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Family Ruptures and Intergenerational Transmission of Stress

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Family Ruptures and Intergenerational Transmission of Stress*

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Abstract

The high and rapidly increasing prevalence of mental illnesses underscores the importance of understanding their causal origins. This paper analyzes one factor at a critical stage of human development: exposure to maternal stress from family ruptures during the fetal period. We find that *in utero* exposure to the death of a maternal close relative has lasting consequences on mental health in adulthood, as captured by 11 and 9 percent increases in the consumption of prescription drugs treating anxiety and depression, respectively, and a 23 percent increase in the average daily dose of medication for Attention Deficit Hyperactivity Disorder (ADHD). Further, children exposed prenatally to the death of a relative up to four generations apart are 20 percent more likely to be born low-birth-weight and 11 percent more likely to be hospitalized for conditions originating in the perinatal period in early childhood. Our results imply large welfare gains from preventing fetal exposure to severe stress; the decrease in consumption of prescription drugs treating depression alone can be valued at nearly $1 billion. More generally, our results point to *in utero* stress exposure as a potential cause of the rising incidence of several mental illnesses.

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1 Introduction

Mental illness generates vast private and social costs. In 2008, the market for prescription drugs treating depression totaled $9.6 billion in the United States, a sales volume exceeded only by cholesterol regulators and pain medications (Dickstein, 2014). In 2013, one in seven school-age boys were treated with prescription drugs for Attention Deficit Hyperactivity Disorder (ADHD), fueling a $9 billion market, which is more than five times larger than the $1.7 billion market just a decade earlier (Visser, 2014). Moreover, estimates suggest that mental illness accounts for over half of the rise in disability receipt among men in the last two decades (Duggan and Imberman, 2009).

The high and rapidly increasing incidence of mental conditions such as depression, anxiety, ADHD, and autism-spectrum disorders has prompted fervent debates regarding their causes and correlates both in popular media and across scientific disciplines. While this question is undeniably complex—a variety of factors are likely important—the understanding of specific causes is necessary for prevention and cost-effective policy design. Existing research has documented correlations between different mental conditions and a range of socioeconomic, hereditary, and environmental factors. Yet, as discussed further in Section 2, the evidence on causal drivers is limited and misperceptions abound. For example, a widely popularized (yet repeatedly refuted) claim that the Measles, Mumps, and Rubella (MMR) vaccine causes autism-spectrum disorders has contributed to a substantial decline in vaccination rates, causing measles to re-emerge in Europe and the U.S. after having been effectively eliminated (see, e.g. McIntyre and Leask (2008)).

In this paper, we focus on one possible causal factor at a critical stage of human development: *in utero* exposure to maternal stress. Specifically, we analyze how a mother's stress resulting from a death in the family during pregnancy affects her unborn child's well-being from birth to adulthood, with a particular emphasis on mental health.

Our focus on the fetal stage is consonant with recent neuroscientific studies showing

that mental illness is related to brain abnormalities that likely arise before birth.[1] Further, two recent studies in economics trace adult mental illness to malnutrition during the fetal stage, using data from Uganda and Iraq (Almond and Mazumder, 2011), as well as Ghana (Adhvaryu et al., 2014).[2]

Our focus on stress is motivated by prior evidence of a correlation between mothers' pregnancy levels of the stress hormone cortisol and their children's mental health.[3] Yet to the best of our knowledge, no existing study establishes evidence of a *causal* link between antenatal exposure to maternal stress—from family bereavement or from other stressors—and later-life mental health.[4] Moreover, the particular stressor that we study is arguably universal: the sudden loss of a loved one plausibly ranks among the stressors with the widest reach in society, affecting nearly everyone, across socio-economic groups and ages, at some point in life.

To investigate whether the uterine environment propagates the impact of this stressor to the unborn child, we leverage administrative data from Sweden. As we detail in Section 3, we start from the universe of children born in Sweden during selected years between 1973 and 2005, and use multigenerational population registers to construct family trees that span four generations, from the child to his/her maternal great-grandparents. Our sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within one year of the child's date of birth. By considering the deaths of different relatives, our approach presents a new measure of the intensity of stress exposure: the strength of the family tie that is severed. We then merge these data with information about the

[1] See, for example, Liu et al. (2012) for depression and Berquin et al. (1998) and Stoner et al. (2014) for ADHD and other autism-spectrum disease.

[2] Consistent with this evidence, epidemiologists have documented a correlation between *in utero* exposure to the Dutch famine of 1944 and the onset of mental disease in adulthood (Susser and Lin, 1992; Susser et al., 1996; Neugebauer et al., 1999; McClellan et al., 2006).

[3] A multitude of epidemiological papers have documented a correlation between antenatal stress and ADHD; see Appendix C for details.

[4] Malaspina et al. (2011) show that exposure to the Six-Day Arab-Israeli War *in utero* increased the likelihood of developing schizophrenia in adulthood. However, their empirical design precludes the isolation of fetal exposure to stress from the other consequences of the war, such as its economic repercussions.

children's mental and physical health throughout childhood and into adulthood stemming from birth records, inpatient and outpatient records, and the prescription drug registry.

For identification, we take advantage of quasi-random variation in the exact timing of bereavement relative to the child's *expected* date of delivery at full-term, as described in Section 4. Intuitively, we exploit the fact that some mothers experience the death of a relative during pregnancy, while others experience such a death shortly after giving birth. While all these children are exposed to the post-natal consequences of the relative's passing (e.g., the associated income shocks), only the former group is exposed to the mother's experience of the death through the uterine environment. By comparing the outcomes of these two groups, we isolate any additional effects of fetal exposure to maternal stress, *relative to the consequences of such exposure shortly after birth.* Our analysis relies on the assumption that the precise timing of death within a narrow time frame of the estimated expected birth date, which is pre-determined at conception, is uncorrelated with other determinants of child well-being, and we provide evidence that there is no significant association between the timing of death and a variety of observable maternal characteristics.

This paper makes two primary contributions, as presented in Section 5. First, to the best of our knowledge, our study is the first to document a causal link between fetal stress exposure and mental health in later life. We find that *in utero* exposure to the death of a relative increases the likelihood that an individual consumes a prescription drug used to treat a mental health condition by 6 percent. This effect is driven by 11 and 9 percent increases in the consumption of drugs treating anxiety and depression, respectively, and persists into adult age. Additionally, we find that fetal exposure to family bereavement raises the average daily dose of ADHD and depression prescription drugs by 23 and 20 percent, respectively. These adverse effects only arise as consequences of the death of a close relative of the mother, suggesting that the severity of stress exposure is important for its mental health consequences.

Second, by following the same children from birth to adulthood, we can trace the onset of adverse effects of exposure to maternal bereavement *in utero*. We document that important physical health consequences are already evident at birth and in early childhood. In particular, we see 20 and 30 percent increases in the likelihoods of low-birth-weight (less than 2,500 grams) and very-low-birth-weight (less than 1,500 grams) births, respectively, a 15 percent increase in the risk of a pre-term birth (less than 37 weeks gestation), and a 10 percent increase in the likelihood of a small-for-gestational-age (SGA) birth. Further, after birth, we find that *in utero* exposure to stress due to the death of a relative increases a child's likelihood of being hospitalized or receiving outpatient treatment during the first five years of life. These impacts are driven by exposure during the first trimester and by treatments for conditions originating in the perinatal period.

Additionally, unlike the mental health consequences we find, we present evidence suggesting that the physical health effects are less sensitive to the severity of stress exposure and seem to fade as the children get older. For example, we find no effects on hospitalizations by ages 10, 18, or 27, or on the consumption of drugs treating physical conditions such as obesity, diabetes, and Cushing's Syndrome. Importantly, our results do *not* imply that stress plays no role in the development of these ailments. Instead, our findings indicate that there are no statistically significant differences between fetal and post-natal exposure to maternal stress for these outcomes, which is in contrast with our evidence that *in utero* exposure to severe stress is particularly harmful for mental health.

In sum, our results show that the death of a relative up to four generations apart during pregnancy has far-reaching consequences for physical health at birth and in early childhood, as well as for mental health into adulthood. We argue that these effects are driven by physiological exposure to maternal stress *in utero* and provide evidence against alternative explanations such as changes in maternal behaviors (e.g., smoking) or physical health conditions (e.g., hypertension) that might produce separate insults

to child health. Our findings suggest large general welfare gains of preventing fetal exposure to severe stress: for example, based on the 2008 figure for the U.S. market, the 9 percent decrease in the consumption of prescription drugs treating depression alone can be valued at nearly $1 billion.

While we do not interpret our findings as having sufficient external validity to be generalizable to all other sources of stress, the causal link between antenatal stress and mental disease that we establish points to increasing stress exposure during pregnancy as one potential cause of the rising incidence of certain mental health conditions. Indeed, as we discuss in Section 6, the prevalence of parent-reported ADHD, for example, has been increasing rapidly over the last decades, in the U.S. as well as in Scandinavia (Visser, 2014; Socialstyrelsen, 2012).[5] Over the same time period, stress has been considered one of the fastest growing health problems in the developed world, especially among women of childbearing age.[6] This issue is of particular importance in the U.S. and other developed countries, where female labor force participation has grown steadily over the last several decades, and many women work full-time during pregnancy.[7]

2 Related literature

Our analysis of exposure to stress in the fetal period contributes to a burgeoning literature in economics documenting long-run impacts of early-life shocks (see Almond and Currie, 2011 for a review). However, while there is evidence on the impacts of

[5] While this dramatic rise may be partially explained by changes in the healthcare system's ability to detect and diagnose the condition, it is also widely thought to reflect a true increase in the underlying prevalence of ADHD in the population.

[6] According to a recent survey conducted by the American Psychological Association, nearly 80 percent of Americans agree that "stress is a fact of life," 32 report regularly experiencing extreme levels of stress, and nearly half of respondents report increasing stress over the last five years, with women and respondents aged 18-33 reporting the highest levels and increases. The report is available at: http://www.apa.org/news/press/releases/stress/.

[7] For example, according to the most recent data from the Survey of Income and Program Participation (SIPP), 56 percent of mothers worked full-time during pregnancy in 2006-2008. See http://www.census.gov/prod/2011pubs/p70-128.pdf for more details.

maternal exposure to physical insults during pregnancy stemming from famines, disease outbreaks, and adverse environmental conditions such as radiation and pollution, the evidence is limited on the consequences of purely psychological stressors.[8]

Moreover, the precise mechanisms through which these effects occur are not well understood, and in several prominent theories, stress plays a key role even in driving the adverse impacts of physical stressors. For example, one hypothesis for why malnutrition during pregnancy harms the unborn child is that nutritional restrictions in the mother inhibit the development of a placental enzyme that is required to convert the stress hormone cortisol into inactive cortisone. As a consequence of maternal malnutrition, the fetus is exposed to excessive amounts of cortisol *in utero*. Overexposure to cortisol, in turn, is believed to lead to a reprogramming of the hypothalamic-pituitary-adrenal axis (HPA), which could lead to impaired fetal development and worse health in adult age.[9] This hypothesis suggests that a rigorous analysis of the causal effects of *in utero* exposure to stress can provide new insights on the determinants of health and human capital formation more broadly.

Our focus on stress relates our paper to Aizer et al. (2009), who implement a sibling fixed effects estimation and show that exposure to elevated cortisol *in-utero* adversely affects cognition at age 7 and educational attainment later in life.[10] Researchers have also exploited quasi-exogenous shocks during pregnancy stemming from extreme inci-

[8]The "fetal origins hypothesis", originally put forth by British epidemiologist David J. Barker, argues that poor nutrition *in-utero* "programs" the fetus to have metabolic characteristics that can lead to future disease in adulthood (Barker, 1990). Economists have exploited a variety of shocks to the *in utero* environment to provide some of the most credible causal evidence in support of the hypothesis. See, e.g., Van den Berg, Lindeboom and Portrait (2006); Almond, Edlund, Li and Zhang (2010); Almond and Mazumder (2012); Hoynes, Schanzenbach and Almond (2012); Scholte, van den Berg and Lindeboom (2012) on malnutrition; Almond (2006); Barreca (2010) on disease outbreaks; Almond, Edlund and Palme (2009); Black, Butikofer, Devereux and Salvanes (2013) on radiation; and Sanders (2012); Isen, Rossin-Slater and Walker (2013) on air pollution.

[9]See Dunkel Schetter (2011) as well as a review of the literature in Jaddoe (2006). Also see Appendix C for a more detailed discussion.

[10]Though this design controls for time-invariant differences between mothers that might be correlated with stress, it does not control for time-varying factors that might lead to variation in cortisol levels across pregnancies within the same mother.

dents such as hurricanes, earthquakes, or terrorist attacks.[11] However, one important limitation of these designs is that it is difficult to separate out the effects of stress from the other consequences of these disasters such as the physical health insults or the economic damages. Additionally, as these events are relatively rare, it is often difficult to generalize the findings from these studies to the broader population; in contrast, we focus on a near-universal stressor, family bereavement.

Our analysis of maternal stress due to the loss of a relative makes our paper closely related to the concurrent work of Black, Devereux and Salvanes (2014), who use Norwegian data and analyze the impacts of deaths of maternal parents during pregnancy in a sibling fixed effects framework. They find small adverse effects on birth outcomes and no effects on long-run economic outcomes such as education and earnings. Our work is complementary as we focus on mental health and use a different empirical strategy. Specifically, our analysis does not involve sibling comparisons; instead we quantify the difference in outcomes between individuals who lose a relative during the fetal period and those who suffer the same loss in the months after birth. As such, we aim to disentangle the effect of intrauterine exposure to maternal trauma from any post-natal effects of maternal stress and, potentially, economic shocks or other consequences of a death in the family. In our context, this methodological distinction may also be particularly important because we provide some evidence of maternal fertility responses, suggesting that sibling comparisons may be biased due to the endogeneity of the existence of younger siblings.[12]

[11] Specifically, see the evidence on hurricanes (Simeonova, 2011; Currie and Rossin-Slater, 2013), earthquakes (Tan et al., 2009; Glynn et al., 2001; Torche, 2011), and the terrorist attacks of September 11 (Berkowitz et al., 2003; Lederman et al., 2004; Lauderdale, 2006; Eskenazi et al., 2007).

[12] Additionally, by expanding the number of relatives when considering deaths—in addition to the maternal parents, we include the child's father, siblings, as well as maternal siblings and grandparents—we are also able to create a novel measure of the severity of antenatal stress. Another related paper is Li Jiong and Sorensen (2010), who use Danish data to compare the Body Mass Index (BMI) of children of mothers who experienced a death during pregnancy to children of those who did not. However, an important limitation is that this study does not account for non-random exposure to death.

3 Data

Our analysis leverages administrative population-level data from Sweden. We start from the universe of children born in Sweden in 1973, 1977, 1983, 1988, 1995, 1999, 2001, and 2005. Using data from the *Multigenerational Register (Flergenerationsregistret)*, we identify each child's siblings, parents, grandparents, aunts and uncles, and maternal great-grandparents. We then use the *Cause of Death Register (Dödsorsaksregistret)* to obtain their death dates and causes. We also obtain information about the mothers' and fathers' educational attainment, labor market income, and marital status measured around the time of conception.

Our baseline sample includes all children who experienced the death of a relative (other than the mother) within one year of his or her date of birth. Put differently, our baseline sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within one year of the child's date of birth. Our data include both live births and stillbirths (at 22 weeks gestation or later), allowing us to examine changes to the composition of live births.

To these data, we merge information from several agencies and registers. Because some registers exist for limited time periods, we observe some outcomes for all cohorts of children in the baseline sample and others for only a subset of cohorts.

From the *Medical Birth Register (Medicinskt Födelseregister)*, we get each child's exact date of birth, birth weight, birth length, head circumference, gestation age, and a variety of diagnosis codes at birth. We also have information on the mother's pregnancy and delivery: tobacco use before and during pregnancy, pregnancy risk factors (diabetes, kidney disease, epilepsy, asthma, hypertension, or urinary infection), caesarean section (c-section) delivery (planned or emergency), induction of labor, any complications at delivery, and the number of days spent in the hospital.

To trace health outcomes after birth and throughout life, we add information from the *National Patient Register (Patientregistret)* and the *Drug Register (Läkemedelsregistret)*. These data provide us with information on inpatient and outpatient records, as

well as purchases of prescription drugs. For all of these, we have the universe of records associated with pre-specified health conditions described below. Inpatient records exist from 1964 to 2010, allowing us to capture hospitalizations throughout childhood and young adulthood. Outpatient records are only available for years 2001 to 2010; thus we are only able to observe these visits through age five in our sample. Additionally, our prescription drug data is for years 2005-2012. For each occasion when a prescription drug was bought, the data contain detailed information about the drug name, active substance, average daily dose, and the drug's exact ATC code.[13] The ATC classification allows us to link the drugs to the conditions they are most commonly used to treat.

To select the inpatient, outpatient, and prescription drug records, we pre-specified certain health conditions before undertaking any analysis. First, we include a set of physical health conditions, which, broadly defined, have been linked to stress *in utero* or after birth in the epidemiological and medical literature: type II diabetes, heart disease, Cushing's syndrome, hypo- and hyperthyroidism, neoplasms, and conditions originating in the perinatal period.[14] Second, we include all mental illnesses. In parts of the analysis, we examine eight specific ATC classification subcategories of these disorders: ADHD, depression, anxiety, bipolar disease, psychotic disorders, sleeping disorders, addiction, and Parkinson's disease.[15]

Finally, for the (older) cohorts that we can follow into adult age, we add data on annual taxable income from the *Income and Taxation Register (Registret över inkomster och taxeringar)*, and calculate labor income at age 30. We also obtain information

[13]The Anatomical Therapeutic Chemical (ATC) Classification System is controlled by the World Health Organization Collaborating Centre for Drug Statistics Methodology (WHOCC), and was first published in 1976.

[14]We are grateful to Johannes Haushofer for help in compiling this list. See Appendix B for exact ICD codes for these conditions, as well as ATC codes for prescription drugs that can be linked to their treatment, when possible. Further, Appendix C for details and references relating to the biological mechanisms through which stress affects human health.

[15]As outlined in Appendix B, the in- and outpatient records also include visits related to health outcomes that might be impacted through a behavioral channel: sexually transmitted disease, injury, suicide, and lifestyle issues. These we do not capture through prescription drugs, either because no prescription drug is used, or because no drug can uniquely be linked to their treatment.

about their marital status from the *Register of the Total Population (Registret över totalbefolkningen)*.

In sum, we create a unique data set that enables us to follow the children from the fetal period to their birth, throughout childhood, and into adult life, all the while tracking health status and prescription drug use.

4 Empirical methodology

Our goal is to examine the causal link between antenatal maternal stress and children's physical and mental well-being at birth and later in life. More concretely, for each individual i, let the causal relationship between an outcome of interest, y_i, and stress exposure *in utero* be given by:

$$y_i = \lambda UteroStress_i + \mathbf{x}_i'\delta + e_i, \tag{1}$$

where x_i is a vector of all other relevant determinants of y_i, and e_i is a random vector of predetermined and unobservable characteristics.

We study a particular stressor—the death of a relative. Such a loss is a traumatic event that induces acute and immediate stress. However, the occurrence of death is likely correlated with unobserved family characteristics. For example, some types of accidental deaths are robustly and negatively associated with socio-economic status (Adda et al., 2011). Additionally, this loss may have many consequences for families aside from stress. For instance, a relative's passing may consitute either a financial burden or a source of income through bequests or insurance payouts. A death in the family may lead to a decline in household productivity and necessitate time away from work for the survivors. If a relative's death is due to a hereditary condition, then it may also provide other family members with information about their own genetic makeup, life expectancy, and expected health costs.

In particular, suppose that instead of estimating equation (1), we run:

$$y_i = \gamma RelativeDeath_i + \mathbf{x}_i'\kappa + u_i, \tag{2}$$

Then, under the assumption of additive separability of the impact of *in utero* stress exposure, we have that:

$$RelativeDeath_i = \alpha_1 UteroStress_i^* \mathbf{1}[c \leq RelativeDeath < b] + \alpha_2 Other_i + \varepsilon_i, \tag{3}$$

where $UteroStress_i$ represents intrauterine exposure to the physiological stress experienced by the mother, and $Other_i$ captures all other consequences and correlates of family bereavement, including shocks to family income, changes to the mother's work schedule, changes to the mother's information regarding her own health status, and any family characteristics that make death more likely. Note that c denotes the child's date of conception, and b denotes the child's date of birth, with $\mathbf{1}[c \leq RelativeDeath < b]$ indicating that the relative's death happened during pregnancy. Thus, in equation (2), γ identifies the combined impact of all these factors associated with the passing of a relative.

Note, however, that equations (2) and (3) show that children whose mothers experience a death shortly after giving birth face the same income shocks and other consequences as the children whose mothers experience a death during pregnancy. But unlike the children who are *in utero* when the death occurs, the former group does not have intrauterine exposure to the physiological stress experienced by the mother.

Now, consider a sample of children who either experience the death of a relative during gestation or in the year after birth such that:

$$S = \{i : \mathbf{1}[c \leq RelativeDeath < b]_i = 1 \,|\mathbf{1}[b \leq RelativeDeath < b + 365]_i = 1\}.$$

For all $i \in \{S\}$, suppose we now estimate:

$$y_i = \sigma \mathbf{1}[c \leq RelativeDeath < b] + \mathbf{x}_i'\eta + \epsilon_i, \tag{4}$$

where all of the variables are defined as above.

We have that:

$$E(y_i|c \leq RelativeDeath < b) = \zeta_1 UteroStress_i + \zeta_2 Other_i + \mathbf{x}_i'\eta$$
$$E(y_i|b \leq RelativeDeath < b + 365) = \zeta_2 Other_i + \mathbf{x}_i'\eta$$

and hence we obtain that

$$E(y_i|c \leq RelativeDeath < b) - E(y_i|b \leq RelativeDeath < b + 365) = \zeta_1 UteroStress_i.$$

Here, ζ_1 captures the impact of intrauterine stress alone and *not* the entire effect of bereavement. Empirically, under the assumption of additive separability and as long as we have captured a true causal effect (i.e., for all $i \in \{S\}$, $E(\mathbf{1}[c \leq RelativeDeath < b]\epsilon_i) = 0$), ζ_1 can be estimated with σ in equation (4) on the sample with $i \in \{S\}$.

However, as discussed further below, we find that exposure to the death of a relative *in utero* reduces gestational age. Since the key treatment variable in equation (4), $\mathbf{1}[c \leq RelativeDeath < b]$, is defined based on the child's actual birth date, b, we face a violation of the excludability restriction. Moreover, there is a mechanical correlation between the length of the pregnancy and the likelihood that the death occurs during it.[16]

To address these issues, we adjust our treatment variable by defining it relative to the *expected* date of birth at full term instead of the actual date of birth. More precisely, we define a child's estimated date of birth as $e_b = c + 280$, that is, 280 days (40 weeks) after the date of conception, c. Unlike the actual date of birth, this expected

[16] See Currie and Rossin-Slater (2013) and Black, Devereux and Salvanes (2014) for more discussion of these issues.

date of birth is pre-determined at the relative's death date.

Consequently, instead of estimating equation (4), we estimate the following equation on the sample with $i \in \{S\}$:

$$y_{iymp} = \beta_0 + \beta_1 \mathbf{1}[c \leq RelativeDeath < e_b]_{iymp} + \psi_y + \phi_m + \rho_p + \mathbf{x'}_i\beta_{\mathbf{3}} + \nu_{iymp}, \quad (5)$$

where $\mathbf{1}[c \leq RelativeDeath < e_b]_{iymp}$ captures "treatment": a discontinuous variable that takes the value of 1 if the relative's death occurs before the child's estimated date of birth at full term, and 0 otherwise. Intuitively, our empirical strategy exploits a discontinuity around the threshold of 280 days after conception, and assigns a child to intrauterine stress exposure if the relative's death occurred before this date.

In this model, y_{iymp} is an outcome of individual i, conceived in year and month (y, m), with a mother residing in municipality p in the year before conception. ψ_y and ϕ_m are year and month of conception fixed effects, respectively, and ρ_p are pre-conception municipality fixed effects. Further, $\mathbf{x}_i$ is a vector of variables capturing mother- and child-specific characteristics, including indicator variables for mother's age at childbirth (five categories: < 20, $20 - 24$, $25 - 34$, > 35), mother's education in the year prior to conception (four categories: <HS, HS diploma, some college, college+), indicator variables for the mother being born outside of Sweden and being married in the year prior to conception, and dummies for parity (three categories: 1, 2, 3+). Additionally, $\mathbf{x}_i$ includes the relative's age and age squared at the time of death. Standard errors are clustered on the mother's municipality of residence in the year prior to conception. Under the identifying assumption discussed below, the estimate of interest, $\hat{\beta}_1$, captures the causal impact of exposure to maternal stress through the uterine environment.

In parts of our analysis, we also analyze trimester-specific impacts, replacing $\mathbf{1}[c \leq RelativeDeath < e_b]_{iymp}$ with three indicator variables capturing whether the death occurred in the expected first, second, or third trimester, respectively.

Notably, by comparing individuals who experience a stressful shock during gestation with those who experience such a shock shortly after birth, our analysis has a distinct advantage over the existing studies in this literature that rely on exposure to war or other disasters. These studies cannot rule out that the documented effects on adult outcomes arise from post-natal differences that were induced by the events that occurred during pregnancy, rather than by the differences in the uterine environment. A compelling feature of our methodology is that our estimates are not contaminated by such post-natal effects—these effects are borne by all children in our sample, while only the treatment group is exposed to maternal trauma *in utero*.

Identifying Assumption This methodology yields an estimate of the causal effect of antenatal maternal stress under the identifying assumption that the exact timing of death within a short time frame around the expected date of birth is uncorrelated with unobserved characteristics of the child or family. Put differently, we assume that there is no selection on unobservables into treatment, where treatment is defined as experiencing death during the first 40 weeks (280 days) after conception.

While less restrictive than assuming no selection into death *per se*, the assumption is nonetheless not innocuous. We therefore subject it to several "plausibility tests," since the exact assumption is inherently untestable. First, we test whether selection into treatment is correlated with a range of characteristics of the mother that are observed prior to conception: the mother's age, first parity birth, the mother's marital status, the mother's educational attainment being below high school, the mother's educational attainment being a college degree or higher, the mother's wage income, and the mother being born outside Sweden. As shown in Appendix Table A1, we do not find any statistically significant correlation except for one small positive relationship between death during pregnancy and the likelihood of the mother being college-educated. These results suggest that if anything, any selection into the timing of death would bias as against finding any adverse effects on child outcomes.

As another way of testing for a spurious correlation between maternal characteristics and the timing of relative death, Appendix Table A2 presents results from specifications that regress our key explanatory variable (an indicator for a death occurring during pregnancy) on the vector of controls included in our models. Column 1 only includes covariates contained in $\mathbf{x}_i$, while Column 2 adds fixed effects for the year and month of conception, as well as the mother's municipality of residence in the year before conception. The results show that most of the controls included in our models do not predict the timing of bereavement. As in Appendix Table A1, we find that more educated mothers are slightly more likely to experience a death during pregnancy relative to their less educated counterparts. However, based on a joint F-test of significance of these covariates, we cannot reject the null hypothesis that all of them are equal zero.

These results are reassuring as they suggest that the timing of a family member's death in relation to the child's expected date of birth is uncorrelated with a variety of maternal and family characteristics. Nevertheless, we also examine the robustness of our results to limitations in types of death causes that have been shown to be more exogenous than others; see Section 5 for details.

In addition to these efforts, several features of our particular empirical setting help assuage potential concerns with violations of the identifying assumption. First, we do not only observe the child's date of birth, but also the child's gestation length. As described above, we do not define treatment relative to the child's actual date of birth, but instead relative to the *expected* date of birth at full term. This date is determined at conception, and hence pre-determined at the time of family rupture. If, in contrast, the child's birth were to affect the probability that a family experiences a death, then this would plausibly occur at the actual birth date and not at the expected one. Second, the extremely rich data implies that the set of unobserved characteristics—and hence the set of characteristics for which a correlation with treatment would be of concern—is very small (although of course non-empty).

Sample and Summary Statistics To estimate each child's date of conception, c, we subtract the number of gestation days from the date of birth, b. As described above, we define the set of treated individuals as those experiencing the death of a relative during the 40 weeks after conception (i.e., in days, the time interval of $[c, c+280]$). Our comparison group includes all children who experience a relative death at any point between the estimated date of birth and one year after their actual birth date.

Table I presents summary statistics. The sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within 280 days after the child's estimated date of conception or in the year after birth.[17] Column one displays statistics for the full sample, while the second and third columns consider the treatment and comparison groups separately. In our sample, mean maternal age at childbirth is about 28 years, and about 32.2 percent of mothers are married in the year prior to conception. Most mothers have a high school degree in the year before conception. Average birth weight is $3,557$ grams, with 3 percent of children born low-birth-weight (less than 2,500 grams) and 5 percent of children born pre-term (less than 37 weeks gestation). Notably, the maternal characteristics are quite similar across the treatment and comparison groups. However, even this simple unadjusted comparison shows that treatment children tend to have slightly worse birth outcomes relative to the comparison group. We next explore the differences between the outcomes of the two groups more rigorously using the methods described above.

5 Results

We present results in chronological order. We start with the analysis of birth outcomes, following with a study of physical and mental health throughout childhood and into adulthood, and then finally examine some measures of adult labor market and marital

[17] We drop all individuals who had a relative death occur prior to conception to avoid issues related to any confounding effects on fertility.

outcomes. We also present some additional results that examine the possibility of alternative explanations besides stress in our analyses, and that test the robustness of our main findings.

5.1 Birth Outcomes

Table II presents the results on the effects of exposure to a relative death *in utero* on average birth weight, indicators for low-birth-weight, very-low-birth-weight (less than 1,500 grams), and high-birth-weight (more than 4,000 grams) births, as well as indicators for a pre-term birth, a stillbirth (at 22 weeks gestation or more), and a perinatal death (stillbirth or a death occuring in the first 28 days of life). In Table III, we report results for additional outcomes: indicators for small-for-gestational-age (SGA) and large-for-gestational-age (LGA), birth length and head circumference (in centimeters), indicators for procedures at delivery (c-section, induction of labor), and an indicator for any ruptures during delivery. All of our analyses include the vector $\mathbf{x}_i$ described above, as well as fixed effects for the year and month of conception and the mother's municipality of residence in the year prior to conception.

Our estimates suggest that *in utero* stress due to family bereavement leads to a small negative effect on average birth weight of 18 grams. However, much of this effect is driven by impacts at the lower end of the birth weight distribution. Prenatally exposed infants are 20 percent more likely to be born low-birth-weight, 30 percent more likely to be born very-low-birth-weight, and 10 percent more likely to be SGA. In contrast, there is only a 4 percent decline in the likelihood of a high-birth-weight birth and no effect on the likelihood of being LGA. These children are also 15 percent more likely to be born pre-term, and the mothers are about 4 percent more likely to have a c-section delivery (although this latter coefficient is only marginally significant at the 10 percent level). We find no statistically significant effects on stillbirths or deaths in the first 28 days of life.

In Figures I and II, we examine how the effect varies with the timing of the death

relative to the expected date of birth. As outcomes, we focus on low-birth-weight in Figure I and on preterm births in Figure II. In these graphs, we plot the coefficients on the effects of the death of a relative during the (expected) 1st, 2nd, and 3rd trimesters of pregnancy, along with 95% confidence intervals. Note that the omitted category is a death occuring after 280 days (40 weeks) of gestation (i.e., post-childbirth in most cases).

Both figures show positive coefficients on exposure to stress during each trimester of pregnancy relative to post-partum, with slightly higher effects during the first trimester. In Table IV and Appendix Table A3 we display trimester-specific effects on the other birth outcomes as well. In general, the effects tend to be similar across the trimesters, although the impacts on birth weight and head circumference seem to be largest when exposure is in the first trimester.[18]

5.2 Physical Health Outcomes Beyond Birth

Having documented that stress *in utero* adversely impacts health at birth, we turn to the analysis of physical health measures later in life. First, we examine the effects on the occurrence of hospitalizations and outpatient visits by certain ages. Our inpatient data exist for years 1964 to 2010 and thus allow us to study hospitalizations into adulthood. Our outpatient data are only available for years 2001 to 2010, and thus we are limited to studying these outcomes through age five.

Table V presents results on the effects of *in utero* exposure to relative death on child hospitalizations by ages one and five. We find that *in utero* stress is associated with an 8 percent increase in the likelihood that a child is ever hospitalized by age one, and a 4

[18]The trimester results also show a reduction in stillbirths, which was not statistically significant when we looked at exposure during the whole pregnancy. This protective effect on stillbirths might suggest that stress impacts the timing of fetal loss; it may be that the decrease in stillbirths is driven by an increase in earlier miscarriages which we do not observe. Note that we have also followed several papers in this literature and examined the sex ratio as a signal of changes to miscarriage rates (e.g., Sanders and Stoecker, 2011; Halla and Zweimüller, 2013). Since male fetuses are more likely to miscarry, a reduction in male births may indicate an increase in miscarriages. However, we do not find statistically significant effects on this outcome.

percent increase in the likelihood he/she is ever hospitalized by age five. In Table VI we show that there are similar increases in outpatient visits occurring at these ages. We explored in detail the diagnoses codes to try to understand which causes are driving these results and found that they are primarily driven by treatments for conditions originating in the perinatal period, as seen in Table VII.[19] In Figures III and IV, and Appendix Tables A4 and A5, we also present the results by trimester of pregnancy and find that these health effects are primarily driven by exposure in the first trimester. Note, again, that since the omitted category in these figures and regressions is family bereavement after 40 weeks of gestation, we are estimating the effects of stress during different points of the pregnancy *relative to the effect of stress post-partum*.

Next, we turn to the Drug Register data. As described in Section 3, these data contain information about prescription drugs bought during 2005-2012. To maximize our sample size, we do not make any age restrictions on these analyses and simply study the consumption of prescription drugs at any age observable in our data. This means that, for example, the 1973 cohort is observed at ages 32 to 39, while the 2001 cohort is observed at ages 4 to 11. While prescription drug use is certainly not the same across these age groups, these differences are controlled for by the inclusion of conception year fixed effects in our regressions.

Appendix Table A6 presents results on the effects of *in utero* stress on the consumption of drugs used to treat the following three health conditions: obesity, diabetes, and Cushing's Syndrome. As discussed in Section 3, the analysis of these particular ailments is motivated by the epidemiological and medical literature. Appendix Section B provides the exact ATC codes employed to associate prescription drugs to diagnoses.

[19] We use the entire set of perinatal conditions, which include all conditions with ICD-10 codes in the range P00-P96. These include the following conditions: 1) Fetus and newborn affected by maternal factors and by complications of pregnancy, labour and delivery, 2) Disorders related to length of gestation and fetal growth, 3) Birth trauma, 4) Respiratory and cardiovascular disorders specific to the perinatal period, 5) Infections specific to the perinatal period, 6) Haemorrhagic and haematological disorders of fetus and newborn, 7) Transitory endocrine and metabolic disorders specific to fetus and newborn, 8) Digestive system disorders of fetus and newborn, 9) Conditions involving the integument and temperature regulation of fetus and newborn, 10) Other disorders originating in the perinatal period.

For each set of drugs we consider two outcomes: an indicator for ever purchasing the drug and the average daily dose.

We find no statistically significant effects of exposure to a relative death during pregnancy on these outcomes. Note our results do *not* imply that stress plays no role in the onset of these conditions—instead, our estimates suggest that stress exposure during the fetal period is not statistically different from stress exposure shortly post-birth in determining these illnesses.

On the whole, our physical health results suggest that the adverse consequences of fetal stress exposure last beyond birth and impact child health through age five. However, the physical health effects seem to fade after early childhood—we find no effects on hospitalizations at later ages (see Appendix Table A7, which shows no significant effects on hospitalizations by ages 10, 18, or 27) or on the onset of conditions such as obesity, diabetes, and Cushing's Syndrome in later life.

5.3 Mental Health Outcomes

Thus far, we have shown that exposure to a stressful event *in utero* has negative consequences for child physical health in early childhood, and that these effects fade away as the children age. We next use the Drug Register data to analyze effects on mental health.

Table VIII presents the results on the effects of family bereavement during gestation on the purchases of prescription drugs commonly used to treat some of the mental health conditions described in Section 3. In these specifications, we find no effects except for a 10 percent increase in the average daily dose of depression medications (results for the other subcategories of mental health conditions are similar, and available upon request).

However, our empirical approach allows us to test whether the effects are different depending on the severity of the stressful event. In particular, the stress associated with the loss of a family member may be greater when this family member is closer to

the mother on the family tree. Thus, to examine more severe stress, we focus on deaths of close relatives of mothers: a sibling, a parent, the child's father, or an own (older) child. In other words, we drop mothers who experience the death of a grandparent.

Table IX presents the results from these regressions, and shows statistically significant increases in the consumption of drugs used to treat mental health conditions. Specifically, there is a 6 percent increase in the likelihood of ever purchasing a drug for any mental health condition. This effect is driven by increases in the purchases of drugs treating anxiety and depression (11 and 9 percent increases, respectively). We also see a 20 percent increase in the average daily dose of drugs for depression. Additionally, while we do not see effects for ADHD on the extensive margin in terms of ever purchasing drugs, we do see a 23 percent increase in the average daily dose.[20] Figure V suggests that these mental health effects are fairly similar across the three trimesters of exposure.

These results indicate that the adverse mental health impacts of exposure to stress *in utero* are stronger when the stress is more severe. In contrast, we find that the "close relative" specifications for our physical health outcomes (birth outcomes, hospitalizations, outpatient visits, and prescriptions for obesity, diabetes, and Cushing's Syndrome) are very similar to the main results presented above.[21] Thus, our results imply that physical health impacts may be less sensitive to the severity of the *in utero* shock relative to the mental health consequences.

Moreover, while the physical health effects seem to diminish as children age, Appendix Table A8 shows that the mental health consequences of severe stress exposure persist into adulthood. In this table, we further limit our analysis to cohorts born in 1973-1988, who are only observed at ages 17-39 in the Drug Register data, and show that the mental health impacts on anxiety and depression are also present for these individuals.

[20] The impact on ADHD is driven by prescription drugs with the active substance methylphenidate, with trade names in the U.S. such as Concerta, Methylin, Ritalin, and Equasym XL, which in Sweden is consumed by 89 percent of all individuals using any prescription drug treating ADHD.

[21] Results available upon request.

Finally, we examine the mechanism through which a death of one of the mother's close relatives may induce greater stress than the death of one of her grandparents. In particular, our measure of stress severity may capture two distinct components. On the one hand, mothers may have closer and more intimate relationships with their parents, siblings, spouses/partners, and children than with their grandparents. Consequently, the passing of the more immediate family member may induce more mourning than the death of a grandparent. Alternatively, the death of a younger relative may simply be more shocking than the death of a grandparent. More precisely, if the prior on the likelihood of a relative's death increases with the relative's age, then the advent of death constitutes a larger deviation from the prior when the deceased relative is younger. In Appendix Table A9, we try to distinguish between these two factors by examining heterogeneity in mental health effects by the relative's age within the "close relative" sample. In these regressions, we include an interaction with an indicator for the relative being younger than 50 years at the time of death. The interaction coefficients are almost all insignificant, and suggest that, if anything, younger relative deaths lead to fewer prescription drug purchases.[22] This suggests that our severity of stress measure captures the strength of the family tie being broken, rather than the deviation from the prior on the probability of the relative dying.

On the whole, these results suggest that experiencing a comparatively stressful event *in utero* is more deleterious for mental health than experiencing such an event shortly post-birth. This finding is consistent with recent neuroscientific research tracing the origins of depression and autism-spectrum diseases such as ADHD to the fetal period (see, for example, Liu et al., 2012 for depression and Berquin et al., 1998; Stoner et al., 2014 for ADHD and other autism-spectrum related illness, as well as the references cited therein).

A related issue is whether these adverse mental health effects are consequences of the physical health insults that we document at birth, or whether there exist separate

[22]We have also estimated regressions interacting with continuous relative's age, and obtained similar results.

effects of intrauterine stress exposure. Indeed, a key feature of the "fetal origins hypothesis" is the possibility of latent health impacts that do not materialize until later life (Barker, 1990). While it is inherently hard to distinguish between these mechanisms, one way to potentially shed some light on this question is to benchmark our effects to previously published estimates of the correlation between birth weight and the mental health conditions we study.

For example, according to Colman et al. (2007), a one standard deviation increase in birth weight is associated with a 0.08 percentage point reduction in the likelihood of suffering from depression or anxiety in adulthood. Our sample has a 563 gram standard deviation in birth weight; thus, our estimated 18 gram decrease in birth weight corresponds to 0.03 standard deviations. A back-of-the-envelope calculation suggests that, if the entire effect on mental health were to operate through birth weight, then we would expect a $0.03 * 0.08 = 0.0024$ effect on the take-up of prescription drugs. In contrast, our estimates are over four times larger, suggesting 0.008 and 0.009 increases in the take-up of drugs treating anxiety and depression, respectively. Of course, this calculation relies on strong assumptions, including that the correlation in Colman et al. (2007) based on a British sample of the 1946 cohort is applicable to our context in Sweden, and that the relationship between mental health and birth weight is linear. Nevertheless, our calculation is at least suggestive that intrauterine stress exposure has distinct effects on mental health that are separate from its impacts on physical health at birth.

5.4 Labor Market and Marital Outcomes

After documenting some adverse physical and mental health effects of *in utero* stress exposure, we would ideally like to assess whether they translate into impacts on other measures of adult well-being, such as earnings and marital stability. Unfortunately, we observe these outcomes imperfectly.

First, we observe annual earnings during 1990-2010. In Sweden, the average age of

completion of the first university degree is 30, which is several years higher than the OECD average.[23] Stable employment is therefore best captured starting around age 30. Thus, although reducing our sample size substantially and sacrificing power, we use data from 2003 and 2007 to measure age-30 earnings for our 1973 and 1977 cohorts. We have two earnings measures, both of which include all employer-reported income exceeding SEK 100 ($15). However, because employers in Sweden also pay (out) sick leave compensation (i.e., disability income), none of our two income measures perfectly captures individual earnings from participation in the labor force.[24]

The fact that our earnings measures include disability income renders us unable to detect many transitions out of work and into disability. This is particularly unfortunate because eligibility for sick leave is determined not only by physical ailments and disabilities, but also by depression and other mental health issues, which are precisely the conditions that we find to be impacted by prenatal stress.[25]

Second, we also observe yearly marital status during 1964-2010. We do not make any age restrictions on these variables and simply study indicators for ever being married and ever being divorced in our data. In Sweden, the average age at first marriage was 32.9 years for women and 35.5 years for men in 2010 (Statistics Sweden, 2011), and above 29 for both genders since the beginning of the 1980s (Persson, 2014). As

[23] See OECD (2013) for comparisons across OECD countries.

[24] Specifically, before December 1992, employers paid the first two weeks of sick leave, after which the employee started claiming benefits from *Försäkringskassan* (equivalent of the Social Security Adminstration). In the case of multiple periods of sick leave, the employer paid for sick leave up to 14 days so long as the employee returned to work for at least one day in between. After 1992, employers also paid out compensation beyond the first 14 days, but later claimed this from the Social Security Administration (Statistics Sweden, 2005; Försäkringskassan, 2013). Our earnings measure *wage income* thus captures all these transfers. Our second earnings measure, *labor income*, also includes parental leave transfers, disability transfers made from the government, and other taxable social insurance payments that are indexed by earnings.

[25] All individuals working in Sweden are eligible for sick leave. To receive benefits, an individual must provide a doctor's certificate by the eighth day of employer's sick leave payment. A certificate must confirm that the individual suffers from a condition that renders her unable to perform regular duties. If the individual cannot perform regular duties, or duties compensated at equal pay, but the individual can perform duties at a lower pay scale, then the employer may not reallocate the individual to those lesser activities, but must pay sick leave benefits. Mental illness such as depression grants the right to sick leave when the ailment reduces the individual's ability to perform regular work duties. See Statistics Sweden (2005) for more information.

such, since we only observe our two oldest cohorts into their 30s, the analysis of these outcomes is limited in both power and relevance.

For completeness, we nonetheless present results on the impact of prenatal stress on age-30 earnings and on the marital outcomes described above. Table X shows that we do not find any impacts on these outcomes. However, for the above-mentioned reasons, we are hesitant to conclude that these outcomes are unaffected by prenatal stress.[26]

5.5 Alternative Channels

Thus far, we have argued that the adverse physical and mental health consequences of family bereavement *in utero* are driven by physiological exposure to maternal stress. In particular, as discussed in detail in Section 4, we posit that the other consequences of a death in the family are netted out when our comparison group consists of children who experience such a death in the year after birth. Additionally, we argue that the severity of stress exposure (as measured by the strength of the family tie that is broken) is important for affecting child mental health. However, our method leaves room for some alternative explanations, which we discuss here.

Maternal Behaviors and Physical Conditions First, it is possible that a fetus is not affected by the stress on its own, but rather by a maternal behavior or physical health condition during pregnancy that is induced by stress. For example, if a woman responds to a stressful event by taking up smoking or developing hypertension, then her child might be affected through these channels. In Appendix Table A10, we examine this potential mechanism in more detail. Specifically, we study whether *in utero* stress exposure is associated with maternal smoking (during pregnancy and regularly) or with the presence of "high-risk" factors. These include the following conditions

[26]For a few of our cohorts, we do have information on 3rd grade math test scores, as well as 9th grade math and English test scores and GPA. However, these measures have been changed over time and are not easily standardized across cohorts. Additionally, we are limited by power due to the small sample sizes in these analyses. We find no statistically significant effects on these outcomes (results available upon request).

during pregnancy: diabetes, kidney disease, epilepsy, asthma, hypertension, or urinary infection. We find no effects on these outcomes, suggesting that our results on child physical and mental health are likely not driven by changes in maternal behaviors or physical conditions.

Differences in Maternal Reactions to Stress Second, the mother's own mental health may respond differently to a stressful event that occurs during pregnancy than to an event occurring after giving birth. For example, relative to pregnant women, mothers of infants may, on the one hand, be less vulnerable as they can divert their attention toward childrearing; on the other hand, mothers of newborns may be prone to post-partum depression, or generally be more sensitive to additional stressors. In Appendix Table A11, we try to examine the plausibility of this mechanism by studying *maternal* mental health outcomes as measured by our prescription variables. We find no evidence that experiencing a close relative's death during pregnancy has a differential effect on maternal mental health relative to experiencing such a death post-childbirth.[27] Thus, our results suggest that the adverse effects of *in utero* exposure to family bereavement are not driven by differences in maternal experiences of the event between pregnancy and post-childbirth, but rather signify the critical nature of the fetal period in propagating the effects of stress.

Differential Income Shocks Third, it may be the case that any income shocks associated with the death of a family member affect the child differently depending on whether the loss occurs during pregnancy or if it happens shortly after childbirth. In the notation of our framework presented in Section 4, this possibility would entail including an extra interaction effect, $UteroStress_i^* \mathbf{1}[c \leq RelativeDeath < b]^* Income_i$ in equation (3), and assuming that this term is additively separable from any other income effects. Then, our estimates would capture both the effect of physiological exposure to maternal stress and the differential impact of income during pregnancy

[27] We also examined all other mental health conditions and found no effects.

relative to post-partum.

This issue is most relevant for income shocks that affect families immediately following the death of a relative—for example, funeral expenses. However, in Sweden, 90% of all estates can fully cover the funeral expenses, and then also leave some inheritance to the surviving relatives (Erixson and Ohlsson, 2014). Therefore, this channel is likely not very relevant in our context.

Moreover, relative to other countries such as the U.S., income shocks—and hence their precise timing—likely matter less in Sweden due to the extensive social security and benefits system. For example, reductions in income should not affect the likelihood that a woman receives prenatal care due to the existence of universal health insurance coverage. In Appendix Table A12, we also present some indirect evidence that differential income effects are likely unimportant in our context. In particular, if income effects were to matter *in utero*, then we would expect them to matter more for lower-income families, which would translate into heterogeneous treatment effects with respect to the socio-economic status of the mother. Appendix Table A12 shows the results from regressions that interact our treatment variable with an indicator for the mother having a high school degree or less at the time of conception. We find no evidence of heterogeneous effects along this margin for our main outcomes of interest.

Inheritances and the Severity of Stress Fourth, we find that adverse mental health effects arise when the deceased is a close relative of the expectant mother (an own older child, a spouse/partner, a parent, or a sibling), but not when the relative is more distant (a grandparent). As discussed above, we interpret this difference as resulting from varying degrees of emotional stress associated with the relative's passing. An alternative interpretation is that the adverse effects are equal, but that a grandparent's death entails a larger income transfer to the family than the death of other relatives. Such an income effect could assuage any adverse effects of stress associated with the passing of a grandparent.

To shed light on this alternative interpretation, three sources of income are relevant: bequests, generation-skipping transfers, and life insurance payouts. Table XI displays these three sources of income following the death of a relative from a deceased spouse, parent, and grandparent, respectively, for the universe of deaths in Sweden occurring from 2002 to 2005.[28] The three leftmost columns display the average amount in Swedish Krona (SEK) in each class of recipients, i.e., *not* the average amount conditional on the amount received being greater than zero. The rightmost column displays the sum of the three income classes.

Column 1 shows the average amount received as inheritance following the death of a relative: SEK 280,000 ($42,562) from a spouse, SEK 30,000 ($4,560) from a parent, and SEK 7,000 ($1,064) from a grandparent.[29] The second relevant possibility to receive income in conjunction with a grandparent's passing is through a generation-skipping transfer. Column 2 shows that the unconditional mean of the generation-skipping transfer to grandchildren is SEK 32,000 ($4,864), an amount roughly similar to the unconditional average inheritance from a parent. While these numbers are averages based on the entire population rather than our sample alone, and while inheritances and generation-skipping transfers only occur for a strict subset of all deaths, these statistics indicate that inheritances and generation-skipping transfers together are likely not much larger when a grandparent dies than when a parent dies. Losing a spouse, however, entails a substantially larger amount.[30] Finally, column 3 shows that insurance payouts are small and uncommon. Together these facts suggest that losing a grandparent does not entail a larger positive income effect than losing other (closer)

[28] We display average amounts for the universe of deaths in Sweden—and not only for our sample—because the bequest data are not linked to our dataset. Moreover, bequests data exist for the years 2002 to 2005 only.

[29] Inheritance from a parent is far more common than inheritance from a grandparent. This is understandable in light of the fact that, in the absence of a will, an individual only inherits from her grandparent if her own parents are deceased. Moreover, less than 20 percent of all deceased in Sweden write a will; further, writing a will only enables transfer of 50% of the assets, while the remainder must be allocated according to the above-mentioned inheritance rules. These amounts presented in the table, however, represent averages across all spouses, children, or grandchildren of all deceased individuals, i.e., the table displays the unconditional amounts.

[30] Losing a spouse likely also entails larger costs, in particular through foregone household income.

relatives.

5.6 Additional Results

Overall, our findings point to important physical and mental health consequences of exposure to stress *in utero*. This section presents some additional results that test the robustness of our main findings.

"Exogenous" Deaths The reliability of our results rests on the assumption that the timing of relative death within a narrow time frame surrounding the expected date of birth is uncorrelated with other factors that might affect child outcomes. We have already shown that this timing is generally uncorrelated with a variety of observable maternal characteristics. Now, we also explore the sensitivity of our findings to sample limitations based on causes of death that are determined to be more exogenous than others.

More specifically, we turn to the work of Adda et al. (2011), who study the effect of parental death around age 18 on children's educational and labor market outcomes. To find plausibly exogenous causes of deaths, Adda et al. (2011) test for a placebo correlation between a death occurring after an outcome is determined. So, for example, a death occurring shortly after age 18 cannot affect scores on a cognitive test taken at a younger age. They determine that the following causes of death pass this exogeneity test: endocrine and metabolic diseases, accidents, and other causes.[31] Appendix Tables A13, A14, A15, and A16 replicate our main findings limiting the sample to only these three causes of death. Although we lose some power with the sample size reductions, the results are qualitatively very similar to the main ones presented above.[32]

[31] Other causes are all causes except infectious and parasitic disease, neoplasms, endocrine and metabolic diseases, mental and behavioral disorders, circulatory system, respiratory system, digestive system, accidents, suicides and homicides.

[32] We unfortunately cannot replicate the method used by Adda et al. (2011) to determine which causes of death are exogenous in our sample. To do this, one needs to have a comparison group of children who do not experience a relative death surrounding the time of their birth. With such a comparison group, one is able to test for placebo effects of deaths occuring shortly after birth on

Adjusting for Multiple Hypothesis Testing Another important concern for our analysis is that we may find spurious effects due to the number of outcomes we consider. To address this issue, we follow Kling, Liebman and Katz (2007) and create two outcome indices: one for physical health and one for mental health. Specifically, the physical health index consists of all the outcomes analyzed in Tables II, III, V, VI, VII, and Appendix Table A6: continuous birth weight, low-birth-weight indicator, very-low-birth-weight indicator, high-birth-weight indicator, pre-term indicator, still-birth indicator, perinatal death indicator, SGA indicator, LGA indicator, birth length, head circumference, c-section indicator, induced labor indicator, any ruptures indicator, any and total hospitalizations by ages 1 and 5, any and total outpatient visits by ages 1 and 5, any and total hospitalizations for perinatal causes by ages 1 and 5, any medications for obesity or diabetes or Cushing's Syndrome, any medications for obesity, average dose for obesity medication, any medication for diabetes, average dose for diabetes medication, any medication for Cushing's Syndrome, average dose for Cushing's Syndrome medication. The mental health index consists of an indicator for ever purchasing a mental health drug, as well as 16 other outcomes comprised of our two measures—an indicator for every purchasing the drug and the average daily dose—per condition (ADHD, anxiety, bipolar disorder, depression, psychotic disorders, addiction, and sleep disorders).

To create the indices, we first orient each outcome such that a higher value represents a better outcome (e.g., the indicator for low-birth-weight is inversed such that we instead consider an indicator for *not* being low-birth-weight). Then, we standardize each oriented outcome by subtracting the comparison group mean and dividing by the comparison group standard deviation. Finally, we take an equally weighted average of the standardized outcomes.

Table XII presents the results from our main specifications using the two indices as outcomes. We show results for all deaths as well as the close relative deaths for which

birth outcomes, for example. However, our sample only contains individuals who experience a relative death in the year surrounding their birth date, and thus we cannot conduct these tests.

we saw mental health effects. Just like our main results, these estimates suggest that physical health is adversely affected by exposure to any relative death *in utero*. Mental health is also impacted, but only in the case of severe stress, as measured by the death of a closer relative.

Maternal Responses to *In Utero* Shocks: Effects on Subsequent Fertility Finally, as our data allow us to observe all births by the mothers in our sample (i.e., we observe all siblings of the sample children), we can study whether our *in utero* shock of interest is correlated with an important maternal behavioral response: fertility. In fact, in recent work studying parental responses to fetal exposure to the Chernobyl accident, Halla and Zweimüller (2013) find that low-education Austrian mothers in high radiation fallout areas during pregnancy reduce their subsequent fertility. They argue that this is a form of compensating behavior because doing so allows mothers to allocate more resources to the affected children.

We examine this behavior in Appendix Table A17, which shows that women who experience a relative death during pregnancy are more likely to have a subsequent child in our data. Since some women in our sample have not yet completed their childbearing years, this effect could be driven by a retiming of births rather than an increase in lifetime fertility. Nevertheless, our findings suggest that, unlike Austrian mothers in the context of Chernobyl, the mothers in our data do not invoke the "quantity-quality" trade-off. If anything, we find evidence of reinforcing behavior, consistent with some other work on this topic (see Almond and Mazumder, 2013).

Additionally, just like Halla and Zweimüller (2013), our analysis suggests caution in the interpretation of estimates from sibling fixed effects designs. The possibility of endogenous subsequent fertility suggests that comparisons of treated children with younger siblings could be biased. Even if there are no spillover effects on other (older) family members, comparing treated children only to their older siblings would still be problematic as it is then difficult to separately identify treatment effects from the

effects of birth order.

6 Conclusion

This paper analyzes whether the uterine environment propagates the impact of stress across generations. We exploit multigenerational registers in Sweden to create family trees that span four generations, and study how ruptures of family ties during pregnancy affect the unborn child. Unlike other studies of shocks to the prenatal environment, our empirical strategy isolates the effect of physiological fetal exposure to stress by comparing the outcomes of children whose relatives die while they are *in utero* to those whose relatives die in the year after birth. Additionally, by studying family bereavement instead of other shocks such as disasters and wars, we present evidence on exposure to a very universal stressor.

We find that *in utero* exposure to the death of a relative up to four generations apart has far-reaching consequences for physical health at birth and in early childhood. We also provide novel evidence that severe antenatal stress—as measured by bereavement of younger and closer family members—has causal impacts on the onset of psychological conditions including anxiety, depression, and ADHD, which last into adulthood. Our findings suggest large general welfare gains of preventing fetal exposure to severe stress: for example, based on the 2008 figure for the U.S. market, the 9 percent decrease in the consumption of prescription drugs treating depression alone can be valued at nearly $1 billion.

While our findings do not necessarily have external validity to all other sources of stress, we believe that we make some important headway toward understanding the potentially far-reaching consequences of stress during pregnancy. This is pertinent in light of the fact that stress is a growing health problem around the world. For example, according to recent survey evidence from the U.S. using a 10-item Perceived Stress Scale, women's average stress levels have increased by about 18 percent between

1983 and 2009 (Cohen and Janicki-Deverts, 2012). Concurrently, over these last few decades, mental health diagnoses and prescription drug use among both children and adults have risen substantially. For instance, a recent study by the Centers for Disease Control and Prevention shows that antidepressant consumption among individuals aged 12 years or older has increased by 400 percent from 1988 to 2008.[33] Certainly, it is likely that some of the growth in antidepressant use is driven by increases in diagnoses and in the availability of prescription drugs. Nevertheless, our results present some of the first evidence on a causal link between these two trends in the population—the prevalence of stress and the incidence of mental health issues—perpetuated by the fetal environment.

The presence of such a causal link may point to novel avenues for curbing the high and rapidly rising private and social costs associated with mental illness. Specifically, if a mother's stress during pregnancy harms her unborn child's mental health in adulthood, measures that help reduce stress during pregnancy may come at low costs relative to their social benefits. For example, although most countries have some kind of family leave policy that facilitates reductions in women's labor supply in the weeks or months following childbirth, regulation allowing women to take protected time off from work during pregnancy may also be important.

Finally, as low socio-economic status women experience higher levels of stress than their more advantaged counterparts, our results suggest that fetal stress exposure may play a potentially important role in the intergenerational transmission of disadvantage (see also Thompson, 2014 for a recent summary of evidence on this point). Future research might explore these conjectures in more detail by examining the effects of specific interventions that reduce pregnant women's stress levels on their children's outcomes, especially among low-income populations.

[33]See http://www.cdc.gov/nchs/data/databriefs/db76.htm for more details.

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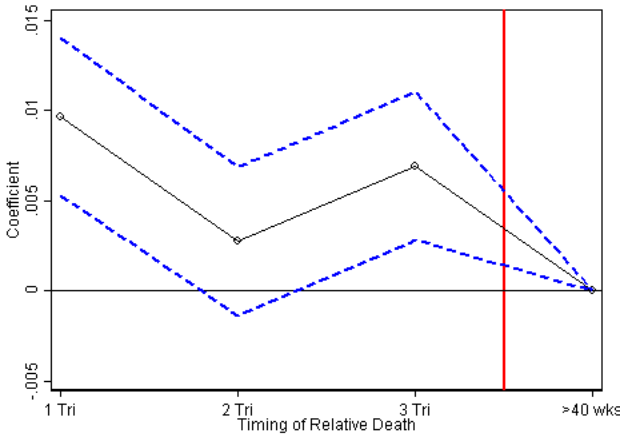
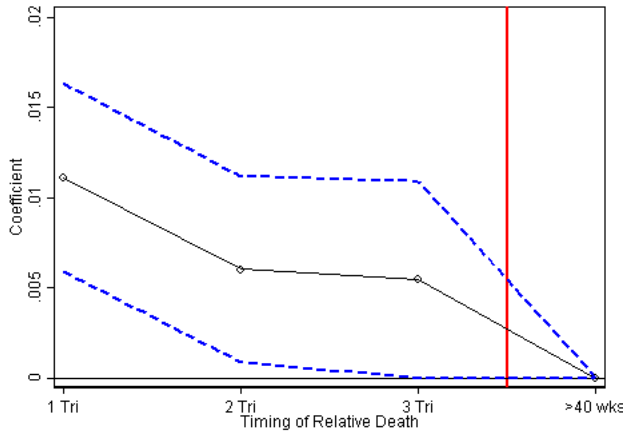
7 Figures

Figure I: Effect of Relative Death on the Incidence of the Child Being Born Low-Birth-Weight



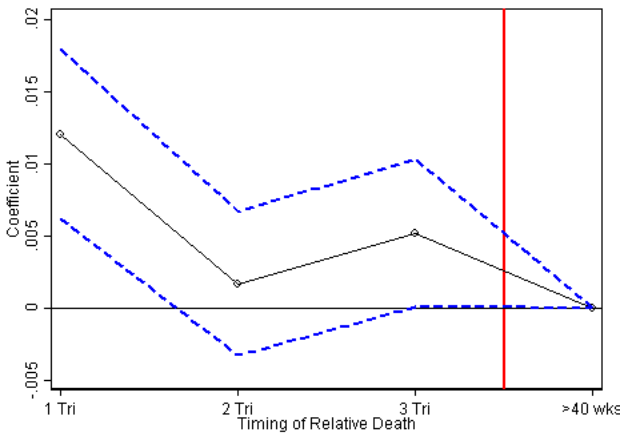
Notes: The sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within 280 days of the child's estimated date of conception or in the year after birth. To assign exposure to treatment, we first calculate each child's estimated date of conception by subtracting the number of gestation days from the date of birth. This figure plots the coefficients (and 95% confidence intervals in dashed blue lines) on the effects of the death of a relative during the 1st, 2nd, and 3rd trimesters of pregnancy. The omitted category is an indicator for the relative death occurring after 280 days (40 weeks) of gestation (i.e., post-childbirth in most cases). The outcome is an indicator for the child being born low-birth-weight.

Figure II: Effect of Relative Death on the Incidence of the Child Being Born Preterm



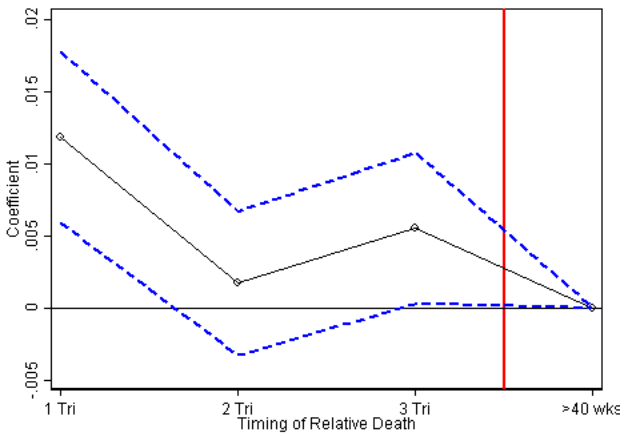
Notes: The sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within 280 days of the child's estimated date of conception or in the year after birth. To assign exposure to treatment, we first calculate each child's estimated date of conception by subtracting the number of gestation days from the date of birth. This figure plots the coefficients (and 95% confidence intervals in dashed blue lines) on the effects of the death of a relative during the 1st, 2nd, and 3rd trimesters of pregnancy. The omitted category is an indicator for the relative death occurring after 280 days (40 weeks) of gestation (i.e., post-childbirth in most cases). The outcome is an indicator for the child being born preterm.

Figure III: Effect of Relative Death on the Incidence of the Child Being Hospitalized for a Perinatal Condition by Age 1



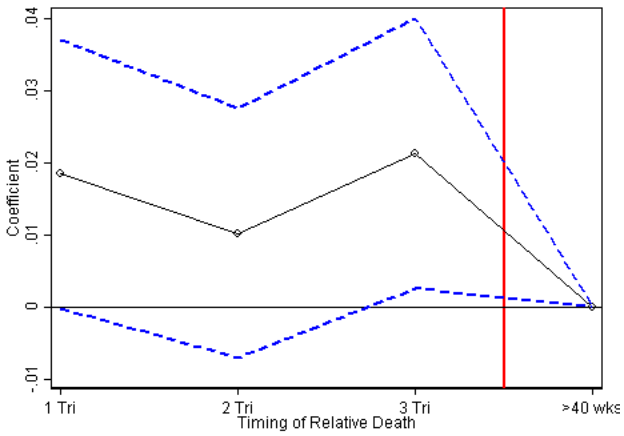
Notes: The sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within 280 days of the child's estimated date of conception or in the year after birth. To assign exposure to treatment, we first calculate each child's estimated date of conception by subtracting the number of gestation days from the date of birth. This figure plots the coefficients (and 95% confidence intervals in dashed blue lines) on the effects of the death of a relative during the 1st, 2nd, and 3rd trimesters of pregnancy. The omitted category is an indicator for the relative death occurring after 280 days (40 weeks) of gestation (i.e., post-childbirth in most cases). The outcome is an indicator for the child being ever hospitalized for a condition arising from the perinatal period by age 1.

Figure IV: Effect of Relative Death on the Incidence of the Child Being Hospitalized for a Perinatal Condition by Age 5



Notes: The sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within 280 days of the child's estimated date of conception or in the year after birth. To assign exposure to treatment, we first calculate each child's estimated date of conception by subtracting the number of gestation days from the date of birth. This figure plots the coefficients (and 95% confidence intervals in dashed blue lines) on the effects of the death of a relative during the 1st, 2nd, and 3rd trimesters of pregnancy. The omitted category is an indicator for the relative death occurring after 280 days (40 weeks) of gestation (i.e., post-childbirth in most cases). The outcome is an indicator for the child being ever hospitalized for a condition arising from the perinatal period by age 5.

Figure V: Effect of Close Relative Death on the Incidence of the Child Consuming Any Mental Health Medications



Notes: The sample includes all children whose mother loses a close family member—a sibling, a parent, the child's father, or an own (older) child—within 280 days of the child's estimated date of conception or in the year after birth. To assign exposure to treatment, we first calculate each child's estimated date of conception by subtracting the number of gestation days from the date of birth. This figure plots the coefficients (and 95% confidence intervals in dashed blue lines) on the effects of the death of a relative during the 1st, 2nd, and 3rd trimesters of pregnancy. The omitted category is an indicator for the relative death occurring after 280 days (40 weeks) of gestation (i.e., post-childbirth in most cases). The outcome is an indicator for the child ever consuming any medications used to treat mental health conditions.

8 Tables

Table I: Summary Statistics

	(1) All	(2) Death During Preg.	(3) Death After Preg.
Mother's age at childbirth	28.30	28.29	28.31
	(4.998)	(4.994)	(5.001)
Mother married pre-concep.	0.323	0.321	0.324
	(0.467)	(0.467)	(0.468)
Mother's ed: <HS pre-concep.	0.183	0.182	0.184
	(0.387)	(0.386)	(0.388)
Mother's ed: HS pre-concep.	0.326	0.321	0.329
	(0.469)	(0.467)	(0.470)
Mother's ed: some college pre-concep.	0.198	0.198	0.197
	(0.398)	(0.399)	(0.398)
Child's Birth Weight (g)	3552.9	3542.7	3560.6
	(563.3)	(573.9)	(555.2)
Child is Low Birth Weight (<2500g)	0.0321	0.0355	0.0296
	(0.176)	(0.185)	(0.169)
Child is Preterm (<37 weeks)	0.0506	0.0550	0.0474
	(0.219)	(0.228)	(0.212)
Observations	63,756	27,339	36,417

Note: The sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within 280 days of the child's estimated date of conception or in the year after birth. To assign exposure to treatment, we first calculate each child's estimated date of conception, c, by subtracting the number of gestation days from the date of birth. We then define the set of treated individuals as those experiencing the death of a relative in the time interval $[c, c + 280]$. Column one displays statistics for the full sample, while the second and third columns consider the treatment and comparison groups separately.

Table II: Effects of Relative Death *In Utero* on Birth Outcomes (1)

	(1) Birwt	(2) LBW	(3) VLBW	(4) HBW	(5) Pret.	(6) Stillb.	(7) Peri.Death
Death During Pregnancy	-18.37***	0.00643***	0.00172***	-0.00744**	0.00745***	-0.000752	-0.000243
	[4.915]	[0.00150]	[0.000665]	[0.00349]	[0.00187]	[0.000477]	[0.000550]
Mean, dept. var	3555.7	0.0316	0.00566	0.196	0.0501	0.00374	0.00606
Obs.	61836	61836	61836	61836	62056	62056	62056

Note: See table I for more information on the sample of analysis. Each column is a separate regression. All regressions include controls for mother's age at childbirth (five categories: < 20, $20-24$, $25-34$, > 35), mother's education in the year prior to conception (four categories: <HS, HS diploma, some college, college+), indicator variables for the mother being born outside of Sweden and being married in the year prior to conception year, dummies for parity (three categories: 1, 2, 3+), and the relative's age at death and age squared. Additionally, all regressions control for fixed effects for the year and month of conception, as well as the mother's municipality of residence during the year prior to conception. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table III: Effects of Relative Death *In Utero* on Birth Outcomes (2)

	(1) SGA	(2) LGA	(3) Length	(4) Head	(5) C-sect	(6) Induced	(7) Rupt.
Death During Pregnancy	0.00271**	-0.000703	-0.0745***	-0.0629***	0.00483*	-0.000254	-0.00698
	[0.00113]	[0.00164]	[0.0217]	[0.0139]	[0.00256]	[0.00197]	[0.00608]
Mean, dept. var	0.0264	0.0360	50.50	34.81	0.125	0.0692	0.534
Obs.	61834	61834	61382	59281	62056	62056	31595

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table IV: Effects of Relative Death *In Utero* on Birth Outcomes (1): Results by Trimester

	(1) Birwt	(2) LBW	(3) VLBW	(4) HBW	(5) Pret.	(6) Stillb.	(7) Peri.Death
Death in 1st Trimester	-29.95***	0.00965***	0.00211*	-0.00816*	0.0111***	-0.000529	-0.000738
	[7.149]	[0.00222]	[0.00120]	[0.00472]	[0.00266]	[0.000731]	[0.000815]
Death in 2nd Trimester	-13.32**	0.00274	0.000456	-0.00801*	0.00601**	-0.000206	0.000220
	[6.663]	[0.00210]	[0.000799]	[0.00471]	[0.00263]	[0.000772]	[0.000850]
Death in 3rd Trimester	-12.47*	0.00692***	0.00255***	-0.00624	0.00544*	-0.00147**	-0.000224
	[6.557]	[0.00210]	[0.000913]	[0.00493]	[0.00278]	[0.000590]	[0.000829]
Mean, dept. var	3555.7	0.0316	0.00566	0.196	0.0501	0.00374	0.00606
Obs.	61836	61836	61836	61836	62056	62056	62056

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table V: Effects of Relative Death *In Utero* on Hospitalizations by Ages 1 and 5

	By Age 1		By Age 5	
	(1) Any Hosp	(2) Tot Hosp	(3) Any Hosp	(4) Tot Hosp
Death During Pregnancy	0.00708***	0.00639	0.00521*	0.0225**
	[0.00247]	[0.00451]	[0.00277]	[0.0107]
Mean, dept. var	0.0858	0.116	0.132	0.216
Obs.	61823	61823	61823	61823

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table VI: Effects of Relative Death *In Utero* on Outpatient Visits by Ages 1 and 5

	By Age 1		By Age 5	
	(1) Any Visits	(2) Tot Visits	(3) Any Visits	(4) Tot Visits
Death During Pregnancy	0.00637*	0.0311**	0.0223***	0.115***
	[0.00375]	[0.0158]	[0.00605]	[0.0360]
Mean, dept. var	0.0734	0.237	0.299	0.859
Obs.	21739	21739	21739	21739

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table VII: Effects of Relative Death *In Utero* on Hospitalizations by Ages 1 and 5 due to Conditions Arising from the Perinatal Period

	By Age 1		By Age 5	
	(1) Any Hosp	(2) Tot Hosp	(3) Any Hosp	(4) Tot Hosp
Death During Pregnancy	0.00624***	0.00557**	0.00629***	0.00513*
	[0.00191]	[0.00238]	[0.00193]	[0.00271]
Mean, dept. var	0.0596	0.0676	0.0599	0.0691
Obs.	61823	61823	61823	61823

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table VIII: Effects of Relative Death *In Utero* on Prescription Use for Mental Health Conditions

	All mental	ADHD		Anxiety		Depression	
	(1) Any RX	(2) Any RX	(3) Avg. dose	(4) Any RX	(5) Avg. dose	(6) Any RX	(7) Avg. dose
Death During Pregnancy	0.00177	-0.0000659	0.0287	0.000964	0.00153	0.00113	0.156**
	[0.00285]	[0.00105]	[0.0439]	[0.00185]	[0.00979]	[0.00177]	[0.0772]
Mean, dept. var	0.199	0.0243	0.757	0.0516	0.126	0.0533	1.541
Obs.	61823	61823	61823	61823	61823	61823	61823

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table IX: Effects of Relative Death *In Utero* on Prescription Use for Mental Health Conditions: The Case of Severe Stress

	All mental	ADHD		Anxiety		Depression	
	(1) Any RX	(2) Any RX	(3) Avg. dose	(4) Any RX	(5) Avg. dose	(6) Any RX	(7) Avg. dose
Death During Pregnancy	0.0167***	0.00287	0.130*	0.00848**	0.0176	0.00907**	0.591***
	[0.00608]	[0.00206]	[0.0710]	[0.00400]	[0.0227]	[0.00413]	[0.208]
Mean, dept. var	0.285	0.0193	0.579	0.0777	0.194	0.0962	2.907
Obs.	18594	18594	18594	18594	18594	18594	18594

Note: See tables I and II for more information on the sample and controls. The sample here is further limited to mothers who experience the death of a sibling, a parent, the child's father, or an own (older) child (we drop mothers who experience the death of a grandparent). Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table X: Effects of Relative Death *In Utero* on Adult Labor Market and Marital Outcomes

	Income, Age 30				Marriage	
	(1) Any Wage	(2) Log Wage	(3) Any Lab.	(4) Log Lab.	(5) Ever Mar.	(6) Ever Div.
Death During Pregnancy	-0.00703	-0.00110	-0.00292	0.00139	0.000964	0.000757
	[0.00922]	[0.0337]	[0.00649]	[0.0221]	[0.00142]	[0.000653]
Mean, dept. var	0.904	11.96	0.945	12.13	0.0617	0.00770
Obs.	6425	5809	6425	6073	61823	61823

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table XI: Inheritances, Generation-Skipping Transfers, and Life Insurance Payouts

	Average amount (SEK), specific transfer class			Total amount (SEK)
Deceased relative	Inheritance	Generation-skipping transfer	Life Insurance Payout	All classes
Spouse	280000	100	3700	283800
Parent	30000	7000	1500	38500
Grandparent	7000	32000	500	39500

Note: The table presents average amounts of the three sources of income following the death of a relative—inheritances, generation-skipping transfers and life insurance payouts—from a deceased spouse, parent, and grandparent, respectively. For each income type, the three leftmost columns displays the average amount in Swedish Krona (SEK) in each class of recipients, i.e., *not* the average amount conditional on the amount received being greater than zero. The rightmost column displays the sum of the three income classes.

Table XII: Effects of Relative Death *In Utero* on Physical and Mental Health Indices

	Physical Health Index		Mental Health Index	
	(1) All Deaths	(2) Close Rel.	(3) All Deaths	(4) Close Rel.
Death During Pregnancy	-0.0118***	-0.0163**	-0.00482	-0.0155*
	[0.00356]	[0.00677]	[0.00401]	[0.00904]
Mean, dept. var	-0.0127	-0.0168	0.00397	-0.0832
Obs.	62072	18690	61823	18594

Note: See tables I and II for more information on the sample and controls. The physical health index consists of all the outcomes analyzed in Tables II, III, V, VI, VII, and A6: continuous birth weight, low-birth-weight indicator, very-low-birth-weight indicator, high-birth-weight indicator, pre-term indicator, stillbirth indicator, perinatal death indicator, SGA indicator, LGA indicator, birth length, head circumference, c-section indicator, induced labor indicator, any ruptures indicator, any and total hospitalizations by ages 1 and 5, any and total outpatient visits by ages 1 and 5, any and total hospitalizations for perinatal causes by ages 1 and 5, any medications for obesity or diabetes or Cushing's Syndrome, any medications for obesity, average dose for obesity medication, any medication for diabetes, average dose for diabetes medication, any medication for Cushing's Syndrome, average dose for Cushing's Syndrome Medication. The mental health index consists of an indicator for ever purchasing a mental health drug, as well as 16 other outcomes comprised of our two measures—an indicator for every purchasing the drug and the average daily dose—per condition (ADHD, anxiety, bipolar disorder, depression, psychotic disorders, addiction, and sleep disorders). See text in Section 5 for more information on how the indices are constructed. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

A Additional Results

Table A1: Correlation Between the Timing of Relative Death and Maternal Characteristics

	(1) M.Age	(2) 1st Par.	(3) M.Mar.	(4) M.Ed:<HS	(5) M.Ed:College+	(6) M. Wage	(7) M. Foreign
Death During Pregnancy	-0.0489	0.00334	-0.00118	-0.000847	0.00748**	1037.5	-0.000442
	[0.0362]	[0.00331]	[0.00324]	[0.00283]	[0.00341]	[1009.2]	[0.00115]
Mean, dept. var	28.30	0.455	0.323	0.183	0.293	113312.5	0.0212
Obs.	63559	63559	63559	62072	62072	42698	63559

Note: See table I for more information on the sample. This table reports the correlation between exposure to relative death during pregnancy and maternal characteristics measured prior to conception. All regressions control for fixed effects for the year and month of conception, as well as the mother's municipality of residence during the year prior to conception. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A2: Do Maternal Characteristics Predict the Timing of the Relative's Death?

	Dep. Var.: Death During Pregnancy	
	(1)	(2)
Mom Age <20	0.00602	0.00829
	[0.0150]	[0.0151]
Mom Age 20-24	0.00397	0.00779
	[0.00765]	[0.00794]
Mom Age 25-34	0.00344	0.00472
	[0.00573]	[0.00580]
1st parity	0.00318	0.00460
	[0.00641]	[0.00648]
2nd parity	0.00431	0.00520
	[0.00645]	[0.00649]
Mom Born Outside Sweden	-0.00244	-0.00252
	[0.0143]	[0.0146]
Mom Married	-0.00465	-0.00145
	[0.00434]	[0.00441]
Mom Ed: <HS	-0.0150**	-0.0122*
	[0.00639]	[0.00641]
Mom Ed: HS	-0.0164***	-0.0140***
	[0.00506]	[0.00523]
Mom Ed: Some College	-0.00888	-0.00980
	[0.00609]	[0.00611]
Relative Age at Death	0.000404	0.000637
	[0.000682]	[0.000685]
Relative Age at Death Sq.	-0.00000544	-0.00000835
	[0.00000507]	[0.00000509]
Fixed Effects	No	Yes
Joint F-Test P-val.	0.180	0.0834
Obs.	62056	62056

Note: See table I for more information on the sample. This table reports results from regressions where the dependent variable is an indicator for a relative death occurring during pregnancy. The explanatory variables are listed in the table. Column 2 also includes fixed effects for the year and month of conception, as well as the mother's municipality of residence during the year prior to conception. The joint F-test p-value is from a test that the coefficients on the listed variables are all equal zero. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A3: Effects of Relative Death *In Utero* on Birth Outcomes (2): Results by Trimester

	(1) SGA	(2) LGA	(3) Length	(4) Head	(5) C-sect	(6) Induced	(7) Rupt.
Death in 1st Trimester	0.00438**	0.00105	-0.107***	-0.0959***	-0.000702	-0.00288	0.00911
	[0.00197]	[0.00206]	[0.0356]	[0.0232]	[0.00405]	[0.00294]	[0.00839]
Death in 2nd Trimester	0.00374**	-0.00227	-0.0438	-0.0565***	0.00484	0.000822	-0.0138*
	[0.00155]	[0.00239]	[0.0272]	[0.0175]	[0.00367]	[0.00280]	[0.00819]
Death in 3rd Trimester	0.000212	-0.000845	-0.0738**	-0.0386**	0.00991**	0.00115	-0.0156**
	[0.00206]	[0.00252]	[0.0296]	[0.0179]	[0.00427]	[0.00286]	[0.00769]
Mean, dept. var	0.0264	0.0360	50.50	34.81	0.125	0.0692	0.534
Obs.	61834	61834	61382	59281	62056	62056	31595

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A4: Effects of Relative Death *In Utero* on Hospitalizations by Ages 1 and 5: Results by Trimester

	By Age 1		By Age 5	
	(1) Any Hosp	(2) Tot Hosp	(3) Any Hosp	(4) Tot Hosp
Death in 1st Trimester	0.0149***	0.0143**	0.0137***	0.0330*
	[0.00369]	[0.00643]	[0.00406]	[0.0170]
Death in 2nd Trimester	0.00367	0.00778	0.000986	0.0288
	[0.00314]	[0.00655]	[0.00381]	[0.0181]
Death in 3rd Trimester	0.00306	-0.00221	0.00137	0.00708
	[0.00347]	[0.00560]	[0.00457]	[0.0134]
Mean, dept. var	0.0858	0.116	0.132	0.216
Obs.	61823	61823	61823	61823

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A5: Effects of Relative Death *In Utero* on Hospitalizations by Ages 1 and 5 due to Conditions Arising from the Perinatal Period: Results by Trimester

	By Age 1		By Age 5	
	(1) Any Hosp	(2) Tot Hosp	(3) Any Hosp	(4) Tot Hosp
Death in 1st Trimester	0.0120***	0.0118***	0.0118***	0.0103***
	[0.00300]	[0.00363]	[0.00303]	[0.00378]
Death in 2nd Trimester	0.00169	0.00206	0.00170	0.00182
	[0.00255]	[0.00353]	[0.00254]	[0.00403]
Death in 3rd Trimester	0.00518**	0.00315	0.00552**	0.00350
	[0.00261]	[0.00309]	[0.00266]	[0.00353]
Mean, dept. var	0.0596	0.0676	0.0599	0.0691
Obs.	61823	61823	61823	61823

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A6: Effects of Relative Death *In Utero* on Prescription Use for Obesity, Diabetes, and Cushing's Syndrome

	All phys	Obesity		Diabetes		Cushing	
	(1) Any RX	(2) Any RX	(3) Avg. dose	(4) Any RX	(5) Avg. dose	(6) Any RX	(7) Avg. dose
Death During Pregnancy	-0.00104	-0.000709*	-0.00218	-0.000297	0.000972	-0.0000583	-0.0000627
	[0.000751]	[0.000424]	[0.00838]	[0.000687]	[0.0474]	[0.0000414]	[0.0000555]
Mean, dept. var	0.0103	0.00285	0.0325	0.00775	0.468	0.0000324	0.0000320
Obs.	62056	61823	61823	61823	61823	61823	61823

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A7: Effects of Relative Death *In Utero* on Hospitalizations by Ages 10, 18, and 27

	By Age 10		By Age 18		By Age 27	
	(1) Any Hosp	(2) Tot Hosp	(3) Any Hosp	(4) Tot Hosp	(5) Any Hosp	(6) Tot Hosp
Death During Pregnancy	0.000354	0.0117	-0.00388	0.0110	0.00215	0.0363
	[0.00390]	[0.0188]	[0.00642]	[0.0347]	[0.00935]	[0.0520]
Mean, dept. var	0.172	0.304	0.259	0.506	0.433	1.033
Obs.	40084	40084	19641	19641	11269	11269

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A8: Effects of Relative Death *In Utero* on Prescription Use for Mental Health Conditions: The Case of Severe Stress, Cohorts 1973-1988 Only

	All mental	ADHD		Anxiety		Depression	
	(1) Any RX	(2) Any RX	(3) Avg. dose	(4) Any RX	(5) Avg. dose	(6) Any RX	(7) Avg. dose
Death During Pregnancy	0.0205**	0.00193	0.0637	0.0136**	0.0364	0.0133*	0.922***
	[0.00929]	[0.00266]	[0.0709]	[0.00593]	[0.0361]	[0.00677]	[0.334]
Mean, dept. var	0.392	0.0117	0.290	0.112	0.283	0.150	4.532
Obs.	11242	11242	11242	11242	11242	11242	11242

Note: See tables I and II for more information on the sample and controls. The sample here is further limited to mothers who experience the death of a sibling, a parent, the child's father, or an own (older) child (we drop mothers who experience the death of a grandparent). Additionally, we only consider cohorts 1973-1988 to capture the incidence of prescription drug purchases in adulthood (at ages 17-39). Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A9: Effects of Relative Death *In Utero* on Prescription Use for Mental Health Conditions: Is Severity of Stress Driven by the Relative's Age at Death?

	All mental	ADHD		Anxiety		Depression	
	(1) Any RX	(2) Any RX	(3) Avg. dose	(4) Any RX	(5) Avg. dose	(6) Any RX	(7) Avg. dose
Death During Pregnancy	0.0234***	0.00309	0.136*	0.0104**	0.0246	0.0115***	0.734***
	[0.00681]	[0.00219]	[0.0745]	[0.00419]	[0.0221]	[0.00433]	[0.231]
Relative Age Less 50	0.0264**	0.00141	0.0554	0.00419	0.0205	0.00775	0.228
	[0.0120]	[0.00417]	[0.137]	[0.00690]	[0.0482]	[0.00822]	[0.344]
Relative Age Less 50*Death During Pregnancy	-0.0377**	-0.000719	-0.0300	-0.0114	-0.0387	-0.0144	-0.815
	[0.0176]	[0.00543]	[0.231]	[0.0108]	[0.0649]	[0.0113]	[0.507]
Mean, dept. var	0.285	0.0193	0.579	0.0777	0.194	0.0962	2.907
Obs.	18594	18594	18594	18594	18594	18594	18594

Note: See tables I and II for more information on the sample and controls. The sample here is further limited to mothers who experience the death of a sibling, a parent, the child's father, or an own (older) child (we drop mothers who experience the death of a grandparent). Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A10: Effects of Relative Death *In Utero* on Maternal Pregnancy Behaviors and Characteristics

	(1) Highrisk	(2) Smoked During Preg	(3) Smokes Regularly
Death During Pregnancy	0.00114	0.00404	-0.000168
	[0.00258]	[0.00352]	[0.00294]
Mean, dept. var	0.141	0.0922	0.174
Obs.	62056	25333	51863

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A11: Effects of Relative Death *In Utero* on the *Mother's* Prescription Use for Mental Health Conditions: The Case of Severe Stress

	All mental	ADHD		Anxiety		Depression	
	(1) Any RX	(2) Any RX	(3) Avg. dose	(4) Any RX	(5) Avg. dose	(6) Any RX	(7) Avg. dose
Death During Pregnancy	-0.00497	0.000368	0.0120	-0.00939*	-0.00937	-0.00620	-0.0985
	[0.00694]	[0.00129]	[0.0250]	[0.00545]	[0.0368]	[0.00568]	[0.251]
Mean, dept. var	0.602	0.00662	0.0813	0.182	0.433	0.241	5.352
Obs.	18594	18594	18594	18594	18594	18594	18594

Note: See tables I and II for more information on the sample and controls. The sample here is further limited to mothers who experience the death of a sibling, a parent, the child's father, or an own (older) child (we drop mothers who experience the death of a grandparent). Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A12: Effects of Relative Death *In Utero* on Main Outcomes: Heterogeneity by Maternal Education

	(1) LBW	(2) Pret.	(3) Any Per. Hosp. 1	(4) Any Per. Hosp. 5	(5) Any Mental, Close Rel.
Death During Pregnancy	0.00542***	0.00659***	0.00478*	0.00469*	0.0238**
	[0.00186]	[0.00249]	[0.00264]	[0.00268]	[0.00986]
Mom Low Ed (HS or less)	0.00831***	0.00796***	0.0102***	0.0106***	0.0420***
	[0.00192]	[0.00271]	[0.00266]	[0.00266]	[0.00961]
Mom Low Ed*Death During Preg	0.00194	0.00170	0.00288	0.00315	-0.0117
	[0.00274]	[0.00374]	[0.00358]	[0.00360]	[0.0149]
Mean, dept. var	0.0316	0.0501	0.0596	0.0599	0.285
Obs.	61836	62056	61823	61823	18594

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A13: Effects of Relative Death *In Utero* on Birth Outcomes: "Exogenous Deaths"

	(1) Birwt	(2) LBW	(3) VLBW	(4) HBW	(5) Pret.	(6) Stillb.	(7) Peri.Death
Death During Pregnancy	-16.68**	0.00709**	0.00109	-0.00432	0.00863**	0.000249	0.000603
	[7.774]	[0.00321]	[0.00118]	[0.00550]	[0.00348]	[0.00115]	[0.00143]
Mean, dept. var	3515.5	0.0358	0.00689	0.173	0.0535	0.00493	0.00836
Obs.	18572	18572	18572	18572	18658	18658	18658

Note: See tables I and II for more information on the sample and controls. The sample is further limited to mothers who experience a relative death from causes determined to be exogenous in Adda et al. (2011). These are deaths from endocrine and metabolic causes, accidents, and other causes. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A14: Effects of Relative Death *In Utero* on Hospitalizations by Ages 1 and 5: "Exogenous Deaths"

	By Age 1		By Age 5	
	(1) Any Hosp	(2) Tot Hosp	(3) Any Hosp	(4) Tot Hosp
Death During Pregnancy	0.000821	-0.00498	0.000452	0.00807
	[0.00457]	[0.00711]	[0.00544]	[0.0210]
Mean, dept. var	0.0665	0.0915	0.111	0.186
Obs.	18566	18566	18566	18566

Note: See tables I and II for more information on the sample and controls. The sample is further limited to mothers who experience a relative death from causes determined to be exogenous in Adda et al. (2011). These are deaths from endocrine and metabolic causes, accidents, and other causes. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A15: Effects of Relative Death *In Utero* on Prescription Use for Mental Health Conditions: Severe Stress and "Exogenous Deaths"

	All mental	ADHD		Anxiety		Depression	
	(1) Any RX	(2) Any RX	(3) Avg. dose	(4) Any RX	(5) Avg. dose	(6) Any RX	(7) Avg. dose
Death During Pregnancy	0.0158*	0.00145	0.0763	0.00904	0.0425	0.0146**	0.937***
	[0.00901]	[0.00316]	[0.0906]	[0.00656]	[0.0368]	[0.00737]	[0.356]
Mean, dept. var	0.368	0.0147	0.394	0.104	0.267	0.140	4.234
Obs.	10335	10335	10335	10335	10335	10335	10335

Note: See tables I and II for more information on the sample and controls. The sample here is further limited to mothers who experience the death of a sibling, a parent, the child's father, or an own (older) child (we drop mothers who experience the death of a grandparent). Additionally, the sample is limited to mothers who experience a relative death from causes determined to be exogenous in Adda et al. (2011). These are deaths from endocrine and metabolic causes, accidents, and other causes. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception. Exact definitions of the prescription drug categories are given in B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A16: Effects of Relative Death *In Utero* on Adult Labor Market and Marital Outcomes: "Exogenous Deaths"

	Income, Age 30				Marriage	
	(1) Any Wage	(2) Log Wage	(3) Any Lab.	(4) Log Lab.	(5) Ever Mar.	(6) Ever Div.
Death During Pregnancy	-0.00686	-0.00110	-0.00316	0.00202	0.00437	0.00262
	[0.00930]	[0.0337]	[0.00647]	[0.0222]	[0.00445]	[0.00219]
Mean, dept. var	0.904	11.96	0.945	12.13	0.196	0.0252
Obs.	6423	5809	6423	6072	18566	18566

Note: See tables I and II for more information on the sample and controls. The sample is further limited to mothers who experience a relative death from causes determined to be exogenous in Adda et al. (2011). These are deaths from endocrine and metabolic causes, accidents, and other causes. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A17: Effects of Relative Death *In Utero* on the Mother's Subsequent Fertility

	Dep. Var: Mother Has Subsequent Children	
	(1) All Deaths	(2) Close Rel.
Death During Pregnancy	0.00785** [0.00332]	0.00832 [0.00709]
Mean, dept. var	0.572	0.459
Obs.	60258	18116

Note: See tables I and II for more information on the sample and controls. Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

B Definitions of Health-Related Outcomes

Diagnosis (ICD) codes For all children and siblings, we get obtain comprehensive inpatient and outpatient medical records for all visits associated with the following diagnosis codes (ICD-10):

- Psychological disease (F00-F99)
- Suicide (X60-X84)
- Type II diabetes (E10-E14)
- Obesity (E65-E68)
- Heart disease (I20-I25, I30-I52)
- Neoplasms (C00-D48)
- Cushing's syndrome (E24)
- Perinatal (P00-P96)
- Deformations at birth (Q00-Q99)
- Drug and alcohol abuse (Z72)
- Thyroid-related issues (E00-E07)
- External cause (S00-T98, V01-Y98)
- Sexually transmitted disease (A50-A64)
- Stroke (I61-I64)

For earlier years, the analogous ICD-9 and ICD-8 codes are applied.

Prescription drug (ATC) codes Prescription drugs are classified according to the Anatomical Therapeutic Chemical Classification System (ATC). To associate certain prescription drugs to mental health diagnoses, we use the classification system below, employed by the National Board of Health and Welfare in Sweden (Socialstyrelsen, 2012):

- Mental health (all): ATC-code begins by "N."
- ADHD: ATC-code begins by "N06BA"

- Bipolar disease: ATC-code begins by “N05AN01”
- Psychotic conditions: ATC-code begins by “N05A," but excluding "N05AN01”
- Depression: ATC-code begins by “N06A”
- Anxiety: ATC-code begins by “N05B”
- Sleeping disorders: ATC-code begins by “N05C”
- Addiction: ATC-code begins by “N07”
- Parkinson: ATC-code begins by “N04”

- Diabetes: ATC-code begins by “A10.”
- Obesity: ATC-code begins by “A08AB01" or "A08AA10.”
- Cushing’s syndrome: ATC-code begins by “J02AB0.”
- Neoplasm: ATC-code begins by “L01.”
- Thyroid: ATC-code begins by “L01.”

C Stress *In Utero*: More References

While it is well established that malnutrition in pregnant women affects the unborn child, the mechanism through which maternal adversity impacts the child is not well understood. One prominent theory proposes a neuro-scientific mechanism in which stress plays a key role (Jaddoe, 2006). It is hypothesized that nutritional restrictions inhibit the development of a placental enzyme that is required to convert the stress hormone cortisol into inactive cortisone. As a consequence of maternal malnutrition, the fetus is thus exposed to excessive amounts of cortisol in utero. Overexposure to cortisol, in turn, is believed to lead to a reprogramming of the hypothalamic-pituitary-adrenal axis (HPA), which could lead to impaired fetal development and worse health in adult age (Jaddoe, 2006).

Substantial evidence from preclinical laboratory studies show that the offspring of prenatally stressed animals displays over activity and impaired negative feedback regulation of the HPA, alternations which have been linked to a diverse spectrum of psychopathology, including schizophrenia and depression (M., 2001; Huizink AC, 2004; Kofman, 2002). Nevertheless, in humans, evidence of an explicit link between maternal stress and long-term disturbance in the HPA is scarce (Kapoor A

and Matthews, 2006). A significant association between measures of prenatal anxiety and individual differences in salivary cortisol has been established in a sample of 10-year-old children from the Avon Longitudinal Study of Parents and Children (ALSPAC)(O'Connor TG, 2005). In another sample, young children whose mothers exhibited higher levels of morning cortisol during pregnancy were found to show higher levels of salivary cortisol (Gutteling BM, 2004, 2005). These results suggest that prenatal anxiety can have lasting effects on HPA functioning in the child, and are consistent with the hypothesis that that prenatal anxiety might constitute a mechanism for an increased vulnerability to psychopathology in children and adolescents.

In humans, researchers have also documented an association between antenatal maternal stress and an increased risk of obstetric complications such as preterm birth, low birth weight, and fetal distress (Crandon, 1979; Lou HC, 1994; Wadhwa PD, 1993), negative reactivity to novelty (Davis EP, 2004), an increase in neonatal crying (Rieger M, 2004), behavioral and/or emotional abnormalities at young ages (O'Connor TG, 2002), a depressed Apgar score (Crandon, 1979; Ponirakis A, 1998), and a higher incidence of ADHD during childhood (Van den Bergh BRH, 2004, 2005). Moreover, in a rare study of the association between maternal stress and non-health related outcomes, researchers established that maternal depression at mid-gestation was associated with a small but significant increase in violent crime in Finland (MakiP, 2003). While these studies establish correlations between antenatal maternal stress and outcomes later in life, the causal link is not clear. The studies assess the level of maternal anxiety and stress using the mother's own rating of symptoms, and some studies also included cortisol measures or an appraisal of recently experienced adverse life events such as divorce, job loss, or marital discord. Because these measures may not be independent of unobserved factors that affect child outcomes, maternal stress may be endogenous.