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Another look at returns to birthweight

Shiko Maruyama [a,*], Eskil Heinesen [b]

[a] University of Technology Sydney, PO Box 123, Broadway, NSW 2007, Australia
[b] Rockwool Foundation Research Unit, Ny Kongensgade 6, DK 1472, Copenhagen K, Denmark

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ABSTRACT

We revisit the causal effect of birthweight. Because variation in birthweight in developed countries primarily stems from variation in gestational age rather than intrauterine growth restriction, we depart from the widely-used twin fixed-effects estimator and employ an instrumental variable – the diagnosis of placenta previa, which provides exogenous variation in gestation length. We find protective effects of additional birthweight against infant mortality and health capital loss, such as cerebral palsy, but in contrast to sibling and twin studies, no strong evidence for non-health long-run outcomes, such as test scores. We also find that short-run birthweight effects have diminished significantly over the decades.

Introduction

A large interdisciplinary literature has documented the association between premature infants (defined by gestational age or birthweight) and various later-life outcomes, from long-term morbidities such as respiratory disorders, neurodevelopmental disabilities, insulin resistance, and hypertension (Institute of Medicine, 2007) to socioeconomic outcomes, such as academic achievement and labor market success, as well as societal costs (Behrman and Rosenzweig, 2004; Almond et al., 2005; Miller et al., 2005; Conley et al., 2006; Black et al., 2007; Figlio et al., 2014; and Bharadwaj et al., 2018a). A key question of policy importance is to what extent the association between prematurity and later-life outcomes reflects causation because a weak causal link implies limited returns to interventions. This is a global and stringent policy issue because advances in neonatal care have resulted in significant worldwide improvements in the survival of premature infants (Institute of Medicine, 2007). The majority of recent causal studies achieve the identification of birthweight effects by comparing twins born to the same mother who differ in their weights at birth. This approach works because this twin fixed-effects estimator allows researchers to focus on the causal effect of interest by holding constant other hard-to-measure factors that may affect both birthweight and later outcomes, such as the mother's health knowledge and risky behaviors.

* Corresponding author.
E-mail address: shiko.maruyama@uts.edu.au (S. Maruyama).

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However, the twin fixed-effects estimator also holds gestational age constant. This means that researchers identify the effect of variations in birthweight driven by variations in intrauterine growth restriction (IUGR) within twin pairs (Behrman and Rosenzweig, 2004; Almond et al., 2005). This fact calls into question the common view that there is a single parameter called the return to birthweight, which can be identified by the twin fixed-effects estimator. Indeed, IUGR is not the primary determinant of birthweight. Almond et al. (2005) clarify this point using US twin data, and state that "despite the significant contribution of gestation length to variation in birth weight, the emphasis of the literature has instead been on intrauterine growth retardation." Kramer (1987) also notes that, in developed countries, "intrauterine growth retardation is far less prevalent. Not only are low birth weight rates much lower, but most low birth weight babies are premature rather than growth retarded." This conceptual issue arises because researchers have used birthweight as a *proxy for neonatal health*. Although the wide availability of birthweight data has motivated its use in the literature, there are numerous determinants of birthweight, from genes to in-utero environment to the timing of birth, which obscure what we mean by the effect of neonatal health. In the monozygotic-twin fixed-effects estimator, the abovementioned determinants of birthweight are all removed (leaving variation in nutrition intake within the twin pair as the cause of birthweight variation), whereas the effects of these determinants all remain in the sibling fixed-effects estimator, and as discussed below, the studies on siblings and twins report substantially different results.

In this paper, we depart from the fixed-effects approach by employing a new instrumental variable (IV) to estimate the effect of variations in birthweight driven predominantly by variations in gestational-age rather than IUGR. Because the overall variation in birthweight in developed countries primarily derives from variation in gestational age rather than IUGR, this approach comprises an important supplement to twin studies. Furthermore, gestation can be directly influenced by medical interventions, at least to some limited extent, whereas genetic factors and much of the in-utero environment cannot. Schulkind and Shapiro (2014), Lalumia et al. (2015), and Borra et al. (2016) find evidence for shifting the timing of birth to shorten gestation, whereas Jürges (2017) studies the effect of delaying birth.

Our IV is the diagnosis of an obstetric complication called placenta previa, in which the placenta is abnormally positioned in the lowest part of the uterus. The condition is rather unpredictable and often results in lower birthweight by premature birth. There are a small number of known risk factors, but our data allow us to control for them and thus avoid bias due to confounders. Placenta previa occurs merely as an abnormal position of the placenta, hence it has little long-term impact on children except for effects via immaturity, whereas most other serious obstetric and neonatal conditions, such as maternal cancer and malformation of the fetus, have significant direct consequences. This IV approach therefore enables us to infer the causal effect of birthweight that reflects maturity at birth but not IUGR, genetic factors, or congenital anomaly.

We take advantage of administrative birth records in Denmark between 1981 and 2013. We investigate an extensive set of outcomes: mortality, morbidity, hospitalization, test scores, labor market outcomes, income, disability pension, teen pregnancy, birthweight of the subjects' children, criminal tendency, height, and weight. Utilizing the data from more than three decades, we also study the dynamics of the birthweight effect across and within birth cohorts.

Because of the different approaches for identification, our results are remarkably different from those of sibling and twin studies. Most of these studies have found significant long-term birthweight effects on health, cognitive, and socioeconomic outcomes, with magnitudes often similar to or even larger than ordinary least squares (OLS) estimates (Conley and Bennett, 2000; Conley and Bennett, 2001; Behrman and Rosenzweig, 2004; Black et al., 2007; Currie and Moretti, 2007; Oreopoulos et al., 2008; Royer, 2009). At the same time, when infant mortality and infant health are studied, whereas sibling fixed-effects estimates tend to be similar to OLS estimates (at least qualitatively), most of the twin fixed-effects estimates yield substantially smaller birthweight effects relative to their OLS counterparts (Almond et al., 2005; Conley et al., 2006; Black et al., 2007; Oreopoulos et al., 2008). These findings of twin studies appear to imply that the correlation between birthweight and infant health is largely non-causal, yet that birthweight does have long-term effects.

Our results are summarized as follows. First, additional birthweight has significant positive causal effects on child health by reducing mortality and permanent health capital loss, such as neurodevelopmental disability and mental retardation. Second, the effect of birthweight on health diminishes as a child grows older. Third, for most non-health outcomes of adolescents and young adults, such as test scores and crime tendency, we find no significant effect. Fourth, the effect of birthweight on infant health has diminished significantly over the last three decades. Our results indicate that the main driver behind the long-term effect of immature birth is permanent health capital loss incurred around birth. If an infant survives without permanent disabilities, birthweight is no longer a critical initial condition. This conclusion emphasizes significant returns to perinatal interventions to reduce premature birth and avoid long-term morbidities. This is consistent with the causal study of neonatal care by Bharadwaj et al. (2013). Explaining the differences between our findings and findings in the twin studies is beyond the scope of this work, but we replicate twin fixed-effects estimates to show that the patterns found in the twin studies hold in our data of twins as well.

We provide extensive discussion of the validity of our IV. We show that placenta previa is one of the most unpredictable obstetric complications. It is also rather unpredictable compared to various birth outcomes, which include IVs widely used in the literature. There are only a few known risk factors for placenta previa, and they are observable in our data and hence can be controlled in our regression analysis. We also show that our results are robust to omitting these risk factors from our regressions. Even when the IV validity does not hold exactly, our

bias simulation shows that large bias is unlikely under mild assumptions due to the large explanatory power of placenta previa in the first-stage regression. We also address concerns about the effects of cesarean section and behavioral response in subsequent pregnancies.

Empirical framework

Setup

For the statistical analysis of birthweight effect, we use the following model,

$$y_i = X_i\theta_x + \theta_{BW}\ln BW_i + e_i \quad (1)$$

where y_i represents an outcome of child i; X_i is a vector of observable variables at the beginning of pregnancy including a constant term; $\ln BW_i$ is child i's birthweight in logs; θ's are unknown parameters; and e_i is an error term that captures unobserved determinants of y_i. We use the log of birthweight as our main treatment variable following Black et al. (2007) who examine the explanatory power of various birthweight measures, such as BW_i, $\ln BW_i$, and indicators for low birthweight, and find that $\ln BW_i$ fits best for their outcome variables. A non-linear relationship between y_i and $\ln BW_i$, however, may well exist. For example, the high risk of very large birthweight has been widely documented in the literature. We nevertheless rely on the linear specification for several reasons. First, the linear model facilitates comparison of birthweight effects across various outcomes. Second, it also facilitates comparison with previous studies, since it is a standard specification in the literature (Black et al., 2007; Oreopoulos et al., 2008). Third, the use of instruments and fixed effects is straightforward. Fourth, Black et al. (2007) report that different measures of birthweight tend to provide consistent results. We also find that our results change little if we use non-log birthweight. Lastly, the log of BW_i has an intuitive interpretation – diminishing returns to birthweight. We also report the results of three specifications with different low birthweight dummies: (1) birthweight less than 3,500 g (approximately the average birthweight in our sample), (2) birthweight less than 2,500 g (low birthweight, or LBW), and (3) birthweight less than 1,500 g (very low birthweight, or VLBW). These models allow us to examine whether birthweight effects are concentrated around the very low end of the birthweight distribution.

Placenta previa

The OLS estimate of θ_{BW} in (1) will have no causal interpretation if unobserved determinants of birthweight affect y_i. We instead conduct IV regressions with the diagnosis of placenta previa as the instrument that provides exogenous variation in the maturity of newborns at birth.

The placenta develops during pregnancy to connect the developing fetus to the uterine wall of the mother. The placenta normally implants in the upper uterine segment. In placenta previa, the placental tissue overlies or is proximate to the cervix. The incidence is estimated to be 3.5–4.6 per 1000 births (Faiz and Ananth, 2003). Women with placenta previa often present with painless hemorrhage, and placenta previa can be confirmed by ultrasound. A placenta previa that completely overlies the cervix requires a cesarean delivery. The likelihood of vaginal delivery increases with the distance between the placenta and the cervix. For stable patients, it is standard to schedule cesarean delivery after 36 weeks of gestation to decrease the risk of neonatal death, whereas significant bleeding may necessitate earlier preterm delivery. Placenta previa leads to low birthweight predominantly as a result of the higher frequency of premature birth,[1] but also because of intrauterine growth restriction by nutritional deficiency, though the latter channel appears to be small and remains controversial (Valero de Bernabé et al., 2004). See Oyelese and Smulian (2006) and Lockwood and Russo-Stieglitz (2015) for a review of placenta previa.

For placenta previa to be a valid IV, two more conditions must be satisfied. First, the "assignment" of placenta previa to a mother must be unrelated to unobserved factors that are relevant to the outcome. Second, the IV must not affect y_i except via the causal channel concerned. The first condition is likely to be satisfied because the occurrence of placenta previa is highly unpredictable (Lockwood and Russo-Stieglitz, 2015). The pathogenesis of placenta previa remains unknown. Proposed hypotheses relate it to previous pregnancies and uterine surgery, but placenta previa occurs to first pregnancy as well. Epidemiological research has identified a few risk factors: increased maternal age, multiple pregnancy, increased parity, prior cesarean delivery, prior pregnancy termination, prior uterine surgery, smoking, male fetus, and cocaine use (Oyelese and Smulian, 2006; Lockwood and Russo-Stieglitz, 2015).[2] The clinical information available in the Danish fertility register allows us to control for these known factors.[3] This is in sharp contrast to many other obstetric complications that significantly affect birthweight, such as infection and premature rupture of membranes, for which the medical literature has identified strong risk factors related to unobservable maternal variables such as mental stress, alcohol intake, and health literacy.

The second condition is also likely to be satisfied because while many neonatal and obstetric complications originate from pathological factors in the body of the mother or fetus (e.g., maternal cancer and malformation of the fetus), placenta previa occurs merely as an abnormal position of the placenta, which is a temporary organ that is discarded after birth. Placenta previa may lead to serious hemorrhage and complications, which are obviously

[1] Nørgaard et al. (2012) study singleton pregnancies in Denmark in 2001-2006 and report that the incidence rates of low birthweight (<2,500 grams) for pregnancies with and without placenta previa are respectively 18.4% and 4.0%, and the rates of preterm births (<37 weeks) for pregnancies with and without placenta previa are 31.7% and 5.1%.

[2] Little evidence has been found for association with alcohol consumption (Macones et al., 1997; Aliyu et al., 2011).

[3] Maternal cocaine use is also purported to be an independent risk factor for placenta previa (Macones et al., 1997). We are unable to control for cocaine use, but a large bias is unlikely because of the rarity of cocaine use. Only about 1% of 16-34-year-old Danish women had used any amount of cocaine within a year according to surveys in 2000, 2005, 2008, and 2010 (Danish Health and Medicines Authority, 2012, p. 17).

of clinical importance, but hemorrhage is maternal rather than fetal and the incidence of serious complications can be regarded as negligible for our purposes.[4] The effect of cesarean section (controlling for gestation) on child health is controversial, but most studies to date have been observational and do not address selection bias. A few recent causal studies find insignificant or somewhat protective causal effects of cesarean section (Hannah et al., 2000; Jensen and Wust, 2015).[5]

Another potential source of bias concerns the possibility that, even if placenta previa occurs purely randomly, the experience of placenta previa might cause the mother to take more precautions during subsequent pregnancies (e.g., more frequent checkups), or it might even influence the next fertility decision. These possibilities imply a systematic difference in the underlying characteristics of groups with and without placenta previa. To address this potential bias, we conduct the IV estimation based only on the first-child sample and confirm that main results do not change. See Appendix A2 for the results of the first-child sample estimation. Lastly, the exclusion restriction can be violated by differences in hospital quality. Good hospitals may be able to detect even a very minor degree of placenta previa early and reduce the risk of premature birth by providing additional checkups and advice, which can have a separate protective effect on health outcomes. To address this concern, we create dummies for 40 major hospitals and home births and include them in our regression analysis to control for hospital fixed effects.[6] County fixed effects are also included and expected to reduce a similar concern regarding cross-county variation in the quality of prenatal care. A series of further sensitivity analyses regarding the validity of our IV discussed in Section Further Evidence for the Reliability of the IV Estimation also indicate the robustness of our conclusions.

Causal framework - what is fixed and what is not?

The causal study of birthweight is problematic not only because of confounders in e_i but also because of the use of birthweight as a proxy for neonatal health, which obscures the interpretation of estimated causal effects. To see this point, let pp_i denote child i's indicator for birth with placenta previa. The intuition of the identification can be seen in the form of the standard Wald estimator,

$$\frac{E(y_i|X_i, pp_i = 1) - E(y_i|X_i, pp_i = 0)}{E(\ln BW_i|X_i, pp_i = 1) - E(\ln BW_i|X_i, pp_i = 0)}$$

which highlights the fact that our IV estimator of the birthweight effect, $\hat{\theta}^{IV}_{BW}$, is defined by the exogenous variation provided by placenta previa, arguably estimating the effect of the maturity of newborns at birth. Because our causal estimates are driven dominantly by the timing of birth, our birthweight effect concerns the "natural" development of the fetus. For example, very low birthweight infants typically exhibit prematurity in many organs including the brain, lungs, and eyes, and the effects of such immaturity are captured in our birthweight effect. Not captured in our estimate are the effects of genetic factors, congenital malformation, extreme nutritional deprivation, maternal infection, and maternal behavior (e.g., alcohol abuse).

Another possible formulation is to examine the effect of gestation rather than birthweight. This might appear reasonable because placenta previa predominantly lowers birthweight because of its effect on gestation. We nonetheless prefer to focus on birthweight for two reasons. First, it remains controversial whether placenta previa affects intrauterine growth, and this possibility precludes us from claiming our IV results as entirely the effect of gestation. Second, past studies have almost exclusively investigated the effect of birthweight (with Oreopoulos et al. (2008) as a notable exception), and our focus on birthweight makes our findings comparable to the literature. In either case, however, gestation is the main determinant of birthweight, and therefore our conclusions hold the same. In addition, we estimate "reduced-form" regressions in which we regress outcome variables on the placenta previa indicator rather than $\ln BW$. As reported in Appendix A2, the results are consistent with the IV results.

Although our IV estimation relies on the linear parametric regression, (1), it is reasonable to interpret our IV estimates in the spirit of local average treatment effects (LATEs) in the Rubin causal model with potential outcomes (Angrist et al., 1996), or rather as average causal response functions, since our treatment, $\ln BW_i$, is continuous and we condition on a large number of covariates (Angrist and Pischke, 2009, page 175-188). This interpretation involves two different margins of effect heterogeneity: the level of birthweight and the determinants underlying birthweight. Regarding the former, our results do not necessarily hold for infants with exceptionally high or low birthweight, but as reported below, placenta previa occurs in almost the entire birthweight distribution, which means that our estimator is not driven by a very particular part of the distribution. Regarding the latter margin of heterogeneity, our IV reflects changes in the natural development of the fetus. The focus on this margin makes our estimates policy relevant, as discussed earlier. The fact that placenta previa does not increase birthweight implies that the monotonicity assumption holds, which is essential in this interpretation of the results (Angrist et al., 1996).

Lastly, we do not model causal pathways after birth, following the majority of previous studies, hence $\hat{\theta}^{IV}_{BW}$ captures various channels such as physiological mechanisms

[4] For example, Nørgaard et al. (2012) report that the neonatal mortality rates of singleton pregnancies with and without placenta previa in Denmark are 1.2% and 0.7% respectively. This difference is statistically significant at 95% but becomes insignificant if maternal characteristics are controlled for.

[5] Consistent with this, Nørgaard et al. (2012) report a smaller neonatal mortality rate of pregnancies with placenta previa that are delivered by cesarean section compared to the overall mortality rate of pregnancies with placenta previa.

[6] A concern in using hospital fixed effects is the fact that the choice of the hospital might be endogenous because the detection of placenta previa and the risk of premature birth may incline mothers toward larger hospitals. To address this concern, we re-estimate all the regressions without hospital dummies and the results turn out to be almost identical. The results without hospital dummies are available upon request.

and the effect generated by the behavioral response of the child and parents. This paper instead aims to provide new insights by studying an extensive set of outcomes in the same econometric framework.

Sibling and twin fixed-effects approach

A popular approach to avoid bias due to omitted variables is the fixed-effects estimator. In case of the sibling fixed-effects estimator, Eq. (1) becomes

$$y_{ij} = X_{ij}\theta_x + \theta_{BW}\ln BW_{ij} + a_j + \eta_{ij} \quad (2)$$

where y_{ij} represents an outcome of child i born to mother j, the term a_j refers to mother-specific time-invariant unobservables, and η_{ij} is the residual term. Compared to $\hat{\theta}_{BW}^{OLS}$, the sibling fixed-effects estimator, $\hat{\theta}_{BW}^{FE}$, is less prone to suffer from omitted variable bias, but there may remain time-varying factors in η_{ij} that are correlated with birthweight. For example, the mother's relationship with the husband, health knowledge, and expectation and affection toward the new child are time-varying and difficult to measure, and may influence birthweight at each pregnancy (Behrman and Rosenzweig, 2004). If these factors also affect the child's future outcome, $\hat{\theta}_{BW}^{FE}$ is biased. Interaction between siblings is another potential confounder for $\hat{\theta}_{BW}^{FE}$, causing the violation of SUTVA (Stable Unit Treatment Value Assumption, see Angrist et al., 1996). Furthermore, if the first child's outcome, y_{1j}, influences the second child's baseline controls, X_{2j}, $\hat{\theta}_{BW}^{FE}$ is biased due to the violation of strong exogeneity. Note also that even when all the assumptions are satisfied, $\hat{\theta}_{BW}^{OLS}$ and $\hat{\theta}_{BW}^{FE}$ have different interpretation because many gene-related unobservable factors are removed by a_j.

Given these sources of bias, the twins fixed-effects estimator has become the workhorse approach in the recent literature. It is argued that the twin fixed-effects estimator provides a clearer causal framework to obtain an unbiased estimate of θ_{BW} because monozygotic twins share the same genes, the same in-utero environments, and the same gestation. Heinesen et al. (2015) discuss two problems with this approach. First, the mechanism that causes differences in intrauterine growth rate (and thereby birthweight) between twins is unclear and we do not know what $\ln BW_{ij}$ proxies for. Second, it may suffer from bias due to not only postnatal interaction but also "in-utero interaction" between twins. Our IV approach offers an alternative way to understanding returns to birthweight by avoiding these econometric difficulties inherent in fixed-effects estimation.

Data

Administrative data and population selection

We draw data from Danish administrative registers. The birth register contains population data for newborns with person identifiers of the newborns and biological parents and a range of clinical and demographic variables about the mother and neonate. We construct each mother's fertility history by linking her pregnancy records. The birth register is considered to be of high quality with regard to validity and coverage (Blenstrup and Knudsen, 2011).

We study births from 1981 onwards because many variables in Danish registers are available only after 1980. The following are excluded from our analysis: stillbirths, children whose mother identifier is missing, children born overseas, and adopted children and their biological siblings (in total less than 1%). We retain children whose father is not identified in our population (1% of our final sample) by including an indicator for missing fathers. A very small number of observations with missing values, highly unrealistic values, and other data problems are discarded. For clear interpretation, we also exclude multiple pregnancies from the main analysis because their distribution of birthweight is substantially different. Singleton siblings of twins are retained.

The rest of this section describes the variables used in the analysis. For further details of the dataset construction, see Appendix A1.

The instrument

An indicator for placenta previa is constructed by combining the birth register and the hospital admission register. The placenta previa indicator takes the value of one if either of the two registers indicates placenta previa. The two registers provide consistent information in the vast majority of cases. In our data from 1981 to 2013, 7913 births are associated with placenta previa, and its incidence is 0.41%, which is within the range found in the literature (e.g., 0.35–0.46% in Faiz and Ananth, 2003). We find no apparent time trend.

Control variables

We obtain from the birth register the following control variables: the sex of the child, year- and month-of-birth dummies, hospital dummies, the mother's age at conception, birth order, smoking habits, the histories of past pregnancies, past cesarean sections, past spontaneous and induced abortions, and past stillbirths. From the hospital admission register, we construct a variable for the number of days spent by the mother in the hospital during the 180 days around the conception, except for pregnancy-related admission, to control for the mother's general health.

The other demographic and socioeconomic variables are constructed from various registers: indicators for the mother's highest level of education completed (less than 9 years, 9 years, upper secondary, low and medium tertiary, and high tertiary education); marital status; coresidence status of the biological mother and father; immigrant status; the mother's working status (an indicator for whether she was working most of the year) and labor income in the previous year; the father's information (the indicator for missing identifier, age at conception, labor income and working status in the previous year, and education indicators); household wealth income and public transfer in the previous year; and county dummy variables. Regression analysis also includes interaction terms between conception month dummies and the mother's income and work

Table 1
Characteristics of Children and Parents.

	Births without placenta previa		Births with placenta previa	
All singleton births (1981–2013)				
N and proportions	1,937,355	99.59%	7913	0.41%
Birthweight (in grams)	3495.2	(564.7)	2943.3	(761.0)
Log(Birthweight)	8.143	(0.187)	7.944	(0.321)
Birthweight < 3500 g (birthweight less than average)	0.479	(0.500)	0.771	(0.421)
Birthweight < 2500 g (low birth weight, LBW)	0.037	(0.190)	0.237	(0.425)
Birthweight < 1500 g (very low birth weight, VLBW)	0.006	(0.077)	0.052	(0.222)
Gestation (in days since conception)	279.6	(12.6)	260.9	(22.4)
Female	0.487	(0.500)	0.455	(0.498)
Birth order	1.797	(0.927)	1.963	(1.033)
Mother's age at conception	28.12	(4.94)	29.97	(5.15)
Parity (number of past pregnancies)	1.146	(0.931)	1.356	(1.120)
Any past cesarean section	0.091	(0.287)	0.170	(0.376)
Cesarean section during the past 2 years	0.012	(0.107)	0.022	(0.145)
Number of past cesarean sections	0.101	(0.338)	0.204	(0.494)
Number of past abortions recorded (both spontaneous and induced)	0.330	(0.718)	0.490	(0.886)
Indicator for stillbirth in the past	0.008	(0.090)	0.014	(0.118)
Number of days in hospital between 90 days before and after conception (excl. obstetrics-related admission)	0.169	(2.203)	0.199	(1.498)
Indicator for mother's smoking during pregnancy (available only for 1991 and onwards)	0.200	(0.400)	0.208	(0.406)
Cohabitation with biological father on Jan 1 before birth	0.850	(0.357)	0.852	(0.355)
Married at delivery (may not be with biological father)	0.545	(0.498)	0.586	(0.493)
Mother's education				
Less than 9 years	0.045	(0.208)	0.063	(0.243)
9 years (reference group)	0.228	(0.420)	0.232	(0.422)
Upper secondary education	0.414	(0.493)	0.380	(0.485)
Tertiary education (low and medium)	0.226	(0.418)	0.237	(0.426)
Tertiary education (high)	0.087	(0.281)	0.087	(0.282)
Mother working in previous year	0.812	(0.391)	0.818	(0.386)
Mother's labor income in previous year (measured in millions of 2010 Danish Krone, DKK1,000,000)	0.179	(0.135)	0.185	(0.142)
Father's information missing	0.010	(0.099)	0.013	(0.115)
Father's age at conception	30.85	(5.82)	32.38	(6.11)
Father's education				
Less than 9 years	0.064	(0.244)	0.081	(0.273)
9 years (reference group)	0.174	(0.379)	0.155	(0.362)
Upper secondary education	0.484	(0.500)	0.470	(0.499)
Tertiary education (low and medium)	0.170	(0.376)	0.175	(0.380)
Tertiary education (high)	0.108	(0.310)	0.119	(0.324)
Father working in previous year	0.903	(0.296)	0.915	(0.278)
Father's labor income in previous year (DKK1,000,000)	0.273	(0.199)	0.284	(0.206)
Household wealth income in previous year (DKK1,000,000)	0.044	(0.346)	0.046	(0.188)
Household public transfer in previous year (DKK1,000,000)	0.066	(0.081)	0.069	(0.084)
Immigrant indicator (either parent or both)	0.141	(0.348)	0.139	(0.346)

Notes: Based on singleton births only. Standard deviations are in parentheses. Parental income variables are measured in millions of 2010 Danish Krone (DKK1,000,000 ≈ EUR134,000, Nov 12, 2015), deflated by the consumer price index. The variables regarding smoking, maternal education, and the father's characteristics are not observed for every child. In the regression analysis, these variables are set to zero when missing, and indicators for a missing value are used. In this table, the summary statistics of each variable are based on observations for which the variable is not missing.

status to account for the effect of pregnancy on the previous year's labor supply.

Table 1 shows the summary statistics of the characteristics of children and parents broken down into births with and without placenta previa. The group with placenta previa exhibits a 15.7% lower birthweight and a 6.7% shorter gestation. Table 1 also shows mean differences for the known risk factors for placenta previa, such as maternal age, male fetus, past cesarean sections, and past abortions. Some variables, such as income variables and the father's age, exhibit unexpected non-negligible differences between the two groups, but these differences are predominantly due to their association with maternal age. Thus, our data show a pattern largely consistent with the medical literature.

Placenta previa, birthweight, and size for gestation

Fig. 1 compares the birthweight distribution by placenta previa status, illustrating a higher frequency of low birthweight neonates when placenta previa complicates pregnancy. The variance is also larger, although the distribution still covers the entire range of birthweight.

Unknown risk factors for placenta previa that are not controlled even by our extensive control variables may exist. However, we argue that such unknown factors are unlikely to bias our IV estimates significantly because even those known risk factors together explain only an extremely tiny part of the incidence of placenta previa. In Table 2, we run OLS regressions with the full set of covariates for placenta previa, other selected obstetric

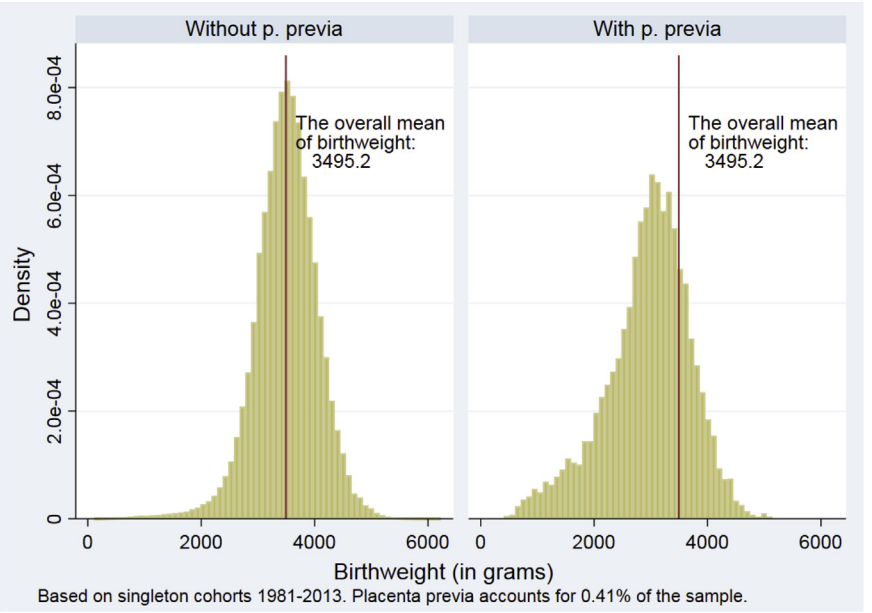

Fig. 1. Distribution of Birthweight.

Table 2
Predictability of Selected Obstetric Complications and Birth Outcomes.

Dependent variable:	Incidence	R^2
Placenta previa	0.41%	0.0020
Placental abruption	0.49%	0.0026
Breech position	3.73%	0.0102
Prolonged labor	26.67%	0.0927
Low birthweight (BW < 2500 g)	3.83%	0.0250
Preterm (<37 weeks)	4.65%	0.0290
Born in 1st quarter	24.24%	0.0059
Born in 4th quarter	23.62%	0.0080
Multiple birth	1.75%	0.0150
Baby female	48.66%	0.0001

Notes: The results are based on OLS linear probability regressions with the cohorts born 1981–2013. The number of observations is 1,945,268. Each regression includes the full set of control variables; see Model [1] in Table 4 for the detailed specification. In the multiple birth regression, the unit of observations is a delivery with at least one live-born child rather than a singleton birth, and $N = 1{,}979{,}919$. Some controls are not used due to the nature of the outcome variables: e.g., monthly dummies in quarter-of-birth models and sex in the models of multiple birth and sex.

complications, and birth outcomes, and report their R^2 coefficients to compare the predictability of placenta previa and other outcomes. We also include outcomes that have been widely used as an IV in the literature: multiple birth in Rosenzweig and Wolpin (1980), the quarter of birth in Angrist and Krueger (1992), and the sex of the child in Angrist and Evans (1998). The results show that birth events such as preterm birth, low birthweight, and multiple pregnancy are roughly ten times more predictable than placenta previa. The sex of the child is the only outcome that clearly dominates our instrument, and the fact that we can explain only 0.2% of the variation in the occurrence of placenta previa indicates the very high degree of randomness of placenta previa.

The fact that placenta previa occurs in almost the entire birthweight distribution (Fig. 1) and the fact that the extensive set of socioeconomic and demographic variables has almost no explanatory power for the incidence of placenta previa (Table 2) imply that our estimates can be interpreted as average causal response over nationally representative sample rather than over a very specific sample.

Whether placenta previa alters the size for gestational days is essential information in understanding what exogenous variation the placenta previa IV captures. Fig. 2 illustrates that, conditional on gestational days at birth, placenta previa makes little difference in birthweight. Placenta previa tends to reduce birthweight slightly around average gestational days at birth, but the opposite is true for premature births, the main interest of our study. This figure requires caution because it is not the growth curve widely used in the obstetric context; it is created from birth outcomes and the timing of birth may be influenced by intra-uterine growth and placenta previa. Nevertheless, the difference by placenta previa status shown in Fig. 2 is not comparable to the average birthweight reduction due to placenta previa of around 16%, providing support that our IV estimation captures predominantly variation in prematurity rather than intra-uterine growth.

Outcome variables

We investigate three broad sets of outcome variables. The first set includes outcomes related to health, such as mortality, morbidity, and hospital admission. The second set consists of socioeconomic outcomes: educational attainment, student/working status, income, disability pension, teen pregnancy, the birthweight of their children, and criminal offense. Table 3 summarizes these outcome variables with their definitions, overall means, and means conditional on low birthweight. These mean values illustrate the worse outcomes of low birthweight infants. The table also shows the population used for each outcome.

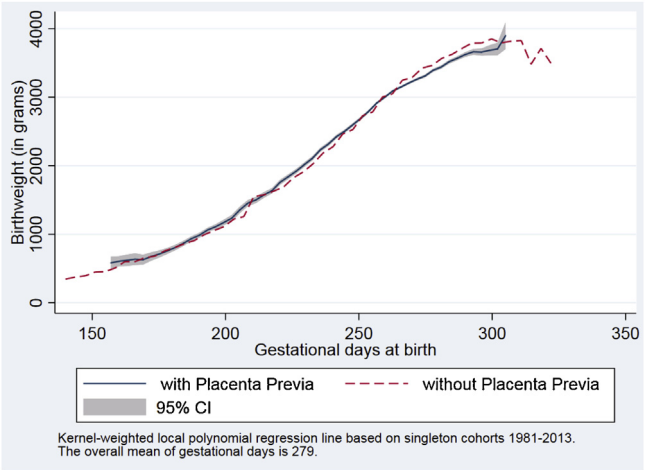

Fig. 2. Size for Gestational Days by Placenta Previa Status.

Most outcomes are analyzed conditional on the child's survival up to a particular age. Although this conditioned sample may give rise to selection bias, such bias will not significantly change our results because of the very low child mortality rate in Denmark. The birth cohort used for each outcome is determined by the availability of data and the number of years after birth necessary to observe the outcome variable.

The third set of outcomes are variables constructed from the military conscription register. All men in Denmark are required to attend an examination session for military conscription once they turn 18, and we observe military qualification, IQ, height, weight, and BMI. However, there are exceptions for attendance requirement, and because of the concern for potential selection bias, we only present a summary of these results, providing detailed explanation and discussion in Appendix A3.

Results

Before turning to the estimates of the birthweight effect, we discuss the first-stage regressions of $\ln BW_i$. Table 4 reports selected coefficient estimates based on the cohorts born 1981–2013 from [1] OLS without controls, [2] OLS with controls, and [3] OLS with sibling fixed effects. Reported in parentheses are standard errors robust to arbitrary heteroskedasticity and correlation within each grandmother cluster. Placenta previa reduces birthweight by 17.7–20.0%, and the coefficients are statistically significant at the 0.1% level. Models [1] and [2] show that including the controls reduces the estimated effect of placenta previa on $\ln BW_i$ only marginally, from 20% to 19%. The F statistics for the relevance of this IV in the three models are 3020, 2889, and 1816, respectively, so the issues of a weak instrument and finite sample bias are not a concern. Moreover, this strong correlation implies that minor violations of the exogeneity condition are unlikely to cause significantly biased estimates (Wooldridge, 2010; Eq. 5.36, p. 108). We provide further quantitative discussion on the size of potential bias in Section Further Evidence for the Reliability of the IV Estimation.

The other determinants of birthweight identified in the literature show the expected signs (Valero de Bernabé et al., 2004). Birthweight tends to be low for female newborns of mothers with past cesarean sections, poor health, and smoking habits. Birthweight increases with birth order, particularly from the first to the second child. Socioeconomic factors also have significant predictive power for birthweight. Cohabitation with the father increases birthweight, and so does being married, though to a lesser extent. Birthweight increases with parental education, particularly with maternal education. Birthweight is low if the father is not working. The same relationship holds for the mother's work status, although it is not reported because its interaction with conception month dummies requires considerable space. Birthweight decreases with maternal age, although this is again not reported due to a large number of age dummy variables. Lastly, immigrants and families with social welfare dependence tend to have low birthweight babies.

Returns to birthweight: health outcomes

Table 5 reports the estimates of the birthweight effect on health outcomes. Each row is dedicated to one outcome, reporting estimated coefficients on $\ln BW$. We report results from five models: (1) OLS with no other control; (2) OLS with the control variables; (3) OLS with sibling fixed effects; (4) IV regression; and (5) IV regression with sibling fixed effects. The full set of control variables is included in all regressions except for (1). Our preferred model is (4) because a valid IV yields consistent estimates even without fixed effects. Moreover, as discussed above, fixed effects may lead to biased estimates and may complicate interpretation. Nevertheless, the comparison of (4) and (5) serves as a robustness check for our IV estimation. Reported in the parentheses are standard errors robust to arbitrary heteroskedasticity and clustering by the grandmother identifier, which allows statistical dependence among siblings and cousins and provides more conservative standard errors than clustering by mother

Table 3
Definitions and Summary Statistics of Outcome Variables.

Outcome variables	Birth cohort	Mean: Overall	Mean: <2500 g	Population conditional on:
Health outcomes				
Infant mortality (within 365 days of birth)	1981–1995	0.006	0.066	Live birth
Infant mortality (within 365 days of birth)	1996–2013	0.003	0.048	Live birth
5 minute APGAR score	1981–1995	9.876	9.368	Live birth
5 minute APGAR score	1996–2013	9.865	9.431	Live birth
Received CPAP (Continuous Positive Airway Pressure) in neonatal ward	2002–2013	0.033	0.328	Live birth
Neurodevelopmental disability by 2nd birthday	1981–1995	0.002	0.018	Survival up to 2nd birthday
Neurodevelopmental disability by 2nd birthday	1996–2011	0.002	0.017	Survival up to 2nd birthday
Mental retardation by 10th birthday	1981–1991	0.005	0.020	Survival up to 10th birthday
Mental retardation by 10th birthday	1992–2003	0.006	0.019	Survival up to 10th birthday
Number of days in hospital by 2nd birthday	1981–1995	6.53	24.09	Survival up to 2nd birthday
Number of days in hospital by 2nd birthday	1996–2011	4.69	25.35	Survival up to 2nd birthday
Hospital admission: 2nd to 5th birthday	1981–1993	0.171	0.256	Survival up to 5th birthday
Hospital admission: 5th to 10th birthday	1981–1993	0.174	0.238	Survival up to 10th birthday
Hospital admission: 10th to 15th birthday	1981–1993	0.141	0.178	Survival up to 15th birthday
Hospital admission: 15th to 20th birthday	1981–1993	0.165	0.193	Survival up to 20th birthday
Purchased medication for asthma: before the 3rd birthday	1995–2004.6	0.515	0.624	Survival up to 3rd birthday
Purchased medication for asthma: 3rd to 6th birthday	1995–2004.6	0.230	0.325	Survival up to 6th birthday
Purchased medication for asthma: 6th to 9th birthday	1995–2004.6	0.123	0.178	Survival up to 9th birthday
Educational attainment				
Completed Grade 9 by the year of age 16	1981–1997	0.846	0.744	Observed Jan 1 before age 17
Standardized score of national exam at Grade 9 (≈ age 16): Mean of 4 mandatory subjects	1986–1997	0.016	−0.122	Observed Jan 1 before age 17 and exam scores of at least 3 subjects at age 15, 16, or 17
Standardized exam score: Danish (oral)	1986–1997	0.029	−0.063	Same as above
Standardized exam score: Danish (written)	1986–1997	0.048	−0.073	Same as above
Standardized exam score: English	1986–1997	0.006	−0.082	Same as above
Standardized exam score: Mathematics	1986–1997	0.049	−0.174	Same as above
Standardized exam score: Science	1986–1997	0.018	−0.098	Same as above
Other socioeconomic outcomes				
Income (excl. public transfer, in 2010 DKK) in the year of age 24	1981–1989	144,239	135,275	Observed Jan 1 before age 24
Worked or student in year of 22nd birthday	1981–1990	0.873	0.800	Observed Jan 1 before age 22
Receipt of disability pension during the three calendar years of age 19 to 21	1981–1992	0.011	0.039	Observed Jan 1 before age 21
Number of weeks on disability pension: the three calendar years of age 19 to 21	1981–1992	1.318	5.000	Observed Jan 1 before age 21
Total amount of disability pension (in 2010 DKK): 3 calendar years of age 19 to 21	1981–1991	4,651	17,517	Observed Jan 1 before age 21
Teen motherhood (pregnancy started before 20th birthday, abortions included)	1981–1993	0.049	0.060	Women, survival up to 20th birthday
Teen fatherhood (pregnancy started before 20th birthday, abortions included)	1981–1993	0.012	0.014	Men, survival up to 20th birthday
Birthweight of the first child by age 22: Women	1981–1992	3392.5	3,180.2	Women, 1 st child by the year of age 22
Birthweight of the first child by age 22: Men	1981–1992	3,386.4	3,330.1	Men, 1 st child by the year of age 22
Any criminal sentence by 22nd birthday	1981–1992	0.122	0.125	Survival up to 22nd birthday
Confinement by 22nd birthday	1981–1992	0.045	0.049	Survival up to 22nd birthday
Any violent crime charge by 22nd birthday	1981–1992	0.038	0.038	Survival up to 22nd birthday

Notes: All statistics are based on singleton births only. The two mean values reported are the overall mean and the mean for low birthweight children (birthweight less than 2,500 g). Neurodevelopmental disability comprises cerebral palsy (CP), loss of vision, and hearing impairment. Hospital admission after age 10 excludes admissions related to pregnancy and birth. (DKK100,000 ≈ EUR13,400, Nov 12, 2015).

identifier.[7] Reduced-form regressions in which we replace ln*BW* with the placenta previa indicator are also estimated and reported in Appendix A2.

Rows [1A] and [1B] in Table 5 show the effect of birthweight on one-year mortality based on the cohort born 1981–1995 and the cohort born 1996–2013, respectively. The OLS coefficient of −0.0831 in Row [1A] implies that a 10% increase in birthweight is associated with a reduction in one-year mortality by approximately 8.3 deaths per 1000 births. Adding controls slightly increases the estimate, and using sibling fixed effects almost doubles the magnitude. Use of the placenta previa IV considerably increases standard errors, but its three estimates are similar and statistically significant, and their magnitudes are between the OLS estimates with and without fixed effects. Using fixed effects and the IV leads to a larger estimate, probably because the simple OLS is downward biased due

[7] We ignore issues of multiple hypothesis testing. It will not affect our main conclusion (dominatingly statistically significant effects on health outcomes and insignificant effects on non-health outcomes).

Table 4
Determinants of Birthweight – First-Stage Regressions with Selected Variables.

Dependent variable: ln*BW*	[1] OLS, no controls	[2] OLS with controls	[3] Sibling FE
Placenta Previa	−0.200****	−0.188****	−0.177****
	(0.00364)	(0.0035)	(0.0042)
Female		−0.0346****	−0.0375****
		(0.00026)	(0.00029)
First child		−0.0400****	−0.0314****
		(0.00061)	(0.00073)
Birth order		0.0117****	0.0197****
		(0.00050)	(0.00074)
Any past cesarean section		0.0171****	0.0557****
		(0.0018)	(0.0020)
Cesarean section during the past 2 years		−0.00468****	0.000693
		(0.0015)	(0.0018)
Number of past cesarean sections		−0.0372****	−0.0382****
		(0.0016)	(0.0017)
Number of days in hospital around conception		−0.000728****	−0.000177**
		(0.000077)	(0.000080)
Mother smoked during pregnancy		−0.0573****	−0.0188****
		(0.00047)	(0.00066)
Cohabitation status		0.00849****	0.00652****
		(0.00047)	(0.00056)
Married		0.00135****	0.00105**
		(0.00033)	(0.00046)
Father's age		−0.0000794**	−0.000594****
		(0.0000374)	(0.000099)
Parental education (reference: 9 years)			
Mother: Less than 9 years		−0.00721****	
		(0.00095)	
Mother: Upper secondary		0.0158****	
		(0.00047)	
Mother: Tertiary (low/medium)		0.0246****	
		(0.00056)	
Mother: Tertiary (high)		0.0253****	
		(0.00074)	
Father: Less than 9 years		−0.00564****	−0.000877
		(0.00080)	(0.00185)
Father: Upper secondary		0.00617****	0.00386****
		(0.00046)	(0.00096)
Father: Tertiary (low/medium)		0.0122****	0.00467****
		(0.00057)	(0.00128)
Father: Tertiary (high)		0.0146****	0.00030
		(0.00068)	(0.00159)
Father working		0.0025****	0.0024****
		(0.00059)	(0.00073)
Father's labor income (in DKK 1000,000 ≈ EUR 134,000)		−0.000088	−0.00101
		(0.000875)	(0.00124)
Household annual wealth income (in DKK 1000,000 ≈ EUR 134,000)		0.00165**	0.00036
		(0.00067)	(0.00060)
Household annual public transfer (in DKK 1000,000 ≈ EUR 134,000)		−0.0575****	−0.0200****
		(0.00255)	(0.00323)
Either parent immigrant		−0.0178****	−0.0134****
		(0.00056)	(0.0020)
F statistics for significance of IV	3020.0	2878.3	1800.2
R^2	0.0046	0.0805	0.0880
N	1,945,268	1,945,268	1,576,311

Notes: The overall mean of ln*BW* is 8.143. The cohort born 1981–2013 is used. Standard errors robust to heteroskedasticity and grandmother clusters are in parentheses. The three models reported are: [1] OLS with no control; [2] OLS with controls; and [3] OLS with sibling fixed effects. Control variables used but not reported in the table are: year- and month-of-birth dummies; county dummies; major hospital dummies, indicators for the mother's age at conception (one dummy for every two years); indicators for parity (1, 2, 3, and 4+); indicators for the mother's past stillbirth and past abortions (1 and 2+); the mother's labor income and working status in the previous year; and indicators for missing father identifier and missing education information of the mother and father. The mother's working status and income are interacted with month-of-conception dummies. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$, **** $p < 0.001$.

to unobserved genetic factors related to body size. The infants of smaller parents tend to be smaller even if completely healthy, and such attenuating unobserved factors are in naïve OLS estimates but not in the fixed-effects and IV estimates.

A comparison of Rows [1A] and [1B] reveals a substantial reduction in magnitude over time in all models. The main contributing factor to this reduction is the trend in infant mortality – a decrease from 0.62% in the first period to 0.32% in the second period – reflecting advances in neonatal care

Table 5
Estimated Coefficients on ln*BW* – Health Outcomes.

OLS without controls	OLS	OLS with sibling FE	IV	IV with sibling FE
[1A] Infant mortality, cohort 1 (1981–1995) $\overline{Y}$ = 0.0062, *N* = 855,041				
−0.0831****	−0.0855****	−0.164****	−0.133****	−0.137****
(0.00167)	(0.00172)	(0.00348)	(0.0142)	(0.0266)
[1B] Infant mortality, cohort 2 (1996–2013) $\overline{Y}$ = 0.0032, *N* = 1,089,688				
−0.0651****	−0.0677****	−0.123****	−0.0355****	−0.0297*
(0.00149)	(0.00155)	(0.00294)	(0.00826)	(0.0160)
[2A] 5 minute APGAR score, cohort 1 (1981–1995) $\overline{Y}$ = 9.876, *N* = 847,194				
0.579****	0.579****	0.909****	1.561****	1.470****
(0.00985)	(0.0102)	(0.0209)	(0.102)	(0.176)
[2B] 5 minute APGAR score, cohort 2 (1996–2013) $\overline{Y}$ = 9.865, *N* = 1,084,201				
0.498****	0.500****	0.793****	0.972****	1.023****
(0.0109)	(0.0114)	(0.0220)	(0.0862)	(0.135)
[3] Received CPAP in neonatal ward (2002–2013) $\overline{Y}$ = 0.033, *N* = 711,746				
−0.256****	−0.256****	−0.286****	−1.011****	−1.024****
(0.00263)	(0.00269)	(0.00521)	(0.0403)	(0.0635)
[4A] Neurodevelopmental disability by age 2, cohort 1 (1981–1995) $\overline{Y}$ = 0.0025, *N* = 846,964				
−0.0186****	−0.0189****	−0.0247****	−0.0308****	−0.0240*
(0.000785)	(0.000808)	(0.00156)	(0.00,877)	(0.0129)
[4B] Neurodevelopmental disability by age 2, cohort 2 (1996–2011) $\overline{Y}$ = 0.0024, *N* = 973,479				
−0.0143****	−0.0146****	−0.0190****	−0.0217***	−0.0260**
(0.000654)	(0.000680)	(0.00130)	(0.00736)	(0.0104)
[5A] Mental retardation diagnosis by age 10, cohort 1 (1981–1991) $\overline{Y}$ = 0.0045, *N* = 583,355				
−0.0182****	−0.0179****	−0.0255****	−0.0275**	−0.0569***
(0.000897)	(0.000916)	(0.00198)	(0.0117)	(0.0218)
[5B] Mental retardation diagnosis by age 10, cohort 2 (1992–2003) $\overline{Y}$ = 0.0058, *N* = 742,962				
−0.0159****	−0.0153****	−0.0210****	−0.00701	0.0133
(0.000790)	(0.000820)	(0.00167)	(0.00900)	(0.0124)
[6A] Days in hospital before 2nd birthday, cohort 1 (1981–1995) $\overline{Y}$ = 6.53, *N* = 846,964				
−20.05****	−19.65****	−24.94****	−44.35****	−39.56****
(0.179)	(0.184)	(0.321)	(1.820)	(2.879)
[6B] Days in hospital before 2nd birthday, cohort 2 (1996–2011) $\overline{Y}$ = 4.69, *N* = 973,479				
−21.13****	−20.85****	−25.18****	−37.78****	−34.96****
(0.171)	(0.176)	(0.299)	(1.186)	(1.613)
[7A] Hospitalization: 2nd to 5th birthday (1981–1993) $\overline{Y}$ = 0.171, *N* = 712,737				
−0.113****	−0.102****	−0.119****	−0.185****	−0.128
(0.00280)	(0.00287)	(0.00646)	(0.0422)	(0.0831)
[7B] Hospitalization: 5th to 10th birthday (1981–1993) $\overline{Y}$ = 0.174, *N* = 709,896				
−0.0869****	−0.0764****	−0.0855****	−0.133***	−0.0940
(0.00276)	(0.00284)	(0.00636)	(0.0422)	(0.0825)
[7C] Hospitalization: 10th to 15th birthday (1981–1993) $\overline{Y}$ = 0.141, *N* = 708,091				
−0.0538****	−0.0408****	−0.0383****	−0.0904**	−0.0717
(0.00250)	(0.00258)	(0.00592)	(0.0387)	(0.0777)
[7D] Hospitalization: 15th to 20th birthday (1981–1993) $\overline{Y}$ = 0.165, *N* = 705,355				
−0.0523****	−0.0274****	−0.0116*	−0.0611	−0.0341
(0.00261)	(0.00269)	(0.00620)	(0.0401)	(0.0819)
[8A] Medication for asthma: before the 3rd birthday (1995–2004.06) $\overline{Y}$ = 0.515, *N* = 590,135				
−0.144****	−0.0971****	−0.111****	−0.338****	−0.434****
(0.00363)	(0.00370)	(0.00827)	(0.0569)	(0.101)
[8B] Medication for asthma: 3rd to 6th birthday (1995–2004.06) $\overline{Y}$ = 0.230, *N* = 585,663				
−0.124****	−0.0935****	−0.103****	−0.278****	−0.265***
(0.00330)	(0.00342)	(0.00736)	(0.0528)	(0.0879)
[8C] Medication for asthma: 6th to 9th birthday (1995–2004.06) $\overline{Y}$ = 0.123, *N* = 581,857				
−0.0615****	−0.0553****	−0.0595****	−0.160****	−0.163**
(0.00266)	(0.00276)	(0.00609)	(0.0430)	(0.0733)

Notes: The description of each outcome variable is followed by the birth years of the cohort used, the mean values of the outcome variable, and the numbers of observations in the OLS and first-child regressions, respectively. The effective number of observations of the fixed-effects regression is smaller than that of OLS. Standard errors robust to heteroskedasticity and grandmother clusters are in parentheses. For the list of control variables included in each regression, see the note to Table 4. * p < 0.1, ** p < 0.05, *** p < 0.01, **** p < 0.001.

such as the widespread use of surfactant in the 1990s. There is also a notable contrast between the time trends in the OLS and IV estimates, the IV estimates showing a much larger reduction than the OLS estimates. A possible explanation is that immaturity itself became less important because of improved neonatal care, while unobserved confounders that link birthweight and infant mortality (e.g., congenital malformation) remain at work. Another explanation for declining birthweight effect is the increasing trend in birth-

weight in Denmark.[8,9] Bharadwaj et al. (2018a) compare two birth cohorts 50 years apart and find similar estimates of long-run birthweight effect on income. This finding does not contradict with our finding because they take the standard twin fixed-effects approach, which does not capture the effect of immaturity related to gestation length.

Rows [2A] – [8C] concern other health outcomes: 5 min APGAR score (widely used health score for newborns); continuous positive airway pressure (CPAP, a ventilation therapy); neurodevelopmental disabilities (which comprise cerebral palsy, loss of vision, and hearing impairment); the diagnosis of mental retardation before the tenth birthday; hospital utilization; and medication purchase for asthma. Except for CPAP and asthma, we again report the results in two periods. In most cases, both the OLS and IV results highlight the protective role of extra birthweight for infant health. This is consistent with previous observational studies such as Leonard and Wen (2002) and the Institute of Medicine (2007), though not consistent with twin studies. Compared to OLS, the IV estimates are much larger for APGAR score, CPAP, hospitalization, and asthma, probably because these are outcomes strongly related to premature birth.

There is no time trend in APGAR score, neurodevelopmental disabilities, and mental retardation, unlike infant mortality. This fact is most likely due to the opposing force of improved neonatal care: it protects newborns from avoidable disabilities, but it also saves the lives of neonates who have a higher risk of morbidity. We nevertheless observe substantial reductions in the magnitude of the coefficients on $\ln BW$ (Rows [2A], [2B], [4A] – [5B]), particularly the IV estimates. This constitutes further evidence for the previous finding: the current role of immaturity is not as important as it was in the earlier period. However, we only observe a marginal reduction in IV estimates for Rows [6A] and [6B], probably because hospital days should be considered not only as health outcomes but also health input – improved medical treatment may require low birthweight babies to stay longer in hospital.

We investigate how the birthweight effect varies over time not only across cohorts but also within a cohort. The question of whether the influence of this initial condition at birth grows or diminishes as the child grows older has important implications for early medical interventions (Almond and Currie, 2011a). Rows [7A] to [7D] report the effect of birthweight on hospital admissions across the age groups of children. To delineate the age effect, we fix our population to those born between 1981 and 1993. A comparison of the four rows reveals that, while the hospital admission rate varies little by age, OLS and IV estimates both diminish as a child grows older.[10] Hummer et al. (2014) apply sibling fixed effects to Austrian data and find a similar reduction in the effect of birthweight on hospital stays. Thus, birthweight is crucial to infant mortality and infant health, but the impact of a birthweight "shock" fades out and surviving children catch up.[11] A similar pattern is found in the analysis for the purchase of medication for asthma. Rows [8A] to [8C] show a significant reduction in the estimated coefficients, although the catch-up in this case seems to be driven by significant reduction in the prevalence of asthma drug purchase.

Returns to birthweight: socioeconomic outcomes

The first group of outcomes reported in Table 6 concerns educational attainment. The first outcome shown in Row [1] is whether a child completes the 9th grade by the year the child reaches age 16. As discussed in Appendix A1.4, in Denmark this outcome largely captures the school entry decision at age 7, rather than later educational attainment. Row [1] indicates that an increase in birthweight by 10% raises the likelihood of completing the 9th grade by age 16 by 1.3–1.5% points. The size of this effect can be interpreted by comparing it with the effect of the birth month; children born in January are more likely to start the 1st grade in the year of age 7 than children born in December of the same year. The estimated coefficients in Row [1] indicate that January-born children having 10% less birthweight have the same propensity to complete the 9th grade by age 16 as those born with average birthweight in September and October.

Rows [2] – [2E] are the results for the standardized scores of the national exam held at the end of the 9th grade. The OLS estimates with and without fixed effects all exhibit highly significant positive correlations between birthweight and test scores. This is consistent with many past studies. The OLS estimate in the second column in Row [2] indicates that a 10% increase in birthweight is associated with an increase in mean exam scores of about 0.018 standard deviations. This becomes smaller when sibling fixed effects are applied. Interestingly, the OLS estimates for mathematics are considerably larger than for other subjects – a finding consistent with past studies (e.g., Torche and Echevarría, 2011; Figlio et al., 2014). IV estimates, however, do not indicate that infant maturity has positive causal effects on exam scores. Most IV estimates have negative signs, but their low precision precludes strong conclusions. Except for one estimate (for English), none are statistically different from zero.

The lack of evidence of beneficial birthweight effects on exam scores may appear contradictory with the statistically significant positive birthweight effect on timely Grade

[8] In our data, the population mean of birthweight has increased from 3,350 grams in the early 1980s to near 3,500 grams in 2000 and after, so that the resulting reduction in immature babies may have contributed to the reduction in the birthweight effect.

[9] It might be suspected that the effect of placenta previa on birthweight has a time trend, thus causing a spurious reduction in the birthweight effect, but further examination verifies that this is not the case. In the first-stage regression without fixed effects, the coefficient on placenta previa is −0.191 in the first period and −0.199 in the second period.

[10] To check whether this reduction is driven by attrition due to death, we conduct the same analysis making it conditional on survival to age 20, and find no difference. Attrition plays no role here due to the low mortality rate after age 2.

[11] Gupta et al. (2011) observe "catch-up" in the effect of birthweight on weight and height, but their finding merely reflects the fact that children born preterm remain behind other children until the growth curve flattens out.

Table 6
Estimated Coefficients on ln*BW* – Socioeconomic Outcomes.

OLS without controls	OLS	OLS with sibling FE	IV	IV with sibling FE
[1] Completed Grade 9 by year of age 16 (1981–1997) $\overline{Y}$ = 0.846, *N* = 942,141				
0.151****	0.153****	0.142****	0.135****	0.125**
(0.00240)	(0.00226)	(0.00458)	(0.0332)	(0.0577)
[2] Standardized exam score, Grade 9: overall mean (1986–1997) $\overline{Y}$ = 0.016, *N* = 627,364				
0.303****	0.183****	0.129****	−0.125	−0.122
(0.00607)	(0.00552)	(0.0104)	(0.0851)	(0.130)
[2A] Standardized exam score, Grade 9: Danish (oral) (1986–1997) $\overline{Y}$ = 0.029, *N* = 625,552				
0.136****	0.114****	0.0894****	−0.157	−0.145
(0.00761)	(0.00730)	(0.0158)	(0.114)	(0.198)
[2B] Standardized exam score, Grade 9: Danish (written) (1986–1997) $\overline{Y}$ = 0.048, *N* = 627,260				
0.168****	0.168****	0.127****	−0.0849	−0.0938
(0.00698)	(0.00647)	(0.0125)	(0.100)	(0.157)
[2C] Standardized exam score, Grade 9: English (1986–1997) $\overline{Y}$ = 0.006, *N* = 614,660				
0.195****	0.110****	0.0541****	−0.259**	−0.265
(0.00,785)	(0.00759)	(0.0151)	(0.116)	(0.187)
[2D] Standardized exam score, Grade 9: Mathematics (1986–1997) $\overline{Y}$ = 0.049, *N* = 625,492				
0.551****	0.317****	0.237****	0.0390	−0.0494
(0.00749)	(0.00696)	(0.0133)	(0.108)	(0.167)
[2E] Standardized exam score, Grade 9: Science (1986–1997) $\overline{Y}$ = 0.018, *N* = 599,824				
0.278****	0.142****	0.102****	−0.177	0.0311
(0.00777)	(0.00756)	(0.0164)	(0.123)	(0.209)
[3] Income in the year of age 24, excl. public transfers (1981–1989) $\overline{Y}$ = 144,239, *N* = 457,246				
23064.8****	13,174.7****	17,600.8****	−4447.7	−9764.1
(1579.2)	(1787.2)	(2625.2)	(17,699.5)	(33,437.5)
[4] Worked or was a student in the year of age 22 (1981–1990) $\overline{Y}$ = 0.873, *N* = 517,725				
0.124****	0.0819****	0.0797****	0.0989**	0.0796
(0.00298)	(0.00297)	(0.00708)	(0.0433)	(0.0850)
[5A] Receipt of disability pension over 3 years age 19–21 (1981–1992) $\overline{Y}$ = 0.011, *N* = 641,978				
−0.0359****	−0.0349****	−0.0501****	−0.0511***	−0.0871***
(0.00124)	(0.00127)	(0.00271)	(0.0159)	(0.0298)
[5B] Number of weeks of disability pension age 19–21 (1981–1992) $\overline{Y}$ = 1.318, *N* = 641,978				
−4.676****	−4.601****	−6.890****	−5.837***	−10.32***
(0.168)	(0.172)	(0.371)	(2.067)	(3.897)
[5C] Total disability pension transfers, 3 years age 19–21 (1981–1991) $\overline{Y}$ = 4,651, *N* = 579,123				
−16619.6****	−16468.7****	−24871.6****	−21805.7***	−38693.7**
(648.1)	(662.3)	(1477.2)	(8075.4)	(15,923.6)
[6A] Teen motherhood before 20th birthday (1981–1993) $\overline{Y}$ = 0.049, *N* = 343,566				
−0.0378****	−0.0101****	0.0177**	0.0257	0.207*
(0.00218)	(0.00217)	(0.00761)	(0.0336)	(0.110)
[6B] Teen fatherhood before 20th birthday (1981–1993) $\overline{Y}$ = 0.012, *N* = 361,841				
−0.00719****	−0.00162	0.00648*	0.0248**	0.0430
(0.00102)	(0.00105)	(0.00383)	(0.0125)	(0.0422)
[7A] Birthweight of the first child by age 22: women (1981–1992) $\overline{Y}$ = 3392, *N* = 23,544				
679.3****	688.5****	352.4***	−35.86	142.5
(24.20)	(24.63)	(132.5)	(374.6)	(2029.4)
[7B] Birthweight of the first child by age 22: men (1981–1992) $\overline{Y}$ = 3,386, *N* = 9,839				
370.7****	360.1****	−234.7	−351.0	1797.2
(35.01)	(36.56)	(431.3)	(417.1)	(3772.8)
[8A] Any criminal sentence by 22nd birthday (1981–1992) $\overline{Y}$ = 0.122, *N* = 640,433				
−0.00961****	−0.00405*	0.0342****	0.0376	0.0376
(0.00236)	(0.00234)	(0.00540)	(0.0359)	(0.0683)
[8B] Confinement by 22nd birthday (1981–1992) $\overline{Y}$ = 0.045, *N* = 640,433				
−0.00965****	−0.00512****	0.0222****	0.0470**	0.0584
(0.00150)	(0.00152)	(0.00366)	(0.0221)	(0.0433)
[8C] Any charge of violent crime by 22nd birthday (1981–1992) $\overline{Y}$ = 0.038, *N* = 640,433				
−0.00166	0.00117	0.0172****	0.0310	0.0135
(0.00135)	(0.00138)	(0.00336)	(0.0208)	(0.0429)

Notes: The short description of each outcome variable is followed by the birth years of the cohort used, the mean values of the outcome variable, and the numbers of observations in the OLS and first-child regressions, respectively. The effective number of observations of the fixed-effects regression is smaller than that of OLS. Standard errors robust to heteroskedasticity and grandmother clusters are in parentheses. For the list of control variables included in each regression, see the note to Table 4. DKK100,000 ≈ EUR13,400 (Nov 12, 2015). * p < 0.1, ** p < 0.05, *** p < 0.01, **** p < 0.001.

9 completion. This is probably due to the fact that we treat exam scores as missing for those who did not take the exam or took the exam after the standard age (see Appendix A1.3 for further details of the construction of exam score variables). As discussed earlier, prematurity increases the risk of permanent disabilities. Children who have a permanent disability are less likely to complete Grade 9 by age 16 and less likely to take exams at the standard age.[12]

Rows [3] to [5C] show the results for labor market outcomes. A pattern similar to test scores holds for income at age 24: in Row [3], the OLS results are significant and large, but the IV estimates are insignificant with large standard errors. The IV estimate in Row [4] indicates that a 10% increase in birthweight increases the propensity for an individual to work or study at age 22 by about 1 percentage point, an effect of similar size as the OLS estimate. The IV sibling fixed-effects estimate is not statistically significant. Rows [5A] to [5C] reveal a large effect of birthweight on disability pension receipt at age 19–21. The IV estimates consistently show larger effects than the OLS estimates; for example, the IV estimates without fixed effects indicate that a 10% increase in birthweight reduces the amount of disability pension transfers during the three years by 2181 Danish Kroner (DKK hereafter) (≈ EUR292). These strong long-term effects of birthweight on disability pension receipt are probably due to permanent damage from the perinatal period, such as cerebral palsy and mental retardation. The catch-up effect discussed above does not benefit individuals affected by these permanent disabilities, who typically depend heavily on welfare assistance throughout their entire lives. Note that the results in this table are based on persons born between 1981 and around 1991. As the estimated birthweight effect on infant health is smaller in recent years, the effect of birthweight on social assistance may well be smaller for recent cohorts.

Teen pregnancy caused by risky sexual behavior often contributes to early life poverty and loss of future opportunities. In Row [6A], the OLS estimate shows a negative and highly significant association between birthweight and teen motherhood, but this association tends to become opposite when we apply fixed effects and/or the instrument. However, the IV estimate is not statistically significant, and the IV sibling fixed-effects estimate is only marginally significant at the 10% level. The fixed-effects and IV estimates for males (Row [6B]) are positive, indicating a positive birthweight effect on teen fatherhood, although the IV sibling fixed-effects estimate is insignificant.

The OLS estimates in Rows [7A] and [7B] show a highly significant positive correlation between the birthweight of those born from 1981 to 1992 and the birthweight of their children. Significant positive coefficients are also found in the fixed-effects regressions in Row [7A]. These are consistent with past sibling and twin fixed-effects studies (Black et al., 2007; Currie and Moretti, 2007; and Royer, 2009). However, our IV estimates reveal no robust evidence for any positive effect, although our results are not definitive because of two limitations. First, the outcome we use here is conditional on the child becoming a parent by the 22nd birthday, which is considerably earlier than the national average, so the selection may cause bias. Second, because of the rarity of parents under age 22 and conditioning on sex, the effective number of observations is quite small for the IV estimation, leading to large standard errors, especially when sibling fixed effects are applied.

Rows [8A] – [8C] examine the effect of birthweight on criminal offense before the age of 22. The OLS estimates in Rows [8A] and [8B] show a significant deterrent effect of birthweight on criminal offense, consistent with the finding of Tibbetts and Piquero (1999), but the fixed-effects and IV estimates indicate a *positive* effect of birthweight on crime. The fixed-effects estimates are all positive and significant. The IV estimates are positive but not statistically significant except for one. All in all, these estimates indicate that additional birthweight is unlikely to reduce criminal inclination.

Our IV estimates have large standard errors compared to those of the OLS estimates for the same outcome because placenta previa occurs in only a tiny fraction of the population. For most non-health outcomes, the IV estimates are not statistically significant, and for some of these outcomes, the IV estimates are not statistically different from the OLS estimates. However, it is important to note that the insignificance of the effects of birthweight on non-health outcomes is not due to a small number of observations for these outcomes. The highly significant estimates for health outcomes and disability pension are based on approximately the same number of observations as in the analysis of exam scores and criminal offense, but they are clearly statistically significant.

Returns to birthweight: military conscription variables

As reported in detail in Appendix A3, the outcomes constructed from the military conscription register also offer a contrast between the OLS, fixed-effects OLS, and IV estimates. For attendance at a conscription examination and qualification for military service, OLS and fixed-effects OLS indicate a significant causal effect of birthweight, whereas the results of IV estimates are mixed and often insignificant. Based on the IV point estimates, a 10% increase in birthweight raises the probability that the person will qualify for military service by 0.7–3.2 percentage points. This effect may be partially explained by the effect of birthweight on permanent disability, but not fully in view of the low prevalence rate of permanent disability, suggesting a lasting effect of birthweight on long-term general health (e.g., asthma) and physical strength. For IQ, however, while the OLS and fixed-effects OLS exhibit positive coefficients that are statistically significant at the 0.1% level, the IV estimates are not statistically significant. Similarly, height, weight, and BMI exhibit significant OLS estimates, but the estimates for height and weight become much smaller and insignificant when we use the instrument, and the BMI estimates become insignificant as well. The use of fixed effects makes a substantial difference for the OLS estimates probably because mother-specific factors, such as maternal body

[12] Another possible reason comes from the fact that children who start school later tend to have better exam scores because they are older (Black et al., 2011).

Table 7
Low Birthweight Effect by Threshold.

Outcome variables	OLS			IV		
	BW < 3500 g	BW < 2500 g	BW < 1500 g	BW < 3500 g	BW < 2500 g	BW < 1500 g
Infant mortality: cohort 1	0.00675****	0.0605****	0.261****	0.107****	0.124****	0.462****
	(0.000171)	(0.00130)	(0.00606)	(0.0126)	(0.0140)	(0.0498)
Infant mortality: cohort 2	0.00426****	0.0454****	0.199****	0.0212****	0.0380****	0.208****
	(0.000116)	(0.00109)	(0.00501)	(0.00511)	(0.00900)	(0.0465)
APGAR score: cohort 1	−0.0477****	−0.512****	−1.674****	−1.227****	−1.447****	−5.817****
	(0.00133)	(0.00812)	(0.0363)	(0.0908)	(0.100)	(0.508)
APGAR score: cohort 2	−0.0288****	−0.432****	−1.498****	−0.574****	−1.038****	−5.952****
	(0.00128)	(0.00824)	(0.0359)	(0.0531)	(0.0961)	(0.723)
Neurodevelopment disability by age 2	0.00185****	0.0151****	0.0470****	0.0167****	0.0253****	0.139****
	(0.0000762)	(0.00507)	(0.00235)	(0.00368)	(0.00553)	(0.0309)
Mental retardation by age 10	0.00273****	0.0142****	0.0283****	0.0114**	0.0155**	0.0797**
	(0.000131)	(0.000632)	(0.00234)	(0.00502)	(0.00680)	(0.0356)
Days in hospital before 2nd birthday	1.980****	19.14****	60.33****	26.38****	39.88****	219.6****
	(0.0174)	(0.114)	(0.408)	(0.935)	(1.213)	(12.95)
Hospitalization: 2nd–5th birthday	0.0182****	0.0759****	0.178****	0.139****	0.163****	0.814****
	(0.000913)	(0.00260)	(0.00836)	(0.0321)	(0.0373)	(0.196)
Hospitalization: 5–10th birthday	0.0135****	0.0552****	0.129****	0.100***	0.117***	0.585***
	(0.000923)	(0.00256)	(0.00813)	(0.0318)	(0.0370)	(0.191)
Hospitalization: 10–15th birthday	0.00689****	0.0313****	0.0696****	0.0681**	0.0793**	0.396**
	(0.000850)	(0.00232)	(0.00727)	(0.0291)	(0.0339)	(0.173)
Hospitalization: 15–20th birthday	0.00549****	0.0189****	0.0373****	0.0460	0.0535	0.267
	(0.000908)	(0.00240)	(0.00716)	(0.0302)	(0.0351)	(0.176)
Complete Grade 9 by age 16	−0.0353****	−0.0886****	−0.196****	−0.0974****	−0.122****	−0.608****
	(0.000704)	(0.00213)	(0.00675)	(0.0243)	(0.0302)	(0.155)
Exam score: Overall mean	−0.0513****	−0.0778****	−0.125****	0.0839	0.118	0.626
	(0.00186)	(0.00494)	(0.0141)	(0.0571)	(0.0803)	(0.433)
Exam score: Mathematics	−0.0859****	−0.136****	−0.259****	−0.0262	−0.0369	−0.195
	(0.00234)	(0.00629)	(0.0179)	(0.0722)	(0.102)	(0.537)
Income age 24 (excl. transfers)	−3133.8****	−7790.2****	−22215.6****	3276.9	3812.6	19786.6
	(534.3)	(1172.8)	(2966.1)	(13,040.0)	(15,167.9)	(78,881.1)
Work or student age 24	−0.0169****	−0.0537****	−0.125****	−0.0743**	−0.0850**	−0.431**
	(0.000927)	(0.00271)	(0.00895)	(0.0327)	(0.0373)	(0.192)
Disability pension age 19–21	0.00537****	0.0279****	0.0727****	0.0385***	0.0446***	0.220***
	(0.000266)	(0.00121)	(0.00513)	(0.0121)	(0.0139)	(0.706)
Criminal sentence by age 22	0.00552****	−0.0119****	−0.0341****	−0.0283	−0.0328	−0.162
	(0.000811)	(0.00204)	(0.00553)	(0.0271)	(0.0314)	(0.155)
Confinement by age 22	0.00368****	−0.00470****	−0.0149****	−0.0355**	−0.0411**	−0.203**
	(0.000527)	(0.00136)	(0.00352)	(0.0168)	(0.0193)	(0.0969)
Violent crime by age 22	0.00154***	−0.00625****	−0.0173****	−0.0234	−0.0271	−0.134
	(0.000489)	(0.00121)	(0.00300)	(0.0157)	(0.0181)	(0.0902)

Notes: The description of each outcome variable, shown at the beginning of each row, is abbreviated to save space. For detailed descriptions, birth cohorts used, and the number of observations, see Tables 1, 5, and 6. Standard errors robust to heteroskedasticity and grandmother clusters are in parentheses. For the list of control variables included in each regression, see the note to Table 4. * p < 0.1, ** p < 0.05, *** p < 0.01, **** p < 0.001.

size, have considerable influence on these outcome variables.

Specifications with low birthweight dummies

To examine whether birthweight effects are concentrated around the very low end of the birthweight distribution, we also estimate specifications with three low birthweight dummies instead of $\ln BW_i$: (1) birthweight less than 3,500 g (approximately the average birthweight in our sample), (2) birthweight less than 2,500 g (LBW), and (3) birthweight less than 1,500 g (VLBW). The first-stage regressions for these three dummy variables again confirm the power of our IV: the *F* statistics for the relevance of the IV in the three first-stage regressions are 3383.3, 1588.9, and 295.5, respectively.

Table 7 reports the estimated coefficients on the three low birthweight dummies in the OLS and IV regressions for selected outcome variables. Consistent with the log birthweight specification, the IV estimates provide clear evidence that low birthweight due to prematurity negatively affects early-life health outcomes and increases the risk of permanent disabilities. The effect exists even when the threshold of 3,500 g is used. This is in contrast to very small OLS estimates. Variation in birthweight reflects various factors including genetics, and comparison around the average birthweight does not predict those health outcomes very well, but the IV estimates show that birthweight loss due to prematurity has non-negligible consequences even if the resulting birthweight is around the mean. The size of the effect becomes larger as we use a lower birthweight threshold. Using the 2,500 g cutoff instead of the 3500 one increases the magnitudes typically by around 20%, whereas the use of the 1,500 g threshold increases the effect size dramatically, typically by a factor of four to six compared to the case of the 2,500 g threshold, confirming an unproportionally heavy health burden on very low birthweight newborns.

Table 8
Plausible Size of Bias in Placenta Previa IV Regression.

Dependent variable:	Case 1:		Case 2:		Case 3:	
	$\theta_{BW} = \hat{\theta}^{IV}_{BW}$		$\theta_{BW} = \hat{\theta}^{simple\ OLS}_{BW}$		$\theta_{BW} = \hat{\theta}^{multiple\ OLS}_{BW}$	
	$\hat{\theta}^{IV}_{BW}$	Bias in $\tilde{\theta}^{IV}_{BW}$	$\hat{\theta}^{simple\ OLS}_{BW}$	Bias in $\tilde{\theta}^{IV}_{BW}$	$\hat{\theta}^{multiple\ OLS}_{BW}$	Bias in $\tilde{\theta}^{IV}_{BW}$
Infant mortality (1981–2013)	−0.0820	−0.0013	−0.0731	−0.0108	−0.0755	−0.0081
Hospital days before age 2 (1981–2011)	−41.05	−0.274	−20.87	−18.59	−20.33	−18.91
Exam score: overall mean (1986–1997)	−0.125	0.045	0.303	−0.304	0.183	−0.204

Notes: This table shows implied bias which is calculated by $\mathrm{Cov}(pp, e)/\mathrm{Cov}(pp, lnBW)$, where e_i is obtained from the three hypothesized values of true θ_{BW}. The values of $\mathrm{Cov}(pp, lnBW)$ are −0.00081, −0.00081, and −0.00078 for the three different periods used for the three dependent variables, respectively.

These specifications with birthweight dummies show that our results for the birthweight effect on long-term outcomes are robust in the sense that we still find negative effects of low birthweight on the probability of completing grade 9 by age 16, working or being a student at age 24, and confinement by age 22, and positive effects on receiving disability pension at age 19–21, whereas effects on test scores, income at age 24, and the two other crime variables are insignificant. These findings indicate that the contrast we found between the short-term and long-term outcomes is not an artifact of the log birthweight specification.

Further evidence for the reliability of the IV estimation

We have so far provided a number of arguments for why placenta previa serves as a valid instrument. In this section, we provide further support for our IV estimation by showing the robustness of our results even if the exclusion restriction does not hold exactly.

First, we conduct the IV estimation based only on the sample of first-born children. As discussed earlier, this is to address the concern that the experience of placenta previa might cause the mother to take more precautions during subsequent pregnancies or it might even influence the next fertility decision. As reported in Appendix A2, the results are fairly consistent with our full sample analysis and our main results hold.

Next, we provide a back-of-the-envelope calculation of the plausible magnitude of bias. Consider a simple model:

$$y_i = \theta_0 + \theta_{BW} \ln BW_i + e_i \tag{3}$$

where we use pp_i as an IV for $\ln BW_i$ to obtain the IV estimate, $\tilde{\theta}^{IV}_{BW}$. Since $\mathrm{Cov}(pp, \ln BW) \neq 0$, we can show the following (Wooldridge, 2010, p.108):

$$\mathrm{plim}\, \tilde{\theta}^{IV}_{BW} = \theta_{BW} + \mathrm{Cov}(pp, e)/\mathrm{Cov}(pp, \ln BW) \tag{4}$$

where the last term is the expression for bias. The bias term shows that, even if there is an unobserved determinant of placenta previa and thus the numerator is nonzero, the bias reduces as the first-stage explanatory power of pp_i increases. We cannot directly evaluate the bias term because (4) holds only asymptotically, but we can conduct informal approximation of the bias using the sample analogue of this term, in which $\hat{\mathrm{Cov}}(pp, \ln BW)$ is directly obtained from data. We calculate $\hat{\mathrm{Cov}}(pp, e)$ by using $\hat{e}_i$ that is obtained from Eq. (3) as a residual with a hypothesized value of θ_{BW}. Because of the large number of observations, this approximation should provide a reasonable quantification of the size of the bias, unless the hypothesized value of θ_{BW} is far from the true birthweight effect parameter.

Table 8 reports the "implied bias" based on three hypothesized values of θ_{BW}: (1) the IV estimate, $\hat{\theta}^{IV}_{BW}$; (2) the estimate from the OLS with no control, $\hat{\theta}^{simple\ OLS}_{BW}$; and (3) the estimate from the OLS with controls, $\hat{\theta}^{multiple\ OLS}_{BW}$. The results in Table 8 indicate the robustness of our main findings. The size of the implied bias for infant mortality (shown in the first rows) is small for all three hypothesized values (Cases 1–3) relative to $\hat{\theta}^{IV}_{BW}$ (which is shown in the first column under Case 1). The bias is small both because the numerator is small and the denominator is large. The size of the implied bias for hospital days is around 50% of the $\hat{\theta}^{IV}_{BW}$ under Cases 2 and 3. The last row in Table 8 shows that, for the test score, the bias in this simple IV estimator can be as large as 0.304 because $\hat{\mathrm{Cov}}(pp, e)$ is larger than that of health outcomes. However, even with this size of bias, the relationship between the OLS and IV estimates found in our analysis (i.e., smaller IV estimates than OLS estimates) is unchanged. Note also that this simulation is conservative in the sense that we do not control for the risk factors for placenta previa and other observable medical and socio-demographic factors. When those controls are included, we expect much less potential violation of the exclusion restriction, and thus even smaller bias of our IV estimates than shown in Table 8.

Another way to infer the size of potential bias due to unknown risk factors for placenta previa is to examine how our results are affected if we omit one of the known major risk factors from our IV regression. As shown in Appendix A4, the results of this exercise indicate limited potential bias. In this robustness test, we first confirm that those risk factors reported in the medical literature are statistically significant predictors of placenta previa (the first row in Table A4-1) and that they are statistically significant in the regressions of other outcomes (the rest of Table A4-1). Then, we conduct our IV regressions with and without those risk factors (past cesarean sections, maternal smoking, and maternal body size, one at a time) and compare the estimated coefficients on $\ln BW_i$ (Tables A4-2 to A4-4). We find no significant differences between IV estimates with and without control for these risk factors. In most cases, differences in point estimates are less than 1%, and in all cases less than 10%. The significance and signs are unaffected. Our conclusions, therefore, are unlikely to be an artificial product of an unknown cause of placenta previa. The critical assumption behind this exercise is that the major risk

factors for placenta previa that have been identified in the medical literature are the most important factors (in terms of the predictive power of placenta previa and the size of direct effect on outcomes), and unknown risk factors, if any, are of the same importance or less.

There is also a concern that our results predominantly capture the effect of the cesarean section rather than maturity at birth because serious cases of placenta previa require a cesarean delivery. Although the causal effect of cesarean birth on the child's general outcomes remains controversial, as discussed in Section Placenta Previa, we investigate the potential bias due to cesarean delivery by conducting a sub-sample analysis in which we exclude first-time mothers and examine how the IV estimate varies by past cesarean delivery status. The motivation here is the fact that past cesarean delivery makes cesarean section the default choice for subsequent pregnancy to avoid uterine rupture and other complications; the use of a subpopulation with past cesarean section should consequently yield different results if there is a substantial effect of cesarean section. As reported and discussed in Appendix A5, our main conclusions are unchanged.

Lastly, there is a possibility that the experience of placenta previa itself may change the behavior of the mother by making the mother more careful and attentive toward the child.[13] We argue that this possibility does not change our main results for the following reasons. First, it is presumably the case that very short-run outcomes such as 5 min APGAR score, receiving CPAP in neonatal ward, and neurodevelopmental disability are predominantly driven by medical factors rather than maternal behavior, and then the mother's behavioral change is unlikely to be relevant for most of our very short-run birthweight effects.

Second, the mother's behavioral response does not change our conclusion that additional birthweight has a significant protective effect in the short run because such behavioral response implies downward bias in the magnitude of the protective birthweight effect. Third, our long-run results are biased only if the placenta previa status itself has long-term influences on parental behavior: in other words, the long-term parental investment differs between the same 1.5 kg babies with and without placenta previa. This is highly unlikely because as discussed above, placenta previa is a temporary condition that has no long-term medical consequences for the child and mother except through the channel of birthweight.

Comparison of twins and singletons

We lastly report twin fixed-effects estimates to highlight the differences between the twin approach and our IV approach. We use the population data of twins from the same data source. For clarity of interpretation, we include only twins who are live-born from pregnancies with two fetuses.[14]

Table 9 shows the comparison for selected outcome variables. In each row, the first two columns report the OLS and fixed-effects estimates based on twins. The next four columns show the singleton estimates that have already been presented. In addition to five selected outcomes, the results of infant mortality from Black et al. (2007) are presented in the second row, [1-BDS], for comparison.

Table 9 reveals clear differences in the results for twins and singletons. Rows [1] – [3] report the estimated effects of birthweight on infant health outcomes, and Row [4] reports the effect on receipt of disability pension. Findings from these rows are summarized as follows. First, when we compare the twin and singleton OLS estimators, the former shows considerably larger estimates. This is because twin infants tend to have significantly worse health outcomes than singleton infants. Second, applying fixed effects changes the OLS estimates of twins and singletons in opposite directions. The twin fixed-effects estimates are substantially smaller than the twin OLS estimates, suggesting that birthweight not be as important as it appears from OLS estimates. In contrast, applying fixed effects to singleton infants yields birthweight effects substantially larger than those of OLS, which suggests an opposite conclusion. This contrast is also visible in the results from Black et al. (2007) in Row [1-BDS].

The singleton IV estimator similarly leads to larger birthweight effects than the singleton OLS estimates, hence our IV estimates for these outcomes related to infant health suggest a larger role of birthweight than the twin fixed-effects estimator, although the IV estimates may or may not be larger than the singleton fixed-effects estimates, depending on each outcome.

This observed pattern, however, turns out to be quite different for longer-term outcomes. As shown in Row [5], the positive and significant estimate of the birthweight effect on the national exam score in the 9th grade in the twin OLS becomes larger if fixed-effects are applied, whereas the use of fixed effects for singletons makes the estimate smaller than singleton OLS. Our IV point estimates are negative but not statistically different from zero because of large standard errors. Nevertheless, the large positive twin fixed-effects estimate is clearly outside the 95% confidence interval of the IV estimates.

These patterns of twin estimates seem to be robust across the twin literature. Twin fixed-effects estimates on infant mortality or infant health which are significantly smaller than their OLS counterparts are found in Almond et al. (2005); Conley et al. (2006); Oreopoulos et al. (2008), and Royer (2009). In contrast, twin fixed-effects estimates on educational achievements or other socioeconomic outcomes that are similar to or larger than their OLS counterparts are found in Behrman and Rosenzweig (2004); Black et al. (2007); Oreopoulos et al. (2008); Lin and Liu (2009); Royer (2009); Torche and Echevarría (2011), and Figlio et al. (2014).

[13] This type of compensatory parental investment is found in past studies (Hsin, 2012; Bharadwaj et al., 2018b).

[14] Our data do not allow us to separate monozygotic and dizygotic twins. To address the issue of zygosity, we follow Black et al. (2007) and estimate the same regression models for same-sex and different-sex twins separately. The results turn out to be very similar in both groups.

Table 9
Coefficients on ln*BW* – Difference Between Twin and Singleton Estimators.

Twin, OLS	Twin, Fixed Effects	Singleton, OLS	Singleton, Sibling FE	Singleton, IV	Singleton, Sibling FE IV
[1] Infant mortality (from birth to 365 days) (1981–2013)					
−0.187****	−0.0473****	−0.0755****	−0.132****	−0.0820****	−0.0721****
(0.0063)	(0.0070)	(0.0012)	(0.0021)	(0.0081)	(0.0133)
$\overline{Y}$ = 0.0185, N = 66,108		$\overline{Y}$ = 0.0045, N = 1,944,729			
[1-BDS] 1 year mortality, results from Black, Devereux, and Salvanes (2007), Tables I and III (1967–1997)					
−0.2796****	−0.0411****	−0.1235****	−0.1867****		
(0.00912)	(0.00764)	(0.00,171)	(0.00069)		
$\overline{Y}$ = 0.0311, N = 33,366		$\overline{Y}$ = 0.0062, N = 1,253,546			
[2] Neurodevelopmental disability diagnosis by 2nd birthday (1981–2011)					
−0.0335****	−0.0073*	−0.0166****	−0.0220****	−0.0261****	−0.0202***
(0.0026)	(0.0044)	(0.0005)	(0.0009)	(0.0057)	(0.0076)
$\overline{Y}$ = 0.0061, N = 60,013		$\overline{Y}$ = 0.0024, N = 1,820,443			
[3] Number of days in hospital before 2nd birthday (1981–2011)					
−54.54****	−5.260****	−20.33****	−25.26****	−41.05****	−37.16****
(0.576)	(0.620)	(0.128)	(0.205)	(1.067)	(1.467)
$\overline{Y}$ = 5.54, N = 60,013		$\overline{Y}$ = 5.54, N = 1,820,443			
[4] Receipt of disability pension over 3 years at age 19–21 (1981–1992)					
−0.0420****	−0.0238*	−0.0349****	−0.0501****	−0.0511***	−0.0871***
(0.00763)	(0.0124)	(0.00127)	(0.00271)	(0.0159)	(0.0298)
$\overline{Y}$ = 0.015, N = 14,027		$\overline{Y}$ = 0.011, N = 641,978			
[5] National exam score, Grade 9 (≈ age 16): overall mean (1986–1997)					
0.111****	0.232****	0.183****	0.129****	−0.125	−0.122
(0.0262)	(0.0452)	(0.0055)	(0.0104)	(0.0851)	(0.130)
$\overline{Y}$ = 0.007, N = 17,316		$\overline{Y}$ = 0.016, N = 627,364			

Notes: In each row, the short description of each outcome variable is followed by the birth years of the cohort used. Below coefficients estimates and standard errors, the mean values of the outcome variable and the numbers of observations are shown for twins and singletons, respectively. The effective number of observations in the fixed-effects regressions is smaller than reported in this table. Standard errors robust to heteroskedasticity and grandmother clusters are in parentheses. For the list of control variables included in each regression, see the note to Table 4. *p < 0.1, **p < 0.05, ***p < 0.01, ****p < 0.001.

Conclusion

By applying the diagnosis of placenta previa as an instrumental variable to administrative population data for two million Danish newborns, this study advances our understanding of the causal effect of birthweight related to variation in gestation length with the following results. First, birthweight does have significant positive causal effects on child health. Second, the effect of birthweight on general health diminishes as a child grows older (catch-up effect). Third, a large part of the highly significant correlations between birthweight and non-health outcomes appears to be non-causal. Fourth, the effect of birthweight on infant health in Denmark has diminished significantly over the last three decades.

These results strengthen the foundation for prenatal interventions to reduce premature birth and postnatal interventions to avoid *permanent health capital loss*. These interventions enable us to save more infants and at the same time reduce neurodevelopmental impairment, mental retardation, and hospital stays, which will lead in turn to a considerable saving of medical and societal costs. Such interventions will have even higher returns in countries with limited access to modern perinatal care.

Our results also have implications for the literature on child health and human capital development. This literature emphasizes the persistent importance of a "good start" in life by claiming "dynamic complementarities" between prenatal health shocks and subsequent investments (Heckman, 2007; Currie et al., 2010; Almond and Currie, 2011b). Our results underscore the importance of a "good start," but in a quite different way. Immaturity may affect the life course of surviving infants permanently by causing morbidities that have their origins in the perinatal period. Other causal effects of birthweight also exist, but they are likely to fade out over time, as implied in standard health capital models. Children who survive their infancy and avoid permanent morbidities can catch up, and in this sense, immaturity itself is not a critical initial condition.

More generally, our IV approach highlights the fact that birthweight is a proxy for multi-dimensional neonatal health endowments, and the interpretation of the "birthweight effect" depends on what exogenous variation the proxy reflects. The qualitative contrast between our IV results and the results typically found in the twin fixed-effects studies warns policies that merely target birthweight.

We did not explore potential effect heterogeneity, although it may offer insights into the mechanism behind the estimated birthweight effects. Splitting the sample across various dimensions is a standard approach to studying effect heterogeneity, but the very low incidence rate of placenta previa prevents us from precise estimation in such subgroup analysis, and hence we focused this paper on reconciling findings of various outcomes and findings from different econometric approaches, leaving the investigation of effect heterogeneity for future research.

Finally, reconciling the different findings from our IV approach and the twin studies warrants further investigation. Future research calls for a better understanding of the causal mechanism behind the short- and long-term effects. For the short-term effect, the pathology literature of pre-

mature birth offers the evidence of direct causal pathways. For example, underdevelopment of the lung of premature infants can cause lung injury and consequent brain hemorrhage (Institute of Medicine, 2007), which may result in neurodevelopmental disabilities. On the other hand, we have limited knowledge of the causal mechanism behind the long-term effect. Figlio et al. (2014) study the cognitive development of children in Florida and conclude that the effects of birthweight are set very early in life and are similar across family backgrounds. Cook and Fletcher (2015) report that genes related to neuroplasticity influence the size of the birthweight effect. Nevertheless, the findings from twin studies that a shock at birth has a limited short-term effect but persistent long-term effects are still puzzling because in standard health capital models, in which health capital depreciates over time, the effects of events further in the past should fade out (Almond and Currie, 2011a). The literature often refers to David J. Barker's "fetal origins" hypothesis, which claims that the intrauterine environment, particularly in respect of nutrition, "programs" the fetus to have particular metabolic characteristics which can lead to future adult diseases even if there is no immediate impact (Barker, 1995), but the empirical basis for this hypothesis remains controversial, particularly for immature infants (Institute of Medicine, 2007, p. 387), but also in laboratory mammal studies (Lagisz et al., 2014).

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Appendix A. Supplementary material

Supplementary material related to this article can be found, in the online version, at https://doi.org/10.1016/j.jhealeco.2019.102269.

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