(Breaking) Intergenerational Transmission of Mental Health

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ABSTRACT

We estimate health associations across generations using information on healthcare visits from administrative data for the entire Norwegian population. A parental mental health diagnosis is associated with a 9.3 percentage point (40 percent) higher probability of a mental health diagnosis of their adolescent child. Intensive margin physical and mental health associations are similar, and extended family estimates account for 42 percent of the intergenerational persistence. We also show that a policy targeting additional health resources for the young children of adults diagnosed with mental health conditions reduced the parent–child mental health association by 39 percent.

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I. Introduction

Mental health disorders are one of the leading causes of disability and contribute a sizable portion of the global disease burden, affecting more than one billion people worldwide (Rehm and Shield 2019). They also exhibited an upward trend in recent decades when it comes to both disability-adjusted life years as well as mortality, exerting unprecedented economic burden on societies. For example, in both the United States and Norway in 2013, mental health disorders topped the list of most costly conditions, generating expenditures of approximately $201 billion and NOK32 billion ($3.7 billion USD), respectively (Kinge et al. 2017; Roehrig 2016). The costs in the United States were projected to reach $225 billion by 2019 (Substance Abuse and Mental Health Services Administration 2014) while, by that year, the spending on mental and substance use disorders exceeded NOK65 billion in Norway (Kinge et al. 2023). Moreover, the demand for mental health care has been exacerbated even more by the COVID-19 pandemic. These numbers pertain only to medical spending, while the true economic costs are much greater due to productivity and learning losses, forgone taxes, and externalities imposed on other individuals. One such understudied externality is the intergenerational association between the mental health of parents and their children.

We ask the following questions relevant to understanding the persistence of mental health inequality across generations: What is the association between the mental health of parents and their children? Is this relationship different for mental compared with physical health? To what extent do we understate the intergenerational persistence of mental health conditions by not accounting for dynastic effects generated by members of the extended family? Armed with this knowledge, we then study if a policy that targeted additional health resources toward the young children of adults diagnosed with a mental health condition can mitigate the aforementioned parent–child associations.

We answer these questions by leveraging unique features of the Norwegian medical and social security registries. First, the data cover the full population of Norway, a country where healthcare is highly subsidized and easily accessible to everyone. This limits the scope for selection and increases the external validity of our findings. Second, the data contain family identifiers, which allow us to connect families across four generations. We use this information to expand our intergenerational analysis to dynasties, which include aunts/uncles, spouses of aunts/uncles, siblings of spouses of aunts/uncles, parents' cousins, and spouses of parents' cousins (as in Adermon, Lindahl, and Palme 2021).[1] Third, our health measures are based on primary healthcare visits, that is, general

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1. In the paper, we interchangeably use "extended family" and "dynasty."

practitioners (GPs) and primary care emergency room (ERs) visits, which provide an external and objective measure of one's physical and mental health. This is different from much prior research that relied on self-reported health, which could suffer from recall and subjective perception biases. In contrast, our measure of health through contact with the healthcare system captures a policy-relevant estimate of intergenerational persistence—one that directly triggers costs for taxpayers.

In the second part of the paper, we use a quasi-experiment to estimate the causal effect of an intervention targeting children of parents with a mental health diagnosis on the aforementioned parent–child association. In 2007, the Norwegian Directorate for Children, Youth, and Family Affairs implemented a pilot program in 26 municipalities that received small-scale funds with the goal of finding the best practices and follow-up models for young children of parents with mental health conditions. During the pilot program, chosen municipalities implemented a variety of light-touch changes targeted toward children from birth until school age (age 6), which included new screening tools to detect psychological distress, establishing and educating specialist teams, coordination with childcare centers, or prevention campaigns against substance use. We estimate the effects of this pilot program using a triple differences design where we compare children across treated and matched-control municipalities, over birth cohorts, and by parental mental health status. This enables us to assess if a low-touch public policy can moderate the persistence of mental health conditions across generations.

Our analyses documenting the persistence of mental health conditions across generations show strikingly stable associations. Having either parent with a mental health diagnosis between ages 25 and 30 increases the probability that their child has a mental health diagnosis at ages 13–18 by 9.3 percentage points, or 40 percent of the prevalence of mental health events among children in families where parents are not diagnosed. This estimate is largely unaffected by controlling for the dynastic effects, which themselves all have statistically significant predictive power. The estimated associations for members of the extended family decrease in relationship distance; for example, the estimate for spouses of parents' siblings is 46 percent of the estimate for parents' siblings, which is itself 28 percent of the parent–child association. The correlations are further invariant to controlling for physical health problems of all members of the extended family. Importantly, not accounting for the extended family effects understates the intergenerational persistence in mental health by 42 percent.

Our results are comparable when we consider the intensive margin and use the number of mental-health-related events as an outcome and the number of parental sickness leaves related to mental health diagnoses as a regressor. We can also compare the magnitudes of associations in physical and mental health. They are largely similar and, if anything, appear modestly larger for physical health conditions.[2] The parent–child intensive margin estimates, at about 0.05, are much smaller compared to intergenerational elasticities in socioeconomic outcomes or mental health outcomes based on survey data. For example, using the same cohorts, we find an intergenerational parent–child association in education of 0.45.

Despite being smaller than in the extant literature, our findings are robust. They do not vary significantly when we include additional control variables or when we account for potential measurement error issues. Besides, the intergenerational associations in

2. We do not estimate the extensive margin transmission for non-mental health diagnoses as the prevalence of this outcome for the children generation between ages 13 and 18 is at 99 percent in our data.

mental health conditions are similar for the paternal and maternal lineage, and they are not affected by the age ranges at which we measure the mental health of the parents, the other family members, or the child.

Finally, we find that the 2007 pilot effectively reduced the intergenerational parent–child association in mental health by 39 percent. These effects are likewise robust. They are neither driven by differential pre-trends nor affected by the inclusion of municipality-specific trends accounting for potential changes in the supply of health services. We also verified that other unobservable changes are not biasing the results by executing a placebo exercise using predetermined health conditions that should not be affected by the intervention. The effects of the pilot program are stronger among children who were treated for a longer period of time and at a younger age, as well as for those with college-educated parents.

A. Related Literature

We make contributions to several literatures. First, this is one of a few studies investigating intergenerational associations in health, especially mental health. Prior work has measured intergenerational correlations in general health (Andersen 2021; Björkegren et al. 2022; Fletcher and Jajtner 2021; Halliday, Mazumder, and Wong 2020, 2021) or mortality and longevity (Björkegren et al. 2022; Black et al. 2023). Other papers focused on specific aspects of health, such as anthropometrics or asthma (Akbulut-Yuksel and Kugler 2016), BMI (Classen 2010; Classen and Thompson 2016), birth weight (Currie and Moretti 2007; Royer 2009), or cardiovascular diseases (Lloyd-Jones et al. 2004). When it comes to mental health, the literature is scarcer. Some studies measure general mental health based on the Strengths and Difficulties Questionnaire (Hancock et al. 2013) or similar behavioral and emotional state questions (Bencsik, Halliday, and Mazumder 2021; Johnston, Schurer, and Shields 2013; Vera-Toscano and Brown 2021). In particular, Johnston, Schurer, and Shields (2013) were the first to investigate intergenerational persistence in mental health across three generations. They found that conditional on maternal mental health, grandmothers' mental health is not significantly correlated with a child's mental health. Both Vera-Toscano and Brown (2021) and Bencsik, Halliday, and Mazumder (2021) further compare estimates for physical and mental health. Others zoom in on specific mental health disorders, such as depression (Akbulut-Yuksel and Kugler 2016; Eyal and Burns 2019), anxiety (Eley et al. 2015), ADHD (Cheung and Theule 2016), or substance use (Knight, Menard, and Simmons 2014).[3] Most of these studies rely on survey data and self-reported measures, which complicates estimation and inference (for example, small sample sizes, ordered intervals, and limited observables), as well as interpretation (for example, reporting bias, recall bias, and individual-specific interpretation of the questions). To the best of our knowledge, only Andersen (2021) provides population-level estimates for general health using administrative data on GP visits and hospitalizations. We add to this work by using administrative data, information on extended family, and measuring both physical and mental health status. Table 1 summarizes the main features of the aforementioned papers focusing on the intergenerational persistence of mental health conditions. The table includes information on the data used, the

3. Some of these disorders have a clear genetic rather than purely social component (Thompson 2014). For example, research reports heritability estimates for schizophrenia at 64 percent (Lichtenstein et al. 2009), bipolar disorder at 59 percent (Lichtenstein et al. 2009), autism at 80 percent (Sullivan, Daly, and O'Donovan 2012), and ADHD at 74 percent (Faraone and Larsson 2019).

Table 1
Summary of Prior Findings on Intergenerational Mental Health Correlations

Study (1)	Country (2)	Data (3)	Survey Data (4)	Maximum Sample Size (5)	Health Outcomes (6)	Main Results (7)
Johnston, Schurer, Shields (2013)	UK	1970 British Cohort Study	Yes	8,194	(1) Mother and children (two generations): 9-question subset of the 24-item Malaise Inventory (2) Mother and grandmother (three generations): 9-question subset of the 24-item Malaise Inventory (3) Children (three generations): SDQ questionnaire	(2) Two generations: mother–child mental health IC of 0.13* to 0.19* (SD) (2) Three generations: mother–child mental health IC of 0.31* (SD) and grandmother–child (conditional on mother) of 0.03 (SD)
Hancock et al. (2013)	Australia	Growing Up in Australia: The Longitudinal Study of Australian Children	Yes	4,069	(1) Children: SDQ questionnaire (2) Parents: Kessler K6 scale of nonspecific psychological distress (3) Grandparents: reported by parents based on binary question "Did your father/mother suffer from nervous or emotional trouble or depression?"	(1) Additional 1.9*, 0.9*, and 1.2* SDQ points if mother, father, or both parents had mental health problems, respectively (2) Additional 0.4* (maternal), 0.2 (paternal), 0.5* (maternal), and 0.4 (paternal) SDQ points if grandmothers and grandfathers had mental health problems, respectively

(*continued*)

Table 1 (*continued*)

Study (1)	Country (2)	Data (3)	Survey Data (4)	Maximum Sample Size (5)	Health Outcomes (6)	Main Results (7)
Knight, Menard, Simmons (2014)	USA	National Youth Survey Family Study	Yes	1,725	Annual substance use frequency (alcohol, marijuana, other drugs) of both parents and children	Elasticities ranging from –0.03 (for other drugs use at ages 12–17) to 0.23* (for alcohol use at ages 18–24)
Eley et al. (2015)	Sweden	Twin and Offspring Study of Sweden	Yes	876	(1) Parental anxiety: 20 items from the Karolinska Scales of Personality (2) Children anxiety: items from Child Behavior Checklist (3) Neuroticism based on Eysenck Personality Questionnaire for both parents and children	(1) Anxiety ICCs of 0.02–0.20* (2) Neuroticism ICCs of 0.03–0.21*
Akbulut-Yuksel and Kugler (2016)	USA	NLSY79	Yes	19,165	Indicator of self-reporting being depressed (sometimes, a moderate amount of this or most of the time during past week) for both mother and children	Mother's depression increases the likelihood of child being depressed by 9* and 0.3 percentage points for native-born and immigrant children, respectively
Eyal and Burns (2019)	South Africa	National Income Dynamics Survey	Yes	3,111	Center for Epidemiological Studies Short Depression Scale (CES-D 10) for both parents and children	Parental depression increases likelihood of adolescent depression by 31* to 35* percentage points

(*continued*)

Table 1 (*continued*)

Study (1)	Country (2)	Data (3)	Survey Data (4)	Maximum Sample Size (5)	Health Outcomes (6)	Main Results (7)
Bencsik, Halliday, Mazumder (2021)	UK	British Household Panel Survey and UK Household Longitudinal Survey	Yes	5,292	Mental health index based on 5 questions in the Short Form 12 Survey	Parents–children IC of 0.22*
Vera-Toscano and Brown (2021)	Australia	Household, Income and Labor Dynamics in Australia	Yes	1,960	Mental health index based on five questions in the Short Form 36 Health Survey	Parents–children ICs of 0.18* to 0.21*
This paper	Norway	Administrative health and social security data	No	370,498	Medically diagnosed mental health conditions based on ICPC-2 classification (code P)	(1) Parent–child mental health IC of 0.05*. Extensive margin: 10* percentage points increase in P(diagnosed) (2) Extended family–child mental health IC of 0.09*. Extensive margin: 17* percentage point increase in P (diagnosed)

Notes: This table summarizes findings from prior research on intergenerational correlations in mental health. Column 1 provides study reference, Column 2 gives the country of origin of the data, Column 3 gives the specific data sets used, Column 4 indicates if the data sets are survey-based, Column 5 provides the maximum sample size used in the paper, Column 6 describes the mental health outcomes used, and Column 7 describes the main findings. * implies that the result is statistically significant at least at the 10 percent level. The lack of asterisk implies that the result is not statistically significant at conventional levels.

sample, the health outcomes, and the main results (see also Section IV for a direct comparison of our findings to this literature).

Second, we contribute to the literature on dynastic effects. In that, we replicate the work by Adermon, Lindahl, and Palme (2021) for Norway but extend it beyond educational outcomes and into both mental and physical health domains. This model provides a lower bound on the long-run intergenerational persistence in health and allows for some separation of genetic and social effects. As they highlight, it is important to include members of the extended family in the estimation of intergenerational associations because they can influence a child's outcomes through several pathways. When it comes to mental health, these individuals could model behaviors, provide resources and expertise, or increase awareness about psychological and psychiatric issues. On the other hand, stressors such as neglect, violence, or substance abuse could likewise spill over through family networks. Empirically, we find that dynastic health associations are quantitatively important, with coefficients for the members of the extended family totaling 69–74 percent of the intensive margin parent–child association. This means that focusing solely on parents underestimates the intergenerational persistence by 42 percent. Furthermore, we also document that the association between the education of children and the health of parents does not mediate the educational parent–child correlation.

Third, we add to studies evaluating health interventions in childhood. Hjort, Sølvsten, and Wüst (2017) and Bütikofer, Løken, and Salvanes (2019) document beneficial effects of home visiting programs and checkups in early childhood. Miller and Wherry (2019) and Brown, Kowalski, and Lurie (2020) study the provision or expansion of health insurance, and Hollingsworth et al. (2022) study the expansion of access to hospital care. Bütikofer and Salvanes (2020) study screening and vaccination for infectious disease. Other studies, such as Baranov et al. (2020), show that addressing maternal postpartum depression could be beneficial for children. In this context, we show that a positive health input can lower the persistence of an undesirable intergenerational outcome. This is in line with Erten and Keskin (2020), who show that increasing maternal education in Turkey mediates intergenerational transmission of violence, and with Bütikofer, Dalla-Zuanna, and Salvanes (2022), who show that economic shocks can weaken the intergenerational transmission of earnings. We add to this growing literature by showing that a low-touch intervention targeted at young children with parents who suffer from mental health conditions is able to lower the intergenerational persistence of mental health conditions.

More broadly, our work highlights the potential of early life interventions to improve outcomes across generations and is in line with work documenting short- and long-run mental health benefits of such programs for treated individuals. For example, a preschool program for disadvantaged children in the United States (Head Start) has been shown to improve adolescent mental health (Carneiro and Ginja 2014), while a universal preschool program (Sure Start in the UK) reduced severe mental health diagnoses (Cattan et al. 2021).

Finally, our work connects with the literature on intergenerational persistence and equality of opportunity. Existing studies document correlations in education (Black, Devereux, and Salvanes 2005), preferences (Dohmen et al. 2012), noncognitive skills (Grönqvist, Öckert, and Vlachos 2017), socio-emotional skills (Attanasio, de Paula, and Toppeta 2021), income (Chetty et al. 2014), and wealth (Black et al. 2020). We extend this work to document physical and mental health correlations at the population level across generations.

II. Data

A. Data Sets and Variables

The data used in this paper are compiled from several Norwegian registers, including health and family registers. Unique personal and family identifiers enable us to follow individuals over time and across registers, so that we can construct detailed measures of children's physical and mental health during adolescence, recover the health of their parents and other family members before the children became teenagers, and complement this information with rich background data.

1. Family registers

The family registers cover the entire Norwegian population and enable us to link parents to their children, provided that the parents and the children have been residents of Norway at any point in the period 1992–2015. Parents are identified through the child's birth certificate, which in practice means that the extended family members are related to the child's biological parents. From this information, we construct children's extended horizontal families, including the biological parents, their siblings, the spouses of parents' siblings, parents' cousins, the spouses of parents' cousins, and siblings of spouses of parents' siblings (as in Adermon, Lindahl, and Palme 2021).[4]

2. Health registers

We use two different health registers, one to measure children's health and another to measure the health of parents and the extended family. For children, we use data on visits to general practitioners (GPs) and emergency rooms (ERs) from the Control and Payment of Health Refunds registry (acronym KUHR in Norwegian), which is available between 2006 and 2020. In Norway, GPs and primary care ER doctors are obliged to report all consultations and all activities during these consultations in order to receive payment. These data include two codes. The first one describes what the provider did, including screening or preventive procedures, prescription of medication, treatments, sickness leave notes, analysis of the results of medical tests, or performing other administrative tasks. The second one contains information on the health symptoms or diagnoses assessed by the doctor, which are recorded using the International Classification of Primary Care (ICPC-2). The ICPC-2 codes are composed of one letter, indicating where the symptoms or diseases are located in the body, and two numbers defining the condition.[5] Using this information, we construct variables indicating whether and how many times a child had mental-health-related symptoms or diagnoses during adolescence (that is, had GP or ER visits with an ICPC-2 code starting with the letter "P"). Online Appendix Table A1 further details the classification of the specific mental health conditions we consider. In our main analyses, we measure children's health between ages

4. We do not have information on adoptions. This prevents us from conducting nature–nurture analysis akin to Björklun, Lindahl, and Plug (2006) or Adermon, Lindahl, and Palme (2021).

5. The list of ICPC-2 codes is provided at https://www.ehelse.no/kodeverk-terminologi/icpc-2e--english-version (accessed October 3, 2023).

13 and 18, due to the low prevalence of mental health diagnoses and treatments before age 13 (see Online Appendix Figures A1 and A2). However, our results are robust to measuring health at ages 6–18, while there are very few mental health events for children younger than 6. We consider two outcome variables of interest capturing the extensive (any health event) and intensive (number of health events) margins for mental health conditions. Regarding physical health, we use the same registers, but we only analyze the intensive margin since 99 percent of individuals ages 13–18 have at least one non-mental health event.

In Norway, as in many single-payer healthcare systems, specialist care, and advanced hospital services can only be accessed and reimbursed if the patient previously obtained a referral from a GP or from a primary care ER doctor.[6] These "first-contact" doctors are responsible for the initial examination, treatment, diagnosis, prescription of medication, sickness note validation, and follow-ups with specialists. Therefore, it is unlikely that a patient would be treated or diagnosed by a specialist without any record in the GP or primary care ER data. In this context, information on GP and ER visits should provide us with an assessment of children's health for a near universe of children in Norway.[7]

Since the registry on GP and ER visits is not available before 2006, we measure parental health and the health of the extended family using the sickness leaves registry from Social Security, which is available from 1992 onward. In Norway, all sickness absences lasting longer than three days must be certified by a physician (eight days for public sector workers), and the main health reason for the absence is registered in the data with an ICPC-2 code. This enables us to create variables indicating whether and how many times each parent and member of the extended family, between ages 25 and 30, went on sickness leave due to a mental or a physical health condition. We define the extensive margin variable as any parent or any member of a specific branch of the extended family having sickness leave due to a mental health condition, while the intensive margin variables average the number of sickness leaves for mental health conditions for each group (for example, parents or parents' siblings). Since, beyond parents, the number of members in each branch of the extended family varies, we always control for the size of the branch in the analyses.

3. Additional registers

We augment our family and health data with detailed demographic, educational, and social information, such as the municipality of residence at the child's birth, parents'

6. For the years 2008–2020, where there is overlap between the specialist services (hospitals and mental health clinics) and the KHUR data, 91 percent of the 13–18-year-old adolescents visited the primary healthcare services prior to visiting a specialist.

7. Private clinics and private insurance have very limited use in Norway. Services by private providers that are paid by the public system (through referrals) are included in the KHUR data set. Services of private clinics paid out-of-pocket or through private insurance are not included but represent a negligible fraction of medical visits. In 2003, about 10,000 adults had private health insurance and about 5,000 had insurance through their employer. The number of individuals with employer-sponsored health insurance has increased since then, but this does not extend to children whose outcomes we measure after 2006 (see https://www.finansnorge.no/statistikk/skadeforsikring/helseforsikring/behandlingsforsikring/, accessed October 3, 2023). Moreover, private insurance in Norway is most often used to get immediate help if there are long waiting periods before ultimately getting some treatment also in the public system. All in all, we do not think that private health services could meaningfully affect our estimates.

education, income and country of origin, and children's middle school and middle school grades. This information comes from various administrative registers that we can link to the aforementioned family and health registers thanks to unique personal identifiers. We use these data for heterogeneity analyses, to quantify our intergenerational correlations, and to test the robustness of our results to the inclusion of control variables (see Online Appendix Table A2 for details).

B. Sample Selection

Our sample of analysis starts with the population of children who were born in Norway between 1988 and 2007 and who resided in Norway for at least some time between ages 13 and 18 during the years 2006–2020. This baseline sample consists of 732,437 observations (Online Appendix Table A3). We exclude a few children with an unknown municipality at birth or unknown parents (1,959 observations). Note that since we base the backbone of our family data on birth certificates, we observe both mothers and fathers irrespective of their subsequent presence in a child's life for 99 percent of births. In the main analysis, we restrict the sample to children with parents in our preferred 25–30 age range, which means that we focus on children whose parents were 30 or younger in 1992 and 25 or older in 2004. This ensures that parents' health at ages 25–30 is measured before 2004 to avoid codiagnoses of parents and children ($N = 568{,}253$). We further exclude children with unknown grandparents or great-grandparents, as we need this information to construct horizontal extended family links ($N = 503{,}883$). We then drop children whose parents did not have labor income between ages 25 and 30 and, hence, are not eligible for sick leave (4,907 observations). The two final steps involve observing extended family members. First, we require information on relatives in each generation, that is, parents' siblings or cousins ($N = 447{,}141$). Second, we require information on the spouses of these relatives ($N = 370{,}498$). This last number is our preferred sample used in the main analysis.

C. Descriptive Statistics

Columns 1 and 2 of Table 2 present descriptive statistics on all children born in Norway between 1988 and 2007 for whom we have information on the municipality of birth, their parents, and who were between 13 and 18 years old in 2006–2020 (Row 3 of Online Appendix Table A3). The subsequent two columns focus on our primary sample of interest (last row of Online Appendix Table A3). Panel A presents children's characteristics, Panel B presents the characteristics of their mothers, and Panel C presents the characteristics of their fathers. A few facts are worth pointing out based on this table.

First, both children and parental characteristics are largely comparable across the full and the preferred samples, which means that our main results should generalize to the entire population. To the extent that differences exist, our empirical sample appears to have somewhat worse health characteristics. Second, as already noted above, almost all children in the data have at least one non-mental health event (either through GP or ER) between ages 13 and 18. Third, 24 percent of children have a mental health diagnosis, and 4 percent of children receive care from mental health specialists (most likely certified psychiatrists), suggesting that most mental health care in Norway is delivered by primary care physicians.

Table 2
Descriptive Statistics

	All Children Age 13–18		Analysis Sample	
	Mean (1)	SD (2)	Mean (3)	SD (4)
Panel A: Children				
Any primary care visits	100.00	0.00	100.00	0.00
Any GP visits	94.76	22.29	94.30	23.19
Any ER visits	63.90	48.03	66.75	47.11
Total of primary care visits	13.41	14.42	14.00	14.83
Total of GP visits	11.66	13.16	12.11	13.51
Total of ER visits	1.75	2.74	1.89	2.89
Any MH diagnosis	23.09	42.14	24.00	42.71
Any non-MH diagnosis	99.42	7.58	99.47	7.23
Depression	6.72	25.03	6.96	25.45
Other MH	11.52	31.93	11.95	32.44
Any hospitalization	53.84	49.85	55.47	49.70
Any specialist care visit for MH	4.03	19.67	4.11	19.85
Birth weight (grams)	3,557.07	596.15	3,559.31	595.16
Age at first observation	14.20	1.55	13.94	1.37
Male	0.51	0.50	0.51	0.50
GPA	4.09	0.82	4.05	0.83
At least one parent with college degree	0.36	0.48	0.33	0.47
Panel B: Mother				
Any sick leave (ages 25–30)	0.50	0.50	0.54	0.50
Days of sick leave (ages 25–30)	18.88	33.34	20.22	33.46
Any mental health sick leave (ages 25–30)	0.06	0.24	0.07	0.26
Any musculoskeletal sick leave (ages 25–30)	0.21	0.40	0.23	0.42
Any depression sick leave (ages 25–30)	0.04	0.20	0.05	0.21
Year of birth	1,968.69	5.15	1,970.87	4.10
Annual income	383,428.34	221,928.04	386,559.05	211,462.54
Panel C: Father				
Any sick leave (ages 25–30)	0.26	0.44	0.28	0.45
Days of sick leave (ages 25–30)	8.55	25.78	9.07	26.14
Any mental health sick leave (ages 25–30)	0.03	0.18	0.04	0.19

(*continued*)

Table 2 (*continued*)

	All Children Age 13–18		Analysis Sample	
	Mean (1)	SD (2)	Mean (3)	SD (4)
Any musculoskeletal sick leave (ages 25–30)	0.15	0.35	0.16	0.37
Any depression sick leave (ages 25–30)	0.02	0.14	0.02	0.14
Year of birth	1,966.51	5.28	1,968.87	4.10
Annual income	616,975.73	452,671.12	616,756.86	402,386.02

Notes: The table uses two different samples: the full population of children born in Norway between 1988 and 2007, for whom health is measured and parents are observed (Columns 1 and 2), and the subsample of children included in our main analyses (Columns 3 and 4). For each sample, the table shows means (and standard deviations) of background and health characteristics. Children's GP or ER visits and related diagnoses and treatments are measured between ages 13 and 18, and parents' health is measured between ages 25 and 30. MH denotes mental health.

When it comes to parents, Table 2 shows that sickness leaves are about twice as common among mothers than fathers: 54 percent of mothers in our sample take at least one sickness leave, while only 28 percent of fathers do. Furthermore, mothers take on average 20 sick leave days between ages 25–30, while fathers take only nine. The most common reason for parents' sickness leave is musculoskeletal conditions. In fact, these events are about three to four times more common than having any mental health event: 7 percent of mothers and 4 percent of fathers take at least one sickness leave during ages 25–30 for a mental health reason. The most common mental health condition for both genders is depression. Given the differences in the prevalence of sickness leave across genders, in Section IV.C we document that our associations are similar regardless of whether we use paternal or maternal diagnoses.

Online Appendix Table A4 complements these statistics with information on the health of the extended family. The prevalence of both mental and physical health events varies somewhat across different branches of the extended family; however, this is primarily explained by the fact that some branches have more members than others, thus increasing the probability of any such event occurring. For example, 28 percent and 14 percent of parents' cousins and siblings have a mental health event, respectively, but on average we observe 2.5 times as many individuals in the former compared with the latter group. For this reason, in all dynastic analyses, we control for the number of members in each branch of the extended family.

In Online Appendix Table A5, we further present correlations between parental mental health and that of the extended family, all measured using the sickness leave data. As expected, this table shows that the mental health of parents is mostly positively correlated with the mental health of the extended family, but the higher the likely genetic distance between the two members of a family, the lower this correlation is. Quantitatively, all these correlations, even for siblings, are relatively low and do not exceed 0.04.

At the same time, comparable Pearson correlations between clearly genetically related individuals (for example, parents and parents' siblings at 0.034) and those who are unlikely to be genetically related (for example, parents' siblings and spouses of parents' siblings at 0.032) suggest that social and assortative mating health components could play an important role in the transmission of mental health. Irrespective of the exact reasons why the extended family matters for parental health, these correlations mean that omitting the members of the extended family when studying the intergenerational persistence of health conditions may lead to overstating the links between parents and children. Additionally, if the mental health of the extended family is in itself associated with children's mental health, beyond its correlation with parental mental health, then neglecting the role of the extended family will lead to underestimating the intergenerational persistence. Ultimately, which one of the opposing omitted variable biases dominates is an empirical question. Lastly, in Online Appendix Table A6 we present condition-specific cross-correlations for children and their parents. Here, likewise, we see relatively low Pearson correlation coefficients, suggesting independence of many of the conditions we consider.[8]

D. Data Limitations and External Validity

1. Data limitations

There are two potential limitations of using the sickness leave data to measure parental health status. First, although verified and certified by doctors, only individuals participating in the labor market are eligible for such benefits.[9] Second, not all health events and diagnoses lead to an individual taking sickness leave. In Online Appendix Table A8, we use data for the period 2006–2008 when both the primary healthcare (KUHR) and sickness leave data overlap. In the table, we correlate characteristics of the individuals with their eligibility status for sickness leave (Panel A) and with their probability of taking sickness leave among individuals with a mental health diagnosis in the KUHR data (Panel B) (see Pei, Pischke, and Schwandt 2019).

Among all individuals ages 25–30 years old between 2006 and 2008, only 6 percent were not eligible for sickness leave (Panel A). Hence, constraining our sample to individuals eligible for sickness absence is unlikely to generate a major selection issue, as 94 percent of 25–30-year-olds in Norway have some pensionable income during this

8. The measures in Online Appendix Table A6 are taken from different data registers for parents (sickness leaves) and children (GPs and ERs visits). In Online Appendix Table A7, we present parent–child correlations relying only on the primary healthcare data set based on the visits to GPs and ERs from the Control and Payment of Health Refunds registry (KUHR). In this sample, to avoid simultaneity of diagnoses, we focus on parental measures taken between 2006 and 2012, restricting them to be at most 30 years old at the date of diagnosis. In turn, for children, the measures are taken between 2014 and 2020, restricting them to be between 13 and 18 years old. This table shows that correlations in specific mental health conditions are smaller (except for other categories) than for conditions such as injuries or asthma.

9. To be eligible for sickness leave, individuals must be away from work for at least 20 percent of the working hours due to illness or injury and must (i) be affiliated with the National Insurance Scheme, (ii) be less than 70 years old, and (iii) be working for at least four weeks before becoming ill in work that provides pensionable income. See https://www.nav.no/en/home/benefits-and-services/Sickness-benefit-for-employees#chapter-1 (accessed October 3, 2023). We thus restrict our analysis to individuals who report any pensionable income over the five-year period we consider in our preferred specification.

period and therefore are eligible for sickness leave (only 3 percent of Norwegians never worked by age 30). Moreover, the regression analyses imply that men, older individuals, and those with more GP visits, higher income, and with a college education are more likely to be eligible for sickness leave. Conversely, foreign-born individuals are less likely to be eligible. These demographic differences make sense given that labor force participation is higher among men, older, higher income, and college-educated individuals, and lower among immigrants. Since sickness leave is awarded by a doctor, it also makes sense that ineligibility is negatively correlated with GP visits. Some of these coefficients are large in relative magnitude (due to the small mean), and thus, we acknowledge that despite 94 percent coverage of our sickness leave data, the estimates might not be externally valid for families with severe enough health problems that they prevent labor force participation.

Furthermore, not all medical visits and diagnoses are due to events severe enough to prevent individuals from working (for example, an individual with depression that can be managed pharmaceutically might not be sent on a sickness leave). Thus, the sickness leave registry may underreport the prevalence of low-severity conditions. We study this possibility in Panel B of Online Appendix Table A8, which limits the sample to the population of individuals eligible for sickness leave benefits between 2006 and 2008 and with a mental health event reported in KHUR data. The explanatory variable in this regression is an indicator that takes a value of one if an individual had a mental health diagnosis or symptoms in the primary care data and is observed with a sickness leave. Overall 23 percent of individuals with a mental health event take an absence from work. Assuming that individuals with mental health conditions who do not show up in the sickness leave data indeed have relatively mild conditions and that the parent–child association in mental health increases with the severity of the condition, our intergenerational correlations should be treated as an upper bound of the true associations. The estimates in Panel B further show that individuals with a mental health diagnosis or symptom, but not taking sick leave are more likely to be males and foreign-born individuals. Conversely, it is less common among older, higher-income, and college-educated workers perhaps because of the types of jobs they hold. We also observe that individuals with more GP visits are more likely to be captured in the sickness leave data, which makes sense if this relates to the severity of the condition. To address both of the issues documented in Online Appendix Table A8, in one of the robustness checks we control for the determinants of this selection process. This is akin to a control function approach (see for example, Blundell and Costa Dias 2009). Naturally, we cannot use the sickness leave registry to measure children's health, as it is restricted to the working population only.

2. *External validity*

It is also possible that our intergenerational sample is selected due to the fact that a mental health diagnosis affects the probability that an individual has children or the number of their children. Thus, in Online Appendix Table A9 we focus on individuals who could potentially be included in the parental generation: those 35 or younger in 1992 and 25 or older in 2004. We then regress demographic and health characteristics on an indicator for not being a parent. The table shows a positive association between

fertility and education and income, as well as a negative association between fertility and being foreign-born. We further find, in contrast to the concern outlined above, that individuals with sickness leave or mental health–specific sickness leaves are more (rather than less) likely to be parents. This suggests that, if anything, our empirical sample of parents has on average slightly lower (mental) health capital than the overall population, which is in line with evidence presented in Table 2. Here, we again use a control function approach to address this selection.

3. Other issues

Despite the aforementioned concerns, the administrative records have some advantages compared to the survey data. First, with this data, we can compare the magnitude of intergenerational associations to within-twin correlations. Second, because we have measures of mental health for the extended family, we can compare the associations of children's mental health with genetically related and likely genetically unrelated members of the extended family.

Another advantage of using administrative data is that it may be more robust to the issues of underreporting of mental health conditions. Indeed, Bharadwaj, Pai, and Suziedelyte (2017) show a large degree of underreporting of mental health conditions in the survey compared with administrative records, especially in contrast with other health conditions. Such behavior is consistent with stigmatization of mental health illness.[10]

Thus, given that all children have at least one contact with primary healthcare services during ages 13–18, we do not expect our measures of mental health to be affected by underreporting linked to a lack of access to care, though there might still be some underreporting due to stigma. Moreover, there are universal health services at schools that refer children to medical services if there is a suspicion of a medical condition that needs a further follow-up (Abrahamsen, Ginja, and Riise 2021).

III. Econometric Models of Intergenerational Persistence

We begin our analysis with a model that regresses a child's mental health outcome at time t (Y_{it}) on parental generation mental health at time $t-1$, which is measured prior to the child's diagnosis to rule out codiagnosing. Y_{it} is an indicator variable for whether child i was diagnosed with or treated for a mental health condition between ages 13 and 18. In the simplest model, which was the focus of almost all of the prior literature, only the child's parents (Y^{p}_{it-1}) are considered in the previous generation. Following Adermon, Lindahl, and Palme (2021), however, we expand it to include k members of the parental dynasty. In our application $k=5$ and includes parents' siblings (*sp*), parents' cousins (*cp*), spouses of parents' siblings (*ssp*), siblings of spouses of parents' siblings (*sssp*), and spouses of parents' cousins (*scp*). Independent variables of interest here are likewise indicators of whether a particular member of the child's i extended family was diagnosed with or treated for a mental health condition between

10. Besides, the Likert scale measures often used in survey data can suffer from severe bias (Bond and Lang 2019).

ages 25 and 30. We also consider intensive margin transformations of both dependent and independent variables of interest where we use counts of events rather than indicator variables, and we average the number of health events across all members of a given branch of extended family. We estimate the following equation:

$$(1) \quad Y_{it} = \alpha + \beta_p Y^p_{it-1} + \sum_{\substack{k=sp,cp,\\ ssp,sssp,scp}} \beta_k Y^k_{it-1} + \gamma \mathbf{X}_{it} + \varepsilon_{it}$$

where $\mathbf{X}_{it}$ is a vector of fixed effects for the number of individuals in each component k of the extended family considered, gender of the child, fixed effects for the year of birth and for the year when the child is first observed in the primary healthcare data (KHUR), and indicators for whether it is possible to identify in the data each grandparent and great-grandparent. In select specifications, we also expand $\mathbf{X}_{it}$ to include additional control variables. The parameters of interest in this equation are β_p and β_k, and they describe intergenerational mental health correlations between parents and their children, as well as between members of extended family and the children. ε_{it} represents the unobserved determinants of child i's mental health, and we use Eicker–Huber–White standard errors in the estimations (as in Adermon, Lindahl, and Palme 2021).[11]

IV. Intergenerational Persistence in Health

A. Main Results

We first present our mental health results in Table 3, which is based on Equation 1, and where the dependent variable is an indicator, multiplied by 100, for whether the child had any primary care event related to mental health between ages 13 and 18. We define an event as a GP or ER visit with an administratively recorded mental health diagnosis or symptom. The control group mean of this variable is 23 percent implying that more than a fifth of teenagers in Norway whose parents are not observed with any mental health event between ages 25 and 30 have had at least one mental health event themselves. The dependent variable is regressed on a series of indicators for generation $t-1$ mental health events, all measured at ages 25–30. In Column 1, we correlate parental and child mental health, while in subsequent columns we add each branch of the extended family one at a time. In the final column, we control for non-mental health events of all members of the extended family to account for the correlation between mental and physical health diagnoses and symptoms.

A few notable patterns arise. First, there is a robust association between the mental health of parents and their children, and this coefficient is largely orthogonal to any additive extended family effects. In the full dynastic model, Column 6, parental mental health event is associated with a 9.6 percentage point higher probability of a mental health event for their child, or more than 40 percent of the dependent variable mean. Second, all the remaining coefficients for the mental health of the extended family

11. We have also clustered the standard errors at the family level, where we define family in two ways: (i) sharing at least one great-grandparent and (ii) at the level of the second generation, that is, sharing at least one maternal or paternal grandparent. The conclusions remain unchanged.

Table 3
Dynastic Correlations in Mental Health: Extensive Margin Estimates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Parents' MH	9.805***	9.664***	9.641***	9.589***	9.583***	9.569***	9.257***
	(0.254)	(0.254)	(0.254)	(0.254)	(0.254)	(0.254)	(0.255)
Parents' siblings MH		2.990***	2.925***	2.876***	2.871***	2.848***	2.557***
		(0.208)	(0.208)	(0.208)	(0.208)	(0.208)	(0.210)
Spouses of parents' siblings MH			1.507***	1.469***	1.465***	1.417***	1.181***
			(0.256)	(0.256)	(0.256)	(0.256)	(0.258)
Parents' cousins MH				1.703***	1.665***	1.654***	1.475***
				(0.163)	(0.163)	(0.163)	(0.164)
Spouses of parents' cousins MH					0.679***	0.676***	0.556***
					(0.195)	(0.195)	(0.195)
Siblings of spouses of parents' siblings MH						1.025***	0.883***
						(0.206)	(0.208)
Control for OH	No	No	No	No	No	No	Yes
Mean	22.9						
Sum of coefficients		12.7	14.1	15.6	16.3	17.2	15.9
SE		0.3	0.4	0.4	0.5	0.5	0.5
R^2	0.052	0.052	0.052	0.053	0.053	0.053	0.053
N	370,498	370,498	370,498	370,498	370,498	370,498	370,498

Notes: The outcome is an indicator that takes the value of one if the child had a primary healthcare visit with a mental-health-related symptom or diagnosis, and zero otherwise. The independent variables take value one if a parent or a relative has had a sick leave due to mental health symptoms or diagnoses, and zero otherwise. Outcome variables are multiplied by 100. Child health is measured at ages 13–18, while parental health is measured at ages 25–30. One observation per parent–child pair in all regressions. Controls included in the regressions but excluded from the table are indicators for the number of maternal and paternal siblings, siblings' spouses, cousins, spouses of cousins, and siblings of spouses of parents' siblings, gender of the child, fixed effects for the year of birth and for the year when the child is first observed in the primary healthcare data (KHUR), and indicators for whether it is possible to identify in the data each grandparent and great-grandparent. MH denotes mental health, while OH denotes non-mental health events. Eicker–Huber–White robust standard errors in parentheses. Significance: *$p<0.10$, **$p<0.05$, ***$p<0.01$.

members are statistically significant and generally diminish in size as the familial distance between the child and members of the extended family grows. Nonetheless, taken together, they add up to an additional 7.6 percentage point increase in the probability of a mental health event, or almost 80 percent of the parent–child association. This means that focusing solely on parent–child correlations greatly underestimates the intergenerational persistence in mental health. Importantly, these associations cannot be solely driven by genetics, as it is unlikely that children's genes are correlated with genes of spouses of parents' siblings or spouses of parents' cousins. Rather, these coefficients—at 1.4 and 0.7 percentage points (14.8 and 7.1 percent of the parent–child association), respectively—suggest a social factor in mental health diagnosis and surveillance. Since these are diagnoses and symptoms verified by a medical professional, we do not view them as the results of overdiagnosing or overreporting—or at least to a lesser degree than with self-reported survey data. Finally, the mental health associations change very little when we control for the physical health of all members of the $t-1$ generation. For example, the parent–child association declines from 9.6 percentage points to 9.3 percentage points, or by a mere 3.3 percent (Column 7). This suggests that intergenerational associations in mental health are largely orthogonal to correlations between the parental generation's physical health and the child's mental health.[12]

The extensive margin estimates presented in Table 3 are of policy relevance for at least two reasons. First, they trigger initial medical treatment and medical costs that are plausibly higher than for the follow-up visits (for example, the average length of an initial visit is higher than the length of consultation with an established patient). Second, they motivate the intervention that we study in the second part of this paper aimed at reducing the burden of parental mental health diseases for their children. At the same time, studying only the extensive margin limits the comparability of estimated intergenerational associations with other studies, which tend to prefer log-log or rank-rank elasticities based on continuous rather than binary variables. To this end, Table 4 presents the intensive margin associations, where we substitute indicator variables with counts of mental health events for children and mental health sickness leaves for generation $t-1$. We standardize both the outcome and the input variables to have a mean of zero and a standard deviation of one. We do so because the number of events varies across the branches of the extended family due to their different sizes.[13]

In the fully saturated model (Column 6), we no longer find a statistically significant relationship between the mental health of children and that of spouses of parents'

12. Online Appendix Table A10 includes estimates for the extensive margin mental health associations allowing for interaction effects between mental and physical health conditions of parents. We limit this analysis to parent–child associations for transparency, but results for the extended family paint a very similar story. The interaction term is small and statistically insignificant, and it does not affect the level coefficients, which is consistent with the results in Column 7 of Table 3. We do not observe mental or physical diagnoses for grandparents; however, the estimates in Column 6 of Table 3 remain unchanged when controlling for grandparental longevity, one proxy for their health that we can observe in the data, namely an indicator for whether at least one grandparent died before age 60 (23 percent of the children in the sample have at least one grandparent that died before turning 60; see Online Appendix Table A11). We further note that prior research (albeit based on survey data) suggests that the health of grandparents has no (Johnston, Schurer, and Shields 2013) or very limited (Hancock et al. 2013) additional association with grandchild's mental health (conditional on parental mental health).

13. We are unable to compute rank-rank associations since the prevalence of mental health diagnoses is below 50 percent implying a large number of zeros in the distributions of the number of sickness leaves or visits.

Table 4
Dynastic Correlations in Mental Health: Intensive Margin Estimates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Parents' MH	0.050***	0.050***	0.049***	0.049***	0.049***	0.049***	0.048***	0.031***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.002)
Parents' siblings MH		0.017***	0.017***	0.017***	0.017***	0.017***	0.016***	0.011***
		(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Spouses of parents' siblings MH			0.007***	0.007***	0.007***	0.007***	0.006***	0.004***
			(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.001)
Parents' cousins MH				0.007***	0.007***	0.007***	0.006***	0.005***
				(0.002)	(0.002)	(0.002)	(0.002)	(0.001)
Spouses of parents' cousins MH					0.002	0.002	0.002	0.002
					(0.002)	(0.002)	(0.002)	(0.001)
Siblings of spouses of parents' siblings MH						0.004**	0.003*	0.002**
						(0.002)	(0.002)	(0.001)
Control for OH	No	No	No	No	No	No	Yes	No
Sum of coefficients		0.067	0.073	0.080	0.082	0.085	0.080	0.055
SE		0.003	0.004	0.005	0.005	0.005	0.005	0.004
R^2	0.032	0.032	0.032	0.032	0.032	0.032	0.034	0.032
N	370,498	370,498	370,498	370,498	370,498	370,498	370,498	370,498

Notes: The outcome is the (standardized) number of primary healthcare visits with a mental-health-related symptom or diagnosis in Columns 1–7. The independent variables are the (standardized) number of sick leaves due to mental health symptoms or diagnoses per dynastic category in Columns 1–7. In Column 8 the outcome is the number of primary healthcare visits with a mental-health-related symptom or diagnosis and the independent variables are the average number of sick leaves due to mental health symptoms or diagnoses per dynastic category. Child health is measured at ages 13–18 while parental health is measured at ages 25–30. One observation per parent–child pair in all regressions. Controls included in the regressions but excluded from the table are indicators for the number of maternal and paternal siblings, siblings' spouses, cousins, spouses of cousins, and siblings of spouses of parents' siblings, gender of the child, fixed effects for the year of birth and for the year when the child is first observed in the primary healthcare data (KHUR), and indicators for whether it is possible to identify in the data each grandparent and great-grandparent. MH denotes mental health, while OH denotes non-mental health events. Eicker–Huber–White robust standard errors in parentheses. Significance: *$p < 0.10$, **$p < 0.05$, ***$p < 0.01$.

cousins. However, all the other coefficients remain statistically significant at conventional levels. We likewise see that parent–child associations are not affected by including the mental health of other members of the extended family (Column 1 vs. Column 6) or by controlling for the physical health of generation $t-1$ (Column 7). The point estimate of 0.049 in Column 6 means that a one standard deviation increase in parental mental health sickness leaves is associated with a 0.049 standard deviation increase in the mental health events of their child. The sum of coefficients on the other members of the extended family is 0.037, or 75.5 percent of the preferred parent–child association. This is very similar to our extensive margin findings. Column 8 of this table presents unstandardized correlations, which exhibit the same statistical significance pattern but have different magnitudes as expected.

Studying the intensive margin further allows us to compare directly associations in mental health (Table 4) and non-mental health (Table 5) conditions. In absolute terms, the point estimates are relatively similar, albeit due to large sample sizes in many cases we are able to reject their statistical equality. For example, parent–child associations that are omitting the dynastic components (Column 1) are 0.050 (95 percent confidence interval 0.044–0.056) and 0.062 (95 percent confidence interval of 0.058–0.066) for mental and physical health, respectively. Furthermore, physical health associations exhibit the same set of patterns as those described above for mental health: (i) parent–child associations are unaffected by the inclusion of extended family effects, (ii) extended family effects are decreasing in the familial distance, and (iii) physical health associations are unaffected by including generation $t-1$ mental health controls. We even find that the sum of coefficients for other members of the extended family constitutes 76.3 percent of the relevant parent–child estimate, which is almost identical to the ratio we found for mental health. Based on these results, we conclude that intergenerational mental health associations exhibit patterns similar to non-mental health associations, and thus both should attract the attention of policymakers.

The richness of our data allows us to investigate the role of extended family in intergenerational health persistence. Given that specific members of extended family are unlikely to be genetically related to the children, it could also be of interest to consider magnitudes of the associations for those with and without plausible genetic links. We consider parents (50 percent of genes shared), parents' siblings (25 percent genes shared), and parents' cousins (at most 12.5 percent genes shared) as definitely genetically related, while spouses of parents' siblings, spouses of parents' cousins, and siblings of spouses of parents' siblings are generally unlikely to be genetically related to the children. Dividing extended family into these two groups we observe that, irrespective of the outcome or the margin, the associations for "plausibly unrelated" individuals are always smaller, suggesting that genetics could be one important driver of the persistence. When it comes to magnitudes, the three coefficients for "plausibly unrelated" individuals comprise 18.1 percent, 15.1 percent, and 12.5 percent of the total intergenerational persistence in extensive margin mental health, intensive margin mental health, and intensive margin physical health, respectively. Interestingly, when we consider educational transmission (Table 6), they constitute only 6.0 percent of the persistence. This suggests that "genetically unrelated" members might matter more for the health than for the educational associations, perhaps because mental or physical health issues are comparatively more "visible" within extended family. We come back to the comparison of health and educational associations below.

Table 5
Dynastic Correlations in Non-Mental Health: Intensive Margin Estimates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Parents' OH	0.062***	0.060***	0.060***	0.059***	0.059***	0.059***	0.058***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Parents' siblings OH		0.023***	0.023***	0.023***	0.023***	0.023***	0.022***
		(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Spouses of parents' siblings OH			0.007***	0.007***	0.006***	0.006***	0.006***
			(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Parents' cousins OH				0.009***	0.009***	0.009***	0.008***
				(0.001)	(0.001)	(0.001)	(0.001)
Spouses of parents' cousins OH					0.004***	0.004***	0.004***
					(0.001)	(0.001)	(0.001)
Siblings of spouses of parents' siblings OH						0.003*	0.003*
						(0.001)	(0.001)
Control for MH	No	No	No	No	No	No	Yes
Sum of coefficients		0.083	0.090	0.095	0.100	0.102	0.105
SE		0.002	0.003	0.004	0.004	0.004	0.004
R^2	0.243	0.244	0.244	0.244	0.244	0.244	0.245
N	370,498	370,498	370,498	370,498	370,498	370,498	370,498

Notes: The outcome is the (standardized) number of primary healthcare visits with a non-mental-health related symptom or diagnosis. The independent variables are the (standardized) number of sick leaves due to non-mental health symptoms or diagnoses per dynastic category. Child health is measured at ages 13–18, while parental health is measured at ages 25–30. One observation per parent–child pair in all regressions. Controls included in the regressions but excluded from the table are indicators for the number of maternal and paternal siblings, siblings' spouses, cousins, spouses of cousins, and siblings of spouses of parents' siblings, gender of the child, fixed effects for the year of birth and for the year when the child is first observed in the primary healthcare data (KHUR), and indicators for whether it is possible to identify in the data each grandparent and great-grandparent. MH denotes mental health while OH denotes non-mental health events. Eicker–Huber–White robust standard errors in parentheses. Significance: *$p<0.10$, **$p<0.05$, ***$p<0.01$.

B. Magnitudes

1. Comparison with prior studies

Our intensive margin estimates imply parent–child mental and physical health elasticities of 0.05 and 0.06, respectively. These grow to 0.09 and 0.10 when we consider dynastic persistence. Comparable estimates in the extant literature are about 0.25 for education (Black, Devereux, and Salvanes 2005), 0.34 for income (Chetty et al. 2014), 0.35 for wealth (Black et al. 2020), and 0.42 for both cognitive and noncognitive skills (Grönqvist, Öckert, and Vlachos 2017), implying that the health associations we consider here are orders of magnitude lower than those for socioeconomic outcomes studied extensively in prior work. They are also lower compared to prior health and longevity studies. Using U.S. data, Halliday, Mazumder, and Wong (2021) find self-reported health elasticities of 0.26, while using U.K. data, Bencsik, Halliday, and Mazumder (2021) find physical health elasticity of 0.17. Other work based on administrative data reports somewhat smaller estimates of 0.13–0.15, using hospitalizations for Sweden (Björkegren et al. 2022), and 0.11–0.14, using administrative data for Denmark (Andersen 2021). Black et al. (2023) find intergenerational correlations in longevity in the United States of 0.09–0.14.

Moving on to mental health correlations, the primary focus here, we summarize the results from select prior papers on this topic in Table 1. In the U.K. the correlations range from 0.13 (Johnston, Schurer, and Shields 2013) to 0.22 (Bencsik, Halliday, and Mazumder 2021), which is very similar to findings using Australian data (Vera-Toscano and Brown 2021). Thus, the results using survey data appear to be two to four times larger than our findings based on registry data. We note, however, that some condition-specific analyses indeed found small and insignificant elasticities, but these are harder to compare with our estimates including all diagnoses (Eley et al. 2015; Knight, Menard, and Simmons 2014). Studies that depart from reporting intraclass correlation coefficients or elasticities are perhaps best compared to our extensive margin estimate of 42 percent. In that, Hancock et al. (2013) and Akbulut-Yuksel and Kugler (2016) report smaller effect sizes, while results in Eyal and Burns (2019) for depression are orders of magnitude larger. One reason for this discrepancy could be that the last study is the only one we identified for an upper-middle-income country rather than a highly developed economy.

2. Comparing health and educational associations

We can also compare our results directly with the only study investigating the dynastic effects. Using Swedish administrative data, Adermon, Lindahl, and Palme (2021) document such associations for educational outcomes. In Table 6, we first replicate their analysis using the Norwegian population (Columns 1–6) and then include controls for physical and mental health associations (Columns 7–9) to understand to what extent intergenerational persistence in education might be mediated by intergenerational correlations between children's education and the health of the parental generation. Our outcome variable for children is Grade 10 GPA, and educational input of the generation $t-1$ is the highest grade level completed (or effectively years of schooling) by 2004.[14] Both sets of variables are standardized to have a mean of zero and a standard deviation of one.

14. Education in Norway is mandatory until the last year of middle school (Grade 10) or age 16, and at the end of middle school, each child receives a middle school GPA. It is composed of teacher-awarded grades in each

Table 6
Dynastic Correlations in Education

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Parents	0.450***	0.386***	0.383***	0.377***	0.377***	0.376***	0.373***	0.373***	0.371***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Parents' siblings		0.122***	0.112***	0.106***	0.106***	0.105***	0.104***	0.103***	0.102***
		(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Spouses of parents' siblings			0.024***	0.022***	0.022***	0.016***	0.016***	0.015***	0.015***
			(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Parents' cousins				0.036***	0.035***	0.034***	0.034***	0.034***	0.033***
				(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Spouses of parents' cousins					0.003*	0.003*	0.003*	0.003	0.003
					(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Siblings of spouses of parents' siblings						0.014***	0.014***	0.014***	0.013***
						(0.002)	(0.002)	(0.002)	(0.002)
Control for MH	No	No	No	No	No	No	Yes	No	Yes
Control for OH	No	No	No	No	No	No	No	Yes	Yes
Sum of coefficients		0.508	0.519	0.541	0.543	0.549	0.543	0.541	0.537
SE		0.002	0.002	0.002	0.003	0.003	0.003	0.003	0.003
R^2	0.301	0.311	0.311	0.312	0.312	0.313	0.313	0.313	0.314
N	265,174	265,174	265,174	265,174	265,174	265,174	265,174	265,174	265,174

Notes: The outcome is the (standardized) GPA in the final year (Grade 10) of compulsory schooling. Education in the parents' generations is measured as standardized years of schooling. One observation per parent–child pair in all regressions. Controls included in the regressions but excluded from the table are indicators for the number of maternal and paternal siblings, siblings' spouses, cousins, spouses of cousins, and siblings of spouses of parents' siblings, gender of the child, fixed effects for the year of birth and for the year when the child is first observed in the primary healthcare data (KHUR), and indicators for whether it is possible to identify in the data each grandparent and great-grandparent. MH denotes mental health, while OH denotes non-mental health events. Eicker–Huber–White robust standard errors in parentheses. Significance: $^{*}p<0.10$, $^{**}p<0.05$, $^{***}p<0.01$.

We observe only slightly higher persistence in Norway compared to Sweden; for example, in the fully saturated model (Column 6), our sum of coefficients is 0.549 compared with 0.518 in Sweden. Interestingly, since the parent–child associations are 32 percent larger in Norway than in Sweden, the bias resulting from not including the dynastic coefficients is larger in the latter than in the former country.[15] Tables 4 and 6 further allow us to compare the persistence in educational and mental health outcomes. The results suggest orders of magnitude higher persistence in education than in mental health. For example, the standardized parent–child association (Column 1) in mental health is 0.05, while it is 0.45 for education—nine times smaller for mental health. Moving to intergenerational persistence (Column 6), the sum of coefficients for mental health is 0.085, while for education it is 0.549, or more than six times higher than the mental health persistence. Remarkably, the dynastic educational associations do not change even after controlling for dynastic mental and physical health (Column 9 vs. Column 6). This is despite the fact that these health inputs are positively and significantly correlated with a child's GPA.

3. Comparison with twin, sibling, and cousin correlations

Another way we can quantify our intergenerational correlations is by comparing them to twin, sibling, and cousin intrafamily correlations. This approach has two advantages. First, it uses the same data set rather than two different data sets. Second, it partially allows us to gauge the role of genetics in these health correlations. Online Appendix Table A12 presents these results for twins (Columns 1 and 2), full siblings (Column 3), and cousins through either maternal grandmother or paternal grandfather (Column 4). For comparability, we focus on our intensive margin measure, and we consider mental (Panel A) and physical health (Panel B) events separately. We find same-sex twin correlations at 0.37 and 0.51, respectively. This declines to 0.25 and 0.47 for opposite-sex twins, which are all dizygotic and thus genetically equivalent to full siblings.[16] Based on these two sets of results, we conclude that (i) physical health events have a stronger familial component than mental health events (higher correlations in the former than the latter), and (ii) same-generation twin mental health correlations are much larger than the intergenerational correlations. Moving to full siblings, the correlations decline further, implying that a common in utero environment and the same age could play a role in both mental and non-mental health. Finally, investigating health correlations between cousins, we find an even smaller coefficient than for the full siblings. At the same time, even these smallest within generation t associations are at least three times larger than

middle school subject and of end-of-the-year external exams in either mathematics, Norwegian, or English. The choice of exam subject among the three areas is random. GPA is standardized by cohort.

15. To be precise, the sum of coefficients on other members of the dynasty in Column 6 of Table 6 is 0.172 versus 0.376 for the parent–child association. Comparable numbers in Adermon, Lindahl, and Palme (2021) are 0.233 versus 0.284. This means that bias in intergenerational educational persistence from not including dynastic effects is only 31 percent in Norway but as high as 45 percent in Sweden, even despite the fact that persistence is somewhat higher in Norway.

16. One reason why the same- versus opposite-sex correlations are so similar for physical health (compared to mental health) could be that same-age children are more likely to engage in play and sports activities together, which could lead to accidents and injuries. This should be less prevalent among children of different ages, and indeed our intrafamily physical health correlation declines in Column 3.

corresponding parent–child associations. The twin associations documented in Online Appendix Table A12 are smaller than those found for years of education and long-run earnings in Sweden (Björklund and Jäntti 2012). On the other hand, they are similar to what Andersen (2021) documents for twins' and siblings' general health in Denmark.

C. Robustness of the Intergenerational Associations

We perform a variety of robustness checks to ensure that our estimates are not meaningfully biased. The primary concerns we address relate to measurement error, sample selection, and omitted variables bias.

Although the registry data used in this paper do not suffer from measurement error problems common in surveys (for example, individual-specific interpretation of questions or recall bias), we still need to consider carefully how mental health is being measured. In particular, Online Appendix Table A8 suggests that our measure of mental health among parents is truncated both from the top—the sickest individuals do not work—and from the bottom—the mildest diagnoses do not lead to sickness leaves. Assuming that the milder the mental health conditions, the lower their persistence, the former scenario would render our estimates a lower bound, while the latter an upper bound of the intergenerational associations. Given that we miss relatively fewer diagnoses due to the former rather than the latter scenario, we tend to think about our estimates, which are already low, as an upper bound. Nonetheless, in order to address this concern, we assess the robustness of our findings to control for the variables driving the aforementioned selection (see Blundell and Costa Dias 2009). It is also possible that the estimates of intergenerational persistence attributed to the extended family could in fact be attributed to omitted family characteristics and sorting of relatives into specific locations (see, for example, Adermon, Lindahl, and Palme 2021; Chetty et al. 2014). Thus, we test if these omitted family and residential location characteristics change our estimates by adding various controls to the baseline models. Finally, we also consider if our estimates may be confounded by the propensity of the attending primary healthcare services or by attending specific GPs. We recognize that some of these robustness checks involve including potentially endogenous variables as controls and thus should be interpreted cautiously.

Estimates in Tables 7 and 8 address these concerns for the extensive and intensive margins of mental health, respectively. Column 1 is our baseline specification from Column 6 of Tables 3 and 4 included to ease the comparisons. Column 2 presents the estimates removing the basic set of controls of Column 1. Since higher correlations in child psychological conditions might be explained just by going to the doctor and getting screened more, in Column 3, we augment the baseline specification with fixed effects for the number of visits to primary healthcare services. In Column 4, we further control for the determinants of sickness leave (Online Appendix Table A8) and fertility (Online Appendix Table A9); namely, we include indicators of fathers' and mothers' educational attainments, indicators for whether the mother and the father are foreign-born, and their (quadratic) incomes. Column 5 further controls for potential omitted but predetermined family characteristics at the child's birth and sorting of relatives into specific locations by adding parent and child characteristics to the model (children's birth weight, children's birth rank, indicators for the mothers' and fathers' age at the child's birth, and fixed effects for the mothers' municipality of residence at childbirth). Column 6 additionally includes fixed effects for the cohort–school level indicating

Table 7
Dynastic Correlations in Mental Health: Sensitivity Analysis (Extensive Margin Estimates)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Parents' MH	9.569***	11.025***	8.401***	7.372***	6.785***	6.755***	7.012***	8.865***
	(0.254)	(0.260)	(0.247)	(0.276)	(0.278)	(0.317)	(0.321)	(0.217)
Parents' siblings MH	2.848***	3.717***	2.612***	2.078***	1.865***	1.794***	1.933***	2.498***
	(0.208)	(0.211)	(0.202)	(0.222)	(0.222)	(0.250)	(0.260)	(0.181)
Spouses of parents' siblings MH	1.417***	1.195***	1.302***	0.694**	0.607**	0.673**	0.634**	0.988***
	(0.256)	(0.260)	(0.250)	(0.273)	(0.273)	(0.308)	(0.320)	(0.217)
Parents' cousins MH	1.654***	1.797***	1.466***	0.951***	0.818***	0.881***	0.764***	1.144***
	(0.163)	(0.160)	(0.159)	(0.175)	(0.175)	(0.199)	(0.205)	(0.163)
Spouses of parents' cousins MH	0.676***	0.206	0.615***	0.393*	0.262	0.018	0.155	1.016***
	(0.195)	(0.193)	(0.189)	(0.209)	(0.209)	(0.237)	(0.245)	(0.188)
Siblings of spouses of parents' siblings MH	1.025***	0.593***	0.857***	0.447**	0.329	0.104	0.295	0.690***
	(0.206)	(0.206)	(0.200)	(0.218)	(0.218)	(0.244)	(0.256)	(0.173)

(*continued*)

Table 7 (*continued*)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Baseline controls	X		X	X	X	X	X	X
No controls		X						
Nb. PC visits			X	X	X	X	X	
Selection				X	X	X	X	
Individual					X	X	X	
Schools						X		
GP							X	
Parental age				25–30				30–35
Sum of coefficients	17.2	18.5	15.3	11.9	10.7	10.2	10.8	15.2
SE	0.5	0.5	0.5	0.5	0.5	0.6	0.6	0.4
R^2	0.053	0.008	0.101	0.108	0.114	0.165	0.152	0.049
N	370,498	370,498	370,498	315,173	314,919	263,210	251,763	356,425

Notes: This table presents robustness checks for the result from Column 6 of Table 3. Column 1 replicates this result, Column 2 drops all control variables, Column 3 replicates Column 1 additionally controlling for fixed effects for the number of primary care (PC) visits, Column 4 further adds controls for determinants of selection (Online Appendix Tables A8 and A9), Column 5 further adds individual level controls (birth weight, birth order, indicators for mother's and father's age at the time of child's birth, and fixed effects for mother's municipality of residence at the time of child's birth), Column 6 further adds middle school-by-cohort fixed effects, Column 7 replicates Column 5 but adds the child's GP fixed effects, and finally Column 8 replicates the results from Column 1 but measures parental mental health events at ages 30–35 rather than 25–30. MH denotes mental health. Eicker–Huber–White robust standard errors in parentheses. Significance: $^{*}p<0.10$, $^{**}p<0.05$, $^{***}p<0.01$.

Table 8
Dynastic Correlations in Mental Health: Sensitivity Analysis (Intensive Margin Estimates)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Parents' MH	0.049***	0.055***	0.040***	0.037***	0.034***	0.034***	0.037***	0.051***
	(0.003)	(0.003)	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)	(0.002)
Parents' siblings MH	0.017***	0.021***	0.015***	0.013***	0.012***	0.011***	0.012***	0.013***
	(0.002)	(0.002)	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)	(0.002)
Spouses of parents' siblings MH	0.007***	0.008***	0.007***	0.004**	0.004**	0.002	0.004	0.003**
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Parents' cousins MH	0.007***	0.010***	0.005***	0.002	0.001	0.003	0.001	0.009***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Spouses of parents' cousins MH	0.002	0.003*	0.002	–0.000	–0.000	–0.000	–0.002	0.004**
	(0.002)	(0.002)	(0.001)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Siblings of spouses of parents' siblings MH	0.004**	0.004**	0.002	–0.000	–0.001	0.000	–0.002	0.002
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)

(*continued*)

Table 8 (*continued*)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Baseline controls	X		X	X	X	X	X	X
No controls		X						
Nb. PC visits			X	X	X	X	X	
Selection				X	X	X	X	
Individual					X	X	X	
Schools						X		
GP							X	
Parental age				25–30				30–35
Sum of coefficients	0.085	0.101	0.071	0.055	0.051	0.050	0.050	0.083
SE	0.005	0.005	0.004	0.005	0.005	0.006	0.006	0.005
R^2	0.032	0.004	0.127	0.132	0.137	0.184	0.174	0.028
N	370,498	370,498	370,498	315,173	314,919	264,558	251,763	356,425

Notes: This table presents robustness checks for the result from Column 6 of Table 4. Column 1 replicates this result, Column 2 drops all control variables, Column 3 replicates Column 1 additionally controlling for fixed effects for the number of primary care (PC) visits, Column 4 further adds controls for determinants of selection (Online Appendix Tables A8 and A9), Column 5 further adds individual level controls (birth weight, birth order, indicators for mother's and father's age at the time of child's birth, and fixed effects for mother's municipality of residence at the time of child's birth), Column 6 further adds middle school-by-cohort fixed effects, Column 7 replicates Column 5 but adds child's GP fixed effects, and finally Column 8 replicates the results from Column 1 but measures parental mental health events at ages 30–35 rather than 25–30. MH denotes mental health. Eicker–Huber–White robust standard errors in parentheses. Significance: $^{*}p<0.10$, $^{**}p<0.05$, $^{***}p<0.01$.

where and when children completed middle school. In Column 7, we further control for fixed effects for the first GP the child is allocated to when first observed in the KHUR data after turning 13 years old.

Irrespective of the controls we include, the parent–child mental health association remains statistically significant, but the intergenerational persistence declines by up to 40 percent. This suggests that socioeconomic status (SES), health at birth, place of residence, schools, and GPs—some of which are endogenous—can play a role in explaining some of the persistence of mental health across generations. Considering the extensive margin (Table 7), we observe the largest declines in the persistence when accounting for selection (via SES controls), at 3.4 percentage points. This is followed by intensity of interactions with the healthcare system (primary care visits), at 1.9 percentage points, and individual-level controls (including prenatal health), at 1.2 percentage points. On the other hand, schools and GP fixed effects do not appear to meaningfully mediate the intergenerational transmission. These results should be interpreted with caution, however, since the order at which variables are introduced in the decomposition could matter for their explanatory power.

Finally, Column 8 shows that our main results remain very similar if we consider parental diagnoses at ages 30–35 rather than 25–30. As yet another robustness exercise, Online Appendix Tables A13 and A14 replicate the associations presented in Table 3 but measure child diagnoses at ages 6–18, while parental diagnoses are taken at ages 25–30 (Online Appendix Table A13) or at ages 30–35 (Online Appendix Table A14). The stability of the estimates across different ages of measurement suggests that the specific six-year band in our main analyses does not drive the results.

D. Heterogeneity

We explore multiple sources of heterogeneity in our data. First, we study the stability of the intergenerational associations across observable characteristics of children, including their gender and education of their parents. Second, we consider maternal and paternal lineages, given that some prior research found differences along those lines (Black, Devereux, and Salvanes 2005). Third, we investigate if the associations differ by whether parents or relatives are medical professionals or not. This could be important for measurement error and given that Chen, Persson, and Polyakova (2022) document that access to the medical expertise of parents increases preventative care utilization and decreases substance abuse of the youth.

In Columns 1 and 2 of Online Appendix Table A15, we investigate if the results presented in Table 3 differ by child's gender or parental education, respectively. These results suggest negligible differences by gender. On the other hand, parent–child associations appear lower in families with higher-educated parents. This gradient, however, does not extend in a significant way to other members of the extended family. Overall, we conclude that there is limited heterogeneity in the estimated intergenerational associations across the two dimensions considered here.

It is also possible that the estimates in Table 3 are driven by either the maternal or the paternal lineage. Thus, in Online Appendix Table A16, we modify our analysis allowing for differential correlations through the mother's and father's lineage and for their respective relatives. The estimates show relatively similar correlations for both lineages. Parent–child associations are somewhat larger for fathers than for mothers. On the other hand, only siblings of spouses of the mother's siblings have a significant correlation with

a child's mental health. Overall, we conclude that pooling both paternal and maternal lineages in our main results does not substantively affect the conclusions.

Online Appendix Table A17 presents the results stratifying the correlations by medical expertise of the extended family. We consider the family as having "medical expertise" if at least one relative within our six dynastic categories is a doctor or a nurse (Column 1), a doctor (Column 2), or neither (Column 3).[17] Overall, we find qualitatively similar intergenerational correlations across the three samples. The parent–child association ranges from 9.0 to 9.8 percentage points, while the total "dynastic effect" ranges from 16.5 to 18.5 percentage points. Thus, we conclude that in Norway within-family medical expertise does not moderate the intergenerational associations in mental health. This also alleviates our measurement error concerns, as one could plausibly expect families with greater medical knowledge to have better access to and utilization of mental health services.

V. Targeted Policies and Intergenerational Persistence

A. The Policy Pilot

Having documented the intergenerational associations in mental health, in the second part of the paper our goal is to understand whether a targeted low-touch policy can break the link documented in Section IV between parental and child mental health. On the one hand, since the policy was clearly concerned with children's mental health, it can increase intergenerational associations if more children are being diagnosed due to additional scrutiny. On the other hand, to the extent that these children are effectively treated, we should see improvements in their mental health. Ultimately, it is an empirical question which of these channels dominates.

We answer this question by studying the effects of a pilot program (Modellkommuneforsøket), which was implemented in a few Norwegian municipalities in 2007. This initiative was motivated by the large share of adolescents with mental health conditions in Norway who also have parents with similar health issues. The pilot program aimed to help children navigate through the process of having a parent diagnosed with a mental illness and to prevent children from developing psychological problems. The program required healthcare providers to "help safeguard the need for information and necessary support that minor children (0–18 years old) of patients with mental illness, drug addiction, or severe physical illness or injury may have due to parent's condition" (Skogøy et al. 2018). Hence, the trial did not mandate that children are themselves screened for mental health conditions, but rather, healthcare professionals were mandated to register dependent children (0–18 years) of their patients, to have a conversation with their patient about children's needs, and to offer help in getting appropriate information and care. The Norwegian Agency for Children, Youth, and Families (Bufetat) had the responsibility for the pilot program. Twenty-six municipalities were chosen from the municipalities that applied for the trial, and they were almost equally distributed over Bufetat's five geographic regions. The municipalities included cities, medium-sized, and small units in all regions of Norway.[18]

17. To capture exposure to medical expertise via relatives, we define an individual as being a health professional if they worked as a nurse or a doctor between 2003 and 2014 for at least five years.

18. The municipalities were Aurskog-Høland, Jevnaker, Lørenskog, Skedsmo, Vestre Toten, Arendal, Drammen, Mandal, Vennesla, Askøy, Bergen, Bømlo, Fjell, Førde, Haugesund, Sandnes, Time, Grong, Leksvik, Namsos,

The pilot program was aimed at developing best practice guidelines for early interventions and a systematic follow-up model for children of parents with mental health conditions from pregnancy to school age. The trial targeted minor children and, in particular, children ages 0–6 with more intensive treatment.[19] The municipalities received annual funds from the central government based on activity in accordance with the mandate of the trial (NOK100,000–290,000) and used this funding in varied ways with the goal of most effectively helping the local community. The goal of the trial was twofold. First, the aim was early identification of the target group and early change-focused assistance. This required the development of a system registering the targeted children across different institutions, the implementation of screening tools to detect psychological distress among the targeted children, as well as other targeted programs. By 2010, all pilot municipalities had built up a registration system, and, except for one, all had implemented screening tools for mental health conditions among children. Other policies (which varied across municipalities) included counseling with pediatric nurses, follow-up services by the child welfare services, special pedagogical programs or specialized psychologists in childcare centers, building up and access to family centers, home visits by specialized nurses, PMTO (Parent Management Training–Oregon) training for parents whose children have behavioral problems, parenting program participation (for example, International Child Development Programme), network groups for pregnant mothers, and enhanced health center services for families where there was substance abuse among parents. The second key goal was the establishment and further development of interagency cooperation between different municipal services, such as healthcare centers and childcare centers, and between municipal services and specialist health services. Hence, municipalities implemented organizational changes, such as the creation of personal service coordinators for the children and their parents or the establishment of interdisciplinary discussion teams to work with parents and children. These teams included employees from various public and municipal institutions, such as child welfare services, pedagogical–psychological services of the Norwegian Directorate for Education and Training, childcare center services, social security agencies, and local healthcare services, including psychological services for children and young people. In addition, the pilot municipalities established contact persons in the various specialist health services (GPs, nurses, psychologists, or social workers). Overall, the exact policies and practices varied across municipalities, and specific implementation was only restricted by the aforementioned overarching goals.

The pilot was qualitatively evaluated in 2010 and 2014 (Deloitte 2015; Rambøll 2010), with the conclusion that routines were established and that the competencies and awareness among employees in the municipalities with respect to children with mentally ill parents increased. Since the program was designed to guide the outline of a new law targeting the needs of children of parents with mental health issues, based on the experiences from the trial, a new law was implemented in Norway in 2010 that requires all municipalities to ensure that minor children (0–18 years old) of patients with mental

Steinkjer, Trondheim, Bodø, Fauske, Nordreisa, and Tromsø. We do not have information on which municipalities applied to be part of the pilot program but were rejected.

19. The program was mostly targeted to young children ages 0–6 years, but some measures were also aimed at all children under the age of 18. Since we use children aged 7–18 as one of the control groups, to the extent that the program also affected them in a positive way, our estimates should be viewed as a lower bound.

illness, drug addiction, or severe physical illness or injury have better access to services mapping and following-up their demands (Ot.prp.nr.84 2009).

B. Empirical Strategy

1. Matching procedure

Since only 26 out of 428 municipalities participated in the targeted pilot program, and these municipalities were not chosen randomly, we rely on a triple differences estimation strategy to evaluate its effects on the parent–child mental health associations. To implement this empirical strategy, we first generate a set of control municipalities (spatial variation) matched based on observable pre-intervention characteristics (see for example, Bhalotra, Karlsson, and Nilsson 2017). The best matches, denoted $J_M(i)$, are identified using the Mahalanobis distance metric, that is:

$$(2) \quad J_M(i) = \arg min_j \sqrt{(\mathbf{X}_i - X_j)' S^{-1} (\mathbf{X}_i - X_j)}$$

where $\mathbf{X}_i$ is the vector of observable municipality characteristics, and S denotes the covariance matrix of this vector. Matching is done in random order, with replacement, and each treated municipality is assigned to one or two control municipalities. Online Appendix Figure A3 shows a map of the 26 treated (red) and 22 matched-control (blue) municipalities (four municipalities serve as controls for multiple treated municipalities). Online Appendix Table A18 further shows the characteristics of treated and matched-control municipalities. The results imply that the matching procedure was effective in finding untreated municipalities comparable to the pilot municipalities, except perhaps for municipality income. Compared to all untreated municipalities in Norway, the municipalities that participated in the pilot program tend to have more resources, and their residents are more educated, but at the same time, they have fewer doctors per capita. On the other hand, the selected control municipalities match the treated municipalities along all observable dimensions well.

2. Main empirical model

To identify the effects of the policy, we utilize the fact that, in the treated municipalities, children under age 6 whose parents were diagnosed with a mental health or substance abuse problem were the primary target of the pilot program. Thus, we compare children in treated and control municipalities and exploit differences in age at the start of the program together with differences in parental mental health diagnosis in a triple difference empirical strategy. We estimate the following model:

$$(3) \quad Y_{icm} = \alpha + \beta(E_{ic} \times D_{im} \times MPH_i) + \delta_1 MPH_i + \delta_2(E_{ic} \times MPH_i) + \delta_3(D_{im} \times MPH_i) + \delta_4(E_{ic} \times D_{im}) + \mathbf{X}_i'\boldsymbol{\mu} + \rho_c + \pi_m + \varepsilon_{icm}$$

where Y_{icm} is an indicator for any mental health GP or ER visit between ages 13–18 of child i, born in cohort c, and in municipality m. MPH_i is an indicator of whether the child's father or mother had at least one mental-health-related sick leave between 2000 and 2010. D_{im} is an indicator for whether child i's municipality of residence at birth m is one of the 26 treated pilot municipalities. E_{ic} is an indicator that takes the value one if the child is aged 6 or below in 2007, as the pilot program was primarily targeted toward

0–6-year-old children. ρ_c and π_m are year of birth and municipality of residence at birth fixed effects, respectively. Lastly, controls included in vector $\mathbf{X}_i'$ are the child's gender, indicators for the age of the mother and father at birth, children's birth weight, children's birth rank, and indicators for fathers' and mothers' educational attainments. As the outcomes might be correlated within municipalities across cohorts, the standard errors are clustered at the level of the municipality of residence at the child's birth m (Bertrand, Duflo, and Mullainathan 2004). Our analysis includes all children born in treated or control municipalities between 1996 and 2005.

The parameter of interest is β, which estimates if and how the parent–child intergenerational mental health association is affected by the pilot program. This is an intent-to-treat (ITT) estimate, as we do not observe which children actually directly benefited from the program. Furthermore, it represents the extensive margin effect of being exposed to the program for at least a year, but when discussing the results we also consider the intensive margin based on the expected duration of exposure. In the latter case, we transform the binary variable E_{ic} into a continuous variable that measures how many years a child born in year c was eligible for the pilot program. This variable ranges from 0 to 4.[20]

As a robustness test, we also present results from a modified Equation 3, where we additionally control nonparametrically for the differential supply of health services. In particular, we interact the cohort fixed effects with the following pre-trial municipality characteristics: number of GPs per 1,000 inhabitants, number of school nurses per 1,000 school-age students, number of school doctors per 1,000 school-age students, number of health professionals per 1,000 inhabitants, and population.

3. *Event study*

In order to construct an event study graph, we estimate the effects of the program for each cohort of children born between 1996 and 2005 using the same set of municipalities as in Equation 3. This enables us to assess the credibility of the parallel trends assumption. Our identifying assumption is that in the absence of the program, the difference in children's likelihood of being diagnosed or treated for mental health conditions, between those whose parents do and do not have a mental health event, should have evolved similarly in treated and control municipalities. To assess this empirically we estimate the following equation:

$$
\begin{aligned}
(4)\quad Y_{icm} = \alpha &+ \sum_{\tau=2,\tau\neq 7}^{11} \beta^{\tau}(D_{im} \times MPH_i \times 1[Age_{i,2007} = \tau]) + \delta_1 MPH_i \\
&+ \sum_{\tau=2,\tau\neq 7}^{11} \delta_2^{\tau}(MPH_i \times 1[Age_{i,2007} = \tau]) + \delta_3(D_{im} \times MPH_i) \\
&+ \sum_{\tau=2,\tau\neq 7}^{11} \delta_4^{\tau}(D_{im} \times 1[Age_{i,2007} = \tau]) + \mathbf{X}_i'\boldsymbol{\mu} + \rho_c + \pi_m + \varepsilon_{icm}
\end{aligned}
$$

20. The pilot program was implemented between 2007 and 2010. We assume that the treatment group is children aged 0–6 years. Thus, children born in 1996–2000 were not eligible for the intensive treatment, children born in 2001 were only eligible for one year, children born in 2002 were eligible for two years, children born in 2003 were eligible for three years, and children born in 2004 or 2005 were eligible for the maximum duration of four years. Since the length of treatment is perfectly collinear with age at the start of the treatment, we cannot separate these two effects.

In Equation 4, parameters of interest are β^{τ}, where τ corresponds to the child's age in 2007 at the start of the pilot. As most of the treatment focused on children aged 0–6, we use age 7 in the year 2007 as our reference period. This means that coefficients $\tau > 7$ represent pre-trends, while coefficients $\tau < 7$ represent treatment effects. If the policy mediates the parent–child association in mental health, we expect the latter set of coefficients to be negative.

C. Policy Effects

Table 9 shows the effects of the pilot on the intergenerational parent–child mental health correlations. Panel A presents the extensive margin estimates comparing younger (eligible) versus older (ineligible) children, in treated versus matched control municipalities, and for children with versus without parents suffering from a mental health condition. Panel B presents the intensive margin estimates where we replace the eligibility dummy with the number of years eligible based on the child's birth year and the start of the pilot. Finally, in Panel C, we reestimate specification from Panel A while controlling for time trends in the supply of health services at the municipality level. Column 1 presents estimates for all families, Columns 2 and 3 present heterogeneity by parental education, and Columns 4 and 5 present heterogeneity by child's gender.

Focusing on Column 1 of Panel A, we observe a parent–child mental health association of 9.7 percentage points in the control municipalities and for children who were older than 6 years of age at the start of the program. This is very similar to the 9.8 percentage point parent–child association reported for the full population in Column 1 of Table 3. The second row of Panel A reveals a negative and statistically significant treatment effect of 3.8 percentage points. This means that the policy was successful at moderating the intergenerational association in mental health, and it reduced the parent–child transmission by almost 40 percent. This result is robust to both intensive margin transformation of the treatment variable (Panel B) and to including municipality trends (Panel C).

Columns 2–5 of Table 9 explore heterogeneity across parental education and child's gender. While we do not observe any meaningful differences in the effects of the pilot program across the child's gender, results indicate that the policy was more effective at reducing the parent–child transmission of mental health among families with at least one college-educated parent compared to families where no parent has a college degree.[21] This means that although the pilot program was effective in reducing mental health transmission across generations, it could have actually somewhat increased inequality within generation t across families of different socioeconomic backgrounds. A stronger response to the intervention among college-educated families could be due to greater and/or earlier uptake by those with higher education. For example, college-educated parents might be more likely (or better equipped) to utilize the resources offered to their children (see, for example, Aizer and Stroud 2010; de Walque 2010; Kjellsson, Gerdtham, and

21. The effects by gender are comparable on the extensive margin; however, the intensive margin effect is 77 percent larger for boys compared with girls (despite both being statistically significant at conventional levels). One reason for this could be that boys are particularly responsive to repeated positive inputs, especially in childhood; see, for example, the discussions on differential sensitivity of boys relative to girls to poverty and role models by Autor et al. (2019) and Bertrand and Pan (2013), especially at the lower tail of the distribution of behavioral outcomes (Autor et al. 2023).

Table 9
Effects of the Pilot Program on the Intergenerational Persistence of Mental Health Conditions

	All (1)	College (2)	No College (3)	Males (4)	Females (5)
Panel A: Baseline					
1[Parental MH]	9.730***	8.983***	10.104***	8.699***	10.840***
	(0.461)	(0.532)	(0.671)	(0.669)	(0.905)
1[Parental MH] × (Age 2007 ≤ 6) × Pilot	–3.796***	–6.210***	–2.141	–3.603**	–3.916***
	(0.927)	(1.307)	(1.390)	(1.574)	(1.253)
N	129,683	60,596	69,087	66,074	63,609
Panel B: Duration					
1[Parental MH]	9.801***	8.965***	10.148***	8.649***	11.036***
	(0.466)	(0.545)	(0.656)	(0.730)	(0.943)
1[Parental MH] × Duration × Pilot	–1.114***	–2.425***	–0.568	–1.403***	–0.795*
	(0.277)	(0.488)	(0.341)	(0.507)	(0.403)
N	129,683	60,596	69,087	66,074	63,609
Panel C: Control for Municipality Trends					
1[Parental MH]	9.703***	9.007***	10.076***	8.673***	10.804***
	(0.457)	(0.535)	(0.671)	(0.667)	(0.887)
1[Parental MH] × (Age 2007 ≤ 6) × Pilot	–3.778***	–6.145***	–2.137	–3.610**	–3.866***
	(0.920)	(1.327)	(1.388)	(1.558)	(1.242)
N	129,683	60,596	69,087	66,074	63,609

Notes: The table focuses on all children born in the 26 treated and the 22 matched-control Norwegian municipalities between 1996 and 2005. For all panels, each column corresponds to a separate regression where we compare the mental health of children who were older versus aged 6 or younger at the start of the program, were born in treated versus matched-control municipalities, and had parents with versus without mental health conditions. Children's mental health outcome is an indicator for any mental health GP or ER visit between ages 13–18, multiplied by 100. "1[Parental MH]" is an indicator for parental MH-related diagnoses between 2000 and 2010. "Pilot" is an indicator of whether the child was born in one of the 26 pilot municipalities. "Age 2007 ≤ 6" indicates cohorts of children who were 6 or younger in 2007, namely at the start of the pilot program. In Panel B, "Duration" is the number of years during which each cohort of children was aged 6 or below between 2007 and 2010. This variable equals zero for children born between 1996 and 2000, one for children born in 2001, two for children born in 2002, three for children born in 2003, and four for children born in 2004 and 2005. Controls excluded from the table and included in the model are fixed effects for children's year of birth, indicators for the ages of mothers and fathers at the time of the child's birth, child's birth weight, child's birth order, indicators for father's and mother's educational attainments, children's municipality of birth fixed effects, and interactions between the main dependent variables of interest described above. Panel C expands the set of controls to include interactions between cohort fixed effects with the following pre-trial municipality characteristics: number of GPs per 1,000 inhabitants, number of school nurses per 1,000 school-age students, number of school doctors per 1,000 school-age students, number of health professional per 1,000 inhabitants, and population. There is one observation per parent–child pair in all regressions. Columns 2 and 3 present results by parental education and Columns 4 and 5 by the child's gender. Standard errors are clustered by children's municipality of birth. Significance: *$p < 0.10$, **$p < 0.05$, ***$p < 0.01$.

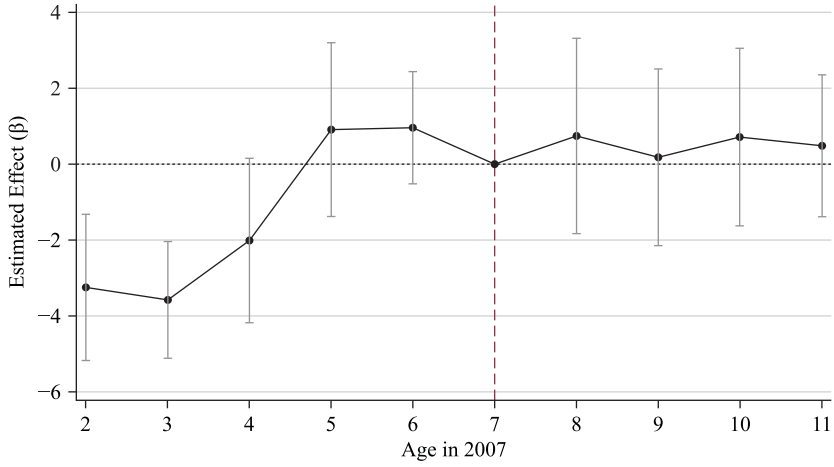

Figure 1

Effects of the Intervention by Age in 2007

Notes: This figure uses the same sample and outcome as Table 9. The figure presents estimates for β^{τ} from Equation 4. Whiskers show 95 percent confidence intervals for each point estimate. Standard errors are clustered by children's municipality of birth.

Lyttkens 2011; Lleras-Muney and Lichtenberg 2005). Nevertheless, we cannot observe the uptake, and we are therefore not able to test whether there is a socioeconomic difference in utilization (first stage). At the same time, there are other explanations for larger effects among college-educated families. For example, it is possible that these families are more responsive to exposure to medical professionals. Another reason could be that college-educated parents with mental health diagnose/symptoms are more malleable to adopting healthier habits, following the guidelines to take medication, or improving investments in health and the home environment.

A causal interpretation of our triple difference coefficients requires the parallel trends assumption to hold. To assess the credibility of this assumption, in Figure 1 we study whether there were any significant differences in the evolution of mental health events between children of parents with and without mental health conditions, across treated and control municipalities, prior to the pilot program launch. Reassuringly, this figure shows no systematic differences in the trend before the pilot program was implemented (ages 7–11 in 2007), thereby providing support in favor of the parallel trend assumption. The figure further shows that the most affected children were those who were the youngest at the start of the program (and they were also exposed the longest). In fact, we do not see any statistically significant benefits for children who were age 5 or 6 in 2007. This could mean three things: (i) that such programs need to be targeted at very young children, (ii) that children benefit from the implemented programs only if there is a longer period of exposure, or (iii) the combination of both. We view this question on timing versus length of treatment as an important extension for future research.[22]

22. We cannot reliably analyze the consequences of the pilot program for children's education, proxied by their middle school GPA, because of data constraints. Children in Norway get a middle school GPA at the end of their

Finally, in Online Appendix Table A19, we present a placebo analysis to assess the credibility of our empirical strategy. For this exercise, we use two sets of outcomes. First, we consider outcomes that are predetermined at the time when the intervention started, namely health outcomes recorded at birth. Second, we use diagnoses at ages 13–18 for outcomes that are unlikely to be directly affected by the intervention, such as fractures or musculoskeletal conditions. We note that while the first set of outcomes are predetermined at the time of the intervention, the second set of outcomes could be indirectly affected by the intervention due to, for example, less neglect in the household as a result of increased mental health monitoring. Columns 1–5 present the estimates for the first set of outcomes, while Columns 6–10 present them for the second set of outcomes. The results show that children in families where parents are diagnosed with mental health disorders have somewhat worse birth and physical health outcomes (although many of these associations are small in magnitude). This is expected given the intergenerational transmission of health documented in the extant literature and discussed here. Importantly, the triple interaction coefficients are never statistically significant (and in most cases they are much smaller than the level effect), suggesting that the policy did not differentially affect health at birth or physical health outcomes. This supports our identifying assumption.

VI. Conclusions

Mental health conditions are costly from both financial and societal perspectives, and they might affect equality of opportunity. Motivated by these concerns, we use unusually rich administrative data from Norway to study intergenerational associations in mental health. On the extensive margin, we document a strong link between the mental health of parents and their children. These associations are replicated when considering intensive margin and are of approximately the same magnitude as physical health associations. Furthermore, we find that a nontrivial share of the intergenerational persistence across generations is due to associations between children and extended family members, such as aunts and uncles. This leads us to believe that the uncovered associations are not driven entirely by genetics and are partially determined by social and environmental factors. Finally, the results are robust to alternative specifications, including adding extensive sets of controls or changing when we measure parental or child mental health.

The (mental) health associations we found using Norwegian data are smaller than those documented in prior literature. This could be due to the fact that Norwegian elasticities are indeed smaller or that prior estimates are upward biased (perhaps due to the use of survey data or hospitalization registers). One argument for the latter

last year of middle school, which is usually at the age of 16. In this context, because we only have information on children's GPA up until 2018, we can only observe the middle school GPA of control cohorts and of the two eldest treated cohorts whose mental health was not impacted by the program as documented in Figure 1. Coherently, an analysis based on these cohorts suggests no effect on the GPA (results available upon request). We have also repeated the analysis using all rather than matched municipalities, and our results are very similar. We prefer the matched control group approach since we documented balance of predetermined covariates (Online Appendix Table A18) in this setting.

explanation is that Norway certainly does not appear to be an outlier among the developed countries when it comes to mental health (see, for example, OECD 2018).

In the second part of the paper, we study if a low-touch public policy targeted at the young children (age 6 or younger) of parents with mental health conditions can moderate these intergenerational correlations. We find that it reduced the parent–child association by 39 percent, suggesting that low-touch interventions, targeted toward young children over multiple years during childhood, can be effective at equalizing mental health opportunities. On the other hand, these gains appear to accrue primarily for children of college-educated parents. Due to data limitations, we leave for future research the question of whether the benefits of this pilot program extend beyond mental health to later-life outcomes, such as education or income.

Our findings have two policy implications. First, from a policy evaluation perspective, we present evidence that low-touch interventions could improve outcomes for children whose parents struggle with mental health disorders. Second, our policy effects present a cautionary tale: although the intervention indeed reduced the average parent–child mental health association, it did so more effectively in higher compared to lower SES families. In that, the pilot reduced intergenerational inequality in mental health outcomes at the cost of increasing intragenerational inequality. This discrepancy highlights the need for policy interventions that could benefit all families irrespective of their education or resources.

Moreover, the documented intergenerational health associations highlight the importance of mapping out extended family links in health outcomes to better understand the multifarious process underlying social mobility. It also points to the possibility for future research to explore the causal links between the extended family's and parent's mental health and child outcomes by analyzing how exogenous shocks to the older generation's health affect child outcomes. Such causal estimates would inform the most cost-effective investments. Related to recent trends, such estimates would allow policymakers to factor potential externalities when assessing the costs of the mental health epidemic that is currently occurring globally (Patel et al. 2007, 2018), especially when we consider the mental health consequences of the COVID-19 pandemic for future offspring of today's adolescents and young adults (Giuntella et al. 2021; Golberstein, Wen, and Miller 2020).

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