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The Effects of Birth Weight on Hospitalizations and Sickness Absences

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Abstract

This study examines the causal effects of birth weight on two health-related outcomes: inpatient hospitalizations and sickness absences, distinguishing between different diagnoses. Our analysis exploits differences within siblings and within twin pairs, using full population Swedish register data on cohorts born between 1973 and 1995, observed through childhood and in adulthood. In childhood, there is a strong relationship between birth weight and days in inpatient care. This is mostly driven by perinatal conditions during infancy, but substantial effects on other conditions are also found. Effects reduce in size when the child grows older. There are also significant and important effects in adulthood, and these are stronger than the ones found in late childhood. In adulthood, the strongest and most consistent effects are obtained for mental conditions. This holds for both hospital visits and sickness absences, but is most striking for hospital visits, where mental diagnoses may account for almost the entire effect of a lower birth weight. Overall, we provide evidence that birth weight does matter for both short- and long-term health outcomes and that the effects may not be smaller than what more traditional OLS regressions suggest.

Key words: Birth weight, childhood health, adulthood health, fetal origins hypothesis

JEL codes: I1, I12

1. Introduction

Substantial evidence suggests that conditions in utero has important and long-lasting effects on health and socioeconomic outcomes. Frequently, the so-called Barker Hypothesis has been a point of departure, according to which fetal growth restriction “programs” the individual to experience an increased likelihood of adult disease (e.g., Barker 1990; Barker 1995).

Following the work of Barker, a large number of researchers have documented associations between, in particular, low birth weight and a range of health problems, including for example cardiovascular and endocrine disease. In general, however, it is unclear whether these associations reflect causality.

Recent studies in economics have used exogenous shocks or within-family comparisons to try to disentangle causal effects of prenatal circumstances (Almond and Currie 2011). While finding strong evidence of effects on education and socioeconomic status, results for health outcomes have been surprisingly mixed. Some studies (e.g., Almond et al. 2005; Almond 2006; Black et al. 2007) have documented effects on outcomes including infant mortality or adult self-reported disability. Other studies (e.g., Oreopoulos et al. 2008; Almond et al. 2009; Field et al. 2009) find no or little evidence of effects on health care visits or on self-reported days of illness in childhood or adolescence.

In this study, we provide new evidence on the relationship between health status at birth and health-related outcomes, throughout childhood and into early adulthood. Our analysis compares the outcomes of siblings and twins, using register data from Sweden on the entire population of individuals born between 1973 and 1995.

In line with much of the previous literature, our focus lies on the effect of birth weight. Our analysis considers two main outcomes: inpatient visits and sickness absences. Since inpatient care means that the individual is admitted to a hospital and normally stays overnight, the first of these variables represents health problems that are, typically, more severe. By exploiting data on specific diagnoses, we contribute to the literature not only by studying the total number of days in care, but also whether the individual was diagnosed with different types of diseases. Virtually no previous study has examined the effect of birth weight on days in sickness absence. Due to its importance, both to the individual’s ability to successfully pursue a labor market career as well as to a country’s public finances, this outcome represents a relevant complement to hospital stays. Again, effects on different types of diagnoses are estimated and contrasted.

2. Background

2.1. Physiological mechanisms

Relationships between, on the one hand, health during the fetal stage and, on the other hand, adult health and well-being have been documented for at least half a century. Whereas the majority of the literature within the field consists of empirical studies documenting correlations, the literature examining the relationship from a more mechanistic perspective emerges as considerably smaller. Existing studies that have contributed to the understanding of the potential mechanisms linking fetal growth restriction (and thus birth weight) to later life outcomes are largely based on experimental studies on animals. Here, two major processes have been identified as responsible for altering the fetal growth process and pathophysiology in adult ages: maternal/fetal malnutrition and hormonal programming (Cottrell and Seckl 2009).

The mechanism linking undernutrition during the fetal stage to later life adversity is the comparatively more frequently encountered one in the literature. In the presence of a lack of nutrients, the fetus will adapt in order to promote survival, something that may imply that the development of certain organs or processes are favored before others. Evidence suggests that nutritional stress may affect the growth of organs such as the pancreas, the heart and the kidneys, as their functional units are programmed already during the fetal stage. A disruption of the developmental trajectory of the kidney creates an increased risk of reduced renal function as well as an increased risk of hypertension. Similarly, a disrupted development of the pancreas may influence the function of the organ throughout the life course, leading to hyperglycemia and impaired insulin secretion, associated with metabolic disease (Gluckman et al. 2008).

2.2. Previous research

A plethora of epidemiological studies has demonstrated associations between measures of fetal health and health as well as socioeconomic outcomes later in life. In general, these studies are not convincing from a causal perspective, since they often regress the future outcomes on measures of early health, potentially problematic since these measures may also be correlated with factors such as mother's biological constraints, socioeconomic status, exposure to infections, and genetics.

Exceptions to the studies of a correlational nature are represented by studies exploiting variation within siblings or twins, as well as exposure to exogenous health shocks during the fetal stage. Examples of the latter include studies examining the outcomes of individuals exposed to environmental insults, such as influenza pandemics. The perhaps most well-known example is represented by the 1918-1919 Spanish flu pandemic, examined in a range of settings, including the US (Almond 2006; Myrskylä et al. 2013) and Sweden (Karlsson, et al. 2014). The results of Almond (2006) suggest a negative long-term influence on socio-economic as well as health outcomes in older ages, the latter largely replicated by Myrskylä and colleagues (2013). Other environmental shocks that have been examined include the 1957 Asian flu (Kelly 2009), the 1944-1945 Dutch hunger winter (e.g., Rooij et al. 2010; Scholte et al. 2012), the 1959-1961 Chinese famine (Chen and Zhou 2007; Almond et al. 2010), and the 1986 Chernobyl disaster (Almond et al. 2009). The iodine-supplementation program implemented in Tanzania in the 1990s (Field et al. 2009) represents one positive shock. In sum, there is evidence that all of these events had effects on human capital or cognitive attainment. Evidence on health has, however, been more mixed. Whereas Rooij et al. (2010) and Scholte et al. (2012) find that the Dutch hunger winter reduced heights and increased hospital visits in older ages, Kelly (2009) finds that heights were affected by the Asian flu only if the mother was a smoker. Almond et al. (2009) find no effects of radioactive fallout on inpatient hospital visits in childhood, and Field et al. (2009) find no effects on self-reported health, as measured at age 10-13. Apart from the diverse contexts, these differences in results may reflect that different indicators of health were used, and that these were measured at different ages.

Studies relying on within-sibling or within-twin variation benefit from the ability to address the particular effect of birth weight. This is especially the case for monozygotic twins, since these are (almost) identical genetically and most often share the same placenta. Existing twin studies represent a multitude of contexts, and have tended to focus on relatively small samples of twins. The literature has examined the influence of differences in birth weight within twin pairs on a range of health outcomes, ranging from the incidence of specific conditions such as ischemic heart disease (IHD) to mortality risk at different ages.

A common result is that estimates from twin models are lower or to a lesser extent statistically significant than those obtained in correlational studies, which fail to properly take into account the selection processes into being born low birth weight. In a review article, Huxley et al. (2002) suggest that associations between birth weight and blood pressure found in the

literature may to a large extent reflect publication bias as well as inappropriate adjustment for confounders, such as those accounted for by comparing twins. There are also studies considering other outcomes within twin pairs, such as diabetes, asthma and ADHD, in some cases reporting significant effects (e.g., Poulsen et al. 1997; Örtqvist et al. 2009; Kindlund et al. 2010; Pettersson et al. 2014). In addition to the common problem of small sample sizes, one crucial limitation of the literature is the inability to follow individuals and to compare outcomes over time.

Studies in economics have reported that birth weight differences within twin pairs are related to infant and child mortality (Almond et al. 2005; Black et al. 2007; Conley and Strully 2012; Oreopoulos et al. 2008; Royer 2009) as well as BMI and height (Black et al. 2007; Saldarriaga 2015). There is also evidence of an effect on education (Black et al. 2007; Oreopoulos et al. 2008; Royer 2009; Xie et al. 2016) and labor market outcomes (Black et al. 2007; Oreopoulos et al. 2008). While most of these studies benefit from using comprehensive registry-based data and sound empirical strategies for achieving causal estimates, it is not always clear how to interpret the results. It is, for example, far from clear how a certain effect on an individual's height may be interpreted in terms of health problems, and at what ages.

3. Data and method

3.1 Data

Our data come from the Swedish Interdisciplinary Panel (SIP) database, administered at the Centre for Economic Demography, Lund University. The database comprises several population-wide registers which have been linked together through personal identifiers. Data is available for all individuals born in Sweden between 1973 and 1995, including their parents and siblings, followed until 2012.

The article relies on information measured at birth, including birth weight and gestational age. Since other studies (e.g., Black et al. 2007) have found that effects of birth weight tend to be non-linear and there is no crucial threshold, our main analysis uses the logarithm of birth weight, controlling for gestational age. For the outcome variables, we rely on data on hospital admissions and discharges, with information on specific diagnoses. Since 1987, this register covers the entire country, but about half of all counties (the administrative regions in Sweden responsible for health care) reported to the register already in 1973. We also rely on data on spells of sickness absence and disability pension, both referred to as sickness absence in the

analysis. Since regulations surrounding the eligibility for sickness and disability benefits have changed over time, we focus on outcomes observed in the most recent year available; 2011. Similar to the hospital data, we have information on the start and end dates of each spell, as well as the underlying medical diagnoses. One caveat with the data on leaves due to sickness and disability is that only spells exceeding fourteen days are observable, due to the data providing agency only taking responsibility for the payment of benefits from this point. As a result, if individuals with poor birth outcomes have a larger tendency not only to experience longer but also shorter sickness absence spells, the estimates we produce may be viewed as lower bounds of the overall effects.

The analysis focuses on several different outcomes, measuring various aspects of an individual's health. First, we examine overall morbidity, measured through the number of days of hospitalization at different ages between 0 and 30, or days on sickness absence in 2011, when the study population is between the age of 29 and 38 years of age. We exclude adult visits or absences related to pregnancy since our interest lies in health and not in the effect of birth weight on fertility.

We proceed to distinguish between major diagnosis types, which previous evidence indicates may be linked to birth weight. These include circulatory conditions (e.g., Barker 1995; Huxley et al. 2007); respiratory conditions (e.g., Seidman et al. 1991; Brooks et al. 2001); endocrine, nutritional and metabolic conditions (e.g., Hales et al. 1991; Curhan et al. 1996); and mental conditions (e.g., Thompson et al. 2001; Manzardo 2011). During infancy, we also consider inpatient visits due to perinatal problems. While there is no specific threshold, these hospitalizations are often a direct consequence of a relatively low birth weight and they may as such be more reflective of hospitals' expectations of health problems rather than of actual health. We create indicators for whether individuals were diagnosed with a certain type of condition and we also consider the number of days at hospital or on sickness leave due to the various conditions.

When examining child outcomes, we focus on outcomes during infancy (age 0), at age 1, 2-5, 6-12 and between the age 13 and 16, respectively. After necessary sample selection criteria have been applied, the sibling sample, consisting of native Swedes born between 1973 and 1995, amounts to 1,060,072 individuals observed from birth. Our twin sample, where we only include complete twin pairs, includes 27,246 individuals. When considering outcomes at ages later than 0, these samples shrink somewhat further, as only those who survived up to the beginning of the age interval of interest will be examined.

The adult sample is represented by native Swedes born between 1973 and 1981 and observed between the age of 18 and 30, amounts to 371,623 individuals after the sample selection criteria have been applied. The samples shrink somewhat further when considering sickness absences, as we only include those surviving up to age 2011. Table 1 shows descriptive statistics for the childhood and Table 2 for the adult outcome samples.

Table 1: Descriptive statistics, childhood sample

	Childhood, siblings	Childhood, twins
Birth year	1986.434(5.740)	1987.315(5.982)
Birth parity	1.937(0.962)	1.935(0.999)
Gestational days	279.073(12.434)	260.053(17.120)
Log birth weight	8.155(0.175)	7.851(0.233)
Hospital days, age 0	1.420(6.851)	5.859(14.785)
Hospital days, age 1	0.520(3.543)	0.654(5.378)
Hospital days/year, 2-5	0.274(2.436)	0.281(1.813)
Hospital days/year, 6-12	0.304(2.248)	0.283(1.632)
Hospital days/year, 13-16	0.244(2.422)	0.234(2.495)
Hospital age 0, perinatal (0/1)	0.046	0.213
Hospital, respiratory (0/1)	0.179	0.192
Hospital, endocrine etc. (0/1)	0.018	0.022
Hospital, circulatory (0/1)	0.006	0.008
Hospital, mental (0/1)	0.024	0.026
Hospital days age 0, perinatal	0.602(4.888)	4.501(12.686)
Hospital days/year, respiratory	0.058(0.941)	0.079(0.999)
Hospital days/year, endocrine etc.	0.019(0.625)	0.024(0.446)
Hospital days/year, circulatory	0.009(0.898)	0.014(1.221)
Hospital days/year, mental	0.031(0.937)	0.032(0.590)
Individuals	1,060,072	27,246

Note: For childhood outcomes observed later than age 0, descriptives refer to individuals surviving up to the beginning of the age interval, since only those are used in the regressions.

Source: Swedish Interdisciplinary Panel, authors' calculations

Table 2: Descriptive statistics, adulthood sample

	Siblings	Twins
Birth year	1976.956(2.502)	1977.020(2.589)
Birth parity	1.868(0.902)	1.970(1.009)
Gestational days	281.071(12.369)	263.532(17.158)
Log birth weight	8.150(0.167)	7.852(0.214)
Hospital days/year	0.324(3.367)	0.362(3.508)
Hospital, respiratory	0.037	0.038
Hospital, endocrine etc.	0.018	0.017
Hospital, circulatory	0.014	0.015
Hospital, mental	0.036	0.036
Hospital, respiratory days/year	0.019(0.775)	0.018(0.296)
Hospital, endocrine etc. days/year	0.016(0.607)	0.015(0.351)
Hospital, circulatory days/year	0.012(0.293)	0.022(1.036)
Hospital, mental days/year	0.181(3.255)	0.241(3.896)
Sickness days	13.185(59.747)	13.319(60.705)
Sickness, respiratory	0.005	0.003
Sickness, endocrine etc.	0.003	0.002
Sickness, circulatory	0.002	0.002
Sickness, mental	0.037	0.034
Sickness, respiratory days	0.172(5.184)	0.125(4.075)
Sickness, endocrine etc. days	0.262(8.364)	0.255(8.298)
Sickness, circulatory days	0.228(8.123)	0.330(10.116)
Sickness, mental days	7.398(46.805)	7.266(47.153)
Individuals	371,623	10,286

Source: Swedish Interdisciplinary Panel, authors' calculations

3.2 Method

The aim of the analysis is to quantify the causal relationship between conditions experienced during the fetal stage, measured through birth weight, and health outcomes through childhood and into adulthood. As highlighted before, simply regressing various outcomes on birth weight is potentially problematic. We circumvent this problem by focusing on a sample consisting of biological siblings as well as twins. Hence, it will be assumed that differences in birth weight within siblings or twin pairs largely can be interpreted as resulting from physiological processes in the fetus, as other factors relating to the mother are held constant. In the case of single-placenta twins (constituting a majority of MZ twins) birth weight differences may be viewed as randomly assigned, and reflecting nutritional supply (Bryan 1992; Bajoria et al. 2001). Like many other studies, we have no information on zygosity or on whether twins have shared a single placenta. We can, however, check the robustness of our

results by restricting attention to same-sex twin pairs, who are more often MZ and therefore more often single-placenta. Doing this, we find results that are very similar to the ones based on twins in general.

Our empirical analysis is based on regressions of the following form.

$$Y_{ij} = \alpha + \beta H_{ij} + X_{ij}\gamma + f_j + \varepsilon_{ij}$$

Here, i is index for child and j for sibling or twin pair. Y is a health outcome observed after birth, such as the number of days in hospital at a certain age. H is the logarithm of birth weight and X is a vector of individual controls. These controls include gestational age, as well as dummies for the birth year, calendar month of birth, birth parity, and sex of the child. They also include dummies for the birth year, calendar month of birth, and educational attainment of the mother. Standard errors are clustered at the family (i.e., mother) level.

Sibling (i.e. biological mother) fixed effects (f_j) allow us to account for any factor that is specific to the family, for example parenting style, lifetime wealth, and biological constraints of the mother. If the mother is subject to different shocks at different pregnancies and these shocks only operate through effects on birth weight, results from these models can be interpreted as causal effects of birth weight. If effects also operate through other channels, the estimated effect of birth weight may be thought of as representing the effects of various shocks affecting the uterus of a certain mother at different times. In other words, effects do not necessarily the effect of birth weight per se. Using the fixed effects approach on twins, we can potentially come closer to a causal effect of birth weight as we account for both genetic and environmental influences to a larger degree. In this case, birth weight cannot be thought of as representing shocks to the entire utero environment, since both twins share the same environment.

4. Results

Throughout the results section, we focus on three types of specifications. First, we run ordinary least squared (OLS) regressions based on the sibling sample. Here, sibling fixed effects are not included and we refer to these regressions as “naïve.” Second, we run regressions adding sibling fixed effects. Third, we run twin fixed effects regressions, based on the twin sample.

4.1 Childhood hospitalizations

We begin our analysis by considering short- and medium-term outcomes in the form of hospitalizations in childhood and adolescence. Results are shown in Table 3. Our naïve OLS specification (column A) reveals a strong relationship between birth weight and inpatient visits early in life. For hospitalizations during infancy (age 0), the coefficient is about -3.2, implying that a ten percent increase in the logarithm of birth weight is associated with 0.32 fewer days of hospital visits. It also implies that a one standard deviation increase in birth weight (0.175 log units) reduces the number of days in inpatient care at the mean by about 40 percent.

Table 3: Regression output, inpatient visits in childhood

	A	B	C
<i># of days/year</i>			
Age 0	-3.247*** (0.083)	-4.525*** (0.140)	-4.534*** (0.663)
Age 1	-0.342*** (0.036)	-0.468*** (0.067)	-0.493* (0.266)
Age 2-5	-0.135*** (0.024)	-0.212*** (0.038)	-0.107 (0.096)
Age 6-12	-0.076*** (0.019)	-0.152*** (0.032)	-0.159* (0.090)
Age 13-16	-0.044** (0.020)	-0.031 (0.035)	0.124 (0.141)
<i>Whether diagnose</i>			
Perinatal, age 0	-0.081*** (0.002)	-0.129*** (0.003)	-0.099*** (0.015)
Respiratory, age 0-16	-0.013*** (0.003)	-0.031*** (0.005)	-0.056** (0.024)
Endocrine etc., age 0-16	-0.021*** (0.001)	-0.031*** (0.002)	-0.026** (0.011)
Circulatory, age 0-16	-0.002*** (0.001)	-0.004*** (0.001)	-0.007 (0.008)
Mental, age 0-16	-0.019*** (0.001)	-0.019*** (0.002)	-0.10 (0.010)
<i># of days/year, diagnose</i>			
Perinatal, age 0	-2.407***	-3.288***	-2.698***

	(0.064)	(0.105)	(0.468)
Respiratory, age 0-16	-0.051***	-0.078***	-0.104*
	(0.011)	(0.020)	(0.058)
Endocrine etc., age 0-16	-0.022***	-0.040***	-0.052**
	(0.004)	(0.010)	(0.023)
Circulatory, age 0-16	-0.032**	-0.084***	-0.145
	(0.016)	(0.030)	(0.120)
Mental, age 0-16	-0.028***	-0.031**	0.015
	(0.009)	(0.014)	(0.037)

Notes: * indicates significance at the 10 percent level, ** significance at the 5 percent level, and *** significance at the 1 percent level. Controls include birth year, birth month, parity, mother's birth year, mother's birth month, mother's education, gestational age and sex. Regressions were clustered at the mother level. Model A is an OLS model based on the sibling sample and Model B adds sibling fixed effects. Model C is a twin fixed effects model based on the twin sample.

Accounting for sibling fixed effects (column B) produces a coefficient of -4.5, which is, in fact, more strongly negative than the OLS estimate. At the mean, this translates into a reduction in inpatient days of 56 percent when birth weight increases by a standard deviation. The fact that this effect is larger (and significantly larger) than its OLS counterpart may potentially reflect parental responses, such as taking a lighter sibling more often to the hospital. However, in a likely scenario where birth weight is only a proxy for underlying health status, another explanation is possible: There are family-specific measurement errors involved when using birth weight to indicate health, and these get cancelled out when applying the sibling fixed effects. A very similar estimate is also obtained when applying the twin fixed effects (column C). Indeed, our results suggest that the relationship between birth weight and early inpatient visits may be viewed as causal.

Birth weight is also related to inpatient visits after the first year of life, albeit with an effect of a substantially smaller magnitude, in absolute as well as relative terms. The OLS specification (column A) suggests that an increase in birth weight is associated with a continuously declining decrease of days in hospital care as the individual grows older. More specifically, at age 1, a one standard deviation increase in birth weight is linked to 0.06 fewer hospital days (11.5 percent reduction at the mean). The corresponding figures at age 2-5 is 0.02 days annually (8.6 percent), with age 6-12 and age 13-16 both at 0.01 days (4.4 and 3.2 percent, respectively). Model B, estimated with sibling fixed effects and thus taking into account everything that is shared between siblings, tends to produce stronger effects, but the qualitative patterns remain. With the exception of ages 13-16, all effects are statistically

significant, suggesting a gradually decreasing influence from birth weight as the individual ages, both in relative and in absolute terms. Turning to the twin sample (column C), we find similar estimates; however, estimated with less precision due to the substantially smaller sample size. The substantive conclusions remain unchanged.

We now turn to examine if birth weight influences the probability of being diagnosed with certain types of conditions, where we consider hospitalizations for perinatal problems at age 0 and hospitalizations for respiratory; endocrine, nutritional and metabolic; circulatory and mental conditions between the age 0 and 16. Similar to the previous results, those results obtained from a more naïve OLS specification (column A) or with sibling fixed effects (column B) are rather similar, but the latter tend to be stronger. Birth weight is negatively related to all diagnoses we study, with sizeable effects, considering the mean probability of each separate outcome. The models with sibling fixed effects predict a reduction in the risk of cause specific hospitalization from a one standard deviation increase in birth weight ranging from 3 percent (respiratory) to 30 percent (endocrine, nutritional and metabolic) and 49 percent (perinatal). Due to a lower precision, the results from the twin sample (column C) are significant to a lesser extent, with significant effects only found for perinatal, respiratory and endocrine conditions. While the reduction in the probability of hospitalization at the mean from a one standard deviation increase in log birth weight (0.233) is similar for endocrine, nutritional and metabolic diseases (28 percent), the corresponding responses for perinatal conditions is smaller (11 percent), while that for respiratory conditions is larger (7 percent).

Turning lastly to the annual number of days spent in inpatient care due to the same conditions, we find that perinatal issues, with statistically significant effects across all model specifications (columns A-C), drive most of the effect on visits at age 0. Still, these hospitalizations do not account for all of the effect of birth weight within twin pairs; regressing an indicator for “number of inpatient days due to non-perinatal issues” on birth weight produces a highly significant effect (regression results not shown). As regards the remaining types of conditions, observed between the ages 0-16, the results largely mirror those that have previously been obtained. More specifically, the effect of birth weight is statistically significant as well as of a similar magnitude across all diagnose types in the naïve OLS (column A) and sibling fixed effect (column B) specifications. For twins (column C), the statistically significant estimates mirror those obtained for siblings.

4.2 Adult hospitalizations

In Table 4, we show results for hospitalizations in adulthood, between the age of 18 and 30. Our naïve OLS estimates (column A) result in an estimate of about -0.16, suggesting that a one standard deviation increase in log birth weight (0.17) is associated with an 8 percent reduction in the annual number of days of hospitalization at the mean. Adding sibling fixed effects, reducing the influence of omitted variable bias, results in a – by now familiar – larger point estimate (0.24), which translates to a 12 percent reduction at the mean from a one standard deviation increase in birth weight.

Table 4: Regression output, inpatient visits in adulthood (age 18-30)

	A	B	C
# of days/year	-0.157*** (0.041)	-0.240*** (0.079)	-0.467 (0.398)
Whether diagnose			
Respiratory	-0.005** (0.002)	-0.000 (0.004)	0.020 (0.022)
Endocrine etc.	-0.001 (0.002)	-0.002 (0.003)	0.009 (0.014)
Circulatory	-0.003** (0.001)	-0.003 (0.003)	0.017 (0.014)
Mental	-0.023*** (0.002)	-0.019*** (0.004)	-0.015 (0.020)
# of days/year, diagnose			
Respiratory	-0.038*** (0.010)	-0.024 (0.018)	0.008 (0.035)
Endocrine etc.	-0.024*** (0.007)	-0.031** (0.014)	-0.004 (0.041)
Circulatory	-0.010*** (0.004)	-0.009 (0.007)	-0.002 (0.121)
Mental	-0.115*** (0.040)	-0.213*** (0.076)	-0.633 (0.399)

Notes: * indicates significance at the 10 percent level, ** significance at the 5 percent level, and *** significance at the 1 percent level. Controls include birth year, birth month, parity, mother's birth year, mother's birth month, mother's education, gestational age and sex. Regressions were clustered at the mother level. Model A is an OLS model based on the sibling sample and Model B adds sibling fixed effects. Model C is a twin fixed effects model based on the twin sample.

Compared to the results reported in the previous section, where the influence of birth weight on hospitalization declined with the individual's age, the estimates suggest an interesting

pattern. While during childhood and adolescence, the influence of birth weight on the annual number of days of hospitalization, at ages 13-16 represented by a point estimate of -0.031, in the ages 18-30 the corresponding estimate has increased to -0.240 (column B). There thus seems to be a resurgence of the effect of birth weight in adulthood. Both according to naïve OLS and sibling fixed effects results, the effect is significantly larger than in late adolescence. Albeit statistically insignificant, a similar story also emerges from the estimates from the twin sample (column C). More specifically, the point estimate of -0.467 is substantially larger than the ones obtained for adolescence, indeed in terms of size being similar to the estimate obtained for infancy.

Turning to the probability of being hospitalized or the number of days being hospitalized for a specific type of diagnosis, OLS results suggest that all types of health problems are affected. Fixed effects results are not too different, but due to a lower precision, there is only significant evidence on mental conditions, when considering the probability of diagnose (considering the number of days, there is also evidence that endocrine, nutritional and metabolic conditions are affected). Both according to OLS and fixed effects estimates, the impact on mental problems is quite sizeable, suggesting that hospitalization due to mental illness represents almost the entire link between birth weight and hospitalization. While the results from the twin sample (column C) mirrors those already discussed, they again suffer from a lack of precision, arguably largely due to a small sample size. The point estimates, however, again suggest the link between birth weight and hospitalization due to mental illness as the main driver of the results.

4.3 Adult sickness absences

Lastly, we consider effects on sickness absences in adulthood, observed during 2011, when the study sample is between the ages of 29 and 38. Given the larger variation in this outcome than in hospitalizations, potential effects of early life health may be easier to detect; on the other hand, if sickness absences are a less severe outcome than hospitalizations, effects may also be weaker and less significant. Results are shown in Table 5. For total number of days, the naïve OLS estimates (column A) are once again indicating that any possible bias resulting from this estimation method underestimates the link between birth weight and later life health, as estimates are smaller than the ones based on sibling fixed effects. According to the sibling fixed effect estimates (column B), a one standard deviation increase in birth weight reduces the predicted number of sick days by about 1.7 per year, corresponding to almost 13 percent

of the mean number of sickness days per year. The magnitude of the effect is thus quite similar to what was encountered for adulthood hospitalizations, suggesting the processes being largely identical. Within twin pairs (column C), there is no evidence of an effect on the number of sickness days, as the coefficient is too imprecisely estimated to be statistically significant. Moreover, the magnitude of the point estimate is considerably smaller than that obtained in the sibling fixed effect specification; with a 3.5 percent reduction in sick days at the mean from a one standard deviation increase in birth weight.

Table 5: Regression output, sickness absences in adulthood (year 2011)

	A	B	C
<i># of days</i>	-9.010*** (0.739)	-12.110*** (1.368)	-2.208 (6.367)
<i>Whether diagnose</i>			
Respiratory	-0.000 (0.001)	-0.001 (0.002)	-0.008 (0.007)
Endocrine etc.	0.001 (0.001)	-0.000 (0.001)	-0.001 (0.006)
Circulatory	-0.000 (0.000)	0.000 (0.001)	0.000 (0.005)
Mental	-0.017*** (0.002)	-0.025*** (0.004)	-0.023 (0.020)
<i># of days, diagnose</i>			
Respiratory	0.013 (0.064)	0.070 (0.122)	-1.069** (0.483)
Endocrine etc.	-0.078 (0.104)	-0.087 (0.198)	0.428 (0.983)
Circulatory	-0.208** (0.101)	-0.235 (0.194)	0.378 (1.120)
Mental	-5.210*** (0.580)	-7.912*** (1.078)	-1.365 (4.965)

Notes: * indicates significance at the 10 percent level, ** significance at the 5 percent level, and *** significance at the 1 percent level. Controls include birth year, birth month, parity, mother's birth year, mother's birth month, mother's education, gestational age and sex. Regressions were clustered at the mother level. Model A is an OLS model based on the sibling sample and Model B adds sibling fixed effects. Model C is a twin fixed effects model based on the twin sample.

We next consider the incidence of sickness absence due to different diagnoses. The naïve OLS (column A) and sibling fixed effect (column B) specifications again suggest that the link between early life health, operationalized through birth weight, and early adulthood health is

largely driven by mental conditions. This is suggested both by the estimates for whether the individual experiences a sickness absence spell due to a mental condition (-0.025, column B) and the number of days of sickness absence (-7.912, column B). Indeed, with the exception of a modest influence on the number of days of sickness due to circulatory conditions (-0.208, column A), all other estimates are both of a small magnitude and statistically insignificant. The findings suggest that half to two-thirds of the relationship between birth weight and the total number of sick days are due to mental problems, a share that, while being large, is smaller than what was previously obtained for hospitalizations. In fact, the share of the birth weight effect that is accounted for by mental illness roughly matches the share of total sickness days that are due to mental illness, which is somewhat above 50 percent.

The twin fixed effect model (column C) again yields estimates that are more erratic. While the coefficient for number of days of sickness due to mental illness indeed suggests that this contributes to about half of the total days of sickness absence, the point estimate is statistically insignificant. Instead, the estimates indicate that the only diagnose type that is significantly influenced by birth weight among twins is respiratory disease.

5. Conclusion

Using large-scale administrative data on birth outcomes, hospitalizations, and sickness absences, this article studied the impact of health at birth, measured by birth weight, during childhood, adolescence and into adulthood. In an attempt to sort out the effects of parental circumstances and behaviors as well as genetic factors, our analysis focused on comparing the outcomes of siblings and twins. In general, our estimates suggest largely similar effects when comparing naïve OLS and sibling fixed effect estimates, indicating that the selection on unobservable characteristics at the sibling level is small. While the twin fixed effect estimates are associated with important advantages, including a greater control over the influence of genes, the smaller sample size clearly limits statistical power, and the degree to which obtained results can be extrapolated to a broader population may potentially be questioned. We view our sibling fixed effects estimates as our preferred estimates, but note that results obtained based on twins point in the same direction.

Overall, the results from our specifications, examining the influence from birth weight on health throughout childhood, adolescence and into early adulthood, suggest a U-shaped pattern. More specifically, with sizeable effects during the first years of life in absolute as well

as in relative terms, the influence diminishes in importance during later childhood and adolescence. In adulthood, past the age of 18 and until age 30, the influence again becomes stronger, with broadly similar conclusions regarding both hospitalizations and sickness absences. While there is a lot of previous evidence on the effects of birth weight and later diagnoses, either in childhood or later in adulthood, our study provides comparable estimates across different ages.

During childhood and adolescence, the disadvantage associated with a lower birth weight is associated with a broad spectrum of diseases, suggesting a generally worse health rather than one single disease mechanism. In childhood, we find significant and important effects on all types of diagnoses we study. In early adulthood, a different pattern is found, with a strong emphasis on mental conditions. Indeed, the effect of birth weight on the number of days of hospitalization in adulthood is almost entirely driven by to mental conditions. Albeit not quite as dramatic, a similar story emerges for sickness absence.

Industrialized countries currently face financial challenges due to rising costs of health care and sickness absences. Here, one key challenge lies in the increasing rates of disability due to mental problems (Prins 2010). Having documented the role of birth weight, we believe our results have important policy implications, pointing at the potential benefit of programs such as WIC (The Special Supplemental Nutrition Program for Women, Infants and Children) in the US, which provides nutritional counselling and supplemental food to pregnant women. Programs like this are not only likely to reduce costs due to physical health problems among children, but also due to mental health problems in the longer run.

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