Family Ruptures, Stress, and the Mental Health of the Next Generation*

Petra Persson†
Maya Rossin-Slater‡

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Abstract

This paper studies how *in utero* exposure to maternal stress from family ruptures affects later-life mental health. We find that prenatal exposure to the death of a maternal relative increases take-up of ADHD medications during childhood and anti-anxiety and depression medications during adulthood. Further, family ruptures during pregnancy depress birth outcomes and raise the risk of perinatal complications necessitating hospitalization during early childhood. Our results imply large welfare gains from preventing fetal stress—$700 million annually in expenditures on antidepressants alone—and suggest that greater stress exposure among the poor may partially explain the intergenerational persistence of poverty.

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†SIEPR and Department of Economics, Stanford University; Research Institute for Industrial Economics, Stockholm. Email: perssonp@stanford.edu.

‡Department of Economics, University of California at Santa Barbara. Email: maya.rossin-slater@ucsb.edu.

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 use Swedish administrative data to 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 the child's mental health.

Our focus on the fetal stage is consonant with two recent studies in economics that 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).[1] Our study offers complementary evidence linking early-life circumstance to adult mental health, but breaks new ground by focusing on stress—which may be more pertinent than malnutrition in modern developed countries such as the U.S. and Sweden—and by tracing health outcomes throughout the time period between the fetal shock and adulthood.

Our focus on stress is influenced by the growing literature documenting persistent intergenerational transmission of socioeconomic status (see, e.g., Solon, 2001; Chetty et al., Forthcoming for

[1] Consistent with this evidence, epidemiological studies 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). Further, recent neuroscientific evidence shows that mental illness is related to brain abnormalities that likely arise before birth, which further emphasizes the importance of the fetal environment. 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 diseases.

evidence from the U.S. and Boserup et al., 2013 for evidence from Scandinavia). As low socioeconomic status women experience higher levels of stress than their more advantaged counterparts,[2] a causal link between fetal stress exposure and mental disease later in life could shed light on one channel through which disadvantage is transmitted across generations.

Our focus on stress is also 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 credible 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 socioeconomic 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 between 1973 and 2011, 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 maternal grandparent, the child's father, or an own (older) child—in the nine months after the child's date of conception or in the year after 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 children's health throughout childhood and into adulthood stemming from birth and inpatient records. We also merge our data to novel, unique data from Sweden's prescription drug registry, which contain the universe of prescription drug purchases with information on the exact substance and dose prescribed.

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 from family bereavement, *relative to the consequences of such*

[2] See the recent discussion in Thompson, 2014 for evidence on self-reported stress levels. Additionally, estimated levels of the stress hormone cortisol have been shown to be negatively correlated with socioeconomic status (Kunz-Ebrecht et al., 2004; Cohen 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.

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 family characteristics (including measures of maternal and paternal socioeconomic status pre-conception and older siblings' health at birth).

This paper makes two primary contributions. 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.[5] As presented in Section 5, we find that *in utero* exposure to the death of a mother's close relative has substantial effects on the consumption of prescription drugs treating mental health conditions both during childhood (around age 10) and in adulthood (around age 35). For children, these effects are driven by an 18 percent rise in the likelihood of purchasing a drug used to treat ADHD and a 24 percent increase in the average daily dose of ADHD medications. For adults, we see 11 and 7 percent increases in the likelihood of consuming prescription drugs for anxiety and depression, respectively, as well as 15 and 10 percent increases in the average daily doses of these medications. The estimated effects are stronger when the deceased is 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 12, 24, and 12 percent increases in the likelihoods of low-birth-weight (less than 2,500 grams), very-low-birth-weight (less than 1,500 grams), and pre-term (less than 37 weeks gestation) births, respectively. 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 for a condition originating in the perinatal period during the first five years of life.

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 after age five, or on the consumption of drugs treating physical conditions such as obesity, diabetes, and heart disease. 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.[6]

[5] Here, we reference the existing literature on humans, which we discuss further in Section 2. Animal studies have provided credible causal evidence of a link between *in utero* exposure to stress and adverse offspring outcomes. See, e.g., the experimental work on rats of Welberg et al. (2001).

[6] Additionally, our cohorts may be too young to detect any effects on physical health conditions such as obesity and diabetes. For example, Barker (1990)'s "fetal origins hypothesis" (described further in a footnote in Section 2) emphasizes latent effects of prenatal malnutrition on these conditions among individuals aged 50-70 years old, whereas the oldest cohorts in our data are only followed until age 40.

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, weight gain, labor force participation) 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 7 percent decrease in the consumption of prescription drugs treating depression alone can be valued at around $700 million annually.

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 one potential reason for why so few children born into disadvantage are able to escape it in adulthood. Indeed, a growing literature has highlighted how early-life health disparities may perpetuate economic inequality in adulthood (Currie, 2011; Aizer and Currie, 2014). Our results, combined with prior research documenting a strong socioeconomic gradient in stress exposure (see Thompson, 2014 for an overview), contribute to this literature by providing novel evidence on how disparities in early-life health may also translate into lasting disparities in adult mental illness.

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 abundant evidence on the impacts of maternal exposure to physical insults during pregnancy, the evidence on the consequences of purely psychological stressors is more limited.[7]

Moreover, the precise mechanisms through which the effects of physical insults operate are not well understood, and, in several prominent theories, stress plays a key role. 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,

[7]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.

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.[8] 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 most closely relates to the work of Aizer, Stroud and Buka (2009), who implement a sibling fixed effects estimation and show that exposure to elevated cortisol *in-utero* adversely affects cognition at age seven and educational attainment later in life. Though this design controls for time-invariant differences between mothers that might be correlated with stress, it cannot fully control for time-varying factors that might lead to variation in cortisol levels across pregnancies within the same mother. Researchers have also exploited quasi-exogenous shocks during pregnancy stemming from extreme incidents such as hurricanes, earthquakes, or terrorist attacks.[9] However, these designs are limited in their ability to separate the effects of *in utero* stress exposure from any post-natal responses, as well as from the physical health and economic insults associated with these events; our methodology is designed to overcome this limitation. 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.

Because our identification strategy exploits exogenous changes in family structure, we also contribute to the literature in economics that broadly analyzes how family structure affects child well-being. In the context of Sweden, it has been shown that the loss of a parent during childhood may hamper educational attainment (Adda, Björklund and Holmlund, 2011). Further, Black, Devereux and Salvanes (2014) study the impacts of deaths of maternal parents during pregnancy using Norwegian data, and find small adverse effects on birth outcomes, and no effects on long-run economic outcomes such as education and adult earnings. Our work is complementary as we focus on mental health; moreover, by including relatives other than maternal parents, we are able to create a novel measure of the severity of antenatal stress exposure.[10]

3 Data

Our analysis uses administrative population-level data from Sweden. We have data on the universe of children born in Sweden from 1973 to 2011, who experienced the death of a relative (other than

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

[9] 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).

[10] Additionally, our methodology is slightly different from the main strategy employed by Black, Devereux and Salvanes (2014): we do not use a sibling fixed effects design, as, in our particular context, we provide some evidence that the presence of younger siblings is endogenous due to maternal fertility responses. 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 fully account for non-random exposure to death.

the mother) in the 40 weeks after their date of conception or in the one year after their date of birth. Put differently, our baseline sample includes all children whose mother loses a family member—a sibling, a parent, a maternal grandparent, the child's father, or an own (older) child—either during her pregnancy or in the year after childbirth. 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. For each relative who died, we have information on the cause and exact date of death. We also have information about the mothers' and fathers' educational attainment, labor market income, and marital status measured around the time of conception.

For each child in our sample, we have data on the exact date of birth, birth weight, birth length, head circumference, gestation (in days), and a variety of diagnosis codes at birth. We also have variables related to the mother's pregnancy and delivery: tobacco use during pregnancy, pregnancy risk factors (diabetes, kidney disease, epilepsy, asthma, hypertension, or urinary infection), caesarean section (c-section) delivery, induction of labor, and any complications at delivery.

To trace health outcomes after birth and throughout life, we add information from inpatient records and the prescription drug registry. For all of these, we have the universe of records associated with pre-specified health conditions described below. Inpatient records exist from 1964 to 2012, while the prescription drug data exist for the years 2005 to 2014. 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.[11] The ATC classification allows us to link the drugs to the conditions they are most commonly used to treat.

To select the inpatient and prescription drug records, we pre-specified certain health conditions before undertaking any analysis.[12] First, we include all mental illnesses. We further pre-specified the eight sub-categories of mental disorders that were recently selected by Sweden's National Board of Health and Welfare to track prevalence and prescription drug use (Socialstyrelsen, 2012): ADHD, depression, anxiety, bipolar disorder, psychotic disorders, sleeping disorders, addiction, and Parkinson's disease. While we pre-specified all eight subcategories for completeness, most of our analysis focuses on ADHD, depression, and anxiety. For many of the other conditions (especially, bipolar disorder), genetic influences are believed to be more important than environmental factors, suggesting that the fetal environment may not matter as much for their etiology.[13]

Second, although our primary focus is mental health, we pre-specified a small set of physical health conditions that 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,

[11] 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.

[12] We have access only to the subset of the inpatient and prescription drug records described here; not to the entire universe of inpatient and prescription drug records for all possible conditions. We are therefore unable to explore health effects beyond the pre-specified ones in our analysis.

[13] Genetic factors are believed to account for $60-80$ percent of the risk of developing bipolar disease, suggesting a strong hereditary component (Barnett and Smoller, 2009).

cholesterol, neoplasms, and conditions originating in the perinatal period.[14] We include all of these for completeness, although our cohorts may be too young to detect any effects on physical health other than conditions originating in the perinatal period.[15]

Finally, for the (older) cohorts that we can follow into adult age, we add data on labor income at ages 29-36. In sum, we create a unique data set that enables us to follow the children from conception to 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 exposure to a family rupture and children's physical and mental well-being at birth and later in life. The death of a relative is a traumatic event that induces acute and immediate stress in the expectant mother. 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 socioeconomic status (Adda, Björklund and Holmlund, 2011). Additionally, this loss may have many consequences for families aside from stress. For instance, a relative's passing may constitute 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. All of these factors can also affect the child after birth.

To identify the impact of antenatal exposure to a family rupture, we must therefore address two challenges: (i) separation of impacts that operate through the uterine environment from other impacts that also operate through the post-natal environment, and (ii) non-random selection into death. We do this by exploiting variation in the exact timing of family rupture relative to the expected date of delivery (at full term). Our analysis essentially compares individuals who experience the death of a relative during gestation with individuals who experience such a death in the year after birth. Thus, while all children included in this analysis are exposed to the post-natal consequences of the relative's passing, only the former group is exposed *through the uterine environment*.

[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. Cushing's syndrome is a condition that occurs when the body is exposed to high levels of the hormone cortisol for a long time. Symptoms include: fatty hump between the shoulders, rounded face, and pink or purple stretch marks on the skin. Appendix C has details and references relating to the biological mechanisms through which stress affects human health.

[15] As outlined in Appendix B, the inpatient 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.

Isolation of Antenatal Effects More concretely, to see how we address (i), let the causal relationship between an outcome of interest, y_i, and the occurrence of a family rupture be given by:

$$y_i = \gamma RelativeDeath_i + \mathbf{x}_i'\kappa + u_i, \tag{1}$$

where $\mathbf{x}_i$ is a vector of all other relevant determinants of y_i, and u_i is a random vector of predetermined and unobservable characteristics. Here, γ captures the combined impact of all pre- and post-natal consequences of the relative's passing.

Now instead consider a sample of children who either experience the death of a relative during gestation, or shortly after birth:

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

where c denotes the child's date of conception, b denotes the child's date of birth, and w denotes a time window after birth (in days), so that $\mathbf{1}[c \leq RelativeDeath < b]_i = 1$ indicates that the family rupture occurred during pregnancy, and $\mathbf{1}[b \leq RelativeDeath < b + w]_i = 1$ indicates that it occurred within w days of the child's birth, respectively.

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

$$y_i = \sigma \mathbf{1}[c \leq RelativeDeath < b]_i + \mathbf{x}_i'\eta + \epsilon_i, \tag{2}$$

where all of the variables are defined as above. Here, σ captures the effect of bereavement *in utero* relative to the effect of bereavement immediately after birth, and *not* the entire effect of bereavement. Comparing individuals who experience a stressful shock during gestation with those who experience such a shock shortly after birth effectively addresses issue (i) above, and 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 environments. 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*.

By separating antenatal effects from post-natal consequences, our estimate captures the impact of the unborn child's physiological exposure to maternal stress through the uterine environment. The extent to which σ isolates *only* the effect of this stress exposure depends on whether other consequences of the family rupture—e.g. positive or negative income effects or changes in household productivity—are the same across the pre- and post-natal periods, or whether some of them have differential impacts during the pre-natal period. To be more precise, two different assumptions on the separability of the effects of a relative's passing translate into two different interpretations of σ:

A1: Strong additive separability. First, interpreting σ in (2) as the impact of intrauterine stress exposure alone is equivalent to coupling model (1) with the following assumption, which we refer to as "strong additive separability":

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

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. Given (1) and (3), 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. Consequently, if $A1$ holds, σ obtained from estimation of (2) on sample S isolates the impact of intrauterine stress caused by the family rupture.

A2: Weak additive separability. Second, if instead 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, then we would interpret σ in (2) as capturing both the effect of physiological exposure to maternal stress and the differential impact of income during pregnancy relative to post-partum (which may interact with the stress exposure). This is equivalent to coupling model (1) with the following, less restrictive assumption, which we refer to as "weak additive separability":

$$\begin{aligned} RelativeDeath_i = \alpha_1 UteroStress_i^* \mathbf{1}[c \leq RelativeDeath < b]_i + \\ \alpha_2 UteroStress_i^* \mathbf{1}[c \leq RelativeDeath < b]_i^* Income_i + \alpha_3 Other_i + \varepsilon_i, \end{aligned} \tag{4}$$

and assuming that the new term is additively separable from any other income effects.

In Section 5, we examine whether there are any additional income effects stemming from the pre-natal period—that is, income effects that do not only operate through the post-natal environment—and find little evidence of their presence. We also examine a range of mechanisms other than maternal stress. As we discuss further in Section 5, all these tests support the interpretation of σ in (2) as largely capturing the impact of intrauterine stress exposure (though we, of course, cannot rule out all other mechanisms with certainty).

Causality Model (2) represents a causal relationship between *in utero* exposure to bereavement and child outcomes if, for all $i \in \{S\}$, $E(\mathbf{1}[c \leq RelativeDeath < b]_i \epsilon_i) = 0$. 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 (2), $\mathbf{1}[c \leq RelativeDeath < b]_i$, 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 date of birth is pre-determined at the relative's death date.

Consequently, instead of estimating equation (2), we estimate the following equation on the sample with $i \in \{S\}$:

$$y_{iymp} = \beta_0 + \beta_1 \mathbf{1}[c \leq RelativeDeath < e_b]_{iymp} + \psi_y + \phi_m + \rho_p + \mathbf{x'}_i\beta_{\mathbf{2}} + \nu_{iymp}, \tag{5}$$

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 model (5), 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 the mother's age at conception (five categories: < 20, $20 - 24$, $25 - 34$, > 35), the mother's education in the year prior to conception (four categories: <HS, HS diploma, some college, college+), indicators 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 due to family rupture through the uterine environment.[17]

In parts of our analysis, we also analyze pregnancy trimester- and month-specific impacts,

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

[17] Equation (5) represents a reduced-form relationship between a relative's death during the mother's *expected* length of the pregnancy and child outcomes. We also present some results from two-stage least squares (2SLS) specifications where we use $\mathbf{1}[c \leq RelativeDeath < e_b]$ to instrument for exposure to death during the mother's *actual* length of pregnancy. In these specifications, the first stage takes the form of:

$$\mathbf{1}[c \leq RelativeDeath < b]_{iymp} = \gamma_0 + \gamma_1 \mathbf{1}[c \leq RelativeDeath < e_b]_{iymp} + \eta_y + \epsilon_m + \theta_p + \mathbf{x'}_i\gamma_{\mathbf{2}} + \zeta_{iymp}, \tag{6}$$

with the 2SLS estimate given by $\hat{\beta}_1/\hat{\gamma}_1$.

replacing $\mathbf{1}[c \leq RelativeDeath < e_b]_{iymp}$ with indicator variables capturing whether the death occurred in the expected first, second, or third trimester or the expected first through ninth months of pregnancy, respectively.

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 timeframe 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 parental characteristics that are observed prior to conception: the mother's age, the father's age, first parity birth, the mother's marital status, each parent's educational attainment (indicators for below high school and college degree or higher), each parent's wage income, and an indicator for the mother being born outside Sweden.[18] As shown in Appendix Table A1, we find little evidence for a systematic relationship between parental characteristics and the occurrence of death during pregnancy.[19] Only two out of eleven coefficients are statistically significant—we find a positive correlation between treatment and 1st parity births and a negative correlation between treatment and the likelihood of the mother being foreign-born—and the magnitudes are relatively small when compared to sample means. The signs of the (insignificant) coefficients on the other parental characteristics such as education and income suggest that, if anything, higher socioeconomic status parents are more likely to experience a death during pregnancy than after childbirth, meaning that any selection into treatment would bias us *against* finding adverse effects on child outcomes.

As a second test of the identification assumption, we link our sample of children to their older siblings (if they exist), and test whether a younger child's *in utero* exposure to the death of a relative has any spurious impacts on his/her older sibling's birth outcomes.[20] In Appendix Table A2 we present results from these specifications where the older sibling's outcomes considered are: continuous birth weight (in grams) and indicators for low-birth-weight (less than 2,500 grams), very-low-birth-weight (less than 1,500 grams), and pre-term birth (less than 37 weeks gestation).

[18] We do not include father characteristics as controls in our main analysis as they are missing for some children in our sample and we want to maximize our sample size. However, results that include father characteristics as controls are generally very similar to those reported here.

[19] Since our analyses compare individuals who experience a relative death *in utero* to those who experience a relative death after birth while controlling for year-of-conception fixed effects, there is a mechanical correlation between the treatment variable and age of the relative—those who die during the mother's pregnancy are mechanically slightly younger than those who die in the year after childbirth. Thus, all of the regressions in Appendix Table A1 control for the relative's age and age squared.

[20] Siblings data are only available to us for children born in selected years: 1973, 1977, 1983, 1988, 1995, 1999, 2001, and 2005.

We test separately for placebo effects in the whole sample and in the sample limited to mothers who experience a parental or sibling death (as we focus on this sub-sample in some of our main analysis). On the whole, we find little evidence of a statistically significant relationship between a younger child's prenatal exposure to a relative's death and the older child's birth outcomes.

Finally, we also examine whether the distribution of relative death dates exhibits any non-random patterns surrounding the child's expected date of birth. One concern is that some relatives may be better able to "hold on" to life with the hopes of seeing the child's birth, implying that the characteristics of families experiencing a death after the child's expected birth date may be different from those of families experiencing a death while the child is still *in utero*. Appendix Figure A1 plots a histogram of the distribution of the distance in days between the relative's death date and the child's conception date. There is no visible sorting pattern around the expected birth date at 280 days post-conception (shown by the vertical red line).

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 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 and less anticipated 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 Table 1 presents summary statistics. 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.[21] 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 31 percent of mothers are married in the year prior to conception. The modal mother has a high school degree in the year before conception. Average birth weight is $3,544$ grams, with 3 percent of children born low-

[21] To estimate the date of conception, c, we subtract the number of gestation days from the date of birth, b.

birth-weight and 5 percent of children born pre-term. 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 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 2 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, 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 Appendix Table A3, 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.

To examine whether the effects are different depending on the severity of the stressful event, these tables are split into three panels. Panel A presents results for our entire analysis sample. Panel B limits the sample to children whose mothers lose *close* relatives, who are defined as those within one generation from the mother—a mother's sibling, a mother's parent, the child's father, or a mother's own older child (i.e., we drop grandparent deaths). Finally, Panel C further limits the sample to children whose mothers experience the death of a parent or a sibling (i.e., a sub-sample of the "close relative" group). The death of a maternal parent or sibling likely generates severe stress for the mother, but leads to fewer other changes to household resources and immediate family structure than the death of the child's father or the mother's own older child would.

Our estimates suggest that *in utero* stress due to family bereavement leads to a small negative effect on average birth weight of 11 grams. However, much of this effect is driven by impacts at the lower end of the birth weight distribution. Prenatally exposed infants are 12 percent more likely to be born low-birth-weight, and 24 percent more likely to be born very-low-birth-weight.

In contrast, there is only a 3 percent decline in the likelihood of a high-birth-weight birth. These children are also 12 percent more likely to be born pre-term, are 0.18 percent shorter, and have 0.1 percent smaller head circumference. The mothers are 3 percent more likely to have a c-section delivery. We find no statistically significant effects on stillbirths or deaths in the first 28 days of life.[22] Additionally, comparing the results across panels suggests that the effects of *in utero* exposure to the death of a relative are similar across different relative types.

In Figure 1 and Appendix Figure A2, we examine whether our estimated impacts are different across the nine months of pregnancy for low-birth-weight and pre-term births, respectively. The graphs present the coefficients (and 95% confidence intervals) from a single regression that includes indicators for exposure to the death of a relative in each of the 9 (expected) months of pregnancy, with the omitted category being exposure after 280 days (40 weeks) of gestation.

Both figures show positive coefficients on exposure to stress during most months of the pregnancy relative to post-partum, with slightly higher effects during the fourth month. In Appendix Tables A4 and A5 we also display trimester-specific effects on all of the birth outcomes. In general, however, the coefficients tend to be quite similar throughout the pregnancy, and with overlapping confidence intervals.

5.2 Physical Health Outcomes Beyond Birth

Having documented that exposure to family bereavement *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 by different ages. Our inpatient data exist for years 1964 to 2012 and thus allow us to study cumulative hospitalizations into adulthood.

Table 3 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 a 3 percent increase in the likelihood that a child is ever hospitalized by age one (column 1), and a 2 percent increase in the likelihood he/she is ever hospitalized by age five (column 3, although this latter coefficient is only marginally significant).[23] We explored in detail the diagnoses codes to try to understand which causes are driving these results and found that they are entirely driven by treatments for conditions originating in the perinatal period, as seen in columns 2 and 4 of Table 3.[24] As with

[22] 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.

[23] We also examined outpatient visits, and found suggestive evidence of similar increases in outpatient visits occurring by these ages, although we have less power due to smaller sample sizes in these analyses (outpatient data is only available for years 2001 to 2012). These results, as well as a description of the outpatient data, are available upon request.

[24] 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 categories of 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

the results on birth outcomes, we do not see substantial differences in effects across relative types (panels A to C). In Appendix Figures A3 and A4, and Appendix Table A6, we also present the results by month and trimester of pregnancy, respectively. The estimates suggest that the health effects may be stronger when exposure occurs during the first trimester, although we again cannot reject the null hypothesis that the coefficients are the same across different months of exposure.

Next, we turn to the prescription drug registry data. As described in Section 3, these data contain information about prescription drugs bought during 2005-2014. We create variables capturing the incidence of prescription drug consumption at different ages throughout childhood and adulthood. Specifically, we focus on drugs consumed around ages 5, 10, 15, 20, 25, 30, and 35. To reduce measurement error and maximize sample size, we focus on the consumption of prescription drugs in three-year age ranges centered around these multiples of five (e.g., ages 4 to 6, 9 to 11, etc.). While some individuals appear in the drug registry data at all three of the ages in a given range (e.g., children born in 2001 appear at ages 4, 5, and 6), others only appear at one or two of the ages (e.g., children born in 1999 appear at age 6 only). To calculate our outcomes, we include everyone who appears in the data at least at one of the ages in any given range.

Appendix Table A7 presents results on the effects of *in utero* exposure to a relative's death on the consumption of any drugs used to treat the following health conditions at the above age ranges: obesity, diabetes, Cushing's Syndrome, hypo- and hyperthyroidism, cholesterol, and heart conditions (i.e., beta blockers).[25] We find little evidence that exposure to a relative death during pregnancy increases the consumption of these prescription drugs at any of our observable ages. If anything, there may be a small negative effect on the consumption of these drugs at ages 24 to 26. 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 any more damaging than stress exposure shortly post-birth.

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 impacts seem to fade after early childhood—in addition to the null effects on the consumption of prescription drugs treating physical health conditions, we also find no effects on hospitalizations at later ages (see Appendix Table A8). Our results do not rule out the possibility of latent physical health consequences for individuals at older ages (Barker, 1990), though; our cohorts are too young to detect such effects.

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.

[25] Appendix B provides the exact ATC codes employed to associate prescription drugs to diagnoses.

5.3 Mental Health Outcomes

We next use the drug registry data to analyze effects on mental health. Figure 2 graphs the coefficients (and associated 95% confidence intervals in dashed vertical lines) from separate regressions where the outcomes are indicators for individuals consuming prescription drugs used to treat any of the mental health conditions described in Section 3 at 5-year age intervals. In sub-figure 2a, which plots the estimates for our entire sample, none of the coefficients is statistically significant. However, a pattern begins to emerge—mental health impacts seem more likely to arise in middle childhood (ages 9 to 11) and adulthood (ages 34 to 36). When we limit the sample to individuals whose mothers experience close relative deaths in sub-figure 2b, the pattern becomes more pronounced, with the coefficient for consuming mental health drugs at ages 9 to 11 now statistically significant. The pattern remains strong in sub-figure 2c when the sample is further limited to maternal parent and sibling deaths.

The above figures capture the incidence of purchasing any mental health drugs; we explore the specific conditions driving these results further in Table 4. In the close relative sample (panel B), we find that the mental health effects in middle childhood are driven primarily by increases in the consumption of ADHD medications—an 18 percent increase in the likelihood of ever purchasing a drug to treat ADHD and a 24 percent increase in the average daily dose. Among adults in their 30s, the effects are concentrated among anti-anxiety and depression medications—we see 11 and 7 percent increases in the likelihood of ever purchasing drugs to treat anxiety and depression, respectively; and 15 and 10 percent increases in the average daily doses of anti-anxiety and depression medications, respectively. Panel C shows that these effects still remain in the sub-sample further limited to individuals whose mothers lose a parent or a sibling. As with the impacts on the physical health outcomes, we fail to detect statistically significant differences in effects across pregnancy months of exposure (see Figure 3 for ADHD drug consumption among 9 to 11 year-olds and Figure 4 for anxiety and depression drug consumption among 34 to 36 year-olds).

The magnitudes of the coefficients indicate that the adverse mental health impacts of exposure to stress *in utero* are larger when the stress is more severe, as captured by the mother losing a closer relative. In contrast, we showed above that the physical health impacts are less sensitive to the severity of stress exposure.

Moreover, while the physical health effects seem to fade by age five, our results suggest that adverse mental health consequences are present among both elementary school aged children and adults in their thirties. Interestingly, however, we find no adverse effects among cohorts whose drug purchases we observe in their twenties.

Discussion and the Role of Schools in Detection To interpret these results, it is important to keep in mind that we do not observe whether drugs were *ever* consumed by certain ages; instead, we observe the prescription drug purchases of some cohorts (i.e., those born in the late 1990s and 2000s) during early and middle childhood, of other cohorts (i.e., those born in the late 1980s and

early 1990s) during high school, and of still others (i.e., those born in the 1970s and early 1980s) during adulthood. Consequently, we cannot rule out that the individuals whom we observe to consume prescription drugs treating anxiety and depression in their thirties also consumed similar drugs in their twenties. However, because all cohorts in our data display adverse physical health effects at birth, this line of reasoning raises the question of why we fail to detect significant effects on drugs treating anxiety and depression among cohorts observed in their twenties, especially given that the share of the Swedish population treated for these conditions has been increasing over time.

When it comes to ADHD, prescription drugs have only been readily available since 2002, when the first prescription drug with the active substance Methylphenidate was permitted for treatment of ADHD in children below age 18.[26] Though treatment rates were low during the first couple of years, the National Board of Health and Welfare has documented a continuous and substantial increase in the prescription rate of this substance since 2005 (Socialstyrelsen, 2012), which is the year when our prescription drug data begins. Thus, intuitively, the x-axes in Figure 2 indicate the age ranges of different cohorts during this "ADHD revolution." We find positive treatment effects on the consumption of ADHD drugs only for cohorts that were in elementary and middle school during this time period. This is consistent with the fact that the drugs were permitted for treatment of ADHD in children only, and not in adults. In practice, however, adults could obtain "off label" prescriptions (Socialstyrelsen, 2012); hence, the regulatory barriers cannot completely explain the absence of effects among adults.

Another possible interpretation of the fact that we only observe impacts on ADHD among school-aged children is that symptoms of ADHD vanish over time. This story is inconsistent, however, with evidence that treatment often continues for many years once it is commenced, indicating that symptoms may not disappear at the end of school age, even among individuals who are treated with the medications.[27] Thus, the absence of effects beyond school age may instead suggest that ADHD is more readily *detected* while children are in school.

In fact, this candidate interpretation relates to school financing rules: in Sweden, schools are financed at the municipal level—direct school fees imposed on parents are prohibited by law—and municipalities often offer schools extra transfers for pupils with special needs. Hence, these rules impose direct financial incentives on school principals and teachers to help parents detect, and commence treatment of, ADHD in their children.[28]

Of course, if all parents with children suffering from ADHD have full knowledge of this condition and seek medical evaluation and care, then the financial incentives of schools should not influence whether ADHD is detected. If, however, mental health conditions among children are imperfectly

[26] In Sweden, Methylphenidate is consumed by 89 percent of all individuals using any prescription drug treating ADHD, with trade names in the U.S. such as Concerta, Methylin, Ritalin, and Equasym XL.

[27] Among individuals in Sweden who begun treatment with an ADHD prescription drug in 2006, at the age of 18 to 24, approximately 50 percent remained on these drugs five years later. The figure is similar in all older age groups where treatment is begun before the age of 55 (Socialstyrelsen, 2012).

[28] All children attending elementary and middle school in Sweden go through free, yearly health check-ups through *Skolhalsovarden* (the School Health Care System), which is part of the public health care system.

detected by parents, then incentivizing a third party that has superior information—schools and the associated School Health Care System—to detect the condition may be important for detection and, ultimately, for treatment. Indeed, when we interact our treatment variable with the share of municipal resources allocated based on special education needs, we obtain a positive (albeit insignificant) coefficient, providing suggestive evidence of this mechanism.[29] This interpretation highlights a key distinction between mental and physical health: detection of mental health conditions such as ADHD is likely more sensitive to information than the detection of other physical ailments that are more readily observable.

If schools help parents detect mental health problems in their children, then the absence of significant effects on ADHD among individuals who were already out of school when the "ADHD revolution" took place may reflect the fact that their mental health issues were never identified in the first place (though possibly present). Once the child is out of school, the burden to seek medical help falls on the young adult or on his or her parents; without adequate information, the condition may go undetected for at least some time. This, too, would be consistent with the fact that mental health aliments "reappear" when we look at individuals who are observed in their thirties and find significant effects on the consumption of prescription drugs treating anxiety and depression.[30]

Severity of Stress Finally, we examine the mechanism through which a death of a close relative of the mother may induce greater stress than the death of a relative who is further away from the mother on the family tree. 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 closer relative 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 mother's parent's or sibling's age within the "maternal parent/sibling death" sample. In these regressions, we include an interaction with an indicator for the mother's parent or sibling being younger than 50 years at the time of death. The interaction coefficient is positive and significant for the purchases of ADHD drugs at ages 9 to 11, suggesting that our severity of stress measure may at least in part capture the magnitude of the deviation from the prior on the probability of the relative dying.[31]

[29] We use a 2012 cross-section of municipal shares devoted to special needs education. The results are available on request.

[30] Adults with ADHD are likely to have other mental health conditions such as anxiety and depression. See `http://www.adaa.org/understanding-anxiety/related-illnesses/other-related-conditions/adult-adhd` for more information.

[31] We have also estimated regressions interacting with continuous maternal parent's or sibling's age, and obtained similar results.

Summarizing the Results on Mental Health On the whole, these results suggest that experiencing a very 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 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 558 gram standard deviation in birth weight; thus, our estimated 11 gram decrease in birth weight corresponds to 0.02 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 $0.02 * 0.08 = 0.0016$ effect on the take-up of prescription drugs. In contrast, our estimates are several times larger, suggesting 0.007 to 0.009 increases in the take-up of drugs treating anxiety and depression at ages 34-36. 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 Adult Labor Market Outcomes

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

We have annual earnings data from 1990 to 2010. In Sweden, the average age of completion of the first university degree is 30, which is several years higher than the OECD average.[32] Stable employment is therefore best captured starting around age 30. Following our analysis of prescription drugs, we create variables capturing labor market outcomes in three-year age ranges: 29 to 31 and 34 to 36. We have two earnings measures, both of which include all employer-reported income

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

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.[33]

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.[34]

For completeness, we nonetheless present results on the impact of prenatal exposure to the death of a relative on these labor market outcomes. Appendix Table A10 shows that we do not find any impacts on earnings at either ages 29 to 31 or 34 to 36. However, for the above-mentioned reasons, we are hesitant to conclude that these outcomes are unaffected by prenatal stress.

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 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, developing hypertension, changing her eating habits, or adjusting her labor supply, then this may adversely affect the child. Additionally, if the mother has to travel to another location

[33]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.

[34]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.

as a result of the relative's death (e.g., to attend the funeral), and if she therefore must give birth in a different hospital than where she had planned to, then the child may be impacted by this sudden hospital change. In Appendix Table A11, we examine these potential mechanisms in more detail. We study whether *in utero* stress exposure is associated with the presence of "high-risk" factors, maternal smoking during pregnancy, pregnancy weight gain (in kilograms), an indicator for the child's hospital of birth being in a different municipality than the mother's municipality of residence (our proxy for unplanned travel), an indicator for the mother having any positive labor income during the year of conception, and the mother's labor income during the year of conception (in SEK). "High-risk" factors include the following conditions 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 A12, 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 parent's or sibling's death during pregnancy has a differential effect on maternal mental health relative to experiencing such a death post-childbirth.[35] 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, through a biological channel, from mother to fetus.

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 that the less restrictive assumption, that of weak additive separability, is appropriate. Then, our estimates would capture both the effect of physiological exposure to maternal stress and the differential impact of income during pregnancy 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 percent of all estates

[35] In these specifications, we study the incidence of consuming mental health medications at any point between 2005 and 2014 when our drug registry data are available (i.e., we do not limit to specific age ranges of the mother). We also examined all other mental health conditions and found no effects.

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 A13, 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 socioeconomic status of the mother. Appendix Table A13 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 that the impacts of *in utero* exposure to family bereavement are stronger for children of less-educated mothers.

Inheritances and the Severity of Stress Fourth, we find that some of the adverse mental health effects arise when the deceased is a close relative of the expectant mother (such as her parent or sibling), but not when we consider deaths of other more distant relatives (namely, grandparents). 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 closer 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. Appendix Table A14 displays these three sources of income following the death of a parent and grandparent, respectively, for the universe of deaths in Sweden occurring from 2002 to 2005.[36] The three leftmost columns display the average amount in 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 30,000 ($4,560) from a parent and SEK 7,000 ($1,064) from a grandparent.[37] The second relevant possibility to receive income in conjunction with a grandparent's passing is through a

[36] 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. We do not observe bequests or life insurance payouts from sibling deaths.

[37] 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.

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. 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) 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.

Two-Stage Least Squares Models As described in Section 4, our key treatment variable is an indicator for a relative's death occurring between the child's date of conception and the *expected* date of birth at 280 days after conception. However, we can also use this variable to instrument for exposure to death before the child's *actual* date of birth. Appendix Table A15 presents results from two-stage least squares (2SLS) specifications for our main outcomes of interest. As the instrument (relative death before expected birth date) is different from the actual exposure variable (relative death before actual birth date) for only about 1 percent of the individuals in our data, the first stage is very strong with a coefficient of around 0.97. The 2SLS results are quite similar to the main ones we present above.

"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 may affect child outcomes. We have already shown that this timing is generally uncorrelated with a variety of observable parental characteristics, and that there are no placebo effects on older siblings' birth outcomes. 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, Björklund and Holmlund (2011), who study the effect of parental death around age 18 on children's educational and labor market outcomes in Sweden. To find plausibly exogenous causes of deaths, Adda, Björklund and Holmlund (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.[38] Appendix Table A16 presents results for our main outcomes where we limit 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.[39]

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 31 outcomes analyzed in Tables 2 and 3, and Appendix Tables A3 and A7.[40] The mental health index consists of indicators for ever purchasing a mental health drug at ages 9 to 11 and ages 34 to 36, as well as $16 \times 2 = 32$ 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, sleep disorders, and Parkinson's disease) and per age group (9 to 11 and 34 to 36).

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 5 presents the results from our main specifications using the two indices as outcomes. We show results for all deaths as well as the maternal parent/sibling deaths for which we saw the strongest 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 the mother's parent or sibling.

[38] 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.

[39] 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, we would need to have a comparison group of children who do not experience a relative death surrounding the time of their birth. However, our sample contains only individuals who experience a relative death within a limited time frame of childbirth.

[40] The outcomes are: 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 hospitalizations by ages 1 and 5, any hospitalizations for perinatal causes by ages 1 and 5, any medication for any physical health condition, any medication 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, any medication for hypo- and hyperthyroidism, average dose for hypo- and hyperthyroidism medication, any medication for cholesterol, average dose for cholesterol medication, any beta blocker medication, average dose for beta blocker medication.

Maternal Responses to *In Utero* Shocks: Effects on Subsequent Fertility Finally, we study whether our *in utero* shock of interest is correlated with an important maternal behavioral response: fertility. This analysis is motivated by recent work studying parental responses to fetal shocks. For example, Halla and Zweimüller (2013) find that low-education Austrian mothers who were exposed to radiation fallout from the Chernobyl accident during pregnancy reduced their subsequent fertility. The authors interpret this response as a form of compensating behavior as the mothers were able to allocate more resources to the affected children.

We examine maternal fertility 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 negatively affects 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 ADHD during childhood and anxiety and depression in 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 7 percent decrease in the consumption of prescription drugs treating depression alone can be valued at $700

million per year.

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.[41] 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 poor women are subject to more stress than women who have more resources, our results suggest that fetal stress exposure may play a potentially important role in the intergenerational transmission of disadvantage. 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 mental health, especially among low-income populations.

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

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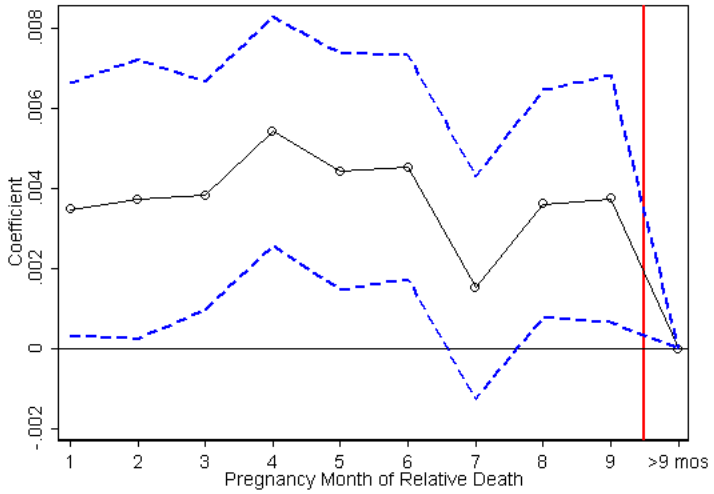
7 Figures

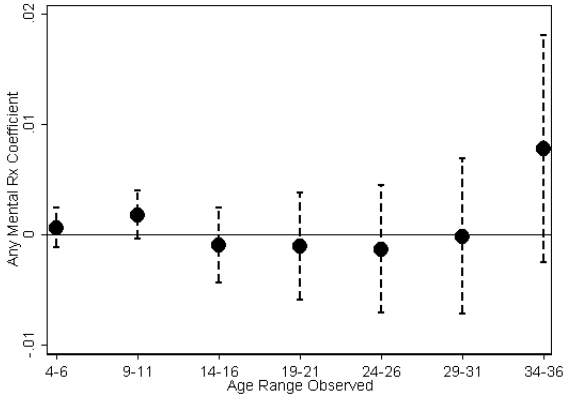
Figure 1: Effect of Relative Death on the Incidence of the Child Being Born Low-Birth-Weight



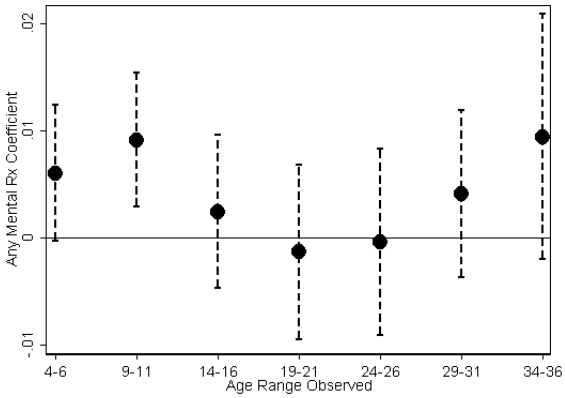
Notes: The sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within 280 days of the child's estimated date of conception or in the year after birth. To assign exposure to treatment, we first calculate each child's 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-9th months 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 2: Effect of Relative Death on the Incidence of the Child Consuming Any Mental Health Medications by Age

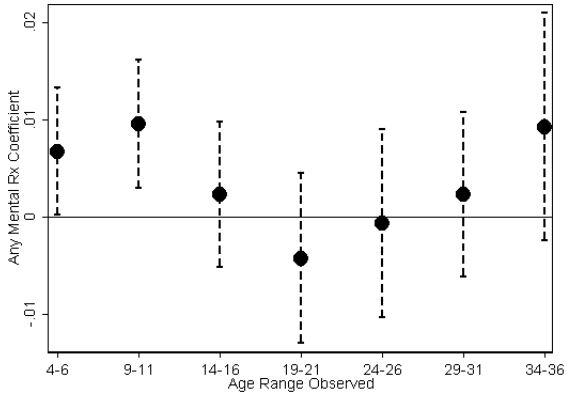
(a) All Deaths



(b) Close Relative Deaths

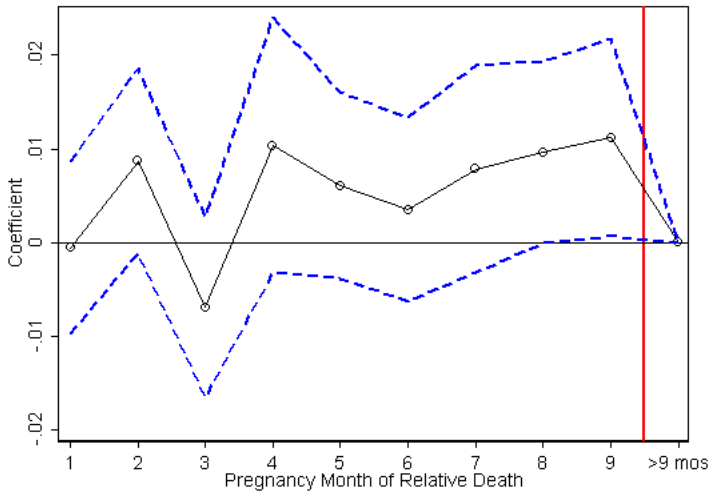


(c) Maternal Parent/Sibling Deaths



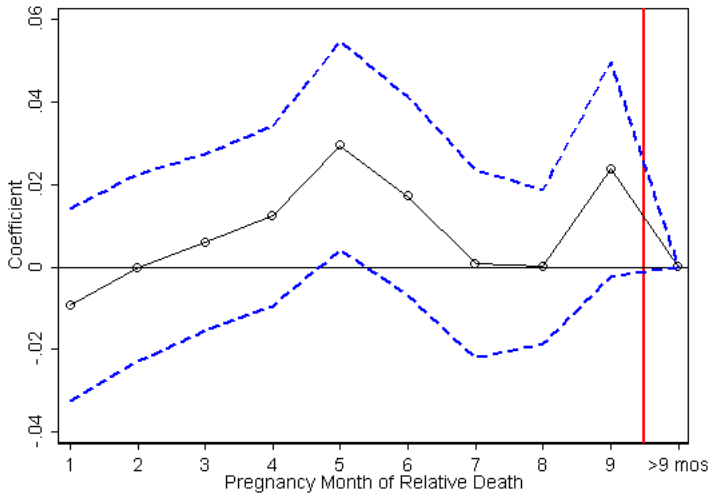
Notes: See notes under Figure 1 for more information on the sample. These figures plot the coefficients (and 95% confidence intervals in vertical lines) on the effects of the death of a relative on the likelihood that the child consumes any mental health medications at different age intervals. Each of the three panels present results from a sample including a certain set of relative deaths. Intuitively, the samples capture different degrees of proximity in the family tree between the expectant mother and the deceased, and hence different intensities of stress exposure.

Figure 3: Effect of Maternal Parent/Sibling Death on the Incidence of the Child Consuming Any ADHD Medications at Ages 9-11



Notes: The sample includes all children whose mother loses a parent or a sibling 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-9th months 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 ADHD at ages 9-11.

Figure 4: Effect of Maternal Parent/Sibling Death on the Incidence of the Child Consuming Any Anxiety or Depression Medications at Ages 34-36



Notes: The sample includes all children whose mother loses a parent or a sibling 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-9th months 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 anxiety or depression at ages 34-36.

8 Tables

Table 1: Summary Statistics

	(1) All	(2) Death During Preg.	(3) Death After Preg.
Mother's age at conception	27.88 (5.058)	27.92 (5.061)	27.86 (5.056)
Mother married pre-concep.	0.311 (0.463)	0.308 (0.462)	0.313 (0.464)
Mother's ed: <HS pre-concep.	0.177 (0.382)	0.174 (0.379)	0.179 (0.383)
Mother's ed: HS pre-concep.	0.314 (0.464)	0.308 (0.462)	0.318 (0.466)
Mother's ed: some college pre-concep.	0.202 (0.401)	0.205 (0.404)	0.199 (0.399)
Child's Birth Weight (g)	3543.9 (557.9)	3537.2 (564.7)	3549.0 (552.7)
Child is Low Birth Weight (<2500g)	0.0323 (0.177)	0.0346 (0.183)	0.0305 (0.172)
Child is Preterm (<37 weeks)	0.0497 (0.217)	0.0534 (0.225)	0.0469 (0.211)
Observations	296,557	127,406	169,151

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 2: Effects of Relative Death *In Utero* on Birth Outcomes

	(1) Birwt	(2) LBW	(3) VLBW	(4) HBW	(5) Pret.	(6) Stillb.	(7) Peri.Death
A. All Relative Deaths							
Death During Pregnancy	-11.47***	0.00392***	0.00124***	-0.00501***	0.00617***	-0.0000696	0.0000635
	[2.067]	[0.000633]	[0.000269]	[0.00150]	[0.000838]	[0.000160]	[0.000268]
Mean, dept. var	3546.3	0.0320	0.00511	0.188	0.0494	0.00166	0.00402
Obs.	288337	288337	288337	288337	289087	289087	289087
B. Close Relative Deaths							
Death During Pregnancy	-10.08***	0.00358**	0.000740	-0.00460*	0.00517***	-0.000141	0.000104
	[3.563]	[0.00140]	[0.000526]	[0.00258]	[0.00145]	[0.000268]	[0.000465]
Mean, dept. var	3523.0	0.0372	0.00603	0.179	0.0511	0.00166	0.00534
Obs.	84584	84584	84584	84584	84817	84817	84817
C. Maternal Parent/Sibling Deaths							
Death During Pregnancy	-10.76***	0.00420***	0.00119**	-0.00444*	0.00542***	-0.000166	0.000445
	[3.780]	[0.00146]	[0.000519]	[0.00265]	[0.00150]	[0.000279]	[0.000495]
Mean, dept. var	3525.8	0.0365	0.00576	0.180	0.0504	0.00174	0.00520
Obs.	80956	80956	80956	80956	81177	81177	81177

Note: See table 1 for more information on the sample of analysis. Each column in each panel is a separate regression. Panel A uses the entire sample of analysis. In Panel B, we drop children of mothers who experience the death of a grandparent. In Panel C, we only include children of mothers who experience the death of a parent or a sibling. All regressions include controls for the mother's age at conception (five categories: < 20, $20-24$, $25-34$, > 35), maternal 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 3: Effects of Relative Death *In Utero* on Hospitalizations by Ages 1 and 5

	By Age 1		By Age 5	
	(1) Any Hosp	(2) Any Hosp-Peri.	(3) Any Hosp	(4) Any Hosp-Peri.
A. All Relative Deaths				
Death During Pregnancy	0.00192**	0.00285***	0.00133	0.00288***
	[0.000924]	[0.000720]	[0.00122]	[0.000720]
Mean, dept. var	0.0737	0.0486	0.113	0.0488
Obs.	288606	288606	288606	288606
B. Close Relative Deaths				
Death During Pregnancy	0.00107	0.00238**	0.000831	0.00233**
	[0.00174]	[0.00109]	[0.00223]	[0.00109]
Mean, dept. var	0.0660	0.0347	0.105	0.0350
Obs.	84676	84676	84676	84676
C. Maternal Parent/Sibling Deaths				
Death During Pregnancy	0.00140	0.00247**	0.000645	0.00242**
	[0.00183]	[0.00111]	[0.00224]	[0.00111]
Mean, dept. var	0.0659	0.0348	0.105	0.0351
Obs.	81036	81036	81036	81036

Note: See tables 1 and 2 for more information on the sample and controls. "Any Hosp-Peri." refers to an indicator for ever being hospitalized for a condition originating in the perinatal period. 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 4: Effects of Relative Death *In Utero* on Prescription Use for Mental Health Conditions at Ages 9-11 and 34-36

	ADHD, Ages 9-11		Anxiety, Ages 34-36		Depression, Ages 34-36	
	(1) Any RX	(2) Avg. dose	(3) Any RX	(4) Avg. dose	(5) Any RX	(6) Avg. dose
A. All Relative Deaths						
Death During Pregnancy	0.00103	0.0476	0.00563*	0.0239	0.00577*	0.382*
	[0.000946]	[0.0323]	[0.00309]	[0.0181]	[0.00348]	[0.227]
Mean, dept. var	0.0228	0.660	0.0681	0.215	0.113	4.642
Obs.	129488	129488	31577	31577	31577	31577
B. Close Relative Deaths						
Death During Pregnancy	0.00620***	0.172**	0.00719**	0.0304	0.00736*	0.472*
	[0.0774]	[0.0440]	[0.00358]	[0.0210]	[0.00436]	[0.246]
Mean, dept. var	0.0244	0.713	0.0674	0.205	0.112	4.559
Obs.	20380	20380	22907	22907	22907	22907
C. Maternal Parent/Sibling Deaths						
Death During Pregnancy	0.00648***	0.169**	0.00864**	0.0390*	0.00915**	0.553**
	[0.00210]	[0.0811]	[0.00367]	[0.0223]	[0.00441]	[0.259]
Mean, dept. var	0.0238	0.702	0.0666	0.204	0.111	4.546
Obs.	19605	19605	21763	21763	21763	21763

Note: See tables 1 and 2 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 Appendix B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

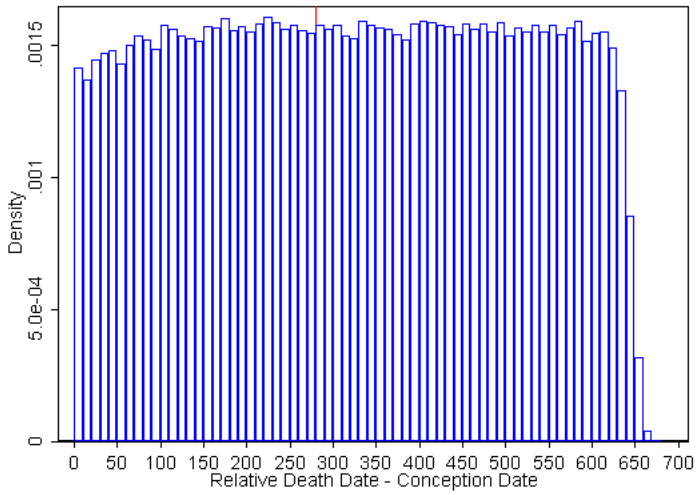
Table 5: Effects of Relative Death *In Utero* on Physical and Mental Health Indices

	Physical Health Index		Mental Health Index	
	(1) All Deaths	(2) Mom Parent/Sib	(3) All Deaths	(4) Mom Parent/Sib
Death During Pregnancy	-0.00685***	-0.00714***	-0.00313	-0.0117***
	[0.00141]	[0.00261]	[0.00210]	[0.00438]
Mean, dept. var	-0.00583	-0.0241	-0.000814	0.00238
Obs.	289087	81177	142547	41368

Note: See tables 1 and 2 for more information on the sample and controls. The physical health index consists of all the outcomes analyzed in Tables 2, A3, 3, and A7: 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 hospitalizations by ages 1 and 5, any hospitalizations for perinatal causes by ages 1 and 5, any medication for a physical health condition, any medication 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, any medication for hypo- and hyperthyroidism, average dose for hypo- and hyperthyroidism medication, any medication for cholesterol, average dose for cholesterol medication, any beta blocker medication, average dose for beta blocker medication. The mental health index consists of indicators for ever purchasing a mental health drug at ages 9-11 and ages 34-36, as well as $16 \times 2 = 32$ 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, sleep disorders, and Parkinson's disease) and per age group (9-11 and 34-36). 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

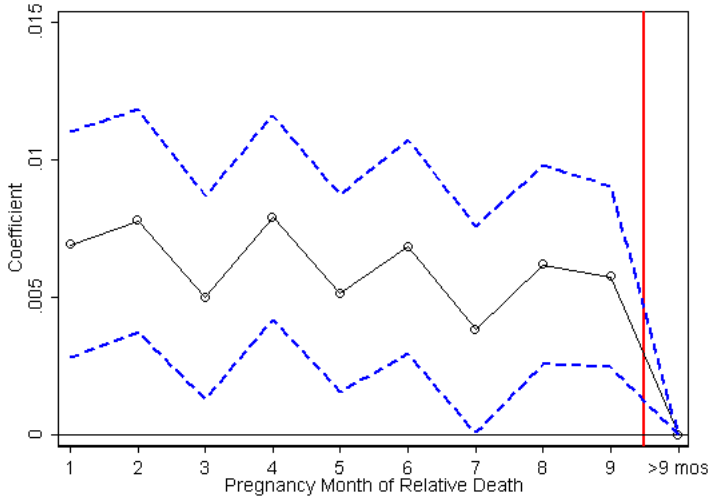
A Additional Results

Figure A1: Distribution of Relative Death Dates Around Child's Expected Birth Date



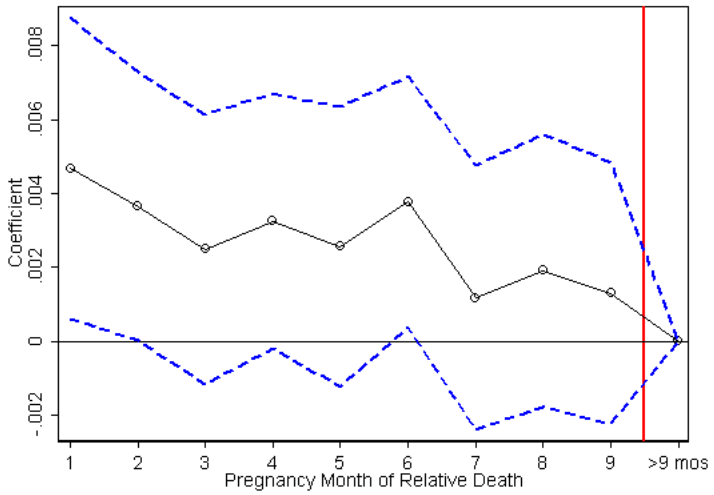
Notes: The sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within 280 days of the child's estimated date of conception or in the year after birth. The graph plots a histogram of the distribution of the distance in days between the relative death date and the child's conception date. The vertical red line depicts the expected birth date at 280 days post-conception.

Figure A2: Effect of Relative Death on the Incidence of the Child Being Born Pre-term



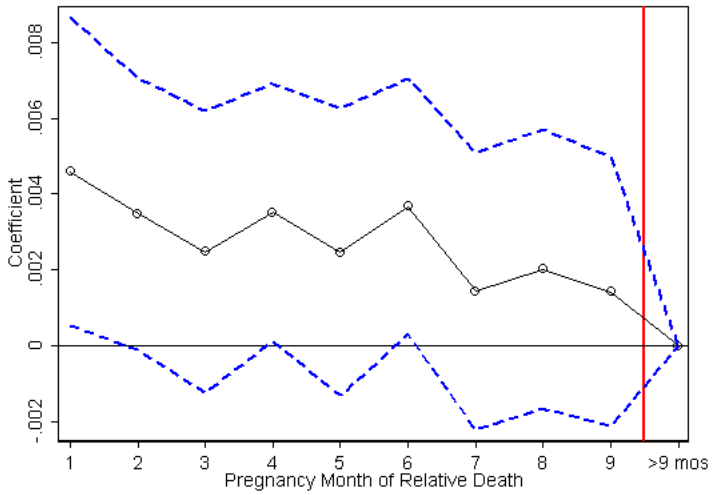
Notes: The sample includes all children whose mother loses a family member—a sibling, a parent, a grandparent, the child's father, or an own (older) child—within 280 days of the child's estimated date of conception or in the year after birth. To assign exposure to treatment, we first calculate each child's 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-9th months 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 pre-term.

Figure A3: 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-9th months 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 A4: 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-9th months 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.

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

	(1) M.Age	(2) F.Age	(3) 1st Par.	(4) M.Mar.	(5) M.Ed:<HS	(6) M.Ed:College+	(7) F.Ed:<HS	(8) F.Ed:College+	(9) M. Wage	(10) F. Wage	(11) M. Foreign
Death During Pregnancy	-0.0103	-0.00854	0.0133***	-0.00201	-0.00111	0.00197	-0.000751	0.0000713	388.3	1022.6	-0.00156***
	[0.0155]	[0.0203]	[0.00188]	[0.00177]	[0.00137]	[0.00161]	[0.00154]	[0.00148]	[489.5]	[666.2]	[0.000482]
Mean, dept. var	27.88	30.53	0.496	0.311	0.177	0.307	0.193	0.269	124317.5	208987.8	0.0216
Obs.	295678	293497	295678	295678	289087	289087	278483	278483	191074	187081	295678

Note: See table 1 for more information on the sample. This table reports the correlation between exposure to relative death during pregnancy and parental characteristics measured prior to conception. "M." denotes mothers' characteristics, while "F." denotes fathers' characteristics. 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: Placebo Effects of Relative Death During Pregnancy on *Older* Sibling's Birth Outcomes

	All Deaths				Mom Parent/Sib Deaths			
	(1) Birwt	(2) LBW	(3) VLBW	(4) Pret.	(5) Birwt	(6) LBW	(7) VLBW	(8) Pret.
Death during sib's gestation	-8.236	0.000577	0.000483	-0.00103	-10.32	0.00114	0.00288*	-0.00378
	[7.124]	[0.00233]	[0.000756]	[0.00243]	[11.82]	[0.00384]	[0.00151]	[0.00503]
Mean, dept. var	3517.0	0.0314	0.00427	0.0505	3496.2	0.0311	0.00451	0.0479
Obs.	34665	34665	34665	34767	11303	11303	11303	11332

Note: See table 1 for more information on the sample. In this table we link all of the children in our analysis sample to their older siblings (if they exist). Siblings data is only available for children born in years 1973, 1977, 1983, 1988, 1995, 1999, 2001, and 2005. The table reports the coefficients on the (placebo) effects of a relative death during the younger child's gestation on the older sibling's birth outcomes. All regressions control for fixed effects for the younger child's 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 A3: Effects of Relative Death *In Utero* on Additional Birth Outcomes

	(1) SGA	(2) LGA	(3) Length	(4) Head	(5) C-sect	(6) Induced	(7) Rupt.
A. All Relative Deaths							
Death During Pregnancy	0.000603	0.000184	-0.0449***	-0.0352***	0.00388***	-0.00108	-0.00384
	[0.000623]	[0.000708]	[0.00941]	[0.00602]	[0.00125]	[0.00102]	[0.00289]
Mean, dept. var	0.0267	0.0336	50.46	34.82	0.128	0.0701	0.593
Obs.	288334	288334	286026	278395	289087	289087	120583
B. Close Relative Deaths							
Death During Pregnancy	0.000225	-0.000324	-0.0377**	-0.0352***	0.00542**	0.00132	-0.00155
	[0.00116]	[0.00124]	[0.0162]	[0.0105]	[0.00219]	[0.00155]	[0.00703]
Mean, dept. var	0.0348	0.0348	50.40	34.76	0.131	0.0472	0.550
Obs.	84584	84584	84016	82300	84817	84817	18424
C. Maternal Parent/Sibling Deaths							
Death During Pregnancy	0.0000839	-0.000228	-0.0408**	-0.0368***	0.00452**	0.00115	-0.00273
	[0.00122]	[0.00129]	[0.0170]	[0.0106]	[0.00221]	[0.00156]	[0.00710]
Mean, dept. var	0.0345	0.0348	50.41	34.76	0.130	0.0474	0.553
Obs.	80956	80956	80427	78778	81177	81177	17688

Note: See tables 1 and 2 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 Birth Outcomes: Results by Trimester

	(1) Birwt	(2) LBW	(3) VLBW	(4) HBW	(5) Pret.	(6) Stillb.	(7) Peri.Death
Death in 1st Trimester	-11.93***	0.00382***	0.00131***	-0.00517**	0.00652***	0.0000119	-0.000151
	[3.376]	[0.000939]	[0.000470]	[0.00236]	[0.00144]	[0.000225]	[0.000317]
Death in 2nd Trimester	-10.69***	0.00450***	0.000854**	-0.00539***	0.00653***	0.0000332	0.000326
	[2.563]	[0.000902]	[0.000400]	[0.00191]	[0.00122]	[0.000225]	[0.000390]
Death in 3rd Trimester	-11.79***	0.00349***	0.00154***	-0.00452**	0.00553***	-0.000235	0.0000110
	[2.925]	[0.000965]	[0.000349]	[0.00204]	[0.00117]	[0.000239]	[0.000373]
Mean, dept. var	3546.3	0.0320	0.00511	0.188	0.0494	0.00166	0.00402
Obs.	288337	288337	288337	288337	289087	289087	289087

Note: See tables 1 and 2 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 Additional Birth Outcomes: Results by Trimester

	(1) SGA	(2) LGA	(3) Length	(4) Head	(5) C-sect	(6) Induced	(7) Rupt.
Death in 1st Trimester	0.000846	0.00134	-0.0382***	-0.0409***	0.00212	-0.00309**	0.00359
	[0.000929]	[0.000964]	[0.0142]	[0.0101]	[0.00200]	[0.00143]	[0.00377]
Death in 2nd Trimester	0.000675	-0.000291	-0.0325***	-0.0253***	0.00493***	-0.00189	-0.00732
	[0.000930]	[0.000978]	[0.0116]	[0.00845]	[0.00177]	[0.00134]	[0.00450]
Death in 3rd Trimester	0.000325	-0.000396	-0.0622***	-0.0394***	0.00445**	0.00143	-0.00723**
	[0.000758]	[0.00108]	[0.0131]	[0.00818]	[0.00178]	[0.00162]	[0.00332]
Mean, dept. var	0.0267	0.0336	50.46	34.82	0.128	0.0701	0.593
Obs.	288334	288334	286026	278395	289087	289087	120583

Note: See tables 1 and 2 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 Hospitalizations by Ages 1 and 5: Results by Trimester

	By Age 1		By Age 5	
	(1) Any Hosp	(2) Any Hosp-Peri.	(3) Any Hosp	(4) Any Hosp-Peri.
Death in 1st Trimester	0.00360**	0.00389***	0.00237	0.00379***
	[0.00154]	[0.00119]	[0.00176]	[0.00119]
Death in 2nd Trimester	0.00164	0.00275**	0.00131	0.00277**
	[0.00134]	[0.00113]	[0.00169]	[0.00113]
Death in 3rd Trimester	0.000703	0.00205**	0.000427	0.00218**
	[0.00138]	[0.00103]	[0.00173]	[0.00105]
Mean, dept. var	0.0737	0.0486	0.113	0.0488
Obs.	288606	288606	288606	288606

Note: See tables 1 and 2 for more information on the sample and controls. "Any Hosp-Peri." refers to an indicator for ever being hospitalized for a condition originating in the perinatal period. 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 A7: Effects of Relative Death *In Utero* on Prescription Use for Physical Health Conditions (Obesity, Diabetes, Cushing's Syndrome, Hypo- & Hyperthyroidism, Cholesterol, and Beta Blockers) by Age

	Any Physical Health Prescriptions at Ages...						
	(1) 4-6	(2) 9-11	(3) 14-16	(4) 19-21	(5) 24-26	(6) 29-31	(7) 34-36
A. All Relative Deaths							
Death During Pregnancy	0.000123	-0.000296	-0.000196	-0.0000335	-0.00245*	-0.00120	0.00473
	[0.000344]	[0.000507]	[0.000618]	[0.000853]	[0.00136]	[0.00211]	[0.00302]
Mean, dept. var	0.00439	0.00904	0.0156	0.0240	0.0358	0.0517	0.0703
Obs.	126736	129488	129161	114881	79498	54198	31577
B. Close Relative Deaths							
Death During Pregnancy	0.0000646	-0.000694	-0.000644	-0.000756	-0.00432**	-0.0000882	0.00438
	[0.000984]	[0.00138]	[0.00171]	[0.00154]	[0.00195]	[0.00277]	[0.00322]
Mean, dept. var	0.00465	0.00931	0.0153	0.0240	0.0346	0.0508	0.0711
Obs.	19582	23096	29433	35364	36308	37140	26305
C. Maternal Parent/Sibling Deaths							
Death During Pregnancy	0.000397	-0.000525	-0.000274	-0.00134	-0.00400*	0.0000956	0.00611*
	[0.000940]	[0.00142]	[0.00177]	[0.00161]	[0.00212]	[0.00302]	[0.00338]
Mean, dept. var	0.00417	0.00882	0.0154	0.0242	0.0349	0.0502	0.0706
Obs.	16561	19605	24754	29626	30266	30863	21763

Note: See tables 1 and 2 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 Appendix B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A8: 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	0.0000762	-0.00811	0.00305	0.00920	0.00222	0.0273
	[0.00184]	[0.00847]	[0.00262]	[0.0147]	[0.00444]	[0.0303]
Mean, dept. var	0.177	0.320	0.268	0.527	0.429	1.010
Obs.	186427	186427	111632	111632	40384	40384

Note: See tables 1 and 2 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 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?

	ADHD, Ages 9-11		Anxiety, Ages 34-36		Depression, Ages 34-36	
	(1) Any Rx	(2) Avg. dose	(3) Any RX	(4) Avg. dose	(5) Any RX	(6) Avg. dose
Death During Pregnancy	0.00287	0.0649	0.00629*	0.0306	0.00730	0.471*
	[0.00217]	[0.0822]	[0.00371]	[0.0226]	[0.00459]	[0.276]
Maternal Par/Sib Age Less 50	-0.0114*	-0.478*	-0.00411	0.0653	-0.0156	-0.606
	[0.00620]	[0.244]	[0.0124]	[0.108]	[0.0159]	[0.703]
Maternal Par/Sib Age Less 50*Death During Pregnancy	0.0281***	0.806***	0.0203	0.0743	0.0158	0.703
	[0.00713]	[0.286]	[0.0137]	[0.110]	[0.0144]	[0.726]
Mean, dept. var	0.0238	0.702	0.0666	0.204	0.111	4.546
Obs.	19605	19605	21763	21763	21763	21763

Note: See tables 1 and 2 for more information on the sample and controls. The sample here is further limited to only mothers who experience the death of a parent or a sibling. 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 Appendix B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A10: Effects of Relative Death *In Utero* on Adult Labor Market Outcomes

	Income, Age 29-31				Income, Age 34-36			
	(1) Any Wage	(2) Log Wage	(3) Any Lab.	(4) Log Lab.	(5) Any Wage	(6) Log Wage	(7) Any Lab.	(8) Log Lab.
A. All Relative Deaths								
Death During Pregnancy	0.00302	-0.00960	0.00174	-0.00683	-0.00232	0.0207	-0.00162	0.0158
	[0.00326]	[0.0111]	[0.00297]	[0.00960]	[0.00523]	[0.0177]	[0.00367]	[0.0128]
Mean, dept. var	0.924	11.99	0.949	12.15	0.921	12.26	0.951	12.40
Obs.	30799	28447	30799	29237	12938	11920	12938	12306
B. Close Relative Deaths								
Death During Pregnancy	0.00247	-0.00713	0.00137	-0.0111	-0.00315	0.0213	-0.00104	0.0153
	[0.00408]	[0.0131]	[0.00385]	[0.0112]	[0.00521]	[0.0179]	[0.00379]	[0.0138]
Mean, dept. var	0.926	12.00	0.951	12.16	0.922	12.27	0.951	12.40
Obs.	24723	22903	24723	23504	11697	10782	11697	11128
C. Maternal Parent/Sibling Deaths								
Death During Pregnancy	0.00104	-0.00988	0.000405	-0.0162	-0.00518	0.0203	-0.00248	0.0115
	[0.00426]	[0.0136]	[0.00404]	[0.0117]	[0.00527]	[0.0185]	[0.00375]	[0.0148]
Mean, dept. var	0.927	12.01	0.951	12.17	0.923	12.28	0.952	12.41
Obs.	23536	21821	23536	22391	11105	10249	11105	10575

Note: See tables 1 and 2 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 Maternal Pregnancy Behaviors and Characteristics

	(1) Highrisk	(2) Smoked	(3) Preg. Wgt Gain (kg)	(4) Hosp. not in Muni.	(5) Any Lab. Inc.	(6) Lab. Inc.
Death During Pregnancy	-0.00150	-0.0000469	-0.0162	0.000534	-0.00111	-177.7
	[0.00147]	[0.00143]	[0.0331]	[0.00103]	[0.000813]	[386.6]
Mean, dept. var	0.166	0.0869	13.96	0.117	0.971	158916.6
Obs.	289087	128393	101326	289087	199828	199828

Note: See tables 1 and 2 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 A12: Effects of Relative Death *In Utero* on the *Mother's* Prescription Use for Mental Health Conditions: Maternal Parent/Sibling Deaths 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	0.000164	-0.000272	-0.00161	-0.00363	0.00702	0.00318	0.0662
	[0.00335]	[0.000456]	[0.00951]	[0.00256]	[0.0129]	[0.00209]	[0.0746]
Mean, dept. var	0.335	0.00432	0.0514	0.109	0.230	0.139	2.922
Obs.	81036	81036	81036	81036	81036	81036	81036

Note: See tables 1 and 2 for more information on the sample and controls. The sample here is further limited to only mothers who experience the death of a parent or a sibling. 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 Appendix B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A13: 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 ADHD 9-11	(6) Any Anx/Dep 34-36
Death During Pregnancy	0.00372***	0.00536***	0.00310***	0.00307***	0.00481	0.00940
	[0.000817]	[0.00109]	[0.00107]	[0.00108]	[0.00293]	[0.00822]
Mom Low Ed (HS or less)	0.00853***	0.00759***	0.0100***	0.0101***	0.0101***	0.0178**
	[0.000929]	[0.00118]	[0.00127]	[0.00126]	[0.00383]	[0.00707]
Mom Low Ed*Death During Preg	-0.000135	0.00160	-0.000642	-0.000571	0.00244	-0.00268
	[0.00126]	[0.00165]	[0.00156]	[0.00157]	[0.00505]	[0.0103]
Mean, dept. var	0.0307	0.0483	0.0493	0.0496	0.0235	0.135
Obs.	272907	273597	273469	273469	18852	20387

Note: See tables 1 and 2 for more information on the sample and controls. In columns 5 and 6, the sample is further limited to children of mothers who experience the death of a parent or sibling. 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 Appendix B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A14: 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
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 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 A15: 2SLS Effects of Relative Death *In Utero* on Main Outcomes

	(1) LBW	(2) Pret.	(3) Any Per. Hosp. 1	(4) Any Per. Hosp. 5	(5) Any ADHD 9-11	(6) Any Anx/Dep 34-36
Death Before Childbirth	0.00404*** [0.000651]	0.00635*** [0.000862]	0.00294*** [0.000740]	0.00296*** [0.000740]	0.00667*** [0.00213]	0.00880* [0.00509]
Mean, dept. var	0.0320	0.0494	0.0486	0.0488	0.0238	0.136
First Stage Coef.	0.971	0.971	0.971	0.971	0.972	0.973
First Stage F-Stat	4732830.8	4745576.4	4745350.4	4745350.4	321520.3	358656.9
Obs.	288294	289044	288563	288563	19604	21715

Note: See tables 1 and 2 for more information on the sample and controls. In columns 5 and 6, the sample is further limited to children of mothers who experience the death of a parent or sibling. In these regressions, the explanatory variable is an indicator for the death of a relative occurring between a child's date of conception and date of birth. It is instrumented by an indicator for the death of a relative occurring between a child's date of conception and his *expected* date of birth (at 280 days post-conception). 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 Appendix B.
Significance levels: * p<0.1 ** p<0.05 *** p<0.01

Table A16: Effects of Relative Death *In Utero* on Main Outcomes: "Exogenous Deaths"

	(1) LBW	(2) Pret.	(3) Any Per. Hosp. 1	(4) Any Per. Hosp. 5	(5) Any ADHD 9-11	(6) Any Anx/Dep 34-36
Death During Pregnancy	0.00345***	0.00581***	0.00153	0.00160	0.0120**	0.00848*
	[0.00127]	[0.00145]	[0.00101]	[0.00102]	[0.00528]	[0.00502]
Mean, dept. var	0.0342	0.0498	0.0231	0.0233	0.0268	0.136
Obs.	90159	90470	90337	90337	3625	21746

Note: See tables 1 and 2 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. In columns 5 and 6, the sample is additionally limited to children of mothers who experience the death of a parent or sibling. 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) Mom Parent/Sib
Death During Pregnancy	0.00725**	-0.000994
	[0.00317]	[0.00633]
Mean, dept. var	0.563	0.457
Obs.	60068	17216

Note: See tables 1 and 2 for more information on the sample and controls. In this table we link all of the children in our analysis sample to their older siblings (if they exist). Siblings data is only available for children born in years 1973, 1977, 1983, 1988, 1995, 1999, 2001, and 2005. 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 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.