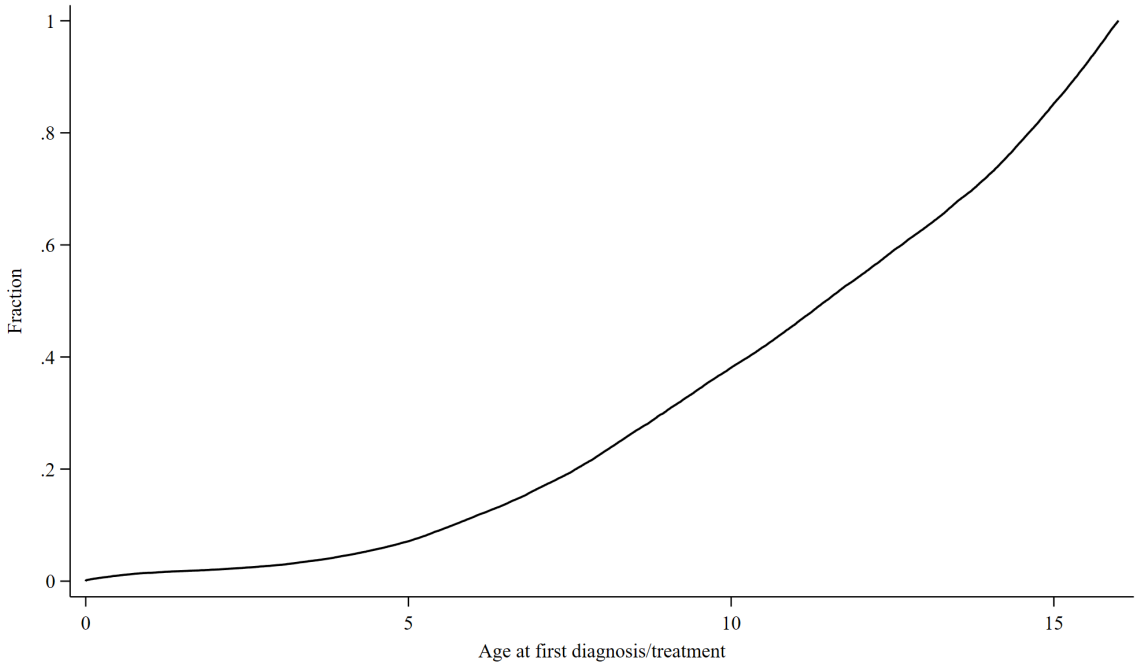
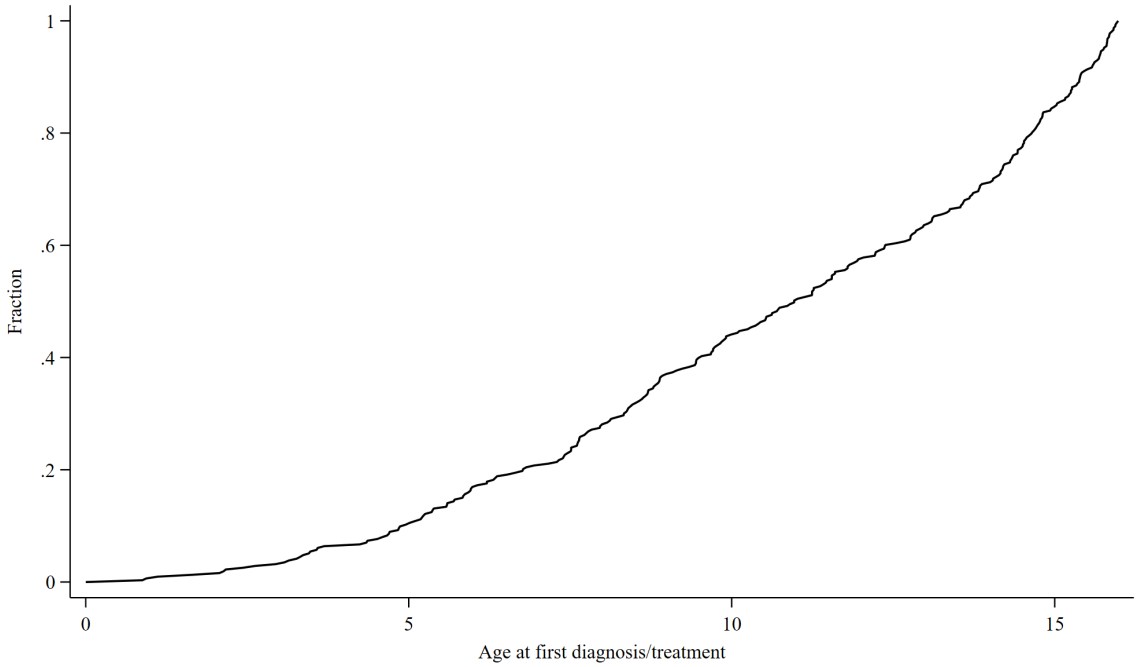


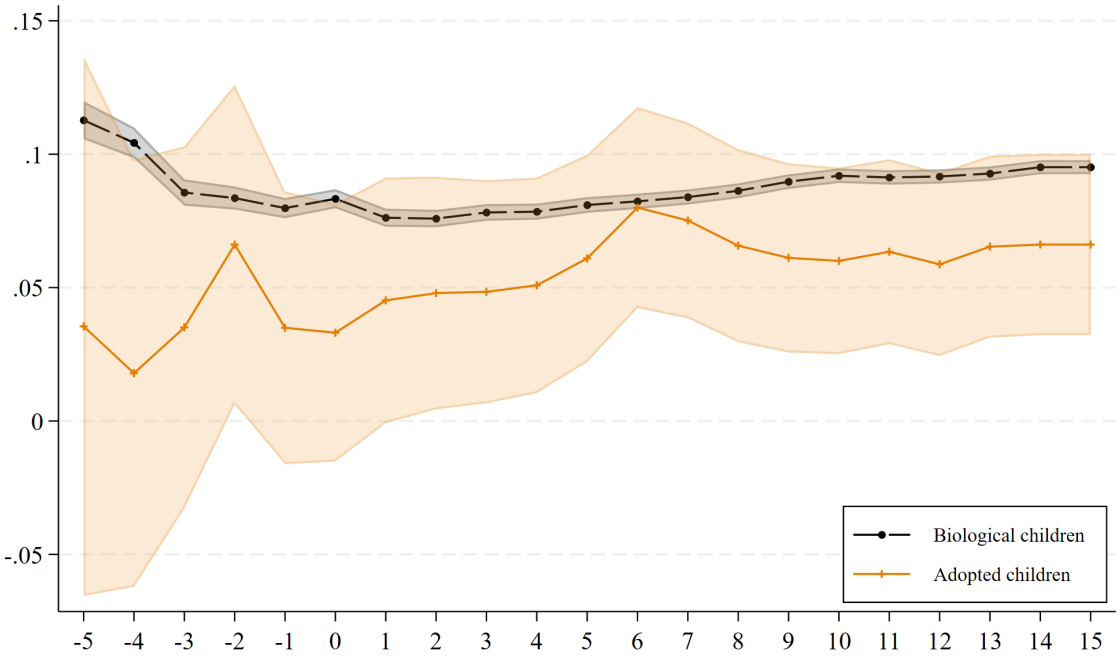
(a) Biological children



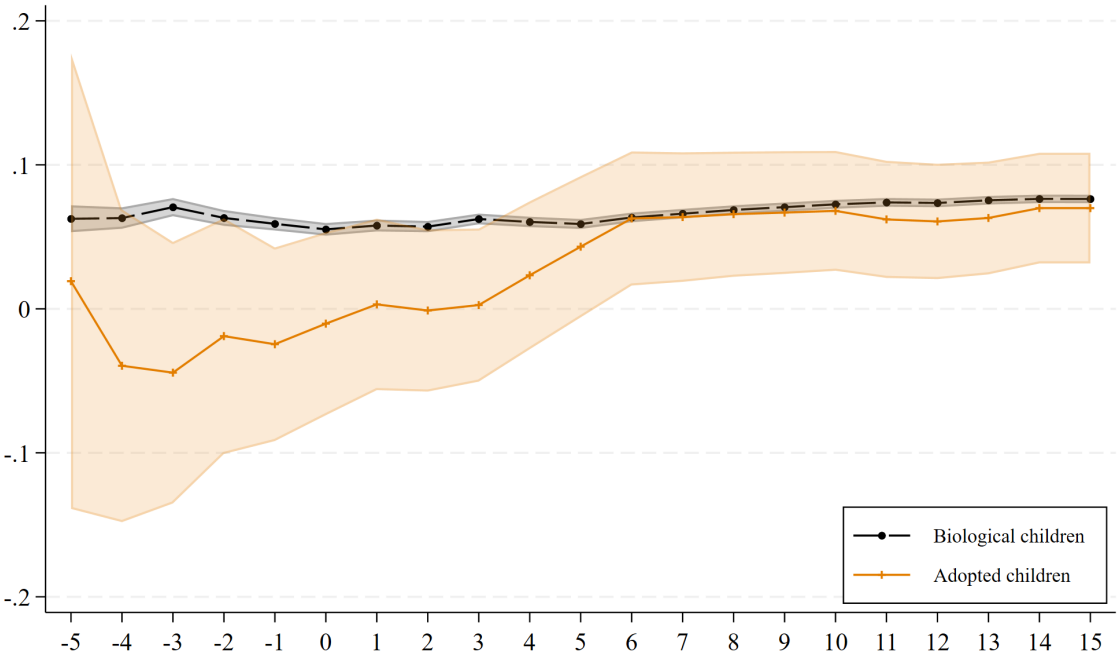
(b) Adopted children

Appendix Figure 1: Cumulative distribution function of age at onset of child mental health

Notes: Sample of first-born children of native parents or children adopted into previously childless native families, born between 1996–2006. The age at onset is the age of the first hospital visit to a psychiatric department with ICD-10 diagnosis codes F00–F99, of the first visit to a privately-practicing psychiatrist or psychologist, or of the first use of psychotropic medications (antipsychotics, antidepressants, lithium, anxiolytics, medication for ADHD, or medication for alcohol and opioid dependence).



(a) Mothers



(b) Fathers

Appendix Figure 2: Intergenerational correlation in mental health as a function of the time window used to measure parental mental health

Notes: Sample of first-born children of native parents or children adopted into previously childless native families, born between 1996–2006. Each dot plots the estimated coefficient from a regression of child mental health or parental mental health and indicators for the birth cohort of the child and of the parent, and the shaded areas represent the corresponding 95% confidence intervals. Child mental health is measured from birth through age 15. Parental mental health is measured from 5 years before birth (or 1995 if later) until the child age indicated on the horizontal axis. The solid line connects the estimates from the sample of biological children, estimated using inverse probability weights. The solid line connects the estimates from the sample of adopted children.

Appendix Table 1: Data sources and definition of mental health

Source	Definition	Years available
Hospital contacts: Danish Psychiatric Central Research Register	ICD-10 codes: F00–F09 (organic disorders), F10–F19 (substance use disorders), F20–F29 (schizophrenia), F30–F39 (mood disorders), F40–F48 (neurotic disorders), F50 (eating disorders), F60 (personality disorders), F70–F79 (intellectual disabilities), F84 (developmental disorders), F90–F98 (behavioral disorders)	1995–2021
Private-practice specialists: National Health Insurance Register (SYSI or SSSY)	SPECIALE codes starting with 63 (psychologist), 24 (psychiatrist), or 26 (child psychiatrist)	1990–2021
Medication use: National Prescription Register (LMDB)	ATC codes: N05A except N05AN (antipsychotics), N06A except N06AX12 and N06AX21 (antidepressants), N05AN (lithium), N05B (anxiolytics), C02AC02, N06BA02, N06BA04, N06BA09 and N06BA12 (ADHD medication), N07BB-N07BB04 (alcohol and opioid dependence)	1995–2021