WORKING PAPERS IN
ECONOMICS AND STATISTICS

Report No 2 | 2025

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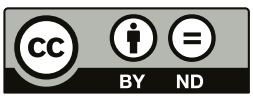
Series editor: Thomas Giebe

ISBN: 978-91-8082-411-8 (pdf)
DOI: https://doi.org/10.15626/ns.wp.2025.2

Report No 2, Department of Economics and Statistics, Linnaeus University, Växjö, 2025



Prenatal Conditions and Midlife Mental Health: Evidence from an Alcohol Policy Experiment

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December 05, 2025

Abstract

We estimate the long-term mental health impact of an alcohol policy experiment on individuals exposed to the policy *in utero*. The policy lasted for 8.5 months and significantly expanded access to alcohol, especially for those under age 21. Using administrative data on healthcare visits, drug prescriptions, and psychological assessments, we show that prenatal policy exposure had a substantial, early, and persistent impact on the mental health of the children of young mothers. The exposed cohorts conceived just before the policy started are 16% more likely to be diagnosed with any mental condition in midlife. We find effects on common midlife conditions such as depression and anxiety, on the ability to cope with psychologically stressful situations at age 18, and on neurodevelopmental disorders that manifest in early childhood. The impact of the policy on midlife earnings is significantly lower among individuals with predicted mental health care needs who reside in areas with lower barriers to accessing mental health care. Overall, our findings indicate that policies increasing access to mental health treatments could substantially improve labor market outcomes, even for conditions with early-life origins.

Keywords: FASD, ADHD, depression, earnings, treatment access, prenatal alcohol, boys

*Comments from Kate Musen, Maarten Lindeboom, Maya Rosin-Slater, Anna Aizer, Anna Chorniy, seminar audience at IIES, Uppsala, Princeton, SOLE-Toronto (2025), were all very helpful.

1 Introduction

Individual health behaviors (e.g., smoking, drinking, or diet) can impact individual long-term health outcomes (Mahler and Yum, 2024; Danesh et al., 2024), but also the health of others (Christakis and Fowler, 2007; Kremer and Levy, 2008; Bharadwaj et al., 2014). A textbook example of behavior with potentially long-run health externalities is alcohol consumption during pregnancy. Since Jones and Smith (1973) alerted the medical profession to the risks related to alcohol use during pregnancy, a large body of research has been dedicated to studying the effect of prenatal exposure to alcohol on cognitive ability, and physical and mental health.[1]

In the United States, self-reported alcohol consumption among pregnant women has declined significantly since the issuance of the first Surgeon General's advisory recommending abstinence from alcohol during pregnancy in 1981 (see Figure 1). However, despite decades of public health initiatives aimed at reducing alcohol consumption during pregnancy, recent estimates continue to indicate a high prevalence in many countries. Europe stands out: while global prevalence (any consumption) is estimated at 10% - Russia (37%), the UK (41%), Denmark (46%), Belarus (47%), and Ireland (60%) top the list. (Popova et al., 2017).[2]

The lingering uncertainties surrounding the causal relationship between prenatal alcohol exposure and child outcomes, especially for consumption levels and patterns common on a population scale (Oster, 2013; Easey et al., 2019), could be a contributing factor. Animal models provide guidance, but it is not straightforward to communicate findings from the lab and translate them into public policy. Assessing the existence and strength of the effects on child development using observational data is on the other hand a formidable challenge: in part because those who drink during pregnancy may differ from those who abstain in many other ways that also affect their children's outcomes; in part because of desirability or recall bias in reported alcohol consumption during pregnancy; and in part because detecting more subtle

[1] e.g., O'Connor, 2001; Barr et al., 2006; Streissguth, 2007; Disney et al., 2008; Sciberras et al., 2017. In the first long-term prospective study, Barr et al. (2006) compared the mental health outcomes of offspring of midpregnancy binge drinkers with non-binge drinkers at age 26 and found, conditional on observables, large increases in odds ratios for three specific conditions measured in interviews using DSM-IV scales: substance disorders, passive-aggressiveness, and antisocial personality disorders or traits. See Rangmar et al. (2015) for a recent Swedish study on individuals diagnosed with FAS.

[2] Other recent estimates suggest that 13 percent of pregnant women in the U.S. report drinking in the past 30 days (Gosdin, 2022; Denny et al., 2020). In Copenhagen, five years after women were recommended to abstain from the time of trying to conceive and throughout pregnancy, 35% reported binge drinking in early pregnancy (Iversen, 2015). In Latin America, the lack of government-specific guidelines makes addressing alcohol consumption in pregnant women more difficult — in Argentina, 75%, and in Chile, 57% of pregnant women reported consuming alcohol during pregnancy (see references in Gimenez et al., 2022, Aros et al., 2006). Alcohol use during pregnancy is believed to be underreported.

effects on child development may require large samples and long follow-up times.

In a recent review, Easey et al. (2019) highlights the methodological shortcomings of existing work[3] and document examples of negative, null, and positive associations between prenatal alcohol exposure and child mental health. They emphasize the need for studies that explicitly are designed to strengthen causal inference. Furthermore, they stress that it is unclear if any associations persist into adulthood. Mental health is of particular interest to economists due its typically early onset, high prevalence, and strong (relative to common physical childhood health conditions) effects on human capital accumulation and labor market outcomes (Currie and Stabile, 2006).

Our contribution to the literature on the effects of prenatal alcohol exposure on long-run mental health is centered around a policy experiment that temporarily, unexpectedly, and sharply increased alcohol availability to young people (under age 21) in two Swedish regions. The policy experiment was implemented prior to the medical community's recognition of the risks associated with alcohol exposure during pregnancy. It lasted for 8.5 months and was discontinued prematurely due to reports of excessive drinking among young people. We focus on the impact of *in utero* exposure to the policy on mental health in midlife — but also test for effects at age 18, and whether access to mental health care in midlife affects the exposed children's earnings trajectories.

Our main measure of mental health is derived from detailed administrative data covering the entire Swedish population, containing information from inpatient and outpatient records, as well as detailed data on mental health drug prescriptions. The health care data allow us to construct a measure of having any mental health condition at ages 42–47, any specific and common midlife conditions, as well as any neurodevelopmental conditions that emerge in childhood.[4]

While comprehensive, a drawback of the health care data is that it only reflects mental health problems severe enough to warrant contact with health care providers in midlife.[5] We therefore complement our analysis with data from psychological assessments at military enlistment. As we show, low values on the primary assessment scale "Psychological Function" (PF) — the ability to "cope with psychologically stressful situations" — are strongly correlated with mental

[3] e.g. residual confounding, attrition, treatment and outcome measurement

[4] Mental disorders account for over 40% of public expenditure on sick leave (Försäkringskassan, 2023b), and anxiety disorders and depression constitute close to half of all mental health-related sick leave spells (Försäkringskassan, 2023a) in Sweden.

[5] Since stigmatization can be a barrier to seeking mental health care (FHM, 2019), the difference between the true prevalence of mental health problems and diagnosed conditions could be substantial.

health diagnoses at enlistment and with having any diagnosed mental conditions, including neurodevelopmental conditions, in midlife. The military psychologists' assessments complement the health care records in two main ways. First, they allow us to extend the baseline midlife observation window and test for the effects of the policy at age 18. Second, since virtually all males participated in the enlistment procedures, they allow us to assess the impact on mental health in an almost unselected sample of young males — irrespective of whether they had been in contact with mental health care providers.[6]

We link the mental health data to information on the time, place, and maternal age at childbirth and provide triple difference-in-differences (DDD) estimates of the impact of prenatal policy exposure. This approach allows us to account for many unobserved contemporaneous and subsequent potential confounding factors (e.g., major plant closures, regional health care treatment practices or waiting times) that could otherwise generate a biased estimate of the policy's impact.

Another key advantage of the policy experiment, in combination with the scale and granularity of the data, is that it gives us the ability to focus on birth cohorts exposed to the policy *in utero* but conceived *before* the policy started. This is an important feature, since alcohol use may affect fertility timing and match quality — i.e., with whom and when one conceives a child. By focusing on children conceived before the policy experiment started, we avoid confounding the estimated impact of *in utero* policy exposure with effects from selection into pregnancy.[7]

We find that prenatal exposure to the policy increases the probability of being diagnosed with any mental condition between the ages of 42 and 47 by an average of 16%. We find no impact on individuals exposed to the policy just after birth, among those conceived just after the policy ended, or on those born in neighboring regions. The main effects are therefore unlikely to be caused by postnatal changes in, e.g., the home environment, or by other changes affecting children of young mothers in general. The largest effects are found for exposure during the second trimester and among men, consistent with a higher vulnerability to adverse early-life conditions among boys (e.g., Little et al., 1986; Wells, 2000; Nilsson, 2017; Grönqvist et al.,

[6]Up to 98 percent participated in the enlistment procedures, and more than 90 percent completed all tests.

[7]Alcohol use increases the risk of having an unplanned pregnancy, meaning that children conceived *during* the policy may be more likely to be born into low-income and unstable households (Joyce et al., 2000; Kaestner and Joyce, 2001; Naimi et al., 2003; Markowitz et al., 2005; Nuevo-Chiquero, 2014). Poorer initial match quality or increased accidental conceptions (Becker, Landes, and Michael, 1977; Weiss and Willis, 1997) may increase family dissolution and/or negatively impact maternal labor market outcomes (Nuevo-Chiquero, 2014; Gallen et al., 2023), which could affect child outcomes in addition to any direct effects of prenatal exposure to the policy.Nilsson (2017) document changes in parental composition and family stability among those conceived *during* the policy experiment.

2020).

To gain a deeper understanding of the mental health challenges experienced by individuals exposed to the policy *in utero*, we examine its effects on specific mental health conditions. We find that prenatal policy exposure increases the likelihood of receiving midlife treatment by 24% for depression and 15% for anxiety disorders. The healthcare data limits our ability to determine the precise timing of onset of these conditions - so it is difficult to know whether these conditions stem from the negative impact of the policy on labor market outcomes (Nilsson, 2017), or if early mental health problems caused the impact on labor market outcomes.

The early emergence of neurodevelopmental disorders - such as Autism Spectrum Disorder and ADHD - is however well-established, as childhood symptom onset is a core criterion for their diagnosis (Wender et al., 2001; Murphy et al., 2016). Moreover, FASD is highly under-diagnosed and frequently misdiagnosed as ADHD (Ehrig et al., 2023).[8] The core symptoms overlap: impulsivity, attention difficulties, hyperactivity, and poor emotional regulation. Such misclassifications is particularly relevant in our context. For FASD, known alcohol consumption during pregnancy is a diagnostic criterion.[9] Since the policy took place years before FAS and FASD diagnoses were introduced - maternal alcohol use during pregnancy was not systematically assessed or recorded, and therefor unlikely to be known with certainty by the children or their doctors. Given the similarity of symptoms, ADHD is likely to be an important fall-back (mis)diagnosis.

We find that prenatal exposure to the policy increase the likelihood of midlife treatment for neuro-developmental disorders by 1.2 percentage points, which translate to high relative effects (50%) due to the low baseline levels for health care contacts due to these conditions in midlife. Consistent with early impacts on mental health, we also observe a 12% increase in the probability of scoring low on the psychological functioning assessment at age 18. Collectively, these findings suggest that prenatal exposure to the policy had significant, early, and lasting effects on mental health.

In the second part of our analysis we focus on the potential protective effects of mental health care access. Nilsson (2017) documents that individuals exposed to the policy *in utero* had substantially worse labor market outcomes at age 32. We first extend his findings by

[8] ADHD is also the most commonly reported mental health co-morbidity for children exposed to maternal alcohol use during pregnancy (Fryer et al., 2007; Popova et al., 2016).

[9] Except for the extreme condition of FAS, which can be diagnosed even without confirmed information on alcohol exposure.

showing that cumulative earnings between ages 26 and 47 decrease by an average of 16%. The negative effects on earnings are largest among those observed with a mental health diagnosis at ages 42–47, but remain economically meaningful and statistically significant across both groups over the life-cycle. We next ask whether the impact on earnings can be mitigated by residing in an area with greater access to mental health treatment. Easier access to care could influence earnings trajectories by, for example, reducing the time it takes to return to work following periods of mental distress and/or lowering the risk that mental distress progresses into more serious psychiatric conditions. Barriers to mental health care access (e.g., waiting times) and treatment practices (e.g., the share of patients receiving prescribed medication conditional on diagnosis) vary substantially across Sweden.[10]

Using the share of neighborhood residents prescribed medication for mental health problems in midlife as a proxy for local treatment access, we find that residing in a higher-treatment-intensity neighborhood cushions the impact of the policy on midlife earnings significantly.[11] A one standard deviation increase in local treatment intensity reduce the impact on earnings by one-third - but only among those with observed or predicted mental health care needs in midlife. In other words, the protective effects of higher local mental health treatment intesity does not seem to be simply caused by other shared neighborhood environmental factors.

The hypothesis that the environment experienced during critical periods of fetal development can have a profound and lasting impact on an individual's health and disease risk (Dobbing, 1970; Barker, 1994) is well recognized in economics. Prenatal and early life conditions have been shown to affect a wide range of outcomes, including cognitive ability and educational achievement (Almond and Mazumder, 2011; Almond et al., 2015; Aizer et al., 2016; von Hinke Kessler Scholder et al., 2014), labor market outcomes (Almond, 2006; Bhalotra and Venkataramani, 2015; Nilsson, 2017; Atella et al., 2023), and adult health (Almond et al., 2012; Conti et al., 2024).[12] Our paper contributes to this literature in two distinct ways:

First, our key contribution is to provide causal estimates showing that prenatal exposure to a policy that sharply increased alcohol consumption led to a higher probability of having a

[10] Waiting times for adult general psychiatric care have been documented at the regional level since 2011 and vary significantly across both time and regions. Some regions rely more heavily on intensive measures (e.g., medication), while others emphasize therapy. Additionally, barriers on both the demand side (e.g., mental health stigma) and the supply side (e.g., wait times) can limit the uptake of mental health treatment, regardless of the care received once contact with the healthcare system has been established.

[11] An important feature of the prescription data is that it covers all prescribed medication, including prescriptions issued by general practitioners in primary care clinics.

[12] See Almond and Currie (2011) and Almond et al. (2018) for two excellent reviews of this literature.

mental disorder later in life. By doing so we contribute to the extensive biomedical literature on the correlation between prenatal alcohol exposure and outcomes in childhood, by using a natural experiment and by focusing on mental health among adults.[13] Our empirical strategy cannot identify the exact dose or consumption patterns responsible for the observed effects.[14] That said, the modal consumption pattern among young people in Sweden at the time (and today) is well documented—typically characterized by binge-drinking (four drinks or more on a single occasion) during weekends and festive occasions. Sweden is not an outlier[15]; similar drinking patterns among young people are common in many other European countries and in the U.S., where about 90 percent of alcohol consumed by youths is in the form of binge drinks.[16].

Second, we also contribute to the still limited body of research that examines the causal impact of prenatal conditions more broadly on mental health. The studies most closely related to our work include Hoek et al. (1998), Brown et al. (1995), Almond and Mazumder (2011), Dinkelman (2017), Persson and Rossin-Slater (2018), and Adhvaryu et al. (2019). Our paper complements these studies through its use of a policy experiment, its ability to demonstrate effects on mental health from late childhood through midlife, and its capacity to link mental health to labor market outcomes across the life-cycle. We add new insights relative to Nilsson (2017) by highlighting the connection between policy exposure, mental health, and labor market outcomes, and by showing that improved access to mental health treatment seems to be a protective factor against the policy's negative effects on labor market earnings.

The remainder of the paper is structured as follows. Section 2 briefly describes the policy. Section 3 details the data and empirical strategy. Section 4 presents the results, and Section 5 summarize and concludes.

2 The Policy Experiment

Sweden has a retail monopoly that regulates all off-premise alcohol sales: beverages with a high alcohol content can only be sold at *Systembolaget*. The policy experiment made strong beer available for purchase in regular grocery stores in two out of 24 Swedish regions[17] between

[13] see Easey et al., 2019 for a recent review focusing on moderate consumption level and offspring mental health.

[14] To do so would require a natural experiment combined with individual-level data on alcohol consumption throughout pregnancy, which is difficult to obtain even today due to social desirability bias.

[15] see e.g. https://www.euronews.com/health/2024/02/17/binge-drinking-rate-in-adolescents-is-double-that-of-adults-in-europe-its-worse-in-these-

[16] `https://www.niaaa.nih.gov/publications/brochures-and-fact-sheets/underage-drinking`

[17] Göteborgs-och Bohuslän and Värmland regions, home to 12% of the population in 1967.

November 1967 and July 1968. The intention was to stimulate a more "continental" alcohol culture by facilitating substitution away from spirits to beverages with lower alcohol content. Whereas the legal age at *Systembolaget* remained at 21, the minimum age for buying strong beer at grocery stores was set to 16. Consequently, the policy unintendedly and sharply increased the availability of relatively high alcohol-content beverages for young people who, up until the onset of the experiment, only had access to medium beer.[18]

We would ideally like to measure changes in alcohol consumption among women in the treated and control regions throughout pregnancy. However, no individual-level data is available to directly measure alcohol consumption during the policy. For this reason, a few key facts is important to bear in mind when interpreting our findings:

First, the policy was implemented well before Jones and Smith (1973), the first Swedish study on prenatal alcohol exposure (Olegård et al., 1979), and recommendations to abstain from alcohol during pregnancy (the first US Surgeon General warning was issued in 1981).

Second, a national survey in 1968 shows that 90% of young women (age 21-23) who consume alcohol had their alcohol debut before age 21, and that less than 10% reported that they completely abstained from alcohol (APU, 1971). In 1967, five percent of high school girls abstained in the three largest cities (Göteborg (Treated), Stockholm (Control), Malmö (Control)), and 8 percent in other cities/towns (APU, 1971).

Third, Nilsson (2017) document that the policy led to a substantial increase in young people's alcohol consumption starting in the quarter of implementation, as measured by several alcohol consumption indicators:

(i) Sales of strong beer in the treatment regions increased dramatically during the policy experiment and declined again after the policy ended (see Figure 2).

(ii) Age-specific police report data show that arrests for drunkenness increased by 15% among individuals under 21 during the policy (with larger effects on girls than boys, and during the early stages of the policy), whereas arrests for this misdemeanor remained unchanged for those older than 21.[19]

(iii) 90% of the children in the exposed cohort were born in municipalities where both the Child Welfare Board (barnavårdsnämnden) and the Temperence Board (nykterhetsnämnden)

[18]Medium beer, with a alcohol content of 3.5% – 4.5 % were sold in regular grocery stores before, during, and after the policy.

[19]The effects on arrests is likely understated since the age specific data is yearly, and due to indications that the police intervened in ways that was not recorded in the offical statistics (SOU 1971:77, p.354)

reported that conditions had deteriorated as a result of the policy.

(iv) A decrease in the share of males (a proxy for spontaneous abortions) in the cohort born by young women and prenatally exposed to the policy from the first trimester.[20]

(v) A change in the parental composition and family instability of children who were conceived by young mothers *during* the early stages of the policy which - through the lens of the family stability models of Becker, Landes, and Michael (1977) and Weiss and Willis (1997) - could be interpreted as an indicator of increased alcohol consumption during the policy.

(iv) The lack of individual level consumption data, in part, likely reflects the unexpected consequences of the policy. In a post-evaluation reservation statement by one of the commissioners of the implementing authority (APU) the "failed" policy is discussed: "While it is clear that total alcohol consumption increased sharply, it is not clear whether the initial consumption increase would be sustained or fade out over time" (our translation, SOU:1971:77, p.354). He further laments that the other commissioners showed no interest in further data collection.

These findings for indirect measures of alcohol consumption lines up well with the contemporary reports of a sharp increase in alcohol consumption in the experimental regions, especially among young people. These reports caused the implementing authority, the Alcohol Policy Commission (APU), to propose an interruption, and on July 15, 1968 the policy experiment was discontinued after only 8.5 months.[21] The following day *Systembolaget* regained it's monopoly on strong beer sales - overnight reducing the number of off-premise outlets of strong beer from just above 1,500 to 26 in Göteborgs-och Bohuslän.[22]

Jointly, the reports, the indicators of increased alcohol consumption and low abstainer rates among young women in combination with the low information setting suggests that the policy could have had an impact on the children exposed to the policy *in utero*. The following section explains how we shed light on the impact on their midlife mental health.

[20]Low secondary birth ratios have previously been used as a proxy for adverse prenatal conditions in economics and elsewhere c.f. Wells (2000) for discussion. The Swedish medical birth register, where prematurity, birth weight etc are collected started in 1973. Induced abortions became legal in 1975.

[21]The policy was intended to last until the end of 1968 and then be evaluated

[22]On-premise sales was in relationship to off-premise sales very low at the time of policy, 13% in Göteborg and Bohuslän, and 7% in Värmland.

3 Data and Empirical Strategy

3.1 Data

Individual-level data from administrative registries allow us to create measures of mental health outcomes between ages 42-47 for all cohorts in our analysis sample. First, we use the National Prescribed Drug Register. The prescription data starts in 2006 and contains prescription information on all drugs dispensed at a pharmacy. We use two Anatomical Therapeutic Chemical (ATC) codes, N05 (Psycholeptics[23]) and N06 (Psychoanaleptics[24]), to measure mental disorders treated with drugs. We complement the drug registry with inpatient and outpatient data from the National Patient Register, covering the universe of hospital and specialized healthcare visits. We use all main diagnosis ICD codes in chapter F to identify visits associated with a mental condition. Our main outcome variable is based on the combination of the health care and prescription records.[25] We create an indicator variable equal to one if an individual at any point between ages 42 and 47 shows up in the data, and zero otherwise.

We also consider more restricted versions of our main outcome, which is important for two reasons. First, we cannot observe the first occasion when an individual received a diagnosis or prescription.[26] However, specific mental health disorders vary considerably in terms of age-of-onset. The median age of onset for mood disorders including depression is around age 30 and anxiety disorders often manifest in late adolescence (Solmi et al., 2022). Neurodevelopmental disorders (e.g. ADHD and Autism Spectrum Disorder), on the other hand, arise during the development period and have an onset of symptoms in childhood as a diagnostic criterion (Wender et al., 2001; Murphy et al., 2016). Studying specific groups of mental disorders therefore allows us to draw some conclusions about when the first symptoms are likely to have appeared. Second, specific mental disorders differ considerably in the type of disability they create, and considering narrower definitions allows us to gain a deeper understanding of the specific mental health challenges faced by individuals exposed to the policy. We create indicators for depression and

[23]This group consists of antidepressants, psychostimulants, nootropics, anti-dementia drugs and combinations with psycholeptics.

[24]This group consists of drugs with antipsychotic actions (i.e. neuroleptics)

[25]The prescription records has the advantage of including medical treatments from e.g. general practitioners in primary care, while the patient records also covers cases where no medication is prescribed, but it does not capture primary care visits.

[26]Appendix figure A2 shows how our main observation window (ages 42-47) outcome variable (ever treated ages 42-47) relates to earlier/later treatments received for ages 34-55 using data on the first and last cohorts in the data.

anxiety, the two most common mental disorders in our sample.[27] We also identify individuals with medication or healthcare visits for ADHD or autism spectrum disorders.[28]

We complement the health care records with a measure of overall psychological function (PF) (*Psykisk funktionssförmåga*) (Lilieblad and Ståhlberg (1977), Carlstedt (1999), FOA (1985)) as measured for enlistees in the year when they turn 18 from the Swedish Military Conscription Register (INSARK). Military conscription was mandatory for males in Sweden, and psychological function assessment is available for $> 90\%$ of men born around the time of the policy experiment.[29]

At enlistment the enlistees meets with an trained psychologist in an semi-structured interview with an average duration of 20-30 minutes (up to 1 hr depending on circumstances).[30] The interview is semi-structured in the sense that certain areas are always covered, but the exact questions are not fixed *a priori*.[31] The psychologists interview has been described as anamnestic – based on the young men's life circumstances and adaptation to stressful events during the preceding years (Lilieblad and Ståhlberg, 1977; Leboeuf-Yde et al., 2006).[32]

Nilsson (2026) provides a detailed account of the history, purpose, content, and correlates of the PF scale with mental health at enlistment and later in life. In summary, Nilsson (2026) argues that the common description in economics of the PF scale as a measure of "non-cognitive ability" may be overly narrow and could obscure information relevant for researchers interested in mental health. The ultimate and stated aim of the PF scale is to provide a prediction of the

[27]We define being treated for anxiety as filling at least one prescription for a drug with an ATC code under chapter N05B between ages 42 and 47. To identify individuals who were treated for depression between ages 42–47 we use prescription drugs under chapter N06A.

[28]We categorize an individual as receiving treatment for one of these conditions between ages 42–47 by using ICD codes F84 (Autism) and F90 (ADHD) in the in- and out-patient registry combined with ATC code N06BA (ADHD) in the drug registry. Since some conditions might be well-managed by midlife (e.g. ADHD) we include subdiagnoses in our main definition. For example, an individual may recieve a main diagnosis for any condition (physical or mental), but the doctor may include information of up to six subdiagnoses which may be of relevance to the main diagnosis, or the treatment of the main diagnosis. In practices including subdiagnoses in the prevalence measures has minuscule impact on our findings.

[29]Only a small proportion of subjects are rejected before appearing at the enrollment center. These cases consist of imprisoned subjects and those who obviously cannot join the military force because of serious disability (such as cerebral paresis) (2% in 1998). In the 1990s more than 95 percent of the young male population have attended the testing, and those who have not, were excluded because of somatic disorders and/or mental retardation (Carlstedt (2000)). In addition, some subjects that during the early stages of the enlistment procedure was classified as obviously medically or intellectually unfit did not reach the interview stage (Leboeuf-Yde et al., 2006. It should also be noted that severe mental health issues is a cause for exemption from military service (Ludvigsson et al., 2022), but not necessarily from the enlistment tests procedure. Only around 2 percent of the male population where exempted from the test procedures entirely, and around 90 percent completed all the tests.

[30](C.f. Lilieblad and Ståhlberg (1977), Wikström (1973))

[31]Semi-structured interview of similar duration is recommended in national guidelines to complement and support clinical diagnosis today(Socialstyrelsen (2019))

[32]The inter-rater reliability of the PF scale is high (.85) Lilieblad and Ståhlberg (1977), and correlates with the end of military service assessment score (Carlstedt (1999)), and labor market outcomes (Lindqvist and Vestman (2011)).

enlistees' ability to "cope with psychologically stressful situations in military service" (Lilieblad and Ståhlberg (1977) p.5). Based on publicly available documents and linked administrative records Nilsson (2026) show that it is clear that the military psychologists are particularly interested in assessing emotional stability and mental health of the conscripts. An important purpose of the psychologists interview at enlistment is to weed out conscripts unfit for military duty and leadership positions. The reason why mental health problems matter is that they undermine soldiers ability to "cope with psychologically stressful situations in military service" because soldiers with mental health problems are perceived to have a lower ability to function in a group during stressful combat situations – when (the lack of) trust in others and trustworthiness are key predictor of breakdown during combat (Andersson and Carlstedt, 2003). The Appendix Figure A3 shows the relationship between the PF score and the presence of psychiatric diagnoses at enlistment and midlife. Having a low PF score is strongly correlated with psychiatric diagnosis at enlistment, but also strongly correlated with having any mental health/neurodevelopmental disorders in midlife. Here, we standardized the score within enlistment cohorts and presents results for both the continuous score, as well as an indicator for having a low or high score.

We also use information from Statistics Sweden's longitudinal database LISA that covers the entire Swedish population aged 16 and over and contains information on labor market outcomes and place of residence since 1990. The LISA data allows us to measure life-time earnings (age 26-47) for all cohorts in the analysis sample. The parish of residence is useful since parish boundaries often overlap with uptake areas and are small enough to measure meaningful variation in treatment practices. The LISA data in combination with the prescription registers allows us to measure mental health treatments in midlife by individuals in the the parish of residence (henceforth neighborhood) (see further details below).

To define policy exposure status we use the Multigenerational register, containing information on parity, age of the mother, region, and month of birth. We restrict the sample to first-order birth, since few women under age 21 are multiparous. The final analysis sample include 353 000 first-born children born between 1964 and 1972 who resided in Sweden in 2000. Using anonymized identifiers, we link the children to the mental health outcome data. Appendix Table A1 provides descriptive statistics for the full analysis sample - split by Region and Exposure window (see next section).

3.2 Empirical Specification

The analysis sample can be divided into five distinct groups using region and month of birth. The first four are similar sized groups born around the policy experiment. Group (I) are individuals born just before the start of the policy experiment. Group (II) consists of individuals who were exposed during pregnancy but conceived before the start of the policy. Group (III) are individuals exposed during pregnancy but conceived after the start of the experiment, and Group (IV) are individuals who were conceived just after the policy experiment ended. Group (V) consist of children born well-before/after the policy in the treated regions, and children born in the control regions (excluding children born in the regions bordering the experiment regions).

The main analysis focus on children of young mothers (below age 21) belonging to Group (II), and we refer to Group (II) as the baseline exposure group throughout. We also provide results for the effects on children of young mothers belonging to groups I, III, and IV. The focus ón Group (II) allows us to rule out effects of the policy on selection into pregnancy. The estimated impact on the other three groups allows us to shed light on alternative explanations behind our main findings; for example, what is the effect of increased alcohol access just after birth (Group (I))? We also provide results after splitting Group (II) into trimester subgroups, to allow the effects of exposure to vary depending on gestational age at the start of the policy.

In our main analysis we use the following difference-in-difference-in-differences (DDD) specification (Gruber, 1994):

$$Y_{i,r,t,m<21} = \alpha_0 + \beta EXPOSURE_{r,t,m<21} + \eta_{r,t} + \eta_{r,m<21} + \eta_{t,m<21} + \varepsilon_{i,r,t,m<21} \quad (1)$$

Where $Y_{i,r,t,m<21}$ indicates if individual i, born in year-month t, in region r to a mother in age group $m < 21$, was ever treated for a mental health condition between ages 42 and 47, and zero otherwise. $EXPOSURE_{r,t,m<21}$ is equal to 1 if the individual was born to a young mother in a treatment region and belongs to Group (II), and zero otherwise.[33] $\eta_{r,t}$ are period-by-region of birth fixed effects, $\eta_{r,m<21}$ are region-by-maternal age group at birth fixed effects, $\eta_{t,m<21}$ are period-by-maternal age group at birth fixed effects. $\varepsilon_{i,r,t,m<21}$ is the error term.

β is the parameter of interest and captures the impact of the policy on mental health out-

[33] Estimated to have been conceived Feb-Oct.67, based on month of birth

comes among the children of young mothers conceived just before the policy started and exposed to it *in utero*, relative to the outcomes of children in the surrounding cohorts, conditional on the fixed effects. The fixed effects ($\eta_{r,t}$, $\eta_{t,m<21}$, $\eta_{r,m<21}$) takes many unobserved and potentially important confounding factors into account. For example, mental health treatment waiting times could vary systematically across regions and could lead to differences in mental health outcomes. $\eta_{r,t}$ accounts for any region specific differences, even when they vary across birth cohorts within a region. Teenage motherhood is a well-known predictor of adverse outcomes. Over the observation period teenage motherhood rates fell, and as a consequence the quality of the average home environment could have deteriorated due to compositional changes in the group of women giving birth before age 21. Any systematic differences in the mental health outcomes among children born by young and old mothers, even when they vary across birth cohorts, or across regions, are taken into account by $\eta_{t,m<21}$ and $\eta_{r,m<21}$.

The identifying assumption for a causal interpretation of the β estimate is that, conditional on the fixed effects, there are no other (time-varying) shocks that coincides with the policy experiment and also only affects the children of young mothers born in the treatment regions. We are not aware of any major and temporary changes that would fulfill these criterias *and* could have a large influence on the mental health of the children. Note that later in life shocks will also have to fulfill the same conditions in order to bias the β estimate, which rules out many potentially confounding factors that could affect mental health in the treatment and control regions differentially (e.g. major plant closures etc.), but that affects adjacent cohorts in a smoother way.

We also assess the plausibility of the identifying assumption indirectly. First, we test for effects of the policy on children born to young mothers in the same period as our baseline exposure group but in the neighboring regions.[34] Specifically, we reestimate Equation (1) on a "placebo" sample that excludes individuals born in the two treatment regions and instead includes all individuals born in the five neighboring regions.[35] The neighboring regions and the two treatment regions are highly economically interdependent, and (since 1999) in the case of Göteborg and Bohuslän, Skaraborg, and Älvsborg, even belong to the same public health care region. So if e.g. cross-border alcohol purchases was a major phenomenon during the policy, or if other temporary and unobserved shocks with only a slightly broader geographical reach (e.g.

[34]Kopparberg, Närke, Skaraborg, Älvsborg, and Halland.

[35]In our main specification we exclude these neighboring regions in order to avoid dilution from (potential) cross-border consumption or other spill-over effects.

temporary fluctuations in waiting times) are causing the observed effects on later life mental health, we we would expect to find impacts on children born in the neighboring regions as well.

Second, we use the following specifications to test to if there is any impact of the policy on mental health outcomes on the three adjacent birth cohorts (I, II, IV - defined above) using the following specification:

$$Y_{i,r,t,m<21} = \alpha_0 + \sum_{g=1}^{G} \beta_g EXPOSURE_{g,r,t,m<21} + \eta_{r,t} + \eta_{r,m<21} + \eta_{t,m<21} + \varepsilon_{i,r,t,m<21} \quad (2)$$

Our prior is that the effects of the policy should be strongest among the prenatally exposed children, but *a priori* there could also be negative effects on mental health outcomes in the other exposure groups. For example, if the changes in alcohol availability has a strong impact on the home environment it is plausible that children born just before the policy also have worse mental health later in life relative to older children or children conceived after the policy experiment ended. Eq.(2) allows us to test for such postnatal exposure effects.[36]

In the final section of the paper we test whether local mental health treatment intensity mitigate the policy's effects on earnings.[37] We follow Kessler and McClellan (2000) and Cuddy and Currie (2024) and define treatment intensity among neighbors based on the treatments received, irrespective of where the prescriber is located. We measure local midlife treatment intensity by using information on the prescription rates between 2006-2008 among slightly older neighbors (cohorts 1960-1963, i.e. born just before the main sample cohorts) in the neighborhood of residence. A drawback of the prescription data is that it only capture intense treatment (i.e. medication rather than e.g. psychotherapy). However, a big advantage is that it also contains treatments prescribed by primary care physicians.

We interact the local treatment intensity measure with the main treatment indicator and report the estimated (β) and (γ) coefficients from the following specification:

[36] Note that one may be worried that the overall effect could stem from the peer effects from a few exposed peers. For example, Aizer (2008) and Gazze et al. (2021) document significant spill-over effects of ADHD diagnoses and individual lead exposure respectively. However, note that our DDD specification will also net out direct spill-over effects if the peers effects is not only constrained to children of young mothers, which seems unlikely. Even if this would be the case presumably Eq.(2) would be able to capture negative spill-over effects, e.g. large negative effects on cohorts born just before or after the prenatally exposed cohorts.

[37] Nilsson (2017) document substantial effects on labor market outcomes at age 32.

$$
\begin{aligned}
ln(Earnings)_{i,j,r,t,m<21} =& \alpha_0 + \beta EXP_{r,t,m<21} + \delta \overline{LocalTreIntensity}_{j,r,t,m<21} + \\
& \gamma[EXP_{r,t,m<21} \times \overline{LocalTreIntensity}_{j,r,t,m<21}] + \\
& \eta_{r,t} + \eta_{r,m<21} + \eta_{t,m<21} + \varepsilon_{i,r,t,m<21}
\end{aligned} \tag{3}
$$

where the $\overline{LocalTreIntensity}_{j,r,t,m<21}$ is the predetermined measure of the neighborhood of residence mental health treatment intensity as specified above. For interpretability of the (β) estimate, we standardize the local treatment intensity variable (mean 0, sd. 1).

The γ estimate is of particular interest, but note that a positive γ estimate – indicating that a higher local treatment intensity mitigates the effects of prenatal exposure on earnings – could reflect both demand side and supply side factors. A positive effect of a high prescription rate of local mental health care providers could reflect lower barriers in access to care and thereby treatments (supply). Alternatively, it could reflect lower demand side barriers (e.g. lower stigmatization of mental health problems) leading to a higher propensity to seek care once mental health deteriorates and thereby higher psychiatric drug rate among the neighbors (demand). In addition, to the extent that mental health problems are more prevalent among disadvantaged groups, a higher share of treated neighbors could also just reflect the socio-economic status of the neighborhood or other unobserved neighborhood characteristics rather than local mental health treatment intensity *per se*. If residing in poorer neighborhoods hampers economic outcomes (due to worse job referral networks/longer distances to jobs, etc.) of the exposed individuals the γ estimate could even be negative. In section 4.3 we provide results from different versions of Eq. (3) designed to allow us to distinguish between alternative interpretations of the estimated γ coefficient.

4 Results

4.1 Main Results

Table 1 present our main results. Column 1 contains the result from estimating Equation (1) using the full main sample. The baseline exposure group has a 5.5 percentage points higher probability of being treated for a mental disorder in midlife. Relative to the mean prevalence of mental health problems in midlife among those born to young mothers, this corresponds to

a 16% increase.

Column 2 provide our first specification test. Here we exclude the children born in the treatment regions and instead test for effects on children born at the same time in the five neighboring regions. There is no effect of the policy on mental health in this "placebo" sample. In other words, the observed effects in Column (1) is not caused by any other temporary (concurrent or subsequent) shock affecting treatment and neighboring region children similarly.

Column 3 shows the results from Equation (2). Children who were born before the start of the experiment, conceived during the experiment, or conceived just after the policy ended are not more likely to be diagnosed with mental health problem in midlife. The Column (3) results are interesting for several reasons: It indicates (i) that the observed effects are not due to shared unobserved factors among children in the treatment regions born by young mothers just a few months apart (e.g. social, school, economic or health care factors). Moreover, (ii) the negative effect on mental health in the main exposure groups is not primary caused by e.g. postnatal changes in the home-environment. If so, we would also expect to see negative effects on the cohorts born just before the policy started.

The concentration of the negative effect on mental health among those exposed to the policy *in utero* is further illustrated by Figure 3. The top panel plots the trends in the share of children receiving mental care after splitting the raw monthly data into 9 month windows based on month of birth, treatment region and age of mother at birth. The bottom panel of Figure 3 provides further nuance, closer to the DDD specification, by plotting the local mean *differences* in the share of children with mental health conditions across children born by young vs old mothers, in the treated and control regions using a narrow bandwidth.

The first thing to note from the two graphs in Figure 3 is that the midlife mental health outcomes of Group II children born by young mothers in the treated regions clearly sticks out - even absent any controls for year-, season-, or region of birth effects. Second, in clear support of the parallel trends assumption, for both the cohorts born before/after the policy experiment started/ended, the children of young mothers in both treated and control region are on average around five percentage points more likely to be have been diagnosed with a mental health condition, in comparison with their same birth-month cohort peers born by an older mother.[38] For the Group II children the difference in midlife mental health increases by around 6-7 percentage points, whereas the corresponding increase in the control regions are around 1

[38]The level differences highlights the, on average, higher mental vulnerability of children born by young mothers.

percentage point. The difference in the relative increase, after accounting for the fixed region, birth-month cohort, and maternal age effects, is reflected in the Table 1, column (1) estimate.

Our baseline specification exposure window reports the average impact of the policy on later mental health irrespective of timing of exposure during gestation. There are however good reasons to suspect that the impact of the policy may vary due to the existence of sensitive and critical periods of development.[39] A first look at such heterogeneity is shown in the bottom graph of Figure 3. Appendix Figure A1, which plots the underlying raw monthly data used to construct Figure 3, shows that the differences in the smoothed means seems to stem from a rapid deterioration in mental health outcomes among children born by young mother in the treatment regions with longer/earlier exposure to the policy *in utero*. In Table 1, column 4, we test statistically for differential effects of the policy depending on gestational age, by splitting the baseline exposure group into three subgroups based on the trimester at the start of the policy. The impact on mental health is strongest for those exposed from the second trimester, consistent with the correlations, conditional on a set of single confounders (i.e. one at a time), documented in Barr et al. (2006).[40]

Appendix Figure A6 presents findings from a robustness check to address the well-known inference problems faced using differences-in-differences estimators. Figure A6 plots the histogram of the 101 estimates generated by reassigning and rolling the baseline exposure window across all birth cohorts (i.e. from Jan. 1964 to Dec. 1972) in the data. The histogram shows that the true baseline exposure window cohorts estimate stands out: none of the other potential assignments generates a β estimate that is larger in absolute terms if we exclude the four expo-

[39]Some previous work (Black et al., 2019; Almond et al., 2009) have found the strongest effect of prenatal shocks in the second trimester, whereas others have found similarly sized effects throughout pregnancy (e.g. Persson and Rossin-Slater, 2018)

[40]However, note that the interpretation of the trimester of exposure results is complicated in our setting by a few factors. First, basing our exposure assignment on the month of birth does not take into account the potential impact of the policy on the risk of premature birth. Consequently, some individuals are assigned to exposure from trimester 2 (3) may have been exposed from the first (second) trimester.[41] Second, abortion by medical intervention was not legal before 1975, but the risk of spontaneous abortion could also increase . Nilsson (2017) finds that the share of boys (a rough indicator of adverse prenatal conditions, c.f. Wells (2000)) decreased most among those exposed from early pregnancy and particularly from the first trimester. Spontaneous abortions could result in a positive sample selection (Dobbing and Sands, 1973), which will bias the estimated impact on mental health among individuals exposed from the first trimester towards zero. The surviving sample will then disproportionately consists of healthier children, and our results will understate the importance of exposure to the policy in the first trimester – the so-called culling effect. Third, the differential effects is not necessarily due to differences in sensitivity to alcohol exposure, but could also occur because of heterogeneous responses to the increase in availability depending on gestational age. Even if information about risks of alcohol was low at the time, it seem reasonable to assume that risky behavior in general decreases with gestation. Fourth, note that the trimester of exposure estimates captures both the timing *and* duration of exposure to the policy. Finally, some of the indicators of increased alcohol consumption reported in Nilsson (2017) suggests that the largest increase in consumption occurred early on during the policy experiment.

sure windows in immediate vicinity of the baseline window (exact p-value <.01). If we include the the four closest windows (which includes most of the main exposure window cohorts) the exact p-value for the main treatment group is .029. Here it may also be of interest to note that the largest point estimates is found for the two cohort windows immediately *after* the baseline window. These exposure windows includes not only those conceived just before the policy, but also the first two birth-month cohorts conceived after the policy started, and thereby reflect not only effects of prenatal exposure but also any effects from e.g. changes in the composition of parents.

4.2 Heterogeneity, Specific Diagnoses, and Psychological Function

Table 2 Panel A, column (1) reiterate the baseline findings for the full sample, while columns (2) and (3) display the impact for the male and female samples respectively. We find that the effect is larger among men, both in absolute and relative terms. Men in the baseline exposure group have a 7 percentage points higher probability of being treated for a mental condition in midlife (28% increase relative to the male sample mean for children of young mothers). For women, the impact of the policy is more muted (3 pp., or 7%).

In Panel B, we find that prenatal exposure increases the probability of being treated for depression by 5.4 percentage points (24%), Anxiety conditions increased by 2.4 pp.(15%). The baseline prevalence of neurodevelopmental disorders (ADHD and autism) in our midlife sample is low, but prenatal exposure to the policy substantially increases the probability of being treated for at least one of these disorder (1.2 percentage points). The relative increase in the effects on the neurodevelopmental disorders are large (50%).

The neurodevelopmental disorder results are particularly interesting for two reasons: First, the onset of symptoms of these conditions in childhood is a key diagnostic criteria - which suggests that (at least some) of the mental disorders has manifested early in life. Second, hyperactivity and deficits in attention are core deficits in children with FASD (Thomas et al., 2010). ADHD is the most commonly reported mental health diagnosis for children exposed to maternal alcohol use during pregnancy (Fryer et al., 2007).[42] ADHD is diagnosed in up to 94% of those with heavy prenatal alcohol exposure (Peadon and Elliott, 2010). Moreover, FASD is

[42]Other work find that more than 90% (50%) of those diagnosed with FASD are also diagnosed with Behavioral disorders (ADHD) (Popova et al., 2016).

underdiagnosed and FASD is often misdiagnosis as ADHD (Ehrig et al., 2023).[43] The relative magnitudes of the impact on the neurodevelopmental disorders should however be interpreted carefully, since we are likely missing a substantial share of individuals with the same condition that by their mid-40s manages their condition without involvement of the health care services.[44]

To further assess the time of onset, in Panel C we presents the impact on psychological function (PF) as measured at age 18. Consistent with the strong correlations between PF and mental health diagnosis in general and neurodevelopmental disorders in particular (see Appendix Figure A2-A4), we find a strong and significant effect on the probability of having a low PF score (a 12% increase) - exactly the part of the distribution where the PF score correlates strongly with mental health diagnosis at enlistment and in midlife. Combined, the presence of significant effects on neurodevelopmental disorders (emerging before enlistment) and a significant increase in the share of children with low PF scores at age 18, indicates that the negative effects on mental health observed in midlife have their antecedents in childhood, before labor market entry. One way to view the findings on effects on "ability to cope with stressful situations" is that it at least partly captures susceptibility to later mental health problems. Since the enlistee outcomes is not dependent on whether the individuals sought care, we see this finding based on trained psychologist assessments as an important contribution and complement to our main health care records findings.

4.3 Easier access to care is associated with a lower impact on earnings

An important contribution of economists to the fetal origins literature is the focus on effects on non-health outcomes and the relevance of subsequent parental or public compensatory investments (Currie and Almond, 2011). In previous work, Nilsson (2017) documents that those exposed to the policy *in utero* had substantially worse labor market outcomes at age 32. Others show that mental health treatment practices vary widely across locations and that they matter for young peoples mental health trajectories in the US (Cuddy and Currie, 2020; Cuddy and Currie, 2024). In Dutch data, Prudon (2025) find that a one-month (0.5 SD) increase in waiting time decreases the probability of employment by two percentage points. In light of

[43] Recall that the policy was implemented prior to the development of the FASD diagnostic criteria (which includes confirmed alcohol consumption during pregnancy). In other words, the neurodevelopmental disorders we measure in midlife is likely to be the best proxy for actual FASD diagnosis available in our data.

[44] In an attempt to alleviate this concern, the outcomes are calculated using not only the incidence of main diagnosis but also add in the occurrence of conditions if they are used as a sub-diagnosis codes, i.e. it also include cases when the individuals is seeking care for e.g. physical conditions. In practice using only main diagnoses or including the sub-diagnosis has minuscule impact on our findings.

these studies, and given the policy impact on mental health, we next test whether local mental health treatment intensity in midlife can mitigate the impact on earnings. Exposed individuals may benefit from better access to care by e.g. reducing the time it takes to return to work following periods of mental distress and/or reduce the risk that mental distress transition into more serious psychiatric conditions.

As a prelude to our main findings, we first show how the policy affected earnings throughout the life-cycle. On average the exposed cohort have 16% lower earnings between ages 26-47 ($p<0.01$). Figure 4 provides the DDD estimates on yearly earnings after splitting the sample by observed mental health at ages 42-47. Throughout the life-cycle, the negative impact on earnings is higher among the diagnosed than among those undiagnosed. However, the differences in the impact on earnings is particularly large in the ages when we observe mental health status. From age 30 up until around age 39 the difference in impact on earnings is rather constant, after which the difference in the impact on earnings starts to widen.[45]

Given this pattern and the earlier findings on the role of local treatment practices we ask: to what extent can a higher local treatment intensity reduce the differences in earnings? We test this hypothesis using Eq. (3) estimated on the full sample, and after splitting the sample by observed and predicted mental health status in midlife. Table 3 Panel A reports the main effects of the policy on earnings at age 42-47 (col.1) and the interaction effects for the full sample, for those never diagnosed at age 42-47 (col.2), and for those diagnosed with mental health problems at age 42-47, (col. 3). First of all, column 1 to 3 shows that prenatal exposure has a strong negative effects on labor market outcomes in midlife. Unsurprisingly, the direct effects on earnings is larger for those with diagnosed mental health problems, than for those without. However, the neighborhood mental health treatment intensity reduce the negative effects of the policy significantly – but only for those diagnosed with a mental conditions in midlife. A standard deviation increase in the share neighbors treated with mental health drugs decreases the impact of the policy on earnings by 11 percent (about 1/3 of the effect).

Panel A splits the sample by observed mental health diagnosis - which may not be unproblematic.[46] In Table 3 Panel B, we therefor instead report results after splitting the data by predicted midlife mental health status. Specifically, we use the psychological enlistment evalua-

[45]This pattern is partly explained by Figure A2 which shows that an increasing share of those treated for mental health issues at ages 42-47 are also treated at younger ages, and that mental health may deteriorate (and thereby result in lower earnings) before the individual visits the doctor.

[46]For example, underdiagnosis, reversed causality, and direct effects of local treatment intensity on the probability of receiving a diagnosis could complicate the interpretation of the Panel A col.2 and 3 findings

tions and predict mental health problems in midlife with a LASSO procedure.[47] One advantage of this approach is that the enlistment takes place before entry into the labor market (and thus avoids concerns of reversed causality); a second is that the underdiagnosis problem is reduced; a third is that we avoid splitting the data based on an outcome variable (being observed with a diagnosis). A drawback is that we need to restrict the estimation sample to males who went through the enlistment procedures. Reassuringly, the overall findings using predicted mental health (Panel B) lines up well with the findings using observed midlife mental health (Panel A).

Panel C provides the same set of results after including a variable to account for differences in underlying mental health status of the neighbors - thus controlling for the variation in treatment intensity caused by differences in underlying mental health of the neighbors.[48] Adding this potentially important neighborhood control varible, if anything, increases the estimated impact of a higher access to treatment for those with a high (predicted) need for mental health treatment.

As discussed above, a key concern with the Table 3 findings could be that the observed mitigating effect of higher treatment intensity is simply reflecting other unobserved neighborhood characteristics correlated with the intensity of treatment. For example, the share of neighbors prescribed mental health drugs could simply reflect worse socio-economic conditions in the neighborhood. If residing in poorer neighborhoods is correlated not only with worse mental health but also with worse job opportunities (longer distance to jobs, worse job referral networks etc.), a higher share of treated neighbors could be *negative* for labor market trajectories. In contrast, we find strong *positive* effects for those with observed or predicted mental health care needs.

Moreover, note that any unobserved local conditions affecting both those with and without mental health problems similarly *cannot* explain the Table 3 results. Comparing across columns in Table 3 it is clear that it is only those with observed or predicted needs of care that are affected by a higher access to care as measured by the share of neighbors prescribed medication. The

[47] Based on the findings in Figure A5 we force the linear lasso procedure to always include indicator variables for enlistment year, and indicator for low PF score and the continuous Statnine PF scale, and allows it to pick from a set of cognitive and physical potential predictors also measured in the enlistment data: standardized Stanine Scale for Cogntive ability, raw subscores for the four cognitive tests, standardized scores for the same tests, the aggregate crystallized and fluid tests. height(cm), weight(kg), to generate a prediction for being observed with a mental health diagnosis/prescription at age 42-47. The Panel B and C results use the 75%-tile of the predicted score to create and indicator for being of high risk of mental health problems in midlife. Appendix Table A2 provides results for using the 66%-tile instead

[48] The proxy for neighbors mental health is constructed in the same way as for the individual prediction for the same cohorts used to construct the treatment intensity variable.

estimated impact of a higher access to care among those without mental health problems is always small and insignificant. The contrast between the impact of higher access across these two groups supports our interpretation - the differential impact indicates that easier access to mental health care, rather than other unobserved neighborhood characteristics, is the cause of the positive influence on earnings.[49]

Despite these asymmetric findings, more research is needed before we would be comfortable to firmly conclude that access to care in midlife plays the key role in mitigating effects of negative prenatal health shocks. Future work should focus on identifying plausibly exogenous sources of variation in access to care or differences in treatment practices across locations - and ideally combine them with plausibly exogenous variation in prenatal environmental conditions to see if the effects of such shocks can be mitigated by better mental health treatments.

5 Conclusions

We test whether a policy experiment that temporarily increased young people's access to alcohol impacted the mental health of children exposed to *in utero*. We find that prenatal exposure to the policy significantly increases the likelihood of being treated for a mental disorder in midlife. In addition to elevated rates of common conditions such as anxiety and depression, we document substantial effects on neurodevelopmental disorders, suggesting that for some individuals, the mental health consequences of prenatal exposure were evident from an early age.

The natural experiment and data we analyze allows us to address many (but not all) of the core challenges linked to the identification of the causal effects of prenatal alcohol exposure on offspring outcomes. A central limitation is our inability to identify what forms of alcohol consumption are responsible for the observed effects. That said, the consumption patterns

[49] Appendix Table A2 provides further robustness checks to these final findings. Overall, these changes in specification does not change our main conclusions. Panel A use the full sample (males and female) while controlling for the predicted mental health care needs of the neighbors - finding similar effects as before. Panel B tests whether local midlife treatment intensity has an effects of the policy on earnings well *before* diagnosis are observed. If local treatment intensity just reflect other unobserved local conditions unrelated to treatment intensity, we could expect that earnings earlier in the career (age 30-35) i.e. well before mental health deteriorates, also could be affected. However, Panel B shows that local midlife treatment intensity does not influence earnings before the individuals reach age 42-47, neither among those later treated or those never treated. In addition, the main effect of policy exposure is also highly similar across the two groups at age 30-35, indicating that the mental health status at that point is more similar across the two groups. In other words, our baseline findings does not seem to be simply caused by other (unobserved) neighborhood characteristics. Panel C tests if the main results are sensitive to alternative cut-offs for the prediction of the high risk of mental health problems index (66th than 75th-percentile). Panel D excludes the top and bottom 1% of the local treatment intensity distribution. Panel E interacts the residual intensity (after netting out predicted underlying neighborhood mental health) with the exposure variable.

among young people in Sweden (and elsewhere) are well-documented (binge drinking during weekends and festivities). Chronic maternal alcoholism, typical among mothers with children with full-blown FAS, among young people are exceedingly rare. Yet, the absence of data on exact consumption patterns of course limits the conclusions we can draw to other settings. In this regard our design is similar to most work in the literature on prenatal shocks more broadly which seldom (if ever) have access to information exact changes in prenatal environmental conditions. Future studies could gather prospective data on large samples of expectant mothers, randomly assign mothers to effective interventions (e.g. cessation encouragement designs), accurately measure changes in consumption, and follow their children into adulthood.

Leaving aside the missing data on individual consumption levels, it remains difficult to directly generalize our findings to other settings due to the differences in the information available today. Most women in developed countries are already aware of the risks of drinking during pregnancy. An introduction of a similar policy in Sweden today would most likely not produce the same impact on offspring outcomes in the *aggregate*. In regions where risks are less widely recognized or communicated, our findings may continue to hold direct policy relevance. Moreover, prenatal alcohol exposure *before* pregnancy recognition is still ubiquitous. In the US, 50% of pregnant women report alcohol use before pregnancy recognition (on average week 5.5, 23%$\geq$ 7 weeks' gestation) while around 10% of women continue to use alcohol throughout pregnancy (Sundermann et al., 2021). Despite these limitations - our work extend and complement previous findings in the literature on prenatal alcohol exposure and the broader literature of *in utero* shocks and subsequent child outcomes in several ways.

We also provide evidence consistent with that greater local mental health treatment access in midlife substantially reduces the policy's negative impact on earnings — but only among individuals diagnosed with, or at high predicted risk of, mental health problems. These final findings suggest that policies aimed at reducing access barriers to mental health care could play an important role in mitigating the impact of adverse prenatal conditions on midlife earnings. Future work should explore possibilities to identify to what extent mental health related policies (e.g. waiting times, treatment practices etc.) could help offsetting the long-term economic consequences of adverse *in utero* conditions.

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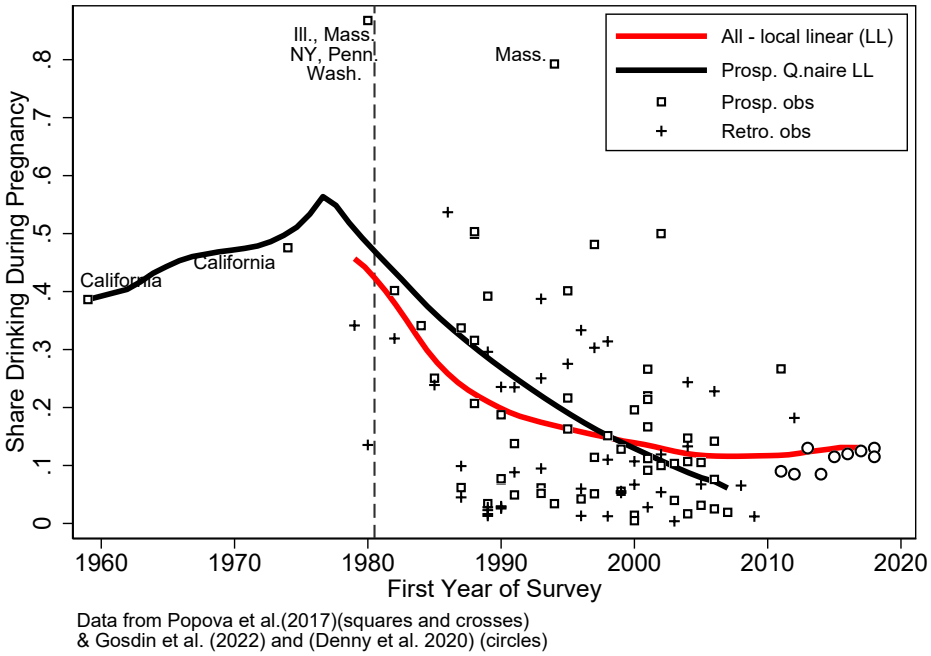
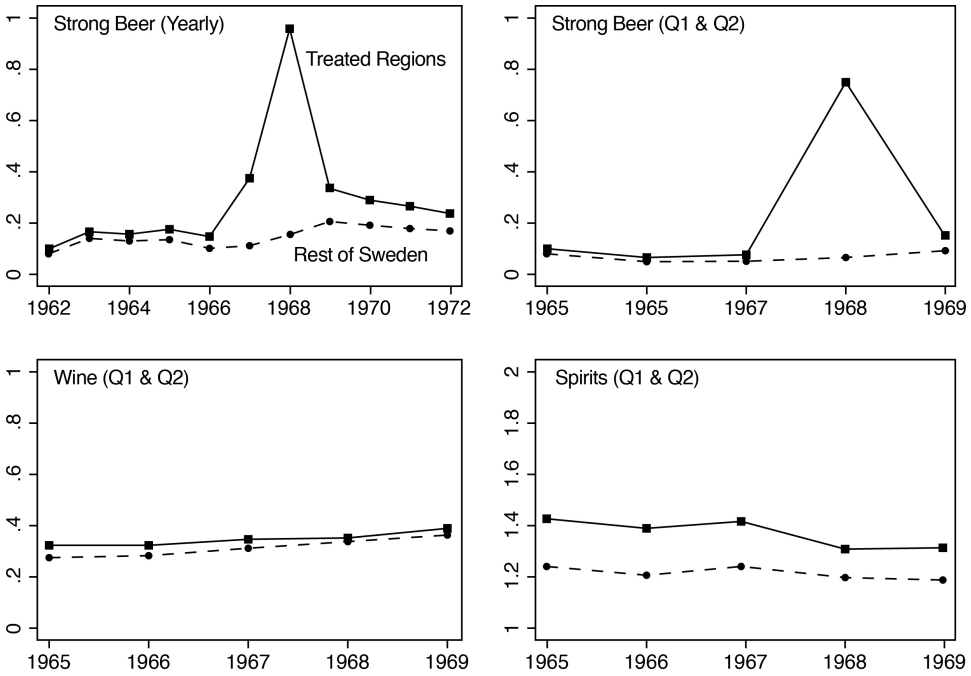
Figure 1: Reported Alcohol Consumption During Pregnancy in the US



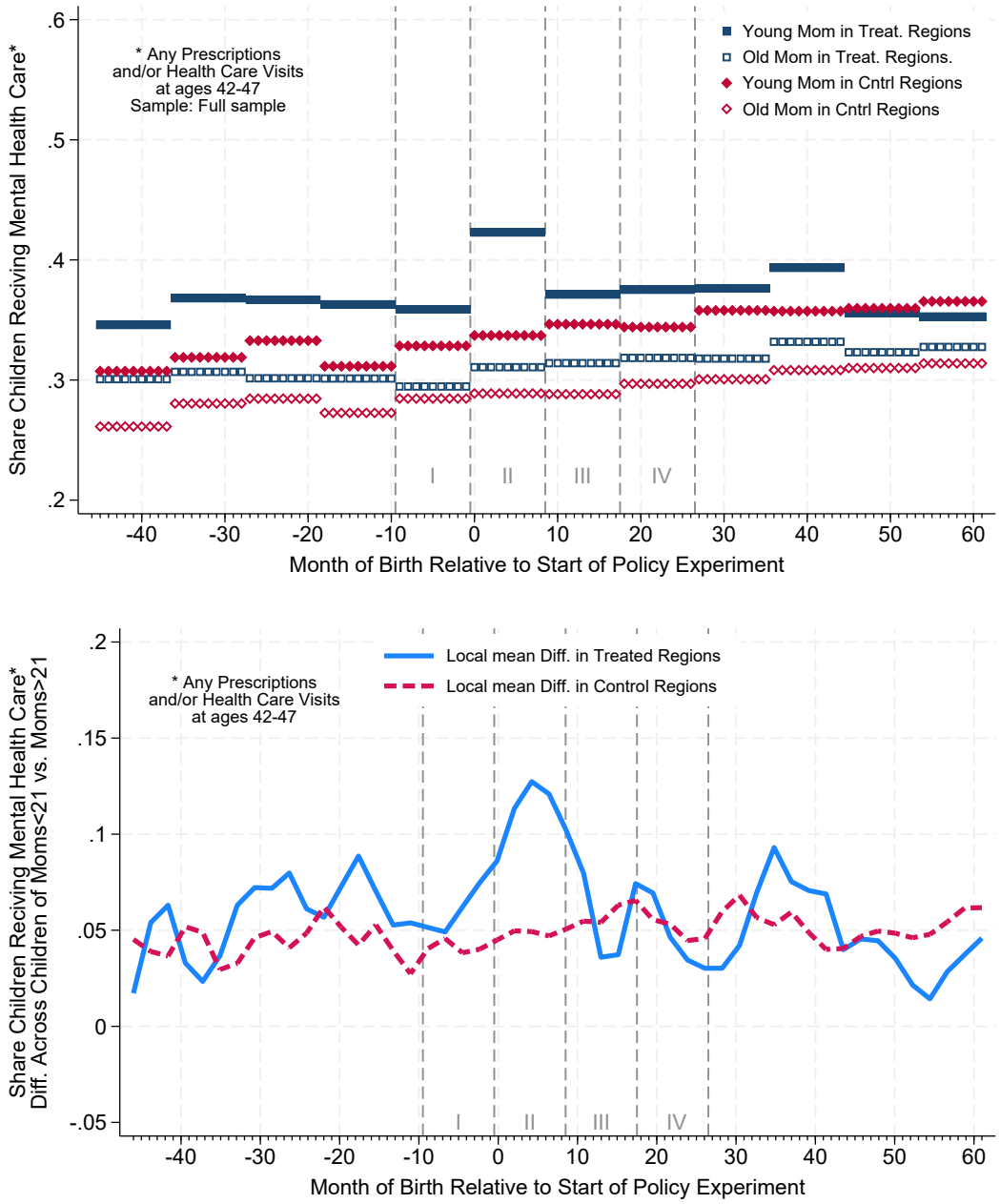
Note: The markers represents share women reporting drinking any amount of alcohol during pregnancy collected in prospective studies (squares) and retrospective studies (crosses) as collected in Popova et al. (2017). The circles are US representative prospective data as surveyed in Gosdin et al (2022) and Denny et al.(2020). The solid lines are the local linear fit for the prospective studies (black) and for all studies (red) after restricting the sample to the the years with more than state available (i.e. post 1979). Many of the studies reported in Popova et al. 2017 report averages over several years, here we use the first year of the survey to plot the means. The dashed line represents the timing of the first US Surgeon general warning to avoid alcohol during pregnancy.

Figure 2: Alcohol Sales in Liters of 100 Percent Alcohol per Capita



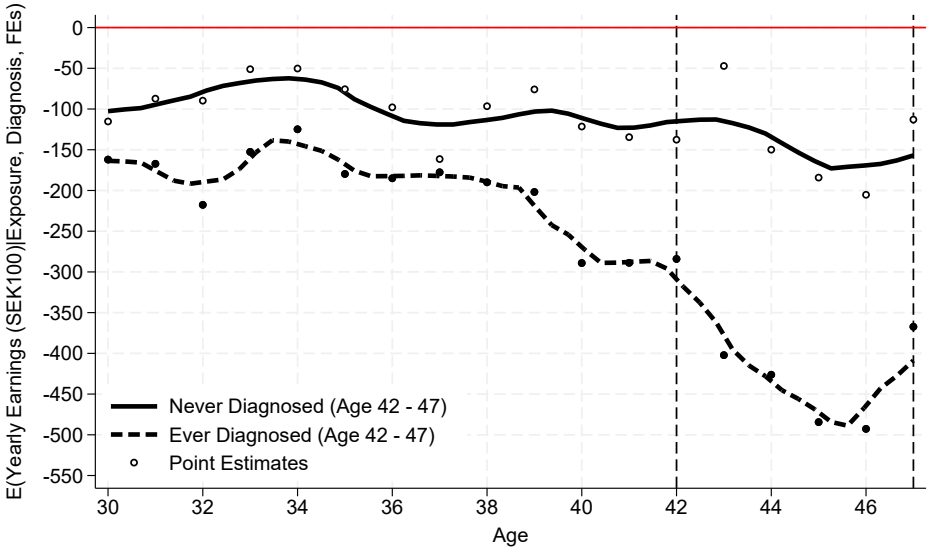
Notes: The annual sales data are from Statistics Sweden (1962–72); quarterly data are from SOU (1971:77). Data are not available for the quarters individually.

Figure 3: Trends in Midlife Mental Health



Notes: The top Figure plots the raw data trend in the share of children with any mental health treatment between ages 42-47 after splitting the data into nine month windows. The nine month averages reflects the share of children born by young and old mothers (below/above age 21 at birth) receiving any mental health care in treated and control regions. **The Bottom figure** plots the difference in the local mean probability of being treated for a mental health condition between individuals born to young (< 21) versus old (≥ 21) mothers, split by region of birth treatment status. The local averages are estimated using a local mean smoother (bandwidth of 1.5 months, triangular weights) on the month of birth data. The figure also highlights the Exposure groups used in Eq.(2) Exposure Group (I) indicates those born just before the policy started, Group (II) – the main exposure group – were conceived before the policy started and exposed to *in utero*, Group (III) were conceived during the policy, and Group (IV) were conceived just after the policy ended. Appendix Figure A1 provides plots of the underlying raw data used to construct these Figures.

Figure 4: The Impact on Earnings by Age and Observed Mental Health Status



Notes: The figure plots DDD points estimates on Yearly Earnings by Age, split by mental health status. The hollow cirlces represent DDD estimates for earnings using the main equation conditional on being observed with any mental condition/treatment at age 42-47. The full circles are the corresponding estimates for the sample with not conditions observed at the same ages. The local averages (solid and dashed lines) are weighted by the inverse of the standard errors of the DDD estimates.

Table 1: Effect of the Policy on Mental Health in Midlife

Outcome:	(1) Any Mental Disorder Age 42-47	(2) Any Mental Disorder Age 42-47	(3) Any Mental Disorder Age 42-47	(4) Any Mental Disorder Age 42-47
Sample	Main	Placebo	Main	Main
Born just before			.0162	.0162
			(.0127)	(.0127)
Exposed *in utero*	.0549***	-.0043	.0562***	
	(.0115)	(.0123)	(.0117)	
Trimester 3				.0466***
				(.0149)
Trimester 2				.0808***
				(.0231)
Trimester 1				.0394**
				(.0189)
Conceived during			-.0085	-.0085
			(.0164)	(.0164)
Conciev. just after			.0051	.0051
			(.0163)	(.0163)
Observations	353 433	369 861	353 433	353 433
R^2	.011	.011	.011	.011
Mean outcome	.34	.33	.34	.34

Notes: *Exposed in utero* is a dummy variable that equals one if the child was conceived before the start of the experiment, exposed to the experiment in utero, and born in a treatment region to a mother who was below age 21 at childbirth, and zero otherwise. Column 1 presents the analysis for our main sample, which excludes the five regions neighboring the two treatment regions. In column 2 we exclude the treatment regions to examine the policy's effect on individuals born to young mothers in the five neighboring regions. In addition to the effect on Group (II), column 3 estimates the effect of being born to a young mother just before the policy started (Group (I)), as well as the effects of being born to a young mother and conceived *during* the experiment (Group (III)) or *after* the experiment had ended (Group (IV)). Column 4 breaks the baseline exposure group into three subgroups based on the onset of prenatal exposure to the policy. All regressions include fixed effects for month-by-year of birth x region of birth, month-by-year of birth x indicator for whether the mother was under age 21, and region of birth x indicator for whether the mother was under age 21, see equation (1). Standard errors, reported in parentheses, are clustered at the level of exposure, which varies by month and year of birth, region, and maternal age group. * $p < 0.10$, **. $p < 0.05$, *** $p < 0.01$.

Table 2: Heterogeneity and Detailed Mental Health Outcomes

PANEL A: The impact by gender			
Sample:	All	Men	Women
Outcomes:	Any Ment.	Any Ment.	Any Ment.
Exposed in utero	.0562***	.0732***	.0293*
	(.0117)	(.0200)	(.0153)
Observations	353 433	182 015	171 418
Sample Mean	.3372	.2616	.4170
R^2	.0109	.0150	.0199
PANEL B: The impact on Specific Mental Conditions			
Sample:	All	All	All
Outcomes:	Depression	Anxiety	ADHD/Autism
Exposed in utero	.0542***	.0250***	.011**
	(.0094)	(.0093)	(.0051)
Observations	353 433	353 433	353 433
Sample Mean	.2242	.1674	.0210
R^2	.0101	.0104	.0088
PANEL C: The impact on ability to cope with stressfull situations as assessed by military psychologist at age 18			
Sample:	Males	Males	Males
Outcomes:	Psychological Function (PF)	Low PF	High PF
Exposed in utero	-.0445	.0429**	-.0412**
	(.0443)	(.0204)	(.0209)
Observations	161 415	161 415	161 415
Sample Mean	-.1986	.3738	.39
R^2	.0360	.0314	.0301

Notes: In Panel A 1–3 the outcome is an indicator for being treated for any mental health condition during ages 42–47. In Panel B the outcome is based on prescriptions for drugs used to treat depression and anxiety whereas column 3 uses both the in- and outpatient data on main *and/or* subdiagnoses for health care visits and the drug registry to identify individuals with autism or ADHD. Panel C shows the standardized continuous stanine scale of the ability to cope with stressful situations (PF), an indicator that equals one if the individual's PF scores is in the bottom (1-4), or top (6-9) of the distribution. Standard errors, clustered at the birth month by region by maternal age group level, in parenthesis. * $p < 0.10$, **. $p < 0.05$, *** $p < 0.01$.

Table 3: Does Local Mental Drug Prescription Intensity Mitigate the Impact on Earnings?

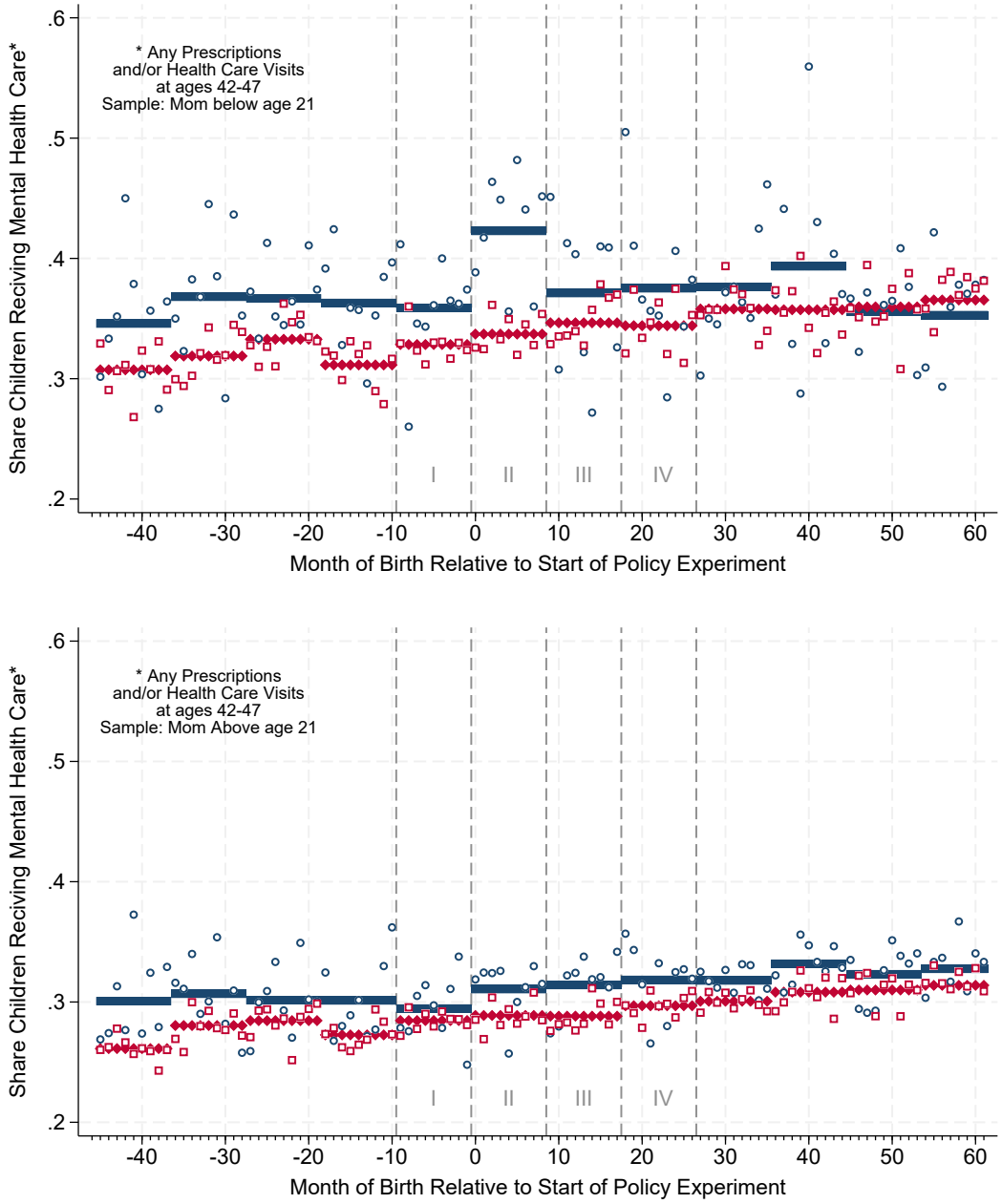
Sample:	Full sample	Poor Mental Health (Age 42-47)	Good Mental Health (Age 42-47)
PANEL A: (log) Earnings ages 42-47 (Split by Observed Mental Diagnosis)			
Exposed *in utero*	-.176***	-.308***	-.081*
	(.039)	(.079)	(.043)
Exposed x Local Prescription Intensity	.041	.112***	.019
	(.028)	(.038)	(.035)
Observations	311,600	92,171	219,428
PANEL B: (log) Earnings ages 42-47 (Split by Predicted Mental Health)			
Exposed *in utero*	-.174***	-.290***	-.104
	(.065)	(.134)	(.070)
Exposed x Local Prescription Intensity	.047	.154**	.005
	(.044)	(.065)	(.044)
Observations	131,371	31,202	100,132
PANEL C: (log) Earnings ages 42-47 (Split by Predicted Mental Health + Controlls for Predicted Local Mental Health Care Demand)			
Exposed *in utero*	-.182***	-.296***	-.111
	(.065)	(.132)	(.071)
Exposed x Local Prescription Intensity	.068	.164**	.015
	(.043)	(.065)	(.045)
Observations	131,371	31,202	100,132

Notes: The table report results for log cumulative earnings ages 42-47 in Panel A, C and D, and for ages 30-35 in Panel B. Local Prescription Intensity is the the share of neighbors treated with mental health medication in midlife (see text for details). Panel A reports results for the full sample. Panel B splits the sample based on predicted midlife mental health care needs using the psychological evaluations at enlistment to predict mid-life mental health care needs (see text for details. Panel C reestimated the Panel B model and controls for the predicted mental health care needs of the neighbors. Standard errors in parenthesis * $p < 0.10$, **. $p < 0.05$, *** $p < 0.01$.

Appendices

A Additional Results

Figure A1: Trends in Midlife Mental Health - Raw Data



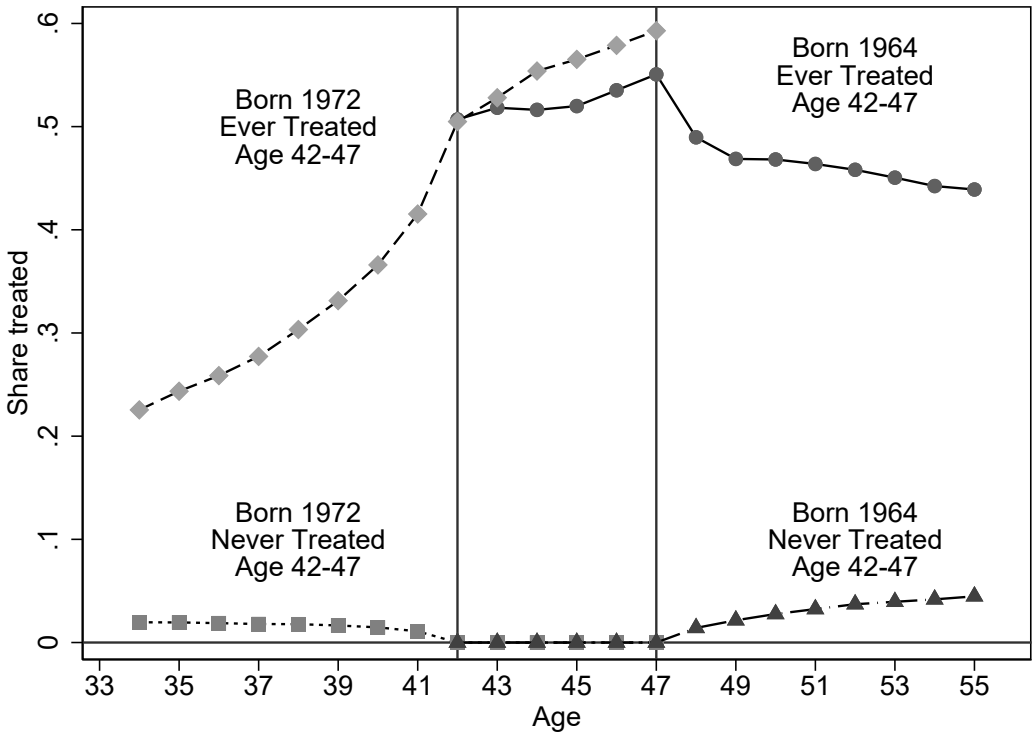
Notes: The top figure plots the raw data trends (share of children with any mental health treatment between ages 42-47) and the corresponding nine month window average for children born by young mothers (below age 21 at birth). **The bottom figure** plots the same trends for children born by older mothers (above age 21 at birth). Red markers are Control region children and blue markers Treated region children. The figure also highlights the Exposure groups used in Eq.(2) Exposure Group (I) indicates those born just before the policy started, Group (II) – the main exposure group – were conceived before the policy started and exposed to in utero, Group (III) were conceived during the policy, and Group (IV) were conceived just after the policy ended. Figure 2 in the main text provides the trends in the differences across young and old mothers in treated and control regions.

Table A1: Descriptive Statistics by Exposure Window and Region

	Treatment Regions Mean (SD)	Control Regions Mean (SD)
Just Before: I	N = 4,675	N = 26,622
Any Condition	0.311 (0.463)	0.296 (0.457)
Depression	0.199 (0.400)	0.187 (0.390)
Anxiety	0.157 (0.364)	0.140 (0.347)
ADHD/Aut.	0.015 (0.123)	0.015 (0.121)
Young Mom	0.262 (0.440)	0.270 (0.444)
Exposed: II	N = 4,349	N = 24,899
Any Condition	0.337 (0.473)	0.301 (0.459)
Depression	0.208 (0.406)	0.187 (0.390)
Anxiety	0.176 (0.381)	0.144 (0.351)
ADHD/Aut.	0.017 (0.128)	0.015 (0.121)
Young Mom	0.235 (0.424)	0.249 (0.432)
Conceived During: III	N = 4,181	N = 23,647
Any Condition	0.328 (0.470)	0.303 (0.459)
Depression	0.211 (0.408)	0.193 (0.394)
Anxiety	0.169 (0.375)	0.142 (0.349)
ADHD/Aut.	0.016 (0.127)	0.017 (0.130)
Young Mom	0.242 (0.429)	0.245 (0.430)
Conceived Just After: IV	N = 3,925	N = 22,206
Any Condition	0.331 (0.471)	0.307 (0.461)
Depression	0.220 (0.414)	0.196 (0.397)
Anxiety	0.170 (0.376)	0.150 (0.357)
ADHD/Aut.	0.017 (0.130)	0.019 (0.137)
Young Mom	0.225 (0.418)	0.221 (0.415)
Concieved Well-before/after: V	N = 35,630	N = 203,299
Any Condition	0.326 (0.469)	0.303 (0.459)
Depression	0.213 (0.409)	0.193 (0.395)
Anxiety	0.164 (0.371)	0.141 (0.349)
ADHD/Aut.	0.018 (0.132)	0.017 (0.130)
Young Mom	0.244 (0.429)	0.248 (0.432)

Notes: The table provides descriptive statistics for main outcome variables in the the main analysis sample measured at ages 42-47, split by exposure group and treatment status based on region and month of birth. See text for definitions of the variables. It also reports the share of children born by a young mother (<21 at birth) in each group.

Figure A2: Mid-life cycle Profiles of share Treated with a Mental Health drug



Notes: The Figure plots the life-cycle profiles for prescriptions of mental health drugs for individuals who ever/never recieved a mental prescription during the ages 42-47 in two birth cohorts at the beginning and end of the baseline sample (1964 and 1972). The y-axis reflects the share of individuals that received treatment at a given age (x-axis), for example - among those ever treated at ages 42-47, around 22 percent were also treated when they were age 34, 30 percent were treated at age 38, etc. Among those not treated at ages 42-47 less than 3 percent were treated at age 34 and age 38 . The prescription data covers the period 2006-2019.

Figure A3: Any Psychiatric Diagnosis at Military Enlistment and Psychological Function Score (PF)

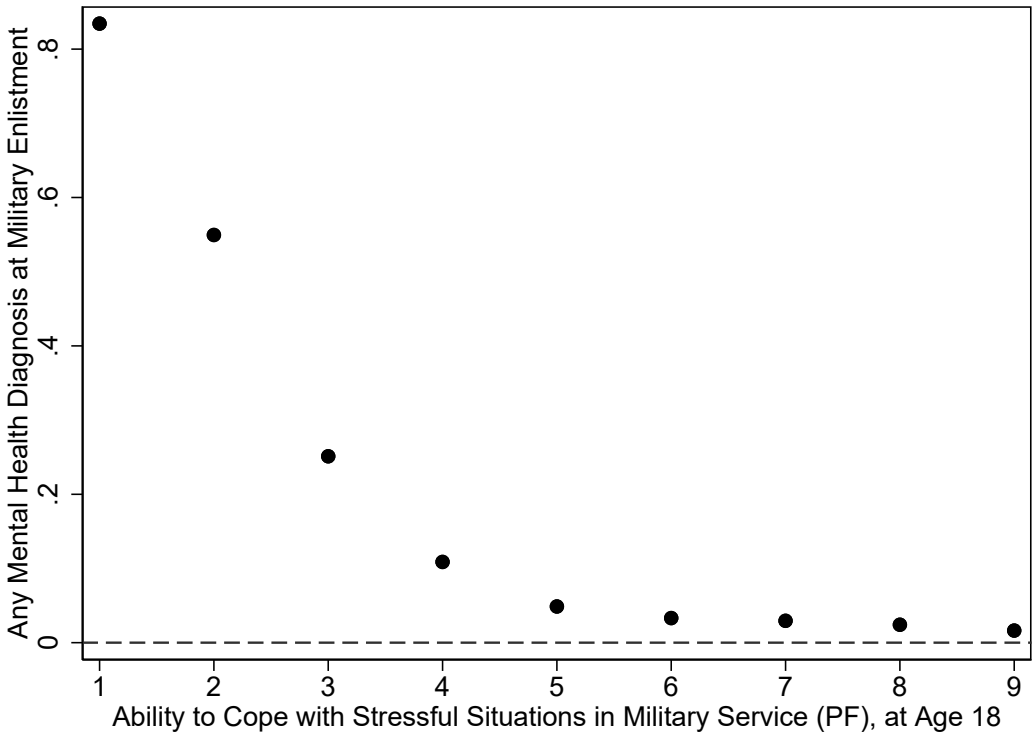
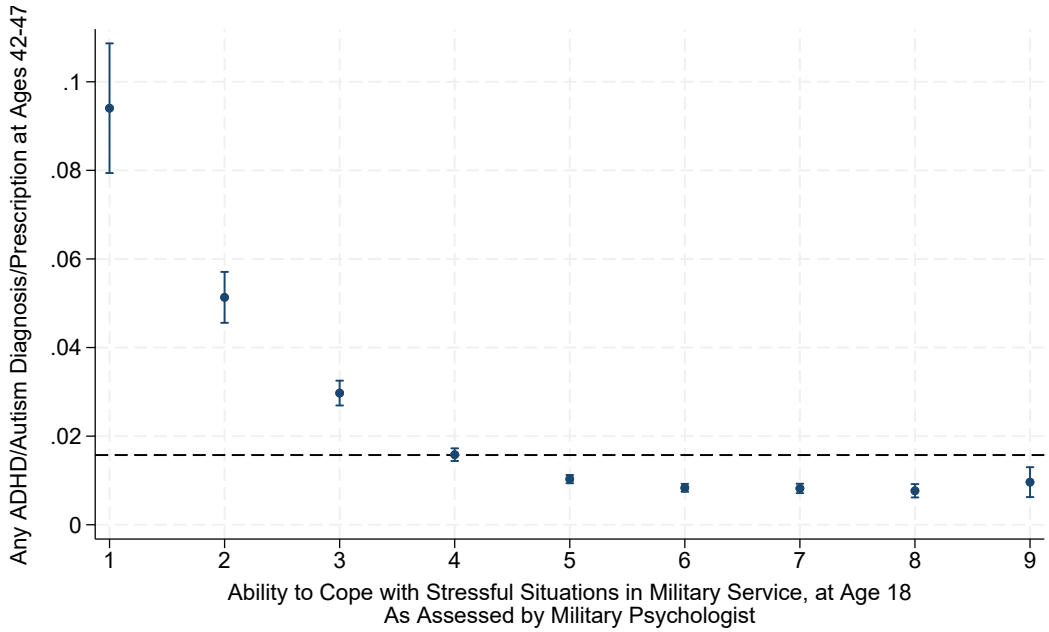
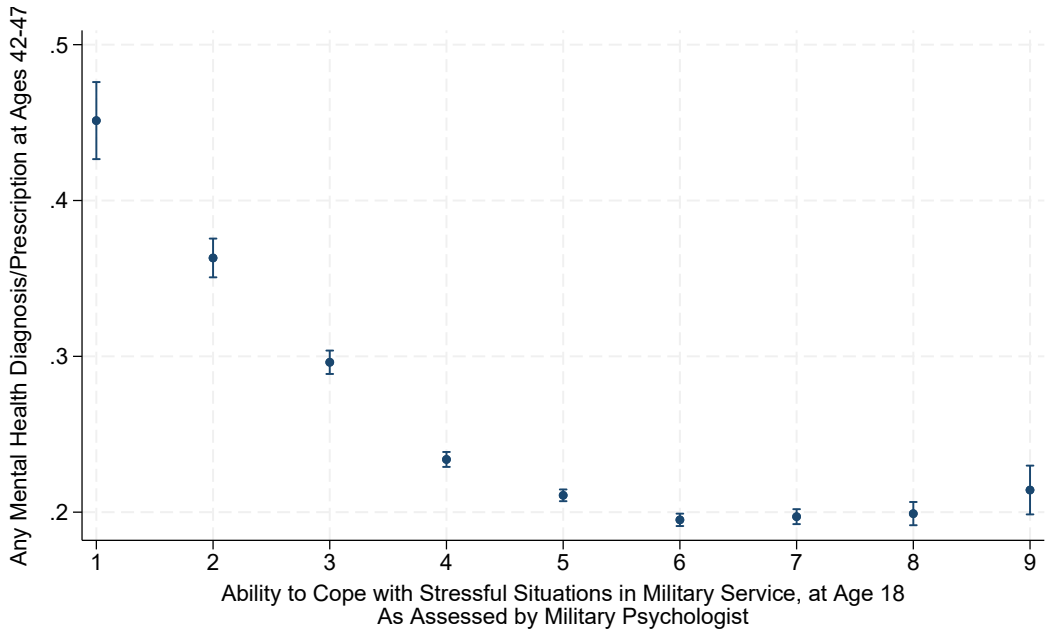


Figure A4: Any Neurodevelopmental Disorder at ages 42-47 and Psychological Function Score (PF)



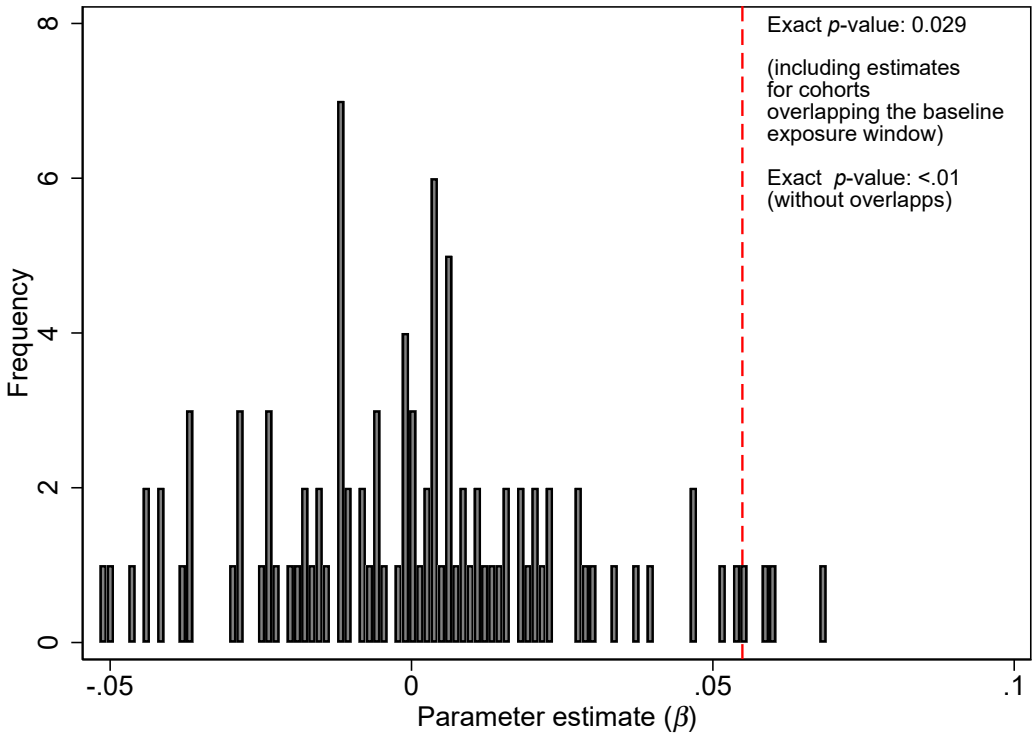
Notes: The Top/Bottom figure plots the indicator for being treated for any mental disorder/Neuropsychological disorders between ages 42–47 against Psychological Function Score as assessed by Military Psychologist at around age 18 (Men only), both variables have been residualized for the year of enlistment and their score on the cognitive test using method proposed by Cattaneo et al., 2024.

Figure A5: Any Mental Disorder at ages 42-47 and Psychological Function Score (PF)



Notes: The figure plots the indicator for being diagnoses with Any Psychiactric Condition at Military Enlistment against Psychological Function Score as assessed by Military Psychologist at around age 18 (Men only), both variables have been residualized for the year of enlistment and their score on the cognitive test using method proposed by Cattaneo et al., 2024.

Figure A6: Rolling Treatment Window Estimates



Notes: The figure plots 101 DDD estimates of the effect of being born to a young (< 21) mother in a treatment region on the probability of being treated for a mental health condition in midlife. We let a 9-month treatment window glide across all birth cohorts in the main sample window - from January 1964 to December 1972. The estimated treatment effect for our baseline exposure group (conceived just *before* the onset of the policy and exposed *in utero*) is indicated by the vertical dashed line. Note that the figure plots the histogram for all possible treatment windows, even if all but one birth cohort belongs to the main exposure window. The exact two-sided p-value is reported in the top right corner, when both including and excluding the four closest exposure windows around the baseline exposure group.

Table A2: Robustness for Local Treatment Intensity Results

Sample:	Full sample	Poor Mental Health (Age 42-47)	Good Mental Health (Age 42-47)
PANEL A: (log) Earnings ages 42-47 (Split by Observed Mental Diagnosis) + Full sample w. Controlls for Predicted Local Mental Health Care Demand			
Exposed *in utero*	-.176***	-.308***	-.081*
	(.039)	(.079)	(.043)
Exposed x Local Prescription Intensity	.041	.112***	.019
	(.028)	(.038)	(.035)
Observations	311,600	92,171	219,428
PANEL B: (log) Earnings ages 30-35 (Split by Future Mental Diagnosis)			
Exposed *in utero*	-.095**	-.110***	-.070*
	(.038)	(.056)	(.058)
Exposed x Local Prescription Intensity	-.052*	-.024	-.055
	(.030)	(.033)	(.049)
Observations	324,505	97,466	227,038
PANEL C: (log) Earnings ages 42-47 (Split by Predicted Mental Health 66%-tile)			
Exposed *in utero*	-.174***	-.287***	-.087
	(.065)	(.111)	(.077)
Exposed x Local Prescription Intensity	.047	.134**	-.020
	(.044)	(.063)	(.049)
Observations	131,371	42,894	88,451
PANEL D: (log) Earnings ages 42-47 (Split by Predicted Mental Health Windsorized (Bottom/Top 1%)			
Exposed *in utero*	-.182***	-.300***	-.106
	(.065)	(.133)	(.070)
Exposed x Local Prescription Intensity	.068	.263**	-.032
	(.043)	(.111)	(.057)
Observations	131,371	30,494	98,624
PANEL E: (log) Earnings ages 42-47 (Split by Observed Mental Diagnosis) + Full sample Main effect interacted with Residual Local Prescription Intensity			
Exposed *in utero*	-.156***	-.259***	-.071*
	(.037)	(.073)	(.040)
Exposed x Residual Prescription Intensity	.048*	.138***	.006
	(.027)	(.040)	(.037)
Observations	311,589	92,169	219,428

Notes: Panel A use the full main sample (men and Women and controls for the predicted mental health of neighbors. Panel B use earnings at age 30-35 as the outcome. Panel C use the 66th-percentile as the cut-off values in the index of individual mental health prediction instead of the 75%-tile. Panel D winsorize the Local prescription intensity variable at the 1% and 99%tiles. Panel E use the residual Local prescription Intensity (Residuals from netting out variation from the predicted underlying mental health of the neighbors). See table 3 for further details.