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Family Ruptures, Stress, and the Mental Health of the Next Generation[†]

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This paper studies how in utero exposure to maternal stress from family ruptures affects later 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 in adulthood. Further, family ruptures during pregnancy depress birth outcomes and raise the risk of perinatal complications necessitating hospitalization. Our results suggest large welfare gains from preventing fetal stress from family ruptures and possibly from economically induced stressors such as unemployment. They further suggest that greater stress exposure among the poor may partially explain the intergenerational persistence of poverty. (*JEL* I12, J12, J13)

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,

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[1]Since the acceptance of this article, it was brought to our attention that there are other related studies on this topic, in particular Class et al. (2011, 2014). At the end of the article, we discuss the relation of our work to these papers. This footnote, as well as the section on the relation of our paper to these earlier papers, has been written post-acceptance and reviewed by the handling Coeditor and Editor of the *American Economic Review*.

which is more than five times larger than the $1.7 billion market just a decade earlier (Visser et al. 2014). Estimates also suggest that mental illness accounts for over one-half of the rise in disability receipt among men in the last two decades (Duggan and Imberman 2009). Moreover, in Sweden (the setting for this paper), mental illness accounts for a larger share of health expenditures on prescription drugs than any other therapeutic class.[2]

The high and rapidly increasing incidence of mental conditions such as depression, anxiety, ADHD, and autism-spectrum disorders has prompted fervent debate 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 I, 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 reemerge in Europe and the United States 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, Fenske, and Nyshadham forthcoming).[3] 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 United States and Sweden, and by tracing health outcomes throughout the time period between the fetal shock and adulthood.

Our emphasis on stress is influenced by a growing literature documenting persistent intergenerational transmission of socioeconomic status (see, e.g., Solon 2001 and Chetty et al. 2014 for evidence from the United States, and Boserup, Kreiner, and Kopczuk 2014 for evidence from Scandinavia). As low socioeconomic status women experience higher levels of stress than their more advantaged counterparts,[4]

[2] See Table 11 in Socialstyrelsen (2013) for Sweden's health expenditures by therapeutic class.

[3] 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, Hoek, and Susser 1999; McClellan, Susser, and King 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.

[4] 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, Kirschbaum, and Steptoe 2004; Cohen, Doyle, and Baum 2006).

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.[5] 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.

To investigate whether the uterine environment propagates the impact of stress to the unborn child, we leverage administrative data from Sweden. As we detail in Section II, 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.[6] 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 III. 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 postnatal 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 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 predetermined 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.

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.[7] As presented in Section IV, we find that in utero exposure

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

[6] This measure is motivated by a psychological literature, which documents that losses of closer family members induce greater levels of self-reported grief and produce stronger cortisol responses (see, e.g., Segal and Bouchard 1993; O'Connor et al. 2012).

[7] Here, we reference the existing literature on humans, which we discuss further in Section I. 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, Sekl, and Homes (2001).

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 a 25 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 13 and 8 percent increases in the likelihood of consuming prescription drugs for anxiety and depression, respectively, as well as 19 and 12 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 year of life.

Our analysis is most closely related to recent work by Black, Devereux, and Salvanes (2016) in Norway, who study the impacts of deaths of maternal parents during pregnancy using a sibling fixed effects methodology. They find small adverse effects on birth outcomes, but no effects on adult body mass index (BMI), educational attainment, or labor market outcomes. Our paper is complementary as we show that, despite the limited impacts on physical health or adult economic outcomes, there are important consequences of in utero exposure to maternal bereavement for childhood and adult mental health. Additionally, by including relatives other than maternal parents in our empirical design, we are able to create a novel measure of the severity of antenatal stress exposure, which we find to be especially relevant for the mental health analysis. Finally, our methodology is slightly different from the main strategy employed by Black, Devereux, and Salvanes (2016): 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.

In sum, our results show that the death of a relative up to three generations apart during pregnancy has far-reaching consequences for physical health at birth and in the first year of life, as well as for mental health during childhood and adulthood. A number of medical studies show that the loss of a loved one is associated with a physiological response in the human body characterized by an increase in the level of the stress hormone cortisol (Irwin et al. 1988; Pfeffer et al. 2007; Dietz et al. 2013; Holland et al. 2014). While it is impossible to rule out all other mechanisms aside from in utero exposure to maternal grief-induced stress, we provide evidence against key alternative explanations such as changes in maternal behaviors (e.g., smoking and weight gain), or physical health conditions (e.g., hypertension), or adverse income effects that might produce separate insults to child health. Our findings suggest large general welfare gains from preventing fetal exposure to severe stress: for example, based on the 2008 figure for the US market, the 8 percent

decrease in the consumption of prescription drugs treating depression alone can be valued at around $800 million annually.

While we recognize that stress from grief is in some ways different from stress induced by economic hardship (e.g., as a result of unemployment or poverty), we believe that our findings may nevertheless be applicable to understanding how economic sources of stress could have intergenerational impacts on mental health. In Section V, we conduct a back-of-the-envelope calculation to understand how exposure to maternal economically induced stress during the fetal stage might affect the mental well-being of the next generation by relying on past research estimating cortisol responses to grief (Irwin et al. 1988; Pfeffer et al. 2007; Dietz et al. 2013; Holland et al. 2014) and to economic shocks like unemployment and poverty (Arnetz et al. 1991; Haushofer and Shapiro 2013). Our calculation suggests that in utero exposure to stress from unemployment may lead to a 17.3 percent increase in the likelihood of ever purchasing a drug to treat ADHD in middle childhood, and 9 and 5.5 percent increases in the likelihoods of ever purchasing drugs to treat anxiety and depression in adulthood, respectively.

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.

I. Hypotheses

The primary contribution of this paper is to shed light on the *mental* health effects of fetal exposure to maternal stress. In this section, we discuss our hypotheses regarding the expected effects on mental health outcomes, as well as the expected timing of the onset of these effects. Our analysis also considers the impacts on physical health at birth and later in life, and analyzes differential effects across gestational age at exposure and with respect to the severity of stress. We provide a brief description of our hypotheses regarding these other impacts; for a longer discussion, see online Appendix B.

A. *Mental Health Outcomes*

The existing evidence on the mental health effects of fetal stress exposure is extremely limited. We are only aware of two recent studies in economics that show that malnutrition in utero may lead to mental and learning disabilities later in life (Almond and Mazumder 2011; Adhvaryu, Fenske, and Nyshadham forthcoming). Both papers focus on adult measures of mental health and neither investigates more precisely where in the life cycle these effects appear.

Further, to the best of our knowledge, no existing study in economics analyzes the impact of stress during the fetal stage, or, more generally, of any in utero shock, on mental health in *childhood*. Our focus on stress is most closely related to the work

of Aizer, Stroud, and Buka (2016), 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.[8] Some of these effects on cognition could potentially be driven by mental health issues, consistent with psychiatric studies showing a correlation between cognitive impairment and the use of ADHD prescription drugs (Simon et al. 2000).

Outside of economics, there is more direct evidence on correlations between mental illness in childhood and adverse conditions during the fetal stage. For instance, recent neuroscientific research traces the origins of depression and autism-spectrum diseases such as ADHD to the fetal period (Liu et al. 2012; Berquin et al. 1998; Stoner et al. 2014). Other epidemiological studies have also established a correlation between mothers' cortisol levels during pregnancy and their children's mental health.[9] Related, Malaspina et al. (2008) provide evidence that exposure to the Six-Day Arab-Israeli War in utero increased the likelihood of developing schizophrenia by age 30.[10]

Thus, taken together, while credible causal evidence on the impact of early-life shocks on mental health is scant, existing evidence does suggest that we may expect mental health effects both in childhood and adulthood. Our analysis specifically focuses on three conditions: ADHD, anxiety, and depression. We focus on ADHD in childhood because it is the most prevalent mental health condition among children in Sweden that can be measured by drug consumption (as well as in many other developed countries like the United States) (Socialstyrelsen 2015), and since medical research has determined that environmental influences including fetal stress exposure are important for its etiology (Berquin et al. 1998; Van den Bergh and Marcoen 2004; Van den Bergh et al. 2005). For adults, we study depression and anxiety, which are also some of the most common mental illnesses in Sweden (Socialstyrelsen 2013), and which have been shown to be related to ADHD diagnosis in childhood.[11]

B. *Timing of the Onset of Mental Health Effects*

Importantly, our data allow us to try to pin down when in the life cycle potential mental health effects appear. Since our analysis uses Swedish prescription registry data to measure these effects, we discuss here the specific institutional context that informs the pattern of results we may expect.

When it comes to ADHD, prescription drugs have only been readily available since 2002 in Sweden, when the first prescription drug with the active substance Methylphenidate was permitted for treatment of ADHD in children below age 18.[12]

[8] 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.

[9] See online Appendix F for details.

[10] An important limitation of this empirical design is that it precludes the isolation of fetal exposure to stress from the other consequences of the war, such as its economic repercussions.

[11] Tables 7, 8, and 12 in Socialstyrelsen (2013) show that depression and anxiety are the most prevalent conditions treated by pharmaceuticals for neurological conditions, after painkillers and sleeping pills. See http://www.adaa.org/understanding-anxiety/related-illnesses/other-related-conditions/adult-adhd for more information on the relationship between ADHD and anxiety and depression.

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

Though treatment rates were low during the first couple of years, Sweden's National Board of Health and Welfare (NBHW) 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 begin.

The NBHW has also documented that both prevalence (share treated) and incidence (share initiating treatment) are highest among individuals aged 10–17 years old during the time period covered by our prescription drug data (Socialstyrelsen 2015).[13] These ages coincide with the end of primary school and the entirety of middle school in Sweden.

The fact that initiation of prescription drugs treating ADHD is most common at these school ages may be explained by the structure of the Swedish school health care system (*Skolhälsovården*). All children attending primary and middle school in Sweden go through free annual health check-ups. Further, according to the most recent guidelines issued by the NBHW in 2002 (Socialstyrelsen 2002), there is a particularly detailed health check-up in grade four (age ten) at which each child's concentration skills and mental health are evaluated. The guidelines also state that all students have the right to further evaluations, and to get help with any mental or concentration issues that are detected at the age of ten.

Additionally, there is reason to believe that Sweden's school financing rules give schools a direct economic incentive to help detect and initiate treatment of children's mental health problems.[14] For example, Hjörne (2012) argues that most evaluations of whether a child has ADHD are initiated by teachers or schools, who alert parents of problems and suggest further evaluation. In sum, given that all children are screened for mental health issues at age ten and the schools' direct incentives in promoting ADHD treatment, it is plausible that the detection of any consequences of in utero stress on ADHD may appear around that age in our data.

With regard to anxiety and depression, the other mental health conditions we focus on, there are fewer specific institutional factors that might guide our expectations. In general, according to the NBHW, nationwide prevalence of prescription drugs treating anxiety and depression in Sweden is higher in older age groups (Socialstyrelsen 2013).[15] This pattern may suggest that detection of any consequences of in utero stress on anxiety and depression may appear at relatively old ages in our sample.

C. *Other Hypotheses*

When it comes to the expected impacts of fetal stress exposure on birth outcomes and physical health in later life, we draw on the large existing literature that points to adverse short- and long-term effects of exposure to *physical* insults during the fetal period (see Almond and Currie 2011 for a review).[16] The evidence on

[13] The considered age groups are 5–9; 10–17; 18–24; 25–34; 45–54; and 55–64.

[14] 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.

[15] See Table 72 for anxiety and Table 74 for depression.

[16] See also, e.g.,Van den Berg, Lindeboom, and Portrait (2006); Almond et al. (2010); Hoynes, Page, and Stevens (2011); Almond, Hoynes, and Schanzenbach (2011); Almond and Mazumder (2013); Hoynes, Schanzenbach, and Almond (2016); Scholte, Van Den Berg, and Lindeboom (2015); Rossin-Slater (2013) on malnutrition; Almond

the consequences of purely *psychological* stressors is more scarce, as studies that exploit variation from extreme and rare events like natural disasters and terrorist attacks are limited in their ability to separate the effects of in utero stress exposure from any postnatal responses, as well as from the physical health and economic insults associated with these events.[17] Our empirical methodology (described in detail in Section III) focuses on a nearly universal stressor designed to overcome these limitations.

An important caveat to the analysis of long-run physical health is that our cohorts, whom we can only follow into their thirties, may be too young to detect any effects on conditions such as obesity and diabetes. Indeed, evidence in support of David J. Barker's "fetal origins hypothesis" (Barker 1990), which argues that poor conditions in utero can lead to latent effects on disease much later in life, comes from studies of adults who are much older than the individuals in our sample.[18]

Additionally, throughout the paper, we explore whether there are any differential effects of exposure to maternal stress across different months or trimesters of pregnancy. The existing literature does not provide a clear picture of whether we should expect in utero exposure to maternal stress to have differential effects across gestational age at the time of shock. While some studies find differential effects with respect to gestational age, other studies, including some that are most closely related to ours (Almond and Mazumder 2011; Mansour and Rees 2012; Currie and Rossin-Slater 2013; Black, Devereux, and Salvanes 2016), fail to find such heterogeneity.

Finally, in contrast with the abundance of studies estimating differential effects across gestational age at the time of shock, the existing literature provides relatively little guidance on whether we might expect to see heterogeneous effects with respect to the intensity of stress exposure. Most closely related to our paper, Aizer, Stroud, and Buka (2016) explore potential nonlinearities in the effect of stress by separately analyzing different quartile ranges of the maternal cortisol distribution. Interestingly, the effects on birth outcomes do not vary with the severity of stress exposure. By contrast, the adverse impacts on cognition, captured by child IQ at age seven and educational attainment, are the largest for the most severe stress. In fact, the effects on cognitive outcomes are not statistically significant in the linear specifications, but are instead driven entirely by the highest quartile of the maternal cortisol distribution. This evidence suggests that mental health and cognition outcomes may be more sensitive to the severity of stress exposure than birth outcomes.

II. 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 the mother) in the 40 weeks after their date of

(2006); Barreca (2010) on disease outbreaks; Almond, Edlund, and Palme (2009); Black et al. (2013) on radiation; Sanders (2012); and Isen, Rossin-Slater, and Walker (2017) on air pollution.

[17] See, for example, 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, 2001 (Berkowitz et al. 2003; Lederman et al. 2004; Lauderdale 2006; Eskenazi et al. 2007). Another recent paper uses in utero exposure to the Super Bowl to identify the effects of prenatal stress on birth outcomes (Duncan, Mansour, and Rees 2015).

[18] See, e.g., Susser and Lin (1992); Almond (2006); Hoynes, Schanzenbach, and Almond (2016).

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), the first date of prenatal care and the number of prenatal visits, Cesarean 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 prespecified 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.[19] 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 prespecified certain health conditions before undertaking any analysis.[20] First, we include all mental illnesses. We further prespecified the eight subcategories of mental disorders that were recently selected by the NBHW to track prevalence and prescription drug use (Socialstyrelsen 2012): ADHD, anxiety, depression, bipolar disorder, psychotic disorders, sleeping disorders, addiction, and Parkinson's disease. While we prespecified all eight subcategories for completeness, our analysis focuses on ADHD, anxiety, and depression, as we discussed in Section I.

Second, although our primary focus is mental health, we prespecified 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, cholesterol, neoplasms, and conditions originating in the perinatal period.[21] We include all of these for completeness, although

[19] 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.

[20] 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 prespecified ones in our analysis.

[21] We are grateful to Johannes Haushofer for help in compiling this list. See online Appendix E 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. Online Appendix F has details and references relating to the biological mechanisms through which stress affects human health.

our cohorts may be too young to detect any effects on physical health other than conditions originating in the perinatal period.[22]

III. Empirical Methodology

Our goal is to examine the causal link between antenatal exposure to the death of a family member and children's physical and mental well-being at birth and later in life. The loss of a relative is a traumatic event that induces acute and immediate stress in the expectant mother (Irwin et al. 1988; Pfeffer et al. 2007; Dietz et al. 2013; Holland et al. 2014). However, the occurrence of death is likely correlated with unobserved family characteristics. For example, some types of accidental deaths are 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 postnatal environment, and (ii) nonrandom 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 postnatal consequences of the relative's passing, only the former group is exposed *through the uterine environment*.

A. *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 postnatal consequences of the relative's passing.

[22] As outlined in online Appendix E, 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.

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

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

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 $\mathbf{1}[c \leq \textit{RelativeDeath} < b]_i = 1$ indicates that the family rupture occurred during pregnancy, and $\mathbf{1}[b \leq \textit{RelativeDeath} < b + w]_i = 1$ indicates that it occurred within w days of the child's birth, respectively.

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

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

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 the issue of distinguishing between antenatal and postnatal effects, 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 postnatal 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 postnatal 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 postnatal 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 postnatal periods, or whether some of them have differential impacts during the prenatal 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":

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

where $\textit{UteroStress}_i$ represents intrauterine exposure to the physiological stress experienced by the mother, and $\textit{Other}_i$ 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 *A*1 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 postpartum (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":

$$
\begin{aligned}
(4)\quad RelativeDeath_i \;=\; & \alpha_1 \, UteroStress_i^{*} \, \mathbf{1}\left[c \;\leq\; RelativeDeath \;<\; b\right]_i \\
& + \alpha_2 \, UteroStress_i^{*} \, \mathbf{1}\left[c \;\leq\; RelativeDeath \;<\; b\right]_i^{*} Income_i \\
& + \alpha_3 \, Other_i + \varepsilon_i ,
\end{aligned}
$$

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

In Section IV, we examine whether there are any additional income effects stemming from the prenatal period, that is, income effects that do not only operate through the postnatal 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 IV, all of 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).

B. *Addressing Endogeneity in Date of Birth*

Model (2) represents a causal relationship between in utero exposure to bereavement and child outcomes if, for all $i \in \{S\}$, $E(\mathbf{1}\left[c \leq RelativeDeath < b\right]_i \epsilon_i) = 0$. 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), $\mathbf{1}\left[c \;\leq\; RelativeDeath \;<\; b\right]_i$, 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.[23]

[23] See Currie and Rossin-Slater (2013) and Black, Devereux, and Salvanes (2016) for more discussion of these issues.

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 predetermined at the relative's death date.

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

$$y_{iymp} = \beta_0 + \beta_1 \mathbf{1}[c \leq \textit{RelativeDeath} < e_b]_{iymp} + \psi_y + \phi_m + \rho_p + \mathbf{x}_i'\beta_2 + \nu_{iymp}, \tag{5}$$

where $\mathbf{1}[c \leq \textit{RelativeDeath} < e_b]_{iymp}$ 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.[24]

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 (four 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 a family rupture through the uterine environment.[25]

In parts of our analysis, we also analyze pregnancy trimester- and month-specific impacts, replacing $\mathbf{1}[c \leq \textit{RelativeDeath} < e_b]_{iymp}$ 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.

[24] We also can estimate models where we use $\mathbf{1}[c \leq \textit{RelativeDeath} < e_b]_{iymp}$ to instrument for exposure to death before the child's *actual* date of birth. 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 two-stage least squares (2SLS) results (presented in online Appendix D) are very similar to those from our main specifications.

[25] 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 2SLS specifications where we use $\mathbf{1}[c \leq \textit{RelativeDeath} < e_b]$ to instrument for exposure to death during the mother's *actual* length of pregnancy. In these specifications, the first stage takes the form of

$$\mathbf{1}[c \leq \textit{RelativeDeath} < b]_{iymp} = \gamma_0 + \gamma_1 \mathbf{1}[c \leq \textit{RelativeDeath} < e_b]_{iymp} + \eta_y + \epsilon_m + \theta_p + \mathbf{x}_i'\gamma_2 + \zeta_{iymp}, \tag{6}$$

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

C. *Identifying Assumption*

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

While less restrictive than assuming no selection into death per se, the assumption is nonetheless not innocuous. We therefore subject it to several "plausibility tests," since the exact assumption is inherently untestable. First, we test whether selection into treatment is correlated with a range of parental characteristics that are observed prior to conception: each parent's age, first parity birth, each parent's marital status, each parent's educational attainment (indicators for below high school, high school degree, some college; with college degree or higher as the omitted category), each parent's wage income, and an indicator for the mother being born outside Sweden.[26] As shown in online Appendix Tables A1 and A2 for maternal and paternal characteristics, respectively, we find little evidence for a systematic relationship between parental characteristics and the occurrence of death during pregnancy.[27] Out of the 16 coefficients reported in these tables, only 2 are statistically significant (we find a positive correlation between treatment and first 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.

We explored the correlation between treatment and first parity births in detail, and conclude that it is mechanically driven by differential seasonality in conceptions by parity that coincides with a seasonal pattern in relative deaths. We discuss this issue at length in online Appendix C. For this reason, all of our analyses include month of conception and parity fixed effects, and we show that our results are also robust to the inclusion of parity $\times$ month of conception interactions in online Appendix D.[28]

A second, and related, concern for our identification assumption is that the death of a relative during pregnancy may cause an increase in miscarriages or fetal or infant deaths, leading to selection in our sample of surviving children. Moreover, there may be differential selection by parity, which could introduce the correlation between treatment and first parity that we see in online Appendix Table A1. While we do not have data on miscarriages, we explore the impacts of treatment

[26] Information on child parity and whether the mother is born outside Sweden comes from the medical birth register; we do not have information on child parity or nativity for fathers. 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.

[27] 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 online Appendix Tables A1 and A2 control for the relative's age and age squared.

[28] The correlation between treatment and the likelihood of the mother being born outside Sweden is driven by a highly skewed distribution of relative deaths in the sample of children of foreign-born mothers that exhibits extra mass of relative deaths around 400–500 days post-conception (i.e., after birth). In online Appendix D, we show that our results are robust to dropping children of foreign-born mothers from our sample.

on stillbirths (at 22 weeks gestation or more), perinatal deaths (stillbirths or deaths in the first 28 days of life), and the sex ratio at birth separately by parity in online Appendix Table A3, finding no statistically significant effects.[29]

As a third 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 outcomes.[30] In online Appendix Table A4, we present results from these specifications where the older sibling's outcomes considered are: an indicator for a low-birth-weight birth (less than 2,500 grams), an indicator for a pre-term birth (less than 37 weeks gestation), an indicator for ever being hospitalized before age 1 for a condition originating in the perinatal period, an indicator for ever consuming drugs treating ADHD between ages 9 and 11, and indicators for ever consuming drugs treating anxiety and depression between ages 34 and 36.[31] These are the main outcomes for which we find effects in Section IV, and we therefore use them as "placebo outcomes" in this analysis. Just as in the main analysis, we focus the placebo analysis of mental health outcomes on a subsample limited to mothers who experience a parental or sibling death. Online Appendix Table A4 shows that there is no statistically significant relationship between a younger child's prenatal exposure to a relative's death and the older child's outcomes.[32]

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 IV and online Appendix D for details.

D. *Sample and Summary Statistics*

Table 1 presents variable means and standard deviations in parentheses. 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.[33] Column 1 displays statistics for our full

[29] We follow several papers in this literature by examining the sex ratio as a signal of changes to miscarriage rates (e.g., Sanders and Stoecker 2015; Halla and Zweimüller 2013). Since male fetuses are more likely to miscarry, a reduction in male births may indicate an increase in miscarriages.

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

[31] When we analyze the indicator for being hospitalized for a condition originating in the perinatal period as an outcome, we limit the sample to siblings born in 1987 or later (as the definition of perinatal conditions is not comparable with earlier years).

[32] We should note that the interpretation of these placebo results is less clear in light of the correlation between treatment and child parity. As discussed above, the correlation between treatment and child parity is mostly mechanical and does not affect our main results. Another concern with this placebo analysis is that we have less power to detect statistically significant effects due to the smaller sample size of cohorts that can be linked to siblings. However, we have replicated our main analysis only using children in the "sibling sample" cohorts (i.e., those who were born in 1973, 1977, 1983, 1988, 1995, 1999, 2001, and 2005). In contrast to the results for older siblings, we find statistically significant deleterious effects of exposure to a relative's death during pregnancy on our main outcomes of interest for children born in these years (results available upon request).

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

TABLE 1—SUMMARY STATISTICS

	Our whole sample (1)	Death during pregnancy (2)	Death after pregnancy (3)	All births in Sweden (4)
Mother's age at conception	27.88 (5.058)	27.92 (5.061)	27.86 (5.056)	28.53*
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)	0.1543*
Mother's ed: HS pre-concep.	0.314 (0.464)	0.308 (0.462)	0.318 (0.466)	0.476*
Mother's ed: some college pre-concep.	0.202 (0.401)	0.205 (0.404)	0.199 (0.399)	0.1435*
Child's birth weight (g)	3,543.9 (557.9)	3,537.2 (564.7)	3,549.0 (552.7)	3,505.1
Child is low birth weight (< 2,500g)	0.0323 (0.177)	0.0346 (0.183)	0.0305 (0.172)	0.0424
Child is preterm (< 37 weeks)	0.0497 (0.217)	0.0534 (0.225)	0.0469 (0.211)	0.0586
Observations	296,557	127,406	169,151	3,988,858

Notes: In the first three columns, 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 1 displays statistics for the full sample, while the second and third columns consider the treatment and comparison groups separately. Column 4 displays related statistics for the universe of births in Sweden during the same time period. Note that the variables marked by * are measured slightly differently in the sample that we use (columns 1–3) than in the universe of births (column 4). In particular, in our sample, all variables indicated by * are measured at conception. In the universe of births, these variables are instead measured at the first prenatal visit. In addition, the three educational attainment categories would not be directly comparable even if they were measured at the same point in time. For our sample (columns 1–3), our dataset contains the official educational attainment variable, matched from records from Statistics Sweden. For the universe of births, we use the variable from the Swedish Board of Health and Welfare, where the educational categories are defined slightly differently. Most importantly, high school attainment includes a broader range of programs than regular three-year high school programs (e.g., various two-year programs). We do not have information about marital status for the universe of births.

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-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. In the subsequent section, we explore the differences between the outcomes of the two groups more rigorously using the methods described above.

Column 4 displays related statistics for the universe of all births in Sweden during the same time period. Relative to the universe of births, average birth weight in our sample is slightly higher, while the likelihoods of pre-term and low-birth-weight

births are slightly lower.[34] Additionally, mothers in our sample are slightly less likely to have a high school degree than all mothers giving birth in Sweden, but this difference is at least partially driven by differences in how educational attainment is measured between the two sources of data.[35]

IV. Results

We present results in chronological order. We start with the analysis of birth outcomes, and then study physical and mental health throughout childhood and into adulthood. 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.

A. *Birth Outcomes*

Table 2 presents the results on the effects of exposure to a relative death in utero on average birth weight, and indicators for low-birth-weight, very-low-birth-weight, and high-birth-weight (more than 4,000 grams) and pre-term births. In online Appendix Table A5, 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), and indicators for procedures at delivery (C-section, induction of labor). 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 maternal 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 subsample 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.

[34] We believe that these differences arise as a result of the fact that our sample, which is conditional on being linked to a relative death, has a slightly smaller share of all births from the earlier years than the later years. The multigenerational register has lower quality data further back in time, and we therefore observe fewer great-grandparent deaths for children born in the 1970s than for those born in the later years. Since birth outcomes have been improving over time, our sample has slightly better infant health measures than the overall population of births.

[35] Specifically, in Table 1, the variables marked by * are measured slightly differently in the sample that we use (columns 1–3) than in the universe of births (column 4). In particular, in our sample, all variables indicated by * are measured at conception. In the universe of births, these variables are instead measured at the first prenatal visit. In addition, the three educational attainment categories would not be directly comparable even if they were measured at the same point in time. For our sample, our dataset contains the official educational attainment variable, matched from records from Statistics Sweden. For the universe of births, we use the variable from the Swedish Board of Health and Welfare, where the educational categories are defined slightly differently. Most important, high school attainment includes a broader range of programs than regular three-year high school programs (e.g., various two-year programs). We do not have information about marital status for the universe of births.

Table 2—Effects of Relative Death In Utero on Birth Outcomes

	Birwt (1)	LBW (2)	VLBW (3)	HBW (4)	Pret. (5)
Panel A. All relative deaths					
Death during pregnancy	−11.47	0.00392	0.00124	−0.00501	0.00617
	[2.067]	[0.000633]	[0.000269]	[0.00150]	[0.000838]
Mean dep. var.	3,546.3	0.0320	0.00511	0.188	0.0494
Observations	288,337	288,337	288,337	288,337	289,087
Panel B. Close relative deaths					
Death during pregnancy	−10.08	0.00358	0.000740	−0.00460	0.00517
	[3.563]	[0.00140]	[0.000526]	[0.00258]	[0.00145]
Mean dep. var.	3,523.0	0.0372	0.00603	0.179	0.0511
Observations	84,584	84,584	84,584	84,584	84,817
Panel C. Maternal parent/sibling deaths					
Death during pregnancy	−10.76	0.00420	0.00119	−0.00444	0.00542
	[3.780]	[0.00146]	[0.000519]	[0.00265]	[0.00150]
Mean dep. var.	3,525.8	0.0365	0.00576	0.180	0.0504
Observations	80,956	80,956	80,956	80,956	81,177

Notes: 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.

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.[36] 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. 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. The lack of heterogeneous treatment effects with respect to our measure of the intensity of stress exposure for birth outcomes is consistent with other studies of maternal cortisol (Aizer, Stroud, and Buka 2016) and stressful shocks like hurricanes (Currie and Rossin-Slater 2013).

In Figure 1 and online Appendix Figure A1, we examine whether our estimated impacts are different across the nine months of pregnancy for low-birth-weight and

[36] High birth weight (defined as more than 4,000 grams) is typically seen as a negative health outcome, which is correlated with a greater incidence of obesity and other adverse conditions like diabetes in later life (see, e.g., Cnattingius et al. 2012). Thus, the decline in the likelihood of a high-birth-weight birth can be seen as a small beneficial effect of in utero stress exposure. However, the magnitude of this decline is much smaller than the corresponding magnitudes of the increases in low-birth-weight and very-low-birth-weight births.

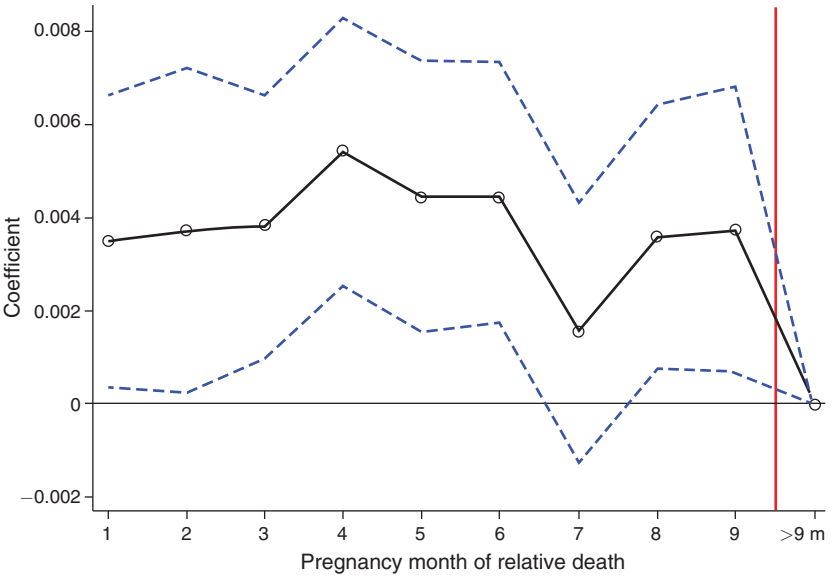

Figure 1. Effect of Relative Death on the Incidence of the Child Being Born Low-Birth-Weight

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 percent confidence intervals in dashed lines) on the effects of the death of a relative during the first through ninth 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.

pre-term births, respectively. The graphs present the coefficients (and 95 percent 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 postpartum, with slightly higher effects during the fourth month. In online Appendix Tables A6 and A7, 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. As discussed in more detail in Section I, the lack of significant differences across the gestational age at exposure is consistent with other recent studies on the effects of in utero shocks on birth outcomes (e.g., Almond and Mazumder 2011; Mansour and Rees 2012; Currie and Rossin-Slater 2013; Black, Devereux, and Salvanes 2016).

B. *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—EFFECTS OF RELATIVE DEATH IN UTERO ON HOSPITALIZATIONS BY AGE 1

	Any hosp. (1)	Tot. hosp. (2)	Any hosp.-peri. (3)	Tot. hosp.-peri. (4)
Panel A. All relative deaths				
Death during pregnancy	0.00192	0.00148	0.00351	0.00294
	[0.000924]	[0.00176]	[0.000892]	[0.00107]
Mean dep. var.	0.0737	0.102	0.0575	0.0646
Observations	288,606	288,606	231,398	231,398
Panel B. Close relative deaths				
Death during pregnancy	0.00107	−0.000250	0.00402	0.00335
	[0.00174]	[0.00291]	[0.00192]	[0.00249]
Mean dep. var.	0.0660	0.0914	0.0595	0.0681
Observations	84,676	84,676	46,500	46,500
Panel C. Maternal parent/sibling deaths				
Death during pregnancy	0.00140	−0.0000993	0.00396	0.00326
	[0.00183]	[0.00299]	[0.00197]	[0.00257]
Mean dep. var.	0.0659	0.0908	0.0595	0.0680
Observations	81,036	81,036	44,634	44,634

Notes: 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. "Tot. hosp.-peri." refers to the total number of hospitalizations for conditions originating in the perinatal period. The sample in columns 3 and 4 is limited to cohorts born in 1987 or later (as the definition of perinatal conditions is not comparable with earlier years). Robust standard errors are clustered on the mother's municipality of residence in the year prior to conception.

Table 3 presents results on the effects of in utero exposure to a relative death on child hospitalizations by age 1. We find that in utero stress is associated with a 3 percent increase in the likelihood that a child is ever hospitalized by age 1 (column 1).[37] 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.[38] As with the results on birth outcomes, we do not see substantial differences in effects across relative types (panels A to C). In online Appendix Figure A2 and online Appendix Table A8, 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.

[37] We also examined outpatient visits, and found suggestive evidence of similar increases in outpatient visits occurring by age 1, although we have less power due to smaller sample sizes in these analyses (outpatient data are only available for years 2001 to 2012). These results, as well as a description of the outpatient data, are available upon request.

[38] The analysis of perinatal conditions is limited to cohorts born in 1987 or later as the definition is not directly comparable to earlier years. For these years, 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: (i) fetus and newborn affected by maternal factors and by complications of pregnancy, labor, and delivery; (ii) disorders related to length of gestation and fetal growth; (iii) birth trauma; (iv) respiratory and cardiovascular disorders specific to the perinatal period; (v) infections specific to the perinatal period; (vi) hemorrhagic and hematological disorders of fetus and newborn; (vii) transitory endocrine and metabolic disorders specific to fetus and newborn; (viii) digestive system disorders of fetus and newborn; (ix) conditions involving the integument and temperature regulation of fetus and newborn; (x) other disorders originating in the perinatal period.

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 1. However, the impacts seem to fade after early childhood: we find no effects on hospitalizations at later ages (see online Appendix Table A9).[39] Though, as we pointed out in Section I, our results do not rule out the possibility of latent physical health consequences for individuals at older ages (Barker 1990); our cohorts are too young to detect such effects.

C. *Mental Health Outcomes*

We next use the prescription drug registry data to analyze effects on mental health. As described in Section II, these data contain information about prescription drugs bought during the period 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.

Figure 2 graphs the coefficients (and associated 95 percent 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 II at five-year age intervals. In panel A of Figure 2, 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 panel B, 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 panel C when the sample is further limited to maternal parent and sibling deaths.

Figure 2 captures 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: a 25 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 13 and 8 percent increases in the likelihood of ever purchasing drugs to treat anxiety

[39] Additionally, we have used our prescription drug registry data to explore effects on the consumption of drugs used to treat any of the following health conditions at ages 4 through 36: obesity, diabetes, Cushing's syndrome, hypo- and hyperthyroidism, cholesterol, and heart conditions (i.e., beta blockers). 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 (see online Appendix Table A10).

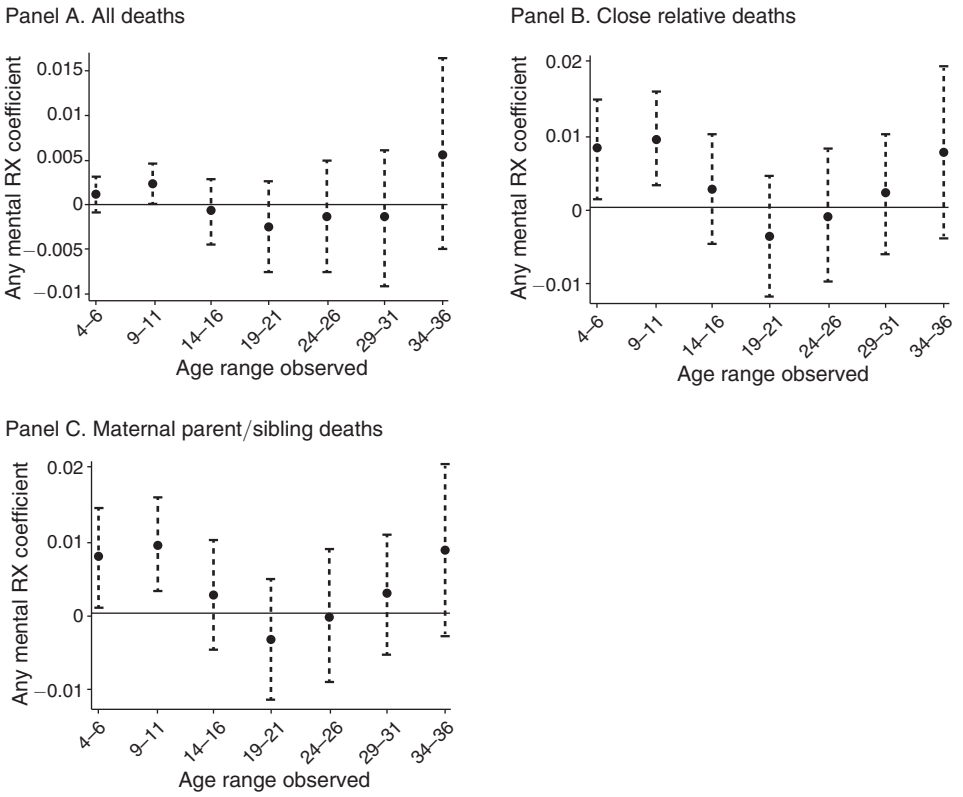

FIGURE 2. EFFECT OF RELATIVE DEATH ON THE INCIDENCE OF THE CHILD CONSUMING ANY MENTAL HEALTH MEDICATIONS BY AGE

Notes: See Figure 1 notes for more information on the sample. These figures plot the coefficients (and 95 percent 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.

and depression, respectively; and 19 and 12 percent increases in the average daily doses of anti-anxiety and depression medications, respectively. Panel C shows that these effects still remain in the subsample 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).

As we discussed in Section I, the age pattern of mental health effects that we find is consistent with certain features of our prescription registry data and the institutional context in Sweden. To interpret our 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.,

Table 4—Effects of Relative Death In Utero on Prescription Use for Mental Health Conditions at Ages 9–11 and 34–36

	ADHD, 9–11		Anxiety, 34–36		Depression, 34–36	
	Any RX (1)	Avg. dose (2)	Any RX (3)	Avg. dose (4)	Any RX (5)	Avg. dose (6)
Panel A. All relative deaths						
Death during pregnancy	0.000973	0.0373	0.00499	0.0202	0.00517	0.404
	[0.000933]	[0.0337]	[0.00306]	[0.0195]	[0.00373]	[0.235]
Mean dep. var.	0.0228	0.667	0.0685	0.217	0.114	4.664
Observations	114,906	114,906	27,641	27,641	27,641	27,641
Panel B. Close relative deaths						
Death during pregnancy	0.00620	0.172	0.00719	0.0304	0.00736	0.472
	[0.00205]	[0.0774]	[0.00358]	[0.0210]	[0.00436]	[0.246]
Mean dep. var.	0.0244	0.713	0.0674	0.205	0.112	4.559
Observations	20,380	20,380	22,907	22,907	22,907	22,907
Panel 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 dep. var.	0.0238	0.702	0.0666	0.204	0.111	4.546
Observations	19,605	19,605	21,763	21,763	21,763	21,763

Notes: 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 online Appendix E.

those born in the 1970s and early 1980s) during adulthood.[40] As we have pointed out, ADHD prescription drugs have only been available in Sweden since 2002, and the prescription rate has been steadily increasing since 2005. Thus, intuitively, the *x*-axes in Figure 2 indicate the age ranges of different cohorts during this "ADHD revolution." The fact that we see the strongest effects on ADHD prescription drug use among cohorts who were aged 9 to 11 during the "ADHD revolution" is also very consistent with Sweden's guidelines that require mental health screenings of children at age 10, and with the direct economic incentives for schools to detect and treat ADHD among students, described in detail in Section I.

In online Appendix Table A11, we attempt to shed more light on this explanation. We split the sample according to the age at which different cohorts would have been at most 11 years old in 2002. Specifically, the first three columns consider the consumption of any mental health drugs, any ADHD drugs, and the ADHD average dose observed at any age between 4 and 14 in our data, while the last three columns consider these outcomes at ages 15 to 36 in our data. Individuals who are at most 14 years old in our data were born in $2005 - 14 = 1991$ or later, and were thus at most 11 years old in 2002. Consequently, only individuals who are represented in the first three columns were likely exposed to a mandated mental health screening and had access to ADHD drugs at the time of the screening. The results demonstrate

[40] In supplementary analyses, we explored whether there are any heterogeneous effects on birth outcomes across these cohorts. We find that these cohorts experience similar adverse impacts on birth outcomes (results available upon request).

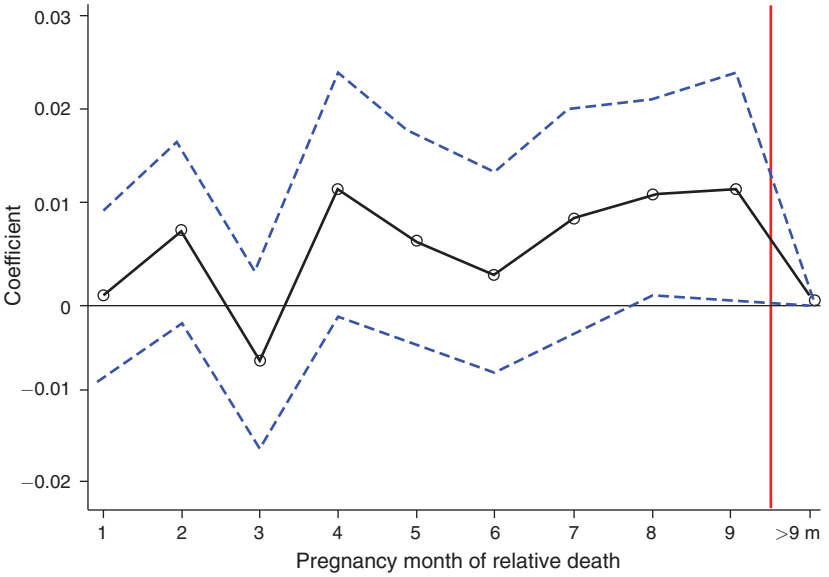

FIGURE 3. EFFECT OF MATERNAL PARENT/SIBLING DEATH ON THE INCIDENCE OF THE CHILD CONSUMING ANY ADHD MEDICATIONS AT AGES 9–11

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 percent confidence intervals in dashed lines) on the effects of the death of a relative during the first through ninth 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.

that, despite the fact that the sample size in the younger age group is only about one-half that of the size of the older age group, the effects on ADHD drug purchases are much stronger for cohorts who are observed at ages 4 to 14 in our prescription data. In other words, we find positive treatment effects on the consumption of ADHD drugs only for cohorts that were in elementary and middle school during the time period when ADHD drugs were available and mental health screenings were mandated in the transition between elementary and middle school.

An alternative 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.[41] Thus, the absence of effects beyond school age may instead suggest that ADHD is more readily *detected* while children are in school, which is again consistent with school financing rules that offer schools extra transfers for pupils with special needs. Indeed, when we interact our treatment variable with the share of municipal

[41] 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 5 years later. The figure is similar in all older age groups where treatment is begun before the age of 55 (Socialstyrelsen 2012).

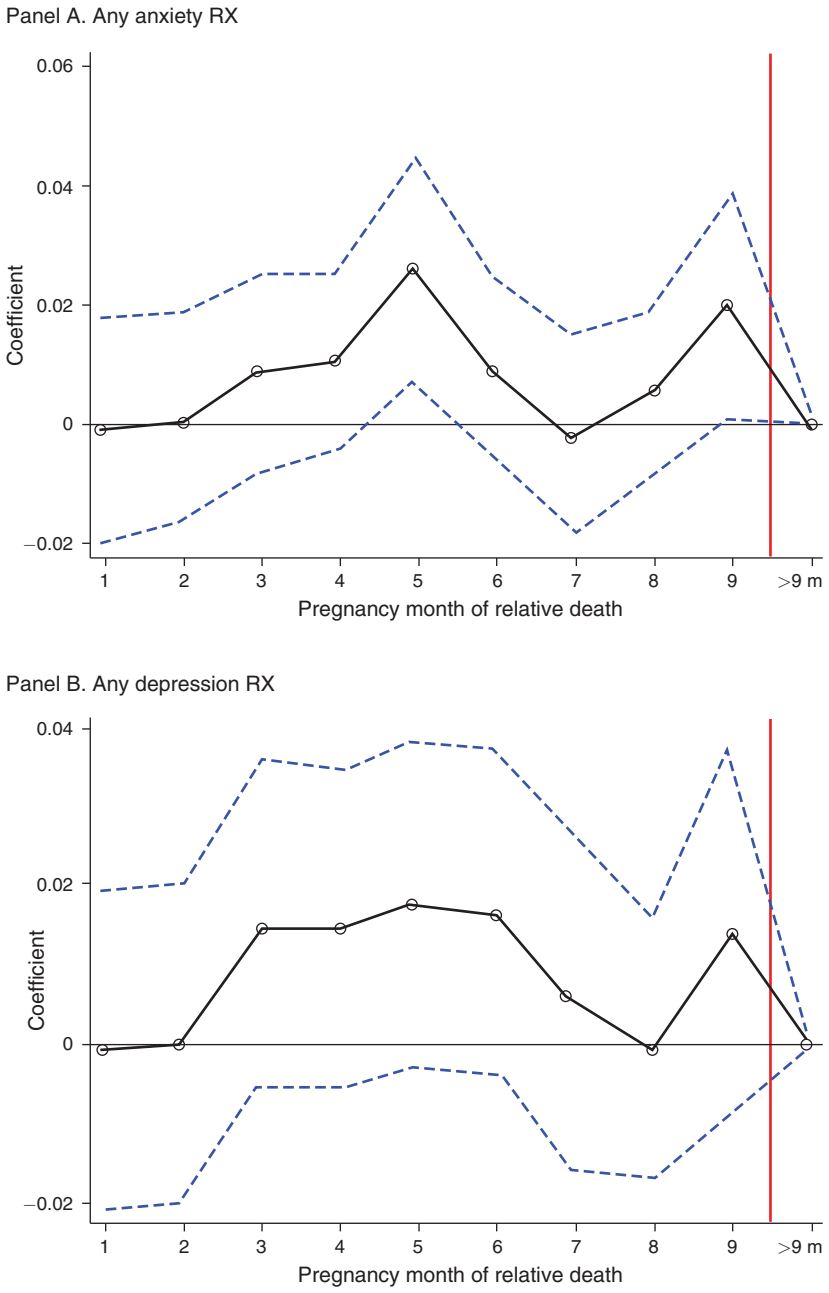

Figure 4. Effect of Maternal Parent/Sibling Death on the Incidence of the Child Consuming Any Anxiety or Depression Medications at Ages 34–36

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. These figures plot the coefficients (and 95 percent confidence intervals in dashed lines) on the effects of the death of a relative during the first through ninth 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 (panel A) or depression (panel B) at ages 34–36.

resources allocated based on special education needs, we obtain a positive (albeit insignificant) coefficient, providing suggestive evidence of this mechanism.[42]

For individuals who were already out of school when the "ADHD revolution" took place, detection of mental health issues may take a longer time. In fact, it may take a "precipitating event," such as marriage or childbirth, for one to seek mental health treatment. Consistent with this idea, in online Appendix Table A12, we show that the effects on the consumption of anti-anxiety and anti-depression drugs at ages 34–36 are driven entirely by individuals who are married during those ages.[43]

Overall, our results suggest that experiencing a very stressful event in utero is more deleterious for mental health than experiencing such an event shortly post-birth. Our estimates also imply 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. The finding that adverse mental health impacts seem to be sensitive to the intensity of the stressor is consistent with Aizer, Stroud, and Buka's (2016) evidence that only the highest levels of maternal cortisol in utero impair children's later cognitive outcomes. In contrast, we showed above that the physical health impacts are less sensitive to the severity of stress exposure (again, consistent with evidence from Aizer, Stroud, and Buka 2016 on birth outcomes).

D. *Magnitudes*

To gauge the plausibility of our estimates, we compare the magnitudes of our effect sizes to those reported in the existing literature. First, our 11-gram decrease in birth weight is within the confidence interval of Black, Devereux, and Salvanes's (2016) 23-gram decrease associated with the death of a maternal parent in Norway. However, we show relatively large effects on the incidence of low-birth-weight and very-low-birth-weight births (12 percent and 24 percent, respectively), while Black, Devereux, and Salvanes (2016) find statistically insignificant impacts on these outcomes. Additionally, Black, Devereux, and Salvanes (2016) report a 12 percent increase in the likelihood of a C-section delivery, while we only find a 3 percent increase for this outcome. The differences between our estimates and those in Black, Devereux, and Salvanes (2016) likely reflect different institutional settings (Sweden versus Norway), and the fact that Black, Devereux, and Salvanes (2016) use a sample of siblings, while we focus on all individuals who experience a relative death in utero or in the year after birth.

It is also informative to compare our estimates for birth outcomes to those found in studies on the effects of natural disasters and terrorist attacks. For example, our 12 percent increase in low-birth-weight births is substantially smaller than the corresponding 40 percent increase in Torche (2011) resulting from exposure to a Chilean earthquake in a "high-intensity" region. Similarly, Eskenazi et al. (2007) find that exposure to the September 11 attacks in New York City was associated with a 44 percent increase in very-low-birth-weight births, a magnitude much higher than

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

[43] There is no effect of treatment on the likelihood of being married (results available upon request). We do not have information on the fertility of the cohorts in our sample, and thus cannot study the effects separately by whether or not they have children.

our estimated 24 percent increase. The fact that the impacts we find are smaller than those reported in these studies suggests that analyses of disasters and attacks may be bundling the effects of multiple "treatments" (i.e., combining stress with the economic and physical health consequences of these events), whereas our research design is more precisely able to isolate in utero exposure to maternal stress.

With regard to mental health, we can compare our estimates to the two existing studies in economics that have examined the impacts of in utero exposure to malnutrition. Almond and Mazumder (2011) find that exposure to Ramadan in utero doubles the likelihood of having a mental disability in adulthood in data from Uganda and Iraq, while Adhvaryu, Fenske, and Nyshadham (forthcoming) show that a one standard deviation increase in cocoa prices (which improves nutrition during pregnancy) leads to a 50 percent decrease in the likelihood of suffering from severe mental distress in adulthood in Ghana. Our 25 percent, 13 percent, and 8 percent impacts on the take-up of ADHD, anxiety, and depression medications, respectively, are considerably smaller. These differences in effect sizes could arise for a number of reasons, including that we are (i) studying different institutional contexts (a high-income country with a large social safety net versus developing countries), (ii) estimating effects of different types of shocks (in utero exposure to maternal stress from bereavement versus malnutrition), and (iii) measuring mental health in different ways (prescription drug take-up versus survey responses). Nevertheless, it is reassuring that our estimates are within the bounds of the recent limited literature in economics on this question.

E. *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 III, 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 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 online Appendix Table A13, we examine these potential mechanisms in more detail. We study whether the death of a relative during pregnancy is associated with changes in prenatal care, the presence of "high-risk" factors (diabetes, kidney disease, epilepsy, asthma, hypertension, or urinary infection), initiation of 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), and an indicator for the mother having any positive wage income during the year of conception or the year after.[44]

In the overall sample, we find no effects on any of these outcomes. When we limit to the close relative and maternal parent/sibling subsamples, we see statistically significant reductions in the adequacy of prenatal care, as measured by the Kotelchuck Index (Kotelchuck 1994).[45] The magnitudes of these estimates are quite small, however: for example, there is a 1 percentage point decline in the likelihood of the mother having adequate prenatal care in the close relative sample, relative to a sample mean of 81 percent. In practice, this effect likely translates into one missed prenatal visit within a small fraction of the treated population (e.g., to attend the relative's funeral).[46] Given that the number of prenatal visits has been shown to have very little effect on children's health at birth (Sikorski et al. 1996; Fiscella 1995; Evans and Lien 2005), we do not think that our main results could be plausibly explained by such a small reduction in prenatal care.

In sum, we believe that changes in pregnancy behaviors and conditions that we can observe are unlikely to drive our estimated effects on birth outcomes, hospitalizations during the first year of life, and mental health in later childhood and adulthood.

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 postpartum depression, or generally be more sensitive to additional stressors. In online Appendix Table A14, 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.[47] 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.

[44] We measure any wage income in the year of conception and the year after to try to capture labor supply during pregnancy. Unfortunately, we cannot look at a more precise measure of labor supply since our wage income data are at an annual level.

[45] The Kotelchuck Index compares the number of prenatal visits received to the number of expected visits, adjusting for gestational age when care began and gestational age at delivery. Adequate prenatal care means that the ratio of observed to expected visits is at least 80 percent. Intermediate prenatal care means that the ratio of observed to expected visits is 50–79 percent.

[46] The death of a relative does not affect the likelihood that a woman is eligible for prenatal care due to the existence of universal health insurance coverage.

[47] 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.

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 III, 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 postpartum.

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 can fully cover the funeral expenses, and then also leave some inheritance to the surviving relatives (Erixson and Ohlsson 2014). Also, immediate income shocks may arise if, for example, when a maternal parent dies, the other maternal parent moves in with her child (the (expectant) mother). In Sweden, however, co-residence between adult children and their parents or other extended family members is very uncommon, largely due to cultural reasons and the fact that the government provides assistance for the care and financial support of the elderly. Therefore, this channel is likely not very relevant in our context.

Moreover, relative to other countries such as the United States, income shocks, and hence their precise timing, likely matter less in Sweden due to the extensive social security and benefits system. In online Appendix Table A15, we 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. Online Appendix Table A15 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.

In sum, while we of course cannot rule out all potential alternative mechanisms, the evidence in this section is suggestive of maternal stress as the primary driver of our main results.

F. *Additional Results*

This section presents two sets of results that test the robustness of our main findings and explore an important maternal behavioral response. In addition, in online Appendix D, we present results from two-stage least squares specifications for our main outcomes of interest; explore the sensitivity of our findings to sample limitations based on causes of death that are determined to be more exogenous than others; explore the heterogeneity in effects by the physical proximity of the mother to the deceased relative; assess an alternative interpretation of our measure of intensity of emotional stress related to the size of inheritances; and perform various additional robustness checks addressing the correlation between treatment, parity, and foreign-born mothers.

Adjusting for Multiple Hypothesis Testing.—First, an important concern for our analysis is that we may find spurious effects due to the number of outcomes we

TABLE 5—EFFECTS OF RELATIVE DEATH IN UTERO ON PHYSICAL AND MENTAL HEALTH INDICES

	Physical health index			Mental health index		
	All (1)	Close (2)	Mom par/sib (3)	All (4)	Close (5)	Mom par/sib (6)
Death during pregnancy	−0.00905	−0.00737	−0.00824	−0.000129	−0.00724	−0.00904
	[0.00175]	[0.00293]	[0.00297]	[0.00188]	[0.00363]	[0.00365]
Mean dep. var.	−0.00854	−0.0185	−0.0165	−0.00172	0.00188	0.00369
Observations	289,087	84,817	81,177	280,699	83,581	79,980

Notes: See Tables 1 and 2 for more information on the sample and controls. The physical health index consists of the 28 outcomes analyzed in Tables 2, 3, A3, A5, A9, and A10: 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 hospitalizations by age 1, total hospitalizations by age 1, any hospitalizations for perinatal causes by age 1, total hospitalizations for perinatal causes by age 1, 7 indicators for ever purchasing a physical health prescription at any of the age categories we consider (4–6, 9–11, 14–16, 19–21, 24–26, 29–31, 34–36), and indicators for any hospitalizations by ages 5, 10, 18, and 27. The mental health index consists of 7 indicators for ever purchasing a mental health drug at any of the main age categories we consider in Figure 2 (4–6, 9–11, 14–16, 19–21, 24–26, 29–31, 34–36), as well as $2 \times 3 \times 7 = 42$ other outcomes comprised of our two measures: an indicator for ever purchasing the drug and the average daily dose, per condition (ADHD, anxiety, depression) and per age group (4–6, 9–11, 14–16, 19–21, 24–26, 29–31, 34–36). See Section IV 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.

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. The physical health index consists of the 28 outcomes analyzed in Tables 2, 3, A3, A5, A9, and A10, described in the notes to Table 5. The mental health index consists of 49 outcomes: 7 indicators for ever purchasing a mental health drug at any of the main age categories we consider in Figure 2 (4–6, 9–11, 14–16, 19–21, 24–26, 29–31, 34–36), as well as $2 \times 3 \times 7 = 42$ other outcomes comprised of our two measures, an indicator for ever purchasing the drug and the average daily dose, per condition (ADHD, anxiety, depression) and per age group (4–6, 9–11, 14–16, 19–21, 24–26, 29–31, 34–36).

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. 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 close relative, and specifically, parent or sibling.[48]

Maternal Responses to In Utero Shocks: Effects on Subsequent Fertility.—Second, we study whether our in utero shock of interest is correlated with an important

[48] The magnitudes of the effect sizes for the two indices are small. This is not unexpected as there are effects for only some parts of the indices, but not others.

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 by reducing the quantity of children that they had.

We examine maternal fertility in online Appendix Table A16, 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 reduce their fertility after an adverse shock during pregnancy, but instead are more likely to have additional children.

While our data do not allow us to better understand the mechanism behind this fertility effect, this 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. This problem is not entirely alleviated by comparing treated children to only their older siblings, as the older siblings are likely to be affected by the endogenous change in family size, and they may be differentially affected than the treated children.

V. Implications for the Costs of Economically Induced Stress

Throughout this paper, we have analyzed the internal validity of our estimates by conducting a variety of robustness checks and indirect tests of mechanisms. However, it is also worth discussing whether our results on the effects of in utero exposure to maternal stress from the death of a relative have any external validity. In particular, in light of evidence on the intergenerational persistence of socioeconomic status in the United States and other developed countries (Solon 2001; Chetty et al. 2014; Boserup, Kreiner, and Kopczuk 2014), and the strong socioeconomic gradient in reported stress levels (Kunz-Ebrecht, Kirschbaum, and Steptoe 2004; Cohen, Doyle, and Baum 2006; Thompson 2014), the question of how economically induced stress can affect individual well-being across generations is of interest to both academics and policymakers.

Although grief-induced stress resulting from the death of a family member and stress stemming from adverse economic shocks are in many ways not the same, both types of stress produce a physiological response in the human body characterized by an increase in the level of the cortisol hormone (which controls the "fight-or-flight" response). Thus, we conduct an exploratory back-of-the-envelope calculation to "translate" our estimates into the costs of economically induced stress. Specifically, we proceed in three steps. First, we use existing studies that quantify the effect of the death of a relative on cortisol levels. Second, we use studies that quantify the impact of adverse economic conditions on cortisol. These two steps together allow us to translate the impact of economic hardship on cortisol into our "relative death scale." In the final step, we use our results to speak to how

in utero exposure to maternal stress from economic hardship may affect long-term mental health.

A. *The Impact of a Relative Death on Cortisol*

Several recent studies show that the death of a loved one is associated with increased cortisol levels. Cortisol levels can be measured in blood (plasma) and in saliva. Because levels estimated in blood are higher than levels estimated in saliva, we distinguish between studies that use these two types of measurements.[49]

Irwin et al. (1988) compare morning plasma cortisol levels of women who experienced the death of a spouse six months earlier with women in a non-bereaved control group. They find that the mean plasma cortisol level is 99.3 $nmol/l$ higher in the bereaved group.

Similarly, Pfeffer et al. (2007) compare the salivary cortisol levels of individuals who lost a parent with individuals in a non-bereaved control group. They find that the mean salivary cortisol level is 2.75 $nmol/l$ higher in the bereaved group, measured four months after bereavement.[50]

B. *The Impact of Economically Induced Stress on Cortisol*

A number of studies present correlational evidence documenting a strong socioeconomic gradient in cortisol. Individuals with lower levels of education, income, and lifetime economic status tend to have elevated cortisol when compared to their more educated, higher income, and higher economic status counterparts (see, e.g., Cohen, Doyle, and Baum 2006; Li et al. 2007).

There are also several studies that present more rigorous quasi-experimental and experimental evidence on this question. In Sweden, Arnetz et al. (1991) find that individuals who were laid off in a mass layoff had blood plasma cortisol levels that were 68 $nmol/l$ higher than individuals who were securely employed, measured one year after the layoff. Comparing to the results in Irwin et al. (1988) discussed above, this study suggests that the impact of economically induced stress through unemployment on cortisol is about 69 ($= 68/99$) percent of the impact of the death of a close relative.

Similarly, in a developing country context, Haushofer and Shapiro (2013) conduct a randomized controlled trial that investigates the impact of poverty on stress by randomly allocating cash transfers to households. They find that cortisol levels are 2.03 $nmol/l$ lower in households that received large transfers ($1,525) than in households that received small transfers ($404). The difference corresponds to a substantial income effect, given that Kenya's GDP per capita was $1,184 in 2012,

[49] Different studies also measure cortisol levels using different units. For the purpose of comparison, we here convert all results that we discuss to $nmol/l$. (Conversion rate: 1 $\mu g/dl = 27.59$ $nmol/l$.)

[50] In addition, several studies show evidence on the impact of bereavement on diurnal cortisol regulation, i.e., the ability of cortisol to be broken down over the course of the day. The evidence suggests that recently bereaved individuals not only have higher morning cortisol levels, but also experience a flatter slope during the course of the day (meaning that cortisol falls less during the day). See Dietz et al. (2013) on the impact on cortisol regulation of the loss of a parent and Holland et al. (2014) on the impact of the loss of a spouse. Further, O'Connor et al. (2012) examine diurnal cortisol production patterns in women who have experienced the death of different relatives and find that more intense grieving is associated with a flatter slope across the day.

at the time of the intervention.[51] Comparing to the results in Pfeffer et al. (2007), this estimate suggests that the effect of economically induced stress through lower income on cortisol is about 74 ($= 2.03/2.75$) percent of the impact of the death of a close relative.

C. *Economically Induced Stress In Utero and Later Mental Health*

Using the estimate above that the impact on cortisol from a layoff is approximately 69 percent of the impact of the death of a close relative, we can calculate how in utero exposure to maternal economically induced stress (resulting from unemployment) might affect the future mental health of the unborn child. This calculation implies that in utero exposure to stress from maternal unemployment induces a 17.3 ($= 0.69 \times 25$) percent increase in the likelihood of ever purchasing a drug to treat ADHD in middle childhood, and a 16.6 ($= 0.69 \times 24$) percent increase in the average daily dose. Further, among adults in their thirties, the calculations suggest that in utero exposure to stress from maternal unemployment leads to 9 ($= 0.69 \times 13$) and 5.5 ($= 0.69 \times 8$) percent increases in the likelihoods of ever purchasing drugs to treat anxiety and depression, respectively; and in 13.1 ($= 0.69 \times 19$) and 8.3 ($= 0.69 \times 12$) percent increases in the average daily doses of anti-anxiety and depression medications.[52]

Of course, these back-of-the-envelope calculations rely on a strong assumption of linearity in the effect of cortisol. Nevertheless, this exercise implies that the effects of economically induced stress on the mental health of the next generation could be quite large.

VI. 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 deaths of family members 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.

We find that in utero exposure to the death of a relative up to three generations apart negatively affects physical health at birth and in the first year of life. We also provide novel evidence that severe antenatal stress, as measured by bereavement of 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 US market, the 8 percent decrease in the consumption of prescription drugs treating depression alone can be valued at $800 million per year.

[51] GDP per capita in current US$ is available at: http://data.worldbank.org/indicator/NY.GDP.PCAP.CD.

[52] If we instead use the estimated relationship between household income and cortisol in the context of Kenya, we obtain very similar impacts of in utero exposure to maternal stress due to poverty.

While our findings may not generalize to all other possible 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 United States using a ten-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.[53] 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 later in life, 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.

VII. Note (Added Post-Acceptance)

Since acceptance of this article, it was brought to our attention that there are other related studies on this topic. This note describes the relation of our work to these studies (the pertinent references have also been added). Class et al. (2011, 2014) use Swedish register data to document associations between maternal bereavement in utero and adverse birth outcomes and hospitalizations for mental health conditions, respectively.[54] Similarly, Khashan et al. (2008, 2011) use Danish register data to document an association between uterine exposure to maternal bereavement and hospitalizations for mental health conditions. These studies compare the outcomes of children whose mothers experienced a close relative death during pregnancy (or in the surrounding years) to the outcomes of children whose mothers did not. They

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

[54] Abel et al. (2014) also use Swedish register data to examine a potential association between maternal bereavement in utero and hospitalizations for mental health conditions, but find no evidence of such an association.

also examine the timing of exposure, by comparing children exposed to maternal bereavement during different stages (e.g., specific months of pregnancy or in the first or second year of life) to children who have no exposure to maternal bereavement.

By contrast, our empirical design compares the outcomes of children whose mothers experienced a relative death within 280 days post-conception to the outcomes of children whose maternal relatives died in the year after their expected date of birth. Further, we explore the heterogeneity of effects across different months of pregnancy, using the group exposed to maternal bereavement in the year after their expected date of birth as the control group (as opposed to a control group that is unexposed, as in the studies referenced above). As we write in Section III, this approach helps us to: (i) separate the impacts of maternal stress that operate through the uterine environment from other impacts (such as income effects) that also operate through the postnatal environment, and (ii) address the concern that the occurrence of death is not a random event and has been shown to be correlated with other family characteristics such as socioeconomic status. Intuitively, *all* children included in our analysis are exposed to the relative's passing, and hence to the postnatal consequences and correlates of this event, but only the treatment group is exposed to the event *through the uterine environment*. Our paper also differs from the referenced studies above in that we measure mental health outcomes using prescription drug data, which enables detection not only of the occurrence of a condition, but also of its severity (as captured by the prescribed dose).

Our approach is similar to that of a much earlier study by Huttunen and Niskanen (1978), who used data from Helsinki, Finland and studied a sample of 335 individuals whose fathers died before age 35 either before their birth or in the year after their birth. They conducted analyses using Student's t-tests and χ^2 tests, finding that, relative to the control group, the treatment group had higher rates of diagnosed schizophrenics treated in psychiatric hospitals and higher rates of individuals committing crimes. They did not find any statistically significant differences between the groups for ten other outcomes that they considered, such as childhood behavior disorders and minor depressive and neurotic disorders. Huttunen and Niskanen (1978, p. 431) further comment:

> *"The number in our total sample and the number of psychiatric cases in the two groups are so small that the present results cannot be considered as conclusive evidence for the proposed hypothesis of the etiological role of maternal stress during pregnancy in psychiatric and behavioral disorders."*

We view our work as building on the earlier Huttunen and Niskanen (1978) paper in the following ways: (i) we assign treatment based on the expected date of birth rather than the actual date of birth, in light of the evidence that in utero exposure to the death of a relative affects gestation length (and hence, the date of birth); (ii) we document impacts of antenatal stress on conditions other than schizophrenia; (iii) we use more recent population-level Scandinavian registry data that provide us with a sample size that is nearly 1,000 times larger than that in Huttunen and Niskanen (1978) and thus lend us much more statistical power; (iv) we use novel prescription registry data to measure mental health outcomes; (v) we study deaths of relatives other than children's fathers, which allows us to test for heterogeneity in effects by

the severity of antenatal stress exposure; and (vi) we use regression models that allow us to control for maternal, child, and relative characteristics, and conduct a variety of additional analyses to test for alternative channels (other than stress), and address issues of multiple hypothesis testing.

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