Demography (2012) 49:525–552
DOI 10.1007/s13524-012-0092-1

Prenatal Health, Educational Attainment, and Intergenerational Inequality: The Northern Finland Birth Cohort 1966 Study

Juho Härkönen · Hande Kaymakçalan · Pirjo Mäki · Anja Taanila

Published online: 28 February 2012

Abstract In this article, we study the effects of prenatal health on educational attainment and on the reproduction of family background inequalities in education. Using Finnish birth cohort data, we analyze several maternal and fetal health variables, many of which have not been featured in the literature on long-term socioeconomic effects of health despite the effects of these variables on birth and short-term health outcomes. We find strong negative effects of mother's prenatal smoking on educational attainment, which are stronger if the mother smoked heavily but are not significant if she quit during the first trimester. Anemia during pregnancy is also associated with lower levels of attained education. Other indicators of prenatal health (pre-pregnancy obesity, mother's antenatal depressed mood, hypertension and preeclampsia, early prenatal care visits, premature birth, and small size for gestational age) do not predict educational attainment. Our measures explain little of the educational inequalities by parents' class or education. However, smoking explains 12%—and all health variables together, 19%—of the lower

J. Härkönen (✉)
Demography Unit, Department of Sociology, Stockholm University, S-106 91 Stockholm, Sweden
e-mail: juho.harkonen@sociology.su.se

J. Härkönen
Swedish Institute for Social Research, Stockholm University, S-106 91 Stockholm, Sweden

H. Kaymakçalan
Department of Science, Bahçeşehir University, Istanbul, Turkey

P. Mäki
Department of Psychiatry, Institute of Clinical Medicine, University of Oulu, Oulu, Finland

P. Mäki
Oulu University Hospital, Oulu, Finland

A. Taanila
Institute of Health Sciences, University of Oulu, Oulu, Finland

A. Taanila
Unit of General Practice, Oulu University Hospital, Oulu, Finland

educational attainment of children born to unmarried mothers. Our findings point to the usefulness of proximate health measures in addition to general ones. They also point to the potentially important role played by early health in intergenerational processes.

Keywords Prenatal health · Education · Social background · Intergenerational inequality

Introduction

Recent social scientific research has shown increasing interest in the effects of early-life conditions on later-life outcomes (e.g., Heckman 2006). A related literature has demonstrated how poor health in childhood predicts weaker socioeconomic attainment in adulthood (e.g., Behrman and Rosenzweig 2004; Black et al. 2007; Case et al. 2005; Case and Paxson 2006; Conley and Bennett 2000; Conley et al. 2003; Currie 2009; Haas 2006; Jackson 2007, 2009; Palloni 2006; Palloni et al. 2008; Currie 2009; Smith 2009; White and Palloni 2009). Most of these studies have relied on measures of general health in childhood. Although they have been successful in adding to our understanding of the role of overall early health in the socioeconomic attainment process, more information is needed on which specific conditions are important in shaping future life chances.

The first objective of this study is to analyze the effects of a selection of prenatal health indicators on educational attainment. Instead of viewing childhood as a single life stage ranging from the fetus to adolescence, we focus on the fetal environment—"the first environment"—which can be of particular importance in influencing later life (Currie 2009:116). Several previous studies have found that birth weight affects health, schooling, and labor market outcomes (e.g., Barker 1991, 2001; Behrman and Rosenzweig 2004; Black et al. 2007; Conley and Bennett 2000, 2001; Conley et al. 2003; Haas 2006; Jackson 2009). However, birth weight is determined by more proximate conditions, at least some of which may independently predict longer-term outcomes.

The measures we use (pre-pregnancy obesity, anemia, mother's antenatal depressed mood, smoking, hypertension and preeclampsia, and early visit to a prenatal clinic), in addition to preterm birth and birth weight for gestational age, are all known correlates of birth outcomes and other short-term outcomes. But do they predict educational attainment in adulthood?

Our second interest lies in examining whether prenatal health contributes to the persistent associations between family background and children's educational attainment. The role of childhood health in the intergenerational transmission of advantage and disadvantage has been increasingly discussed, but only a few studies have empirically assessed these issues.

To analyze these questions, we use data for 8,625 individuals from the Northern Finland Birth Cohort 1966 Study (hereafter referred to as NFBC 1966), an ongoing general-population study. Our data include information on the health of the mother and the child and on the family's sociodemographic characteristics, gathered during pregnancy and at birth, that are linked to educational attainment of the child at age 31. We analyze these data using ordered logit regression models. Before we examine the

data and the results in more detail, we discuss the potential role of prenatal health in socioeconomic attainment and the intergenerational transmission of socioeconomic status. The last section summarizes our findings and discusses their implications.

Prenatal Health and Educational Attainment

Prenatal Health and Educational Outcomes: Pathways

Prenatal health can affect educational outcomes through different avenues, of which the effects on cognitive skills are considered particularly important (Currie 2009). Other effects operate through noncognitive features, which can, among other things, affect children's attention and learning capabilities and shape their interests and potential to proceed to further levels of education (Palloni 2006; Palloni and Milesi 2006). Furthermore, health problems in childhood and adolescence that are caused by fetal conditions can lead students to miss school and thus affect their success in education (Currie et al. 2010).

The underlying causal pathways behind these effects are inherently biological, although they can be shaped by interactions with the genome and the environment. Probably the best-known theory of biological pathways is Barker's hypothesis, which links poor fetal growth to hypertension, cardiovascular disease, stroke, and diabetes through permanent changes in the cardiovascular system that result from the fetus's adaptation to poor nutritional conditions (e.g., Barker 1991, 2001). Similar "biological programming" has been linked to other health, psychobehavioral, and socioeconomic outcomes. An underlying argument is that prenatal conditions can have long-term effects by shaping the development of the fetus at critical periods when the system is most sensitive to environmental inputs (e.g., Gluckman and Hanson 2005). Different stages of pregnancy can be sensitive to different prenatal insults. For example, radiation exposure has the strongest effects between weeks 8 and 25 of pregnancy (Almond et al. 2009; Nowakowski and Hayes 2008), whereas the effects of prenatal exposure to nicotine are the strongest during the last two trimesters (Shea and Steiner 2008; Slotkin 1998).

Following Barker, many studies have stressed the role of fetal nutrition and the physiological responses to it. Other prenatal conditions work through different biological pathways. For example, preeclampsia and prenatal smoking limit blood (and oxygen) flow to the fetus (Ehrenstein et al. 2009; Shea and Steiner 2008; Slotkin 1998), and nicotine can additionally shape brain development by targeting specific neurotransmitter receptors (Slotkin 1998). Maternal stress during pregnancy, on the other hand, can lead to overactivity of the hypothalamic-pituitary-adrenal (HPA) axis and to increased secretions of glucocorticoids, which can alter the function of the HPA axis in the fetus and disturb its brain development (Maccari et al. 2003; Mäki et al. 2010).

The term "programming" suggests rather deterministic avenues from the womb to adult life, but the effects of intrauterine inputs vary according to genetic and social factors in ways that can affect longer-term consequences (Lundborg and Stenberg 2010). A growing body of research analyzes genetic sensitivity to environmental inputs (e.g., Caspi et al. 2007; Kahn et al. 2003; Rutter and Silberg 2002), and another focuses on the epigenetic determination of gene expression (Szyf 2009). Furthermore,

prenatal health effects can be shaped by parents and other significant persons—for example, by providing more (less) attention to children with weaker starting points (Conley et al. 2003; Datar et al. 2010; Stevenson and Fredman 1990). Despite increasing interest, the intrauterine predictors of socioeconomic attainment—and the pathways through which they operate—remain incompletely understood. Nevertheless, previous research has identified some predictors.

Indicators of Prenatal Health and Socioeconomic Outcomes

Measurement of fetal health is difficult, and researchers commonly use measures of birth outcomes and maternal health and behaviors during (and before) pregnancy. Low birth weight (LBW, ≤2,500 g) is a leading indicator. Several studies have found consistent and often substantial effects of LBW on socioeconomic outcomes that generally persist (or even become stronger) after the inclusion of sibling or twin fixed effects (e.g., Behrman and Rosenzweig 2004; Black et al. 2007; Conley and Bennett 2000; Oreopoulos et al. 2008). However, LBW carries some restrictions.

First, birth weight is a function of gestational age and the fetal growth rate, which have different etiologies and different consequences (Kramer et al. 2000). Of these, short gestational age (that is, being born preterm) is the leading, yet less understood, cause of LBW and perinatal morbidity and mortality in developed countries, and may have different long-term consequences than fetal growth restriction (Behrman and Butler 2007; Kiely et al. 1994; Kramer et al. 2000; Paneth 1995). Some studies have looked specifically at the (negative) long-term socioeconomic effects of being born preterm (e.g., Moster et al. 2008); some designs, such as twin studies, have identified the effects of poor fetal growth (e.g., Behrman and Rosenzweig 2004; Black et al. 2007); and others have separately analyzed the effects of both factors (Oreopoulos et al. 2008). Oreopoulos and associates (2008), for example, found that fetal growth had a stronger effect than prematurity on socioeconomic outcomes in early adulthood.[1]

Secondly, LBW is a marker of other prenatal factors that can affect both birth weight (through gestational age, fetal growth, or both) and long-term socioeconomic outcomes. To properly understand the prenatal predictors of later outcomes and to design successful interventions, it is important to identify the underlying behavioral and medical factors responsible (Almond et al. 2005; Oreopoulos et al. 2008). Tobacco use during pregnancy is a major modifiable predictor of both preterm birth and poor fetal growth (Behrman and Butler 2007; Ernst et al. 2001; Kiely et al. 1994; Paneth 1995). Neurological research suggests that fetal exposure to toxins found in tobacco harm neural development in a dose–response fashion, with the last two trimesters being particularly sensitive (Shea and Steiner 2008; Slotkin 1998). Importantly, these negative effects are found independently of birth weight because nicotine and other toxins can affect neurological development directly, as we discussed earlier. A number of studies have reported negative relationships between prenatal smoking and cognitive skills (Ernst et al. 2001) and educational (Case et al. 2005) and labor market success (Jackson 2007), although others have argued that these effects can be accounted for by family background (Batty et al. 2006; Ernst et al. 2001; Lambe et al. 2006; MacArthur et al. 2001).

[1] Interestingly, their findings also suggested that cognitive performance is not the main pathway.

Previous studies have also analyzed the long-term effects of other prenatal insults. From the point of view of estimating causal effects, the most impressive of these have demonstrated long-term socioeconomic implications of such exogenous shocks as prenatal exposure to the 1918–1919 Spanish flu (Almond 2006) or the fallout from the Chernobyl nuclear disaster (Almond et al. 2009). However, given the rarity of such events, these results have limited generalizability, especially for understanding the effects of more common prenatal conditions and behaviors.

The maternal and fetal factors that we analyze have all been linked to birth and short-term child development outcomes, but less is known about their longer-term effects. Negative effects on pregnancy, birth outcomes, and/or short-term child outcomes have been reported for smoking, as discussed earlier; maternal (pre-pregnancy) obesity (Guelinckx et al. 2008); anemia (Allen 2000); hypertensive disorders and their more severe form, preeclampsia (Witlin and Sibai 1997); and mother's antenatal depression, anxiety, and stress (Mulder et al. 2002; Orr and Miller 1995; Torche 2011); whereas early and continuous prenatal care has positive effects on these outcomes (Kiely et al. 1994; Nagahawatte and Goldenberg 2008).

Some research suggests that these conditions and behaviors can have longer-lasting impacts. For example, Heikura and associates (2008) found that maternal obesity was associated with a clearly elevated risk of intellectual disability in children; and Mäki and colleagues reported associations among maternal antenatal depression, criminality (Mäki et al. 2003), and schizophrenia (Mäki et al. 2010), the latter effect being limited only to genetically vulnerable children. There is also observational and some experimental evidence of adverse long-term health, cognitive, and socioemotional outcomes related to anemia in infancy (Lozoff et al. 2006); because children born to anemic mothers have reduced hemoglobin levels, they are themselves at risk of these adverse developmental outcomes. The immediate and short-term effects (limited blood flow to the fetus, and a number of maternal and infant health problems) of pregnancy-associated hypertensive disorders and preeclampsia suggest potentially important effects also in the longer run, as has been reported in the few studies that exist (Ehrenstein et al. 2009).

Summing up, despite increasing interest in the long-term socioeconomic effects of prenatal health, a great deal remains to be learned. Low birth weight can act as a proxy for some prenatal factors that can have long-lasting implications. Given that different prenatal conditions and behaviors can have different pathways through which they operate, it can be difficult to single out the most important ones based on theory alone. Therefore, empirical research is needed to identify which, if any, of the common prenatal factors have long-term implications.

Disparities in Prenatal Health and the Intergenerational Reproduction of Inequality

Social Disparities in Prenatal Health

Socioeconomic disparities in health are already evident during pregnancy and at birth, and can be found from different countries with varying health care systems and levels of social inequality (Kramer et al. 2000). Disparities exist according to

socioeconomic status (SES; measured as education, income, or occupation) but also according to other features, such as family structure (Castro-Martin 2010; Raatikainen et al. 2005).

A clear link exists between different indicators of parents' socioeconomic status and the components of LBW, prematurity, and poor fetal growth. These relationships are not necessarily linear, and the largest increases in LBW and its components are found at the lowest end of the socioeconomic distribution (Kramer et al. 2000; Paneth 1995). Birth-weight disparities have also been reported according to marital status (Castro-Martin 2010; Moser et al. 2003; Pattenden et al. 1999; Raatikainen et al. 2005).

Smoking during pregnancy varies notably across social groups (e.g., Cnattingius 2004); and in Finland, less than 5% of women of higher occupational status smoke during pregnancy, compared with 20%–25% of housewives and working-class women (Jaakkola et al. 2001). Differences are also found for married, cohabiting, and single women, with the first group having the lowest rates of smoking while pregnant (Jaakkola et al. 2001; Kiernan and Pickett 2006).

Similar disparities exist according to other preconception and prenatal health factors. Obesity is increasingly related to low SES (McLaren 2007), and lower-status women are more likely to experience diabetes, pregnancy-associated hypertension, anemia (Korenbrot and Moss 2000), depression, stress, and anxiety during pregnancy (Nagahawatte and Goldenberg 2008). Differences in some of these factors, such as depression, have also been shown for married, cohabiting, and single women (Kiernan and Pickett 2006).

Differential access to or usage of prenatal health care account for only a limited share of disparities in prenatal health and birth outcomes (Case and Paxson 2002; Kramer et al. 2000; Nagahawatte and Goldenberg 2008). In general, behaviors (such as smoking) as well as health and psychosocial conditions (e.g., infections and psychosocial stress) before and after pregnancy have more explanatory power. However, as with health disparities in general, the importance of these factors depends on the outcome. For example, Kramer et al. (2000) (also Korenbrot and Moss 2000; and Paneth 1995) pointed to smoking and anthropometric measures of the mother as important candidates for explaining the links between SES and poor fetal growth and to smoking, infections, and psychosocial stressors for understanding relationships between SES and preterm birth.

In many cases, social group differences in the reception and use of information about behaviors that affect prenatal health seem important. Pickett et al. (2005) found increasing social inequalities in the sudden infant death syndrome (SIDS) after the introduction of a national U.S. campaign, as a result of faster reductions in SIDS among advantaged groups. Similar findings have been reported for prenatal smoking (Cnattingius 2004).

Early Health and the Intergenerational Transmission of Socioeconomic Status

If parents' SES is correlated with early health, which then affects future socioeconomic attainment of the child, can early health explain any of the persistent associations between parents' and their children's SES? This question has recently been raised by several scholars (Case et al. 2005; Case and Paxson 2006; Currie

2009; Currie and Moretti 2007; Palloni 2006; Palloni and Milesi 2006), but has been directly assessed in only few studies.

These analyses have focused on measures of general health or chronic conditions during different stages of childhood and adolescence and, in the case of prenatal health, on birth weight. The general conclusion is that early health explains a nontrivial, yet somewhat limited, share of the associations between parents' and children's SES, although these estimates are likely to be lower bounds. Papers by Palloni and associates (Palloni 2006; Palloni et al. 2008) estimated that in Britain, roughly one-tenth of the share of intergenerational class inequality can be accounted for by child health. Case and Paxson (2006:164) and Currie (2009:114–115) showed calculations suggesting that prenatal and childhood health (e.g., LBW, prenatal smoking, and ADHD) account for only a limited share of the socioeconomic background differences in schooling and earnings.

To play a strong role in the intergenerational transmission of SES, early health should have a strong effect on socioeconomic attainment, and there should be clear social gradients in, or differences in the effects of, early health by social groups (Currie 2009; Kramer et al. 2000). Given the limited measures available in most cases, it is likely that the overall effects of early health conditions are underestimated. Furthermore, the shares of parent–child associations explained by early health may depend on the family background measure used. For example, some key prenatal health indicators can be more strongly associated with education than income. Overall, there is space for better understanding of the role of early health in the intergenerational transmission of socioeconomic position.

Data

The Northern Finland Birth Cohort 1966 Study

Our data come from the Northern Finland Birth Cohort 1966 Study (or NFBC 1966), a prospective study of all children born in the two northernmost provinces of Finland in 1966 (Rantakallio 1969), who were followed up at ages 1, 14, and 31 years (e.g., Ek et al. 2005; Institute of Health Sciences n.d.). Researchers at the University of Oulu recruited mothers at their first visits to a prenatal clinic, or at the maternity clinic. The original sample consisted of 12,231 births (12,058 born alive; 11,870 alive at age 1), which covers 96.3% of all births in the area (Rantakallio 1988). We excluded 163 twin pairs due to the specific developmental trajectories twins often follow.

Of the remaining singletons, we obtained access to health and sociodemographic variables (measured during pregnancy and at birth) and educational attainment at age 31 for 8,625 respondents. Using descriptive statistics from the NFBC 1966 website, we compared our sample with the original sample of all births, using variables that had no or very few missing values. The sample characteristics were remarkably similar on such key variables as parental class and education, mother's marital status, and mother's age at birth. As to birth weight and gestational age, our sample includes individuals that were born slightly heavier (means = 3,490 g and 3,443 g, respectively) and more mature (means =

40.0 and 39.9 weeks, respectively) compared with all births (including twins, stillborns, and those not surviving to age 31).

We use information on health behaviors and conditions during pregnancy as well as data on birth outcomes and on educational attainment. The personnel of local antenatal clinics collected data on the mother's demographic characteristics, behaviors, and health during the 24th to 28th gestational week. Data on birth outcomes come from the registries of the delivery wards. We refer the reader to Rantakallio (1969) for detailed descriptions of these variables. The data on educational attainment at age 31 was linked using personal identifiers from the National Education Registry, which is maintained by Statistics Finland (cf. Isohanni et al. 2001).

A large share of the cases in our sample (47.5%; 4,095 cases) had at least one variable with missing values. We used multiple imputation—with the *ice* (version 1.4.6) package in Stata (Royston 2004, 2005)—to deal with the resulting loss in sample size and the potential bias to our estimates. We followed the procedures recommended by Van Buuren et al. (1999), with each variable having a separate prediction equation. These equations included the dependent and independent variables discussed later, additional measures hypothesized to affect the variable with missing values, and information on the data collection procedure that could result in the missing values. The new data were created using 10 imputations, with 20 regression switching cycles within each imputation. Table 1 presents means and percentages of the original sample, the means and percentages calculated over all the imputed data sets, and the minimum and maximum means and percentages found from these data sets.

Variables

Educational attainment at age 31, our dependent variable, is a five-level ordinal variable (cf. Isohanni et al. 2001). The least educated in our sample had nine years of compulsory education (ISCED 2). After that, they could take either one or two years of vocational training (ISCED 3B and ISCED 3 C), or three years of academically oriented high school education (ISCED 3A). The fourth-highest level of education consists of up to three additional years of postsecondary or lower tertiary education (typically, ISCED 4 and ISCED 5). University level (ISCED 6 and above), or higher tertiary, is the highest level of education.

The availability of proper measures in the NFBC 1966 largely determined our selection of the prenatal health variables. For example, we had no or inconsistent measures of infections and nutrient intake during pregnancy, mother's weight gain, diabetes, alcohol use, or head circumference at birth. Starting with the pre-conception variable, *mother's pre-pregnancy obesity* is measured using a dummy variable that is unity if her pre-pregnancy body mass index (BMI, kg/m^2) exceeded 30 (cf. Laitinen et al. 2001). Following WHO guidelines, we measure *anemia during pregnancy* as hemoglobin concentration below 110 g/l at any of the three measurement points in the NFBC 1966 (3rd, 7th, and 9th months) (Allen 2000; for NFBC, see Jones et al. 1998).

Hypertensive disorders are measured using a four-class variable (no hypertensive disorders being the reference group) (Järvelin et al. 1997). Chronic hypertension was defined as systolic blood pressure higher than 145 mmHg and diastolic blood pressure higher than 90 mmHg through pregnancy and/or clinically defined

Table 1 Means and percentages of the variables in the original and the 10 imputed data sets (means, minima, maxima)

		Imputed		
Variable	Original	Mean	Min.	Max.
Educational Attainment (dependent variable)				
Compulsory (9 years)	13.1	13.1	13.1	13.2
Vocational (10–11 years)	31.0	31.0	31.0	31.1
High school (12 years)	29.1	29.1	29.1	29.2
Lower tertiary	16.4	16.4	16.4	16.5
Higher tertiary	10.3	10.3	10.3	10.3
Prenatal Health				
Mother obese before pregnancy	3.6	4.0	3.9	4.1
Mother anemic during pregnancy	17.3	17.1	16.9	17.5
Mother's smoking: No	82.8	82.4	82.3	82.6
Mother's smoking: light/moderate in first trimester only	4.7	4.8	4.8	4.9
Mother's smoking: light/moderate throughout pregnancy	10.6	10.7	10.6	10.8
Mother's smoking: heavy	2.0	2.0	2.0	2.1
Hypertensive disorders: No	88.2	87.5	87.3	87.9
Hypertensive disorders: Chronic	2.9	2.9	2.8	3.1
Hypertensive disorders: Pregnancy-induced	6.7	7.3	6.9	7.6
Hypertensive disorders: Preeclampsia	2.1	2.2	2.1	2.4
Mother visited prenatal clinic during first trimester	21.9	21.0	20.8	21.3
Mother depressed/very depressed	13.5	13.6	13.6	13.7
Preterm birth (<37 full gestational weeks)	4.7	4.8	4.8	4.9
Small for gestational age (SGA)	2.9	2.8	2.8	2.8
Family Background				
Parental class: Unskilled working class	18.2	18.3	18.2	18.5
Parental class: Farmer	18.4	18.4	18.3	18.5
Parental class: Skilled working class	34.6	34.5	34.3	34.6
Parental class: Lower professional	20.0	19.9	19.8	19.9
Parental class: Higher professional	8.9	9.0	8.9	9.0
Mother's education: Compulsory or less	64.5	64.5	64.5	64.5
Mother's education: Vocational	18.9	18.4	18.4	18.4
Mother's education: General secondary	10.9	10.7	10.7	10.7
Mother's education: High school or more	4.9	4.8	4.8	4.8
Mother unmarried at birth	3.7	3.7	3.6	3.7
Control Variables				
Female	52.2	52.2	52.2	52.2
Birth order	2.9	2.9	2.9	2.9
Mother's age at birth	27.9	27.9	27.9	27.9
Wanted pregnancy?: Yes	64.0	63.9	64.1	63.6
Wanted pregnancy?: No	24.3	24.3	24.2	24.5
Wanted pregnancy?: Later	11.7	11.8	11.7	11.9
Attitudes: Should strive for better life	75.9	76.0	76.3	75.9
Attitudes: Should be happy with conditions	15.0	14.9	14.7	14.9
Attitudes: Society should help more	9.1	9.1	9.0	9.2

Note: $N = 8{,}625$.

Source: Northern Finland Birth Cohort 1966 Study.

hypertension before pregnancy. Pregnancy-induced (gestational) hypertension was defined as systolic/diastolic blood pressure higher than 145/90 mmHg after the 20th week of pregnancy (but not before), and preeclampsia was defined as gestational hypertension with continuous protein in the urine.

Mother's antenatal depressed mood is a dummy variable that is unity if the mother reported being depressed or very depressed during the pregnancy (Jones et al. 1998; Mäki et al. 2003, 2010). The data include information on whether and how much the mother smoked prior to pregnancy, whether and when she changed her smoking habits, and how much she smoked after this change (cf. Rantakallio 1978). Based on the available information, we created four dummy variables indicating *mother's smoking during pregnancy*: no smoking (reference), light or moderate smoking (up to 10 cigarettes/pipefuls daily) only during the first trimester, light or moderate smoking throughout pregnancy, or heavy smoking (more than 10 cigarettes/pipefuls daily). Only few heavy smokers gave up or reduced smoking during the first trimester, thus not permitting the construction of a separate category. The NFBC 1966 also includes information on when the mother paid her *first visit to a prenatal clinic* but, unfortunately, no further information on whether these were done on a regular basis. We thus use a single dummy variable to indicate a visit during the first trimester of the pregnancy (cf. Kiely et al. 1994).

As two birth outcome dummy variables, we use *small for gestational age* (SGA) and *preterm birth*, following the earlier discussion on the components of birth weight. Following recent medical practice, we defined SGA as a birth weight below 2 standard deviations (SD) from the sex and gestational age-specific mean birth weights (Lee et al. 2003), with means and standard deviations derived from Finnish fetal growth charts (Pihkala et al. 1989). We used the common definition of preterm births as those born before the 37th full gestational week, determined from the last menstrual period.

We measure socioeconomic background at birth using information on parents' occupational class, mother's education, and mother's marital status, all at the time of birth.[2] The *occupational class* is measured using the "dominance" principle, in which the partner with the highest class status determines the class of the family. Using the available information, we differentiate between five classes: higher and lower professional, skilled working class, farmers, and unskilled working class (reference group). *Mother's education* is measured using four categories: compulsory school or less (maximum 8 years, reference group); vocational school (additional 1–4 years); general secondary school; and high school or more (including academic education). Note that this measure is different from our dependent variable, due to an educational reform that took place in the early 1970s. *Mother's marital status at birth* is measured using a dummy variable that also acts as a proxy for single motherhood.[3]

Finally, our control variables are *sex* of the child, *mother's age at birth* (up to 18 years, 19–25 years (reference), 26–33 years, and 34 years or more), *birth order* (which we measure as the order of live births),[4] two dummy variables of *mothers'*

[2] The data distributed to us did not include socioeconomic background variables measured at a later time point.

[3] Cohabitation was still rare in Finland in 1966 (Finnäs 1995).

[4] This measure also partly captures any effects of the number of siblings.

attitudes toward self-provision ("one should be happy with conditions," "society should help more," and "one should strive for better life" (reference)),[5] and dummy variables for whether the *pregnancy was unwanted* or *was wanted later*.[6]

Methods

Our empirical analysis consists of three steps. First, we present odds ratios between the health variables and between the health variables and the social background variables. This odds ratio matrix serves to describe relationships between the health variables and the socioeconomic differences in prenatal health.

Second, we turn to analyzing the effects of prenatal health on educational attainment 31 years later. Given our educational attainment variable, ordered logit models are appropriate (e.g., Winship and Mare 1984). With these models, one assumes that the outcome (education) is a manifestation of an underlying latent variable, here interpreted as a propensity for educational attainment. As a result, interpretation of the coefficients is less straightforward than in ordinary least squares (OLS) models. Given our multiply imputed data, we used Stata's *mim* prefix, which calculates corrected standard errors using the so-called Rubin's rules (Royston 2004, 2005). Model fit statistics come from means of the respective ones for each imputed sample.

We estimate four different models. The first ones show the bivariate associations between the prenatal health variables and educational attainment. In the second models, we run separate models for each prenatal health variable, adjusting for family background and the control variables.

The third model includes all variables but the birth outcome measures (preterm birth and SGA), and the fourth and final model includes all the variables. The last two models were estimated to examine whether the effect of the prenatal health on educational attainment operated through the other health or birth outcome variables. We also ran these same models using OLS regression, with years of schooling as the dependent variable (see Table 7 in the Appendix). The results remained mostly robust, and we comment on the differences in the results section. To enhance interpretation of the results, we also present predicted probabilities of attaining different levels of education.

Third, we turn to the question of whether prenatal health contributes to explaining family background (measured by parental class, mother's education, and mother's marital status at birth) inequalities in educational attainment. We do this by comparing parameter estimates of the family background variables with and without prenatal health measures. However, because of neglected heterogeneity, estimates from logit models with and without additional variables are not directly comparable (Winship and Mare 1984:517; Wooldridge 2002:470–472). Therefore, we estimated Y-standardized coefficients, using Stata's *listcoef* command. These estimates are free of bias from neglected heterogeneity and are thus comparable across models (Long and Freese 2001:74). In effect, we analyze how introduction of the prenatal health

[5] In the absence of better measures, this variable can tap into otherwise unmeasured attitudes and behaviors that affect both prenatal health and educational outcomes.

[6] The relatively high rate of unwanted pregnancies corresponds to figures from other countries before the "perfect contraceptive" period (cf. Bumpass and Westoff 1970).

variables changes the relationship between the family background variables and the (latent) educational attainment variable.

Results

Associations Between Measures of Prenatal Health, and Socioeconomic Differences in Prenatal Health

First, we examine associations between the prenatal health variables, and between the family background measures and the prenatal health variables. Because the variables are categorical, these associations are shown as a matrix of odds ratios (Table 2).

One of the strongest relationships is between pre-pregnancy obesity and hypertensive disorders. Pre-pregnancy obesity also has a strong negative association with the child being born as SGA. There is a strong association between preeclampsia and SGA, in line with much previous literature (Witlin and Sibai 1997), and a weaker association between pregnancy-induced hypertension and SGA. However, hypertensive disorders are not associated with preterm birth.

Depressed mothers were more likely to smoke throughout pregnancy. Similar results linking psychosocial problems to continued smoking through pregnancy have been found elsewhere (Pickett et al. 2009). Women who smoked throughout pregnancy were also more likely to have SGA babies. Continued light smoking is additionally related to mother's obesity, visits to prenatal clinics, milder hypertensive disorders (but not preeclampsia), and premature birth. It is also important to point out that some (obesity and hypertensive disorders) of the prenatal health variables are associated with SGA but not with prematurity, while others (anemia and depression) are associated with prematurity but not with SGA. These underline the different etiologies of these two birth outcomes that together determine birth weight.

These associations are generally in line with what is known in the medical literature. It is also worth pointing out that not all measures are related to each other. This suggests that these measures capture different, partly independent, aspects of prenatal health.

Turning to the family background disparities in prenatal health, one can clearly observe many—often gradient-like—differences. Obesity, for example, was less common among the higher classes and among mothers with higher levels of education, and less common among unmarried mothers. Gradient-like socioeconomic patterns can be found for anemia, depression, and early visits to a prenatal clinic. However, unlike in the case of obesity, unmarried mothers were in a less-favorable position according to these measures. Unmarried mothers were also more likely to smoke, continue smoking, and smoke heavily; and were more likely to give birth prematurely and to an SGA child. Prematurity and SGA both show gradient-like disparities by class although not all associations are significant. There is a clear working class/nonworking class division in prenatal smoking in our cohort (cf. Rantakallio 1978). Farmers' wives were less likely to smoke at any point in pregnancy, whereas women from professional classes were more likely to quit smoking. The differences by mother's education are less clear. Better-educated mothers were

Table 2 Associations between the independent variables, odds ratios

			Hypertension				Smoking					
	Obese	Anemia	Chronic	Pregnancy-Induced	Preeclampsia	Depressed	Quit	Light	Heavy	Prenatal Clinic	Preterm Birth	SGA
Anemia	1.02											
Hypertension[a]												
Chronic	6.79***	0.82										
Pregnancy-Induced	1.97**	0.94										
Preeclampsia	2.52**	0.88										
Depressed	1.52**	1.65***	1.26	1.00	0.93							
Smoking[b]												
Quit[c]	0.59	0.93	0.29	0.74	0.88	1.28						
Light	0.49**	1.14	0.33**	0.53**	0.88	1.35**						
Heavy	0.52	1.28	0.37	0.44	0.95	2.02***						
Prenatal Clinic	0.64**	0.80**	0.95	0.96	0.67	0.74**	1.59***	0.82*	0.98			
Preterm Birth	0.72	1.38*	1.48	1.26	1.48	1.50**	1.10	1.72***	1.22	0.92		
SGA[d]	0.20*	1.21	1.30	1.60*	4.85***	1.24	1.13	2.03***	2.28*	0.90	1.44	
Parental Class[e]												
Farmer	1.98***	1.08	2.29***	1.12	0.93	0.74**	0.35***	0.38***	0.31***	0.84	0.81	0.70
Skilled worker	0.48***	0.70***	1.07	0.85	1.30	0.59***	1.40*	0.97	0.62*	1.43***	0.89	0.90
Lower professional	0.57**	0.65***	1.07	0.87	1.25	0.43***	1.26	0.68**	0.63*	1.52***	0.66*	0.66*
Higher professional	0.24***	0.61***	0.72	0.80	0.84	0.36***	0.89	0.39***	0.39**	1.94***	0.63*	0.53*

Table 2 (continued)

			Hypertension				Smoking					
	Obese	Anemia	Chronic	Pregnancy-Induced	Preeclampsia	Depressed	Quit	Light	Heavy	Prenatal Clinic	Preterm Birth	SGA
Mother's education[f]												
Vocational	0.66**	0.76**	0.67*	0.88	1.23	0.67***	1.07	0.63***	0.59*	1.23**	0.65**	0.97
Secondary	0.18***	0.66***	0.40**	0.87	1.39	0.49***	1.44*	0.88	0.76	1.48***	0.69*	0.79
High school	0.15**	0.58**	0.49	0.58*	0.75	0.41***	0.96	0.44***	0.81	1.63***	0.53*	0.92
Unmarried	0.24*	1.89**	0.53	1.05	1.44	5.97***	1.85*	3.53***	4.55***	0.31***	2.29***	1.80*

Notes: Ten multiply imputed data sets were used in the estimation; $N = 8{,}625$.

Source: Northern Finland Birth Cohort 1966 Study.

[a]Reference category: no hypertensive disorders.

[b]Reference category: mother did not smoke.

[c]Quit during first trimester, light smoker before quitting.

[d]SGA: Small for gestational age.

[e]Reference category: Unskilled working class.

[f]Reference category: Mother had compulsory schooling or less.

*$p < .05$; **$p < .01$; ***$p < .001$

less likely to give birth prematurely, but interestingly, no significant associations can be found between mother's education and SGA.

Prenatal Health Conditions and Educational Attainment

Next, we turn to analyzing the effects of prenatal health on educational attainment with ordered logit models. In Table 3, we present results from analyses in which each prenatal health variable is analyzed separately. We present two estimates for each variable. The column on the left shows unadjusted, bivariate associations between each prenatal health measure in question and educational attainment. The column on the right shows the adjusted associations after the family background and control variables have been included in the model.

First, looking at the results from the bivariate models, we find that with the exceptions of hypertensive disorders and SGA, each measure is associated with educational attainment. Children who were born prematurely, or to mothers who were obese prior to pregnancy, suffered from anemia, were depressed, smoked during pregnancy, or did not visit prenatal screening during the first trimester attained, on average, lower levels of education 31 years later.

Table 3 Prenatal health and educational attainment: Separate ordered logit models for each health variable

	Models 1A–1H: Bivariate		Models 2A–2H: Adjusted[a]	
	b	SE	*b*	SE
A. Mother Obese Before Pregnancy	−0.427***	0.100	−0.039	0.106
B. Anemia	−0.281***	0.053	−0.109*	0.055
C. Depressed/Very Depressed	−0.417***	0.057	−0.055	0.063
D. Smoking:				
No (ref.)	—		—	
Light, first trimester only	−0.070	0.093	−0.174	0.094
Light throughout pregnancy	−0.400***	0.066	−0.289***	0.068
Heavy	−0.562***	0.144	−0.424**	0.147
E. Hypertension:				
No (ref.)	—		—	
Chronic	−0.136	0.134	0.005	0.133
Pregnancy-induced	0.088	0.089	0.151	0.087
Preeclampsia	−0.013	0.168	−0.042	0.159
F. Prenatal Clinic Visit, First Trimester	0.259***	0.048	0.083	0.050
G. Preterm Birth	−0.278***	0.094	−0.132	0.095
H. Small for Gestational Age (SGA)	0.064	0.116	−0.136	0.119

Notes: Ten multiply imputed data sets were used in the estimation of these models; $N = 8{,}625$.

Source: Northern Finland Birth Cohort 1966 Study.

[a]Control variables: sex, mother's education, parental class, mother's age at birth, mother's marital status, wantedness of pregnancy, attitudes toward self-provision, and birth order.

*$p < .05$; **$p < .01$; ***$p < .001$

The estimates presented in the third column are weaker and less significant. The only effects that remained significant at the 5% level are those of continued smoking and anemia during pregnancy.[7] Regarding smoking, the estimates also suggest interesting patterns of the timing and intensity of smoking. First, children born to mothers who quit smoking did not attain lower levels of education,[8] whereas those born to mothers who continued smoking fared worse. Furthermore, among those whose mothers smoked throughout pregnancy, children of heavy smokers (more than 10 cigarettes or pipefuls) seem to have fared worse than children of light/moderate smokers. These results are consistent with the neurobiological research discussed earlier.

With the exception of these two prenatal health conditions, many of the variables that feature in the medical literature and could be thought to have long-term effects were only spuriously related to educational attainment. Going beyond this general conclusion, it is worth mentioning that the control variables have different effects on the estimates of the different health conditions (not shown). For example, birth order explained the effect of maternal obesity (obesity risk grows with successive births), whereas the effect of depression became only weakly significant after controlling for whether the pregnancy was wanted. Prematurity was more sensitive to the inclusion of the socioeconomic background variables, while the effects of the other variables lost significance only after inclusion of a fuller set of controls.

The models presented in Table 3 include each prenatal health variable separately. Do the results change after we include all the prenatal variables in the same model? This is examined in the first model in Table 4. Clearly, the answer is no, with one exception: the estimate of prenatal anemia loses significance. However, at closer inspection, it becomes clear that this is due to anemia becoming borderline non-significant from being borderline significant, which partly has to do with the loss in degrees of freedom.[9] Do these effects operate through preterm births or depressed fetal growth? A negative answer to this question is seen from the second model in Table 4, which adds prematurity and SGA into the analysis. Thus, the effects of prenatal smoking (and anemia) operate independently of the other prenatal health and birth outcome variables. In fact, this is unsurprising given that these other measures were not significant predictors of educational attainment after adjusting for family background and the control variables.

Predicted Probabilities

The ordered logit regression analyses presented earlier show robust effects of prenatal smoking, and somewhat less robust effects of anemia, on educational attainment. They do not, however, tell much about the magnitude of the effects. How much less education do children of mothers who smoked or were anemic during pregnancy

[7] In addition, discontinued light smoking, pregnancy-induced hypertension, and a prenatal clinic visit during the first trimester were significant at the 10% level.

[8] However, this estimate was significant at the 10% level throughout the models.

[9] In the OLS specifications, visit to a prenatal clinic was significant at the 5% level in the second model when using years of schooling as the dependent variable (see Table 7 in the appendix), but not when using our ordinal schooling variable. Anemia remained significant at the 5% level in each model, using both outcome variables.

Table 4 Prenatal health and educational attainment: Ordered logit regression

	Model 1:[a] All Prenatal Variables, Without Birth Outcome Variables		Model 2:[a] All Prenatal and Birth Outcome Variables	
	b	SE	*b*	SE
Mother Obese Before Pregnancy	−0.053	0.108	−0.058	0.108
Anemia	−0.103	0.054	−0.102	0.054
Depressed/Very Depressed	−0.044	0.064	−0.043	0.064
Smoking				
No (ref.)	—		—	
Light, first trimester only	−0.175	0.094	−0.174	0.094
Light throughout pregnancy	−0.282***	0.068	−0.276***	0.068
Heavy	−0.417**	0.147	−0.415**	0.147
Hypertension				
No (ref.)	—		—	
Chronic	−0.017	0.136	−0.014	0.132
Pregnancy-induced	0.133	0.087	0.137	0.087
Preeclampsia	−0.045	0.157	−0.033	0.158
Prenatal Clinic Visit, First Trimester	0.081	0.050	0.080	0.050
Preterm Birth			−0.119	0.095
Small for Gestational Age (SGA)			−0.108	0.120
Threshold 1	−1.620	0.073	−1.628	0.073
Threshold 2	0.193	0.070	0.187	0.070
Threshold 3	1.597	0.072	1.591	0.073
Threshold 4	2.864	0.078	2.858	0.078
Log-Likelihood (mean)	−12,392.95		−12,391.67	
Log-Likelihood (max.)	−12,388.30		−12,387.17	
Log-Likelihood (min.)	−12,397.49		−12,395.51	

Notes: Ten multiply imputed data sets were used in the estimation of these models; $N = 8{,}625$.

Source: Northern Finland Birth Cohort 1966 Study.

[a]Control variables: mother's education, parental class, mother's age at birth, mother's marital status, wantedness of pregnancy, attitudes toward self-provision, and birth order.

*$p < .05$; **$p < .01$; ***$p < .001$

attain? We examine this question in Table 5, using predicted probabilities. For the predictions, we use the "gross" estimates from the second models in Table 3, which are adjusted for family background and the controls, but not the other prenatal health and birth variables.[10] We focus on prenatal smoking (Model 2D) and anemia (Model 2B), the two conditions that were statistically significant predictors of educational attainment. The other variables are held at their means.

[10] Given the stability of the estimates of smoking and anemia, predicted probabilities based on the models in Table 4 are practically the same, although with wider confidence intervals.

Table 5 Predicted probabilities and 95% confidence intervals of educational attainment by mother's pregnancy-time smoking status and anemia, with other variables held at their means

	Compulsory (9 yrs.)	Vocational (10–11 yrs.)	High School (12 yrs.)	Lower Tertiary (13–14 yrs.)	Higher Tertiary (16–17 yrs.)
Mother's Smoking Status During Pregnancy					
Nonsmoker	.105	.313	.327	.167	.088
	[.099; .112]	[.303; .324]	[.316; .338]	[.158; .175]	[.082; .094]
Light smoking, first trimester only	.123	.339	.316	.148	.075
	[.103; .143]	[.312; .366]	[.299; .332]	[.129; .168]	[.062; .088]
Light smoking throughout pregnancy	.136	.354	.306]	.136	.067
	[.120; .151]	[.335; .374]	[.292; .321]	[.123; .150]	[.058; .076]
Heavy smoking	.152	.372	.293	.123	.059
	[.115; .189]	[.336; .408]	[.263; .323]	[.096; .151]	[.043; .076]
Mother's Anemia During Pregnancy					
Not anemic	.108	.317	.325	.164	.086
	[.101; .115]	[.306; .328]	[.314; .336]	[.156; .172]	[.080; .092]
Anemic	.119	.333	.318	.152	.078
	[.107; .130]	[.316; .349]	[.306; .330]	[.140; .165]	[.070; .086]

Notes: Predicted probabilities are based on Models 2B (anemia) and 2D (smoking) from Table 3, which include mother's smoking and anemia, respectively, and the family background and control variables. Ten multiply imputed data sets were used in the estimation of these models. $N = 8{,}625$.

Source: Northern Finland Birth Cohort 1966 Study.

We first explore the differences in educational attainment at different levels of mother's prenatal smoking. The difference between children of nonsmokers and of those who quit during the first trimester was not significant (Table 3), so we focus on comparing the children of nonsmokers to those of mothers who continued smoking. Looking at the two extremes of educational attainment (nine years of compulsory school versus 16–17 years with higher tertiary education), Table 5 shows clear differences between children of nonsmokers and children of mothers who continued smoking beyond the first trimester. Although a predicted 10.5% of children of nonsmokers did not continue beyond nine years of compulsory schooling, children of light/moderate smokers were approximately 30% more likely (with 13.6% attaining only compulsory schooling), and children of heavy smokers were approximately 50% more likely (15.2% not continuing) to do so. They were also 24% and 33% less likely to obtain a university degree (6.7% and 5.9% vs. 8.8%, respectively). These differences can be considered large. To compare these effects with those of conventional social background variables, additional analyses not presented here showed that the effect of continued heavy smoking (compared with not smoking) is of very similar magnitude as the (adjusted) effect of being from a skilled versus unskilled working-class family, or of having a mother with the second lowest versus the lowest level of education.

The differences between children of anemic versus nonanemic mothers are, on the other hand, far less important. Although the effect of anemia during pregnancy is significant, the effect size is not very substantial. Therefore, mother's prenatal

smoking not only has the most robust and statistically significant effect, but it is also substantively the most important.

Prenatal Health and the Intergenerational Transmission of Educational Inequality

Does prenatal health contribute to intergenerational inequalities in educational attainment? In this section, we estimate the extent to which intergenerational inequalities by class, mother's education, and family structure can be explained by prenatal smoking, anemia, and all the prenatal health measures collectively.

Table 2 shows that several of our health measures, including mother's prenatal smoking and anemia, are not equally distributed across social groups. Anemia and prenatal smoking were less common among better-educated mothers from higher-class backgrounds (however, smoking was especially uncommon among farmers). There was also a major difference between the prenatal smoking habits of married compared with unmarried mothers. These conditions can thus plausibly contribute to intergenerational inequalities.

We examine whether our health variables can account for differences in intergenerational educational inequality in Table 6. The first column presents ordered logit estimates of the effects of parents' class position, mother's education, and mother's marital status on educational attainment from a model that also includes mother's age at birth, birth order, wantedness of pregnancy, and attitudes toward self-provision as independent variables. The second column shows the Y-standardized coefficient estimates from the same model. The third column gives the Y-standardized coefficient estimates after additionally controlling for anemia, and the fourth column shows the percentage change from the original estimates. The last four columns show the same figures after controlling for prenatal smoking, and for all prenatal health and birth outcome variables, respectively.

Looking at the percentage changes in the Y-standardized coefficients—that is, the share of the family background inequalities explained by the respective variables—it is clear that educational inequalities by class background or maternal education are not accounted for by the prenatal health variables, either individually or collectively. These variables account for at most 6% (and in most cases, less) of the educational attainment differences. Overall, these shares are smaller than the ones estimated by Palloni and colleagues (Palloni 2006; Palloni et al. 2008) for class background gradients in occupational attainment. However, prenatal smoking alone explains 12% of the educational differences between children born to unmarried versus married mothers, and anemia alone accounts for 3% of these differences. Inclusion of the other health variables increases this share to 19%. The stark prenatal health disparities between married and unmarried mothers explain these results.

A conclusion, therefore, of this section of the analysis is that although prenatal health does indeed contribute little to our understanding of intergenerational inequalities according to parental class or education, this does not necessarily apply to all social background differences. Prenatal and childhood measures that both have strong effects on later outcomes and show clear social disparities can contribute to the intergenerational reproduction of socioeconomic inequalities. As we will discuss in the next section, this adds to reasons for being concerned about an increase in early health disparities.

Table 6 Prenatal health and intergenerational educational inequality: *b*-coefficients, Y-standardized coefficients, and percentage changes in Y-standardized coefficients

	Gross[a]	Gross[a]		Adjusted for Anemia During Pregnancy		Adjusted for Smoking During Pregnancy		Adjusted for All Prenatal Health and Birth Outcome Variables	
	b	Y-Standardized	% Δ	Y-Standardized	% Δ	Y-Standardized	% Δ	Y-Standardized	% Δ
Parental Class									
Unskilled working class (ref.)	—	—		—		—		—	
Farmer	0.413***	0.137	—	0.137	0	0.129	−6	0.130	−6
Skilled working class	0.411***	0.137	—	0.135	−1	0.136	−0	0.134	−2
Lower professional	0.650***	0.216	—	0.215	−1	0.214	−1	0.211	−2
Higher professional	1.128***	0.375	—	0.374	−0	0.367	−2	0.364	−3
Mother's Education									
Compulsory or less (ref.)	—	—		—		—		—	
Vocational	0.407***	0.135	—	0.135	−0	0.130	−4	0.129	−5
General secondary	0.717***	0.238	—	0.237	−0	0.236	−1	0.233	−2
High school or more	1.291***	0.429	—	0.427	−0	0.424	−1	0.422	−1
Mother's Marital Status									
Married (ref.)	—	—		—		—		—	
Unmarried	−0.365**	−0.121	—	−0.118	−3	−0.107	−12	−0.098	−19

Note: Ten multiply imputed data sets were used in the estimation of these models. $N = 8{,}625$.

Source: Northern Finland Birth Cohort 1966 Study.

[a]Ordered logit models without prenatal health or birth outcome variables. Independent variables: parental class, mother's education, mother's marital status, mother's age at birth, wantedness of pregnancy, attitudes toward self-provision, and birth order.

***$p < .001$

Conclusions and Discussion

Our results, based on follow-up data from Northern Finland Birth Cohort 1966 Study, confirm that prenatal factors can have long-lasting implications on children's educational attainment. Children whose mothers smoked throughout pregnancy attained clearly less education than those whose mothers did not smoke. Furthermore, the results suggested timing (smoking during the first trimester only did not have a significant effect) and dose–response effects of prenatal smoking, in line with neuroscientific research (Huizink and Mulder 2006; Shea and Steiner 2008; Slotkin 1998). Prenatal smoking alone accounted for up to 6% of the differences in educational attainment by class background and mother's education, but 12% of the lower educational attainment of children born to unmarried mothers (who were single mothers, due to rarity of cohabitation in 1966). These results reflect the large differences in prenatal smoking by marital status in our population, whereas variation by class or maternal education was relatively smaller. We also found a significant, yet weak, effect of prenatal anemia, which contributed little to understanding intergenerational inequalities. Together, our prenatal health measures accounted for up to 6% of the educational attainment differences by class background and mother's education, but up to 19% of the differences by mother's marital status.

Our other indicators of prenatal health (mother's pre-pregnancy obesity, hypertensive disorders during pregnancy, mother's prenatal depression, visits to a prenatal clinic during the first trimester, premature birth, and being born small for gestational age) did not have significant effects after we controlled for sociodemographic characteristics of the mother and the family. All these are predictors of child health and developmental outcomes in the short run, and some have also been shown to have longer-term effects.

A surprising finding was that being born prematurely or small for gestational age did not have effects on educational attainment. Gestational age and the fetal growth rate are the two components of birth weight, which has been found to have long-term effects in several previous studies (cf. Conley et al. 2003; Currie 2009).[11] Our result can be attributable to omitted variables because some studies report stronger effects after sibling or twin fixed effects are controlled for (Behrman and Rosenzweig 2004; Conley and Bennett 2000). Alternatively, children born prematurely or small for gestational age can also receive compensatory attention from parents and other significant adults (cf. Conley et al. 2003). This is suggested by a previous study with the same data, which showed that low-birth-weight children were more likely to be held back a grade or to receive special education, but did not ultimately attain less education (Olsén et al. 1994). However, it is also important to notice that not all previous studies have reported birth-weight effects (Gorman 2002; Kaestner and Corman 1995).

Despite increasing interest in the role of early health in the intergenerational transmission of inequality, our study is among the first to empirically estimate its contributions. The relatively limited social class and educational differences in prenatal smoking —which, however, have widened more recently (Jaakkola et al. 2001)—contributed to its relatively limited importance in explaining intergenerational inequalities by these

[11] Replacing our measure of SGA with a dummy variable for low birth weight (<2,500 g) did not change our results.

background measures. On the other hand, the large differences in prenatal smoking by marital status—approximately 17% of married mothers and 29% of unmarried mothers smoked during pregnancy—that lie behind intergenerational inequalities by family structure are not limited to northern Finland of the 1960s.[12] Recent figures from Finland show even wider disparities, with 8% of married mothers, compared with 23% of cohabiting and 30% of single mothers, smoking during pregnancy (Jaakkola et al. 2001). Similar disparities have been reported for other countries (cf. Kiernan and Pickett 2006). These differences—which remain significant and strong after the inclusion of controls for age, education, and class (not shown)—partly reflect selection into single motherhood (cf. Table 2). However, relationship characteristics can themselves affect prenatal smoking and other health behaviors, since intimate bonds with the father can help in quitting smoking and overtaking other positive health behaviors (cf. Kiernan and Pickett 2006). Overall, family structure emerges as an important family background variable that deserves to be considered in future studies.

A limitation of our study is that our estimates cannot be given causal interpretations. Some studies have found that the association between prenatal smoking and psychological, schooling, and behavioral outcomes disappears after controls for observed and unobserved family characteristics (Batty et al. 2006; Ernst et al. 2001; Lambe et al. 2006; MacArthur et al. 2001; Raatikainen et al. 1992). In such a case, our estimates of the role of smoking in the reproduction of intergenerational inequalities are also biased. However, there are some reasons to expect that our estimates contain a causal component. First, the neuroscientific basis for effects of smoking on brain development is rather solid. Second, our cohort was born at a time (just two years after the landmark U.S. Surgeon General's report) when selectivity to smoking was likely to be lower than more recently. Third, the effects of prenatal smoking were much more robust to the inclusion of control variables than were the effects of the other psychobehavioral variables.[13]

Our study does not—within the limitations of one article—say anything about the biological pathways through which our health measures affect (or fail to affect) educational attainment. We discussed some of these in the second section of this article. Neither do they tell us about their interactive effects with the genome and the environment. This is an area in which recent progress has been made (e.g., Almond et al. 2009; Conley et al. 2003; Jackson 2009; Lundborg and Stenberg 2010; Palloni 2006; Palloni et al. 2008). Nevertheless, more research in this area is clearly welcome. Finally, we might also be underestimating the effects of prenatal health on educational attainment and its role in the process of intergenerational transmission because we naturally could not include many potentially important measures of the fetal environment (cf. Palloni et al. 2008:180).

Regardless of these limitations, our study has demonstrated the usefulness of proximate measures for analyzing the long-term effects of early health. Although

[12] It is worth pointing out that unlike in the United States, the share of children born to unpartnered mothers has remained rather constant. According to Andersson and Philipov (2002:238), 3% of Finnish children were born to single mothers in the 1980s, a figure practically the same as the one in our data (3.7%).

[13] Their effects either disappeared (for mother's depression and attitudes toward self-provision) or were heavily reduced (for wantedness of pregnancy) after the control variables were entered.

general measures such as birth weight are invaluable, identification of more proximate factors can provide a more nuanced understanding of links between health and social position and be beneficial for development of successful interventions. Our results also show that large social disparities in key early-life health variables can produce nonnegligible intergenerational inequalities in socioeconomic attainment. An implication is that if social disparities in prenatal health widen—as seems to be the case for prenatal smoking (Cnattingius 2004) and some other indicators (Korenbrot and Moss 2000)—it can become more important for the intergenerational reproduction of inequality. In such a case, the need for understanding these disparities and the rationale for closing gaps in early health become even stronger.

Acknowledgments We wish to acknowledge financial aid from Emil Aaltonen Foundation and from Signe and Ane Gyllenberg Foundation. This study was largely conducted when J. Härkönen was in the Center for Research on Inequalities and the Life Course (CIQLE) at Yale University. We are indebted to its support. Previous versions of the study have been presented at the RC 28 2008 Summer Meeting (Stanford), Population Association of America 2009 Meeting (Detroit), Oxford University, Stockholm University (SOFI and CHESS), University of Oslo, University of Turku, and Yale University (CIQLE). We thank the seminar participants, and especially two anonymous reviewers, Vida Maralani, Tim Liao, Berkay Özcan, Anette Fasang, Marit Dahlén Gisselmann, Richard Breen, Felicia LeClere, Fabrizio Bernardi, Hannah Brückner, Denny Vågerö, Florencia Torche, Karl Ulrich Mayer, Juha Veijola, and Renee Fox for helpful comments. All remaining errors are ours.

Appendix

Table 7 Effects of prenatal health on years of schooling: OLS estimates with multiply imputed (10) data

	Models 1: Bivariate Associations		Models 2: Health Variables Separately+ Controls[a]		Model 3: All Prenatal Health and Control Variables[a]		Model 4: All Variables	
	b	SE	*b*	SE	*b*	SE	*b*	SE
Mother Obese Before Pregnancy	−0.501***	0.123	−0.039	0.118	−0.056	0.120	−0.059	0.120
Anemia	−0.338***	0.065	−0.131*	0.060	−0.125*	0.060	−0.125*	0.061
Depressed/Very Depressed	−0.485***	0.068	−0.061	0.070	−0.049	0.069	−0.048	0.069
Did Not Smoke (ref.)	—		—		—		—	
Light/Moderate Smoking in First Trimester	−0.112	0.112	−0.201	0.105	−0.204	0.105	−0.203	0.105
Light/Moderate Smoking Throughout Pregnancy	−0.462***	0.078	−0.295***	0.073	−0.287***	0.074	−0.283***	0.073
Heavy Smoking	−0.572**	0.169	−0.384*	0.158	−0.376*	0.159	−0.374*	0.159
Hypertension: No (ref.)	—		—		—		—	
Hypertension: Chronic	−0.146	0.156	0.017	0.141	−0.008	0.143	−0.005	0.143
Hypertension: Pregnancy-Induced	0.107	0.106	0.159	0.096	0.140	0.096	0.143	0.097

Table 7 (continued)

	Models 1: Bivariate Associations		Models 2: Health Variables Separately+ Controls[a]		Model 3: All Prenatal Health and Control Variables[a]		Model 4: All Variables	
	b	SE	*b*	SE	*b*	SE	*b*	SE
Hypertension: Preeclampsia	0.030	0.181	−0.024	0.165	−0.021	0.164	−0.012	0.165
Prenatal Clinic Visit, First Trimester	0.330***	0.058	0.111*	0.056	0.109	0.056	0.109	0.056
Preterm Birth	−0.276*	0.111	−0.097	0.103			−0.084	0.103
Small for Gestational Age (SGA)	−0.000	0.142	−0.117	0.134			−0.090	0.135

Note: Ten multiply imputed data sets were used in the estimation of these models; $N = 8{,}265$.

Source: Northern Finland Birth Cohort 1966 Study.

[a]Additional control variables: mother's education, parental class, mother's age at birth, mother's marital status, wantedness of pregnancy, attitudes toward self-provision, and birth order.

*$p < .05$; **$p < .01$; ***$p < .001$

References

Allen, L. H. (2000). Anemia and iron deficiency: Effects on pregnancy outcome. *American Journal of Clinical Nutrition, 71,* 1280S–1284S.

Almond, D. (2006). Is the 1918 influenza pandemic over? Long-term effects of *in utero* influenza exposure in the post-1940 U.S. population. *Journal of Political Economy, 114,* 672–712.

Almond, D., Chay, K. Y., & Lee, D. S. (2005). The costs of low birth weight. *Quarterly Journal of Economics, 120,* 1031–1083.

Almond, D., Edlund, L., & Palme, M. (2009). Chernobyl's subclinical legacy: Prenatal exposure to radioactive fallout and school outcomes in Sweden. *Quarterly Journal of Economics, 124,* 1729–1772.

Andersson, G., & Philipov, D. (2002). Life-table representations of family dynamics in Sweden, Hungary, and 14 other FFS countries: A project of descriptions of demographic behavior. *Demographic Research, 7,* article 4, 67–144. doi:10.4054/DemRes.2002.7.4

Barker, D. J. P. (1991). Intrauterine origins of cardiovascular and obstructive lung disease in adult life. *Journal of the Royal College of Physicians of London, 25,* 264–266.

Barker, D. J. P. (2001). Fetal and infant origins of adult disease. *Monatschrift Kinderheilkunde, 149,* 2–56.

Batty, G. D., Der, G., & Deary, I. J. (2006). Effect of maternal smoking during pregnancy on offspring's cognitive ability: Empirical evidence for complete confounding in the US National Longitudinal Survey of Youth. *Pediatrics, 118,* 943–950.

Behrman, R. E., & Butler, A. S. (Eds.). (2007). *Preterm birth: Causes, consequences, and prevention*. Washington, DC: National Academies Press.

Behrman, J. R., & Rosenzweig, M. R. (2004). Returns to birthweight. *Review of Economics and Statistics, 86,* 586–601.

Black, S. E., Devereux, P. J., & Salvanes, K. G. (2007). From the cradle to the labor market? The effect of birth weight on adult outcomes. *Quarterly Journal of Economics, 122,* 409–439.

Bumpass, L., & Westoff, C. F. (1970). The "perfect contraceptive" population. *Science, 169,* 1177–1182.

Case, A., & Paxson, C. (2002). Parental behavior and child health. *Health Affairs, 21,* 164–178.

Case, A., & Paxson, C. (2006). Children's health and social mobility. *Future of Children, 16,* 151–173.

Case, A., Fertig, A., & Paxson, C. (2005). The lasting impact of childhood health and circumstance. *Journal of Health Economics, 24*, 365–389.

Caspi, A., Williams, B., Kim-Cohen, J., Craig, I. W., Milne, B. J., Poulton, R., . . . Moffitt, T. E. (2007). Moderation of breastfeeding effects on the IQ by genetic variation in fatty acid metabolism. *Proceedings of the National Academy of Sciences, 104,* 18860–18865.

Castro-Martin, T. (2010). Single motherhood and low birth weight in Spain: Narrowing social inequalities in health. *Demographic Research, 22,* article 27, 863–890. doi:10.4054/DemRes.2010.22.27

Cnattingius, S. (2004). The epidemiology of smoking during pregnancy: Smoking prevalence, maternal characteristics, and pregnancy outcomes. *Nicotine and Tobacco Research, 6,* S125–S140.

Conley, D., & Bennett, N. G. (2000). Is biology destiny? Birth weight and life chances. *American Sociological Review, 65,* 458–467.

Conley, D., & Bennett, N. G. (2001). Birth weight and income: Interactions across generations. *Journal of Health and Social Behavior, 42,* 450–465.

Conley, D., Strully, K. W., & Bennett, N. G. (2003). *The starting gate: Birth weight and life chances*. Berkeley: University of California Press.

Currie, J. (2009). Healthy, wealthy, and wise: Socioeconomic status, poor health in childhood, and human capital development. *Journal of Economic Literature, 47,* 87–122.

Currie, J., & Moretti, E. (2007). Biology as destiny? Short and long-run determinants of intergenerational transmission of birth weight. *Journal of Labor Economics, 25,* 231–264.

Currie, J., Stabile, M., Manivong, P., & Roos, L. L. (2010). Child health and young adult outcomes. *Journal of Human Resources, 45,* 517–548.

Datar, A., Kilburn, M. R., & Loughran, D. S. (2010). Endowments and parental investment in infancy and early childhood. *Demography, 47,* 145–162.

Ehrenstein, V., Rothman, K. J., Pedersen, L., Hatch, E. E., & Toft Sørensen, H. (2009). Pregnancy-associated hypertensive disorders and adult cognitive function among Danish conscripts. *American Journal of Epidemiology, 170,* 1025–1031.

Ek, E., Sovio, U., Remes, J., & Järvelin, M.-R. (2005). Social predictors of unsuccessful entrance into the labour market—A socialization process perspective. *Journal of Vocational Behavior, 66,* 471–486.

Ernst, M., Moolchan, E. T., & Robinson, M. L. (2001). Behavioral and neural consequences of prenatal exposure to nicotine. *Journal of the American Academy of Child and Adolescent Psychiatry, 40,* 630–641.

Finnäs, F. (1995). Entry into consensual unions and marriage among Finnish women born between 1938 and 1967. *Population Studies, 49,* 57–70.

Gluckman, P., & Hanson, M. (2005). *The fetal matrix: Evolution, development and disease*. Cambridge, UK: Cambridge University Press.

Gorman, B. K. (2002). Birth weight and cognitive development in adolescence: Causal relationship or social selection? *Social Biology, 49,* 13–34.

Guelinckx, I., Devlieger, R., Beckers, K., & Vansant, G. (2008). Maternal obesity: Pregnancy complications, gestational weight gain, and nutrition. *Obesity Reviews, 9,* 140–150.

Haas, S. A. (2006). Health selection and the process of social stratification: The effect of childhood health on socioeconomic attainment. *Journal of Health and Social Behavior, 47,* 339–354.

Heckman, J. J. (2006). Skill formation and the economics of investing in disadvantaged children. *Science, 312,* 1900–1902.

Heikura, U., Taanila, A., Hartikainen, A.-L., Olsen, P., Linna, S.-L., von Wendt, L., & Järvelin, M.-L. (2008). Variations in prenatal sociodemographic factors associated with intellectual disability: A study of the 20-year interval between two birth cohorts in northern Finland. *American Journal of Epidemiology, 167,* 169–177.

Huizink, A. C., & Mulder, E. J. H. (2006). Maternal smoking, drinking, or cannabis use during pregnancy and neurobehavioral and cognitive functioning in human offspring. *Neuroscience and Biobehavioral Reviews, 30,* 24–41.

Institute of Health Sciences, University of Oulu. (n.d.). *Northern Finland Birth Cohort 1966 Study.* Retrieved from http://kelo.oulu.fi/NFBC/

Isohanni, I., Järvelin, M.-R., Rantakallio, P., Jokelainen, J., Jones, P. B., Nieminen, P., . . . Isohanni, M. (2001). Juvenile and early adulthood smoking and adult educational achievements—A 31-year follow-up of the northern Finland 1966 Birth Cohort. *Scandinavian Journal of Public Health, 29,* 87–95.

Jaakkola, N., Jaakkola, M. S., Gissler, M., & Jaakkola, J. J. K. (2001). Smoking during pregnancy in Finland: Determinants and trends, 1987–1997. *American Journal of Public Health, 91,* 284–286.

Jackson, M. I. (2007). *The timing of early-life health disadvantage* (Working paper CCPR-007-07). Los Angeles: California Center for Population Research, UCLA.

Jackson, M. I. (2009). Understanding links between adolescent health and educational attainment. *Demography, 46,* 671–694.

Järvelin, M.-R., Elliot, P., Kleinschmidt, I., Martuzzi, M., Grundy, C., Hartikainen, A.-L., & Rantakallio, P. (1997). Ecological and individual predictors of birthweight in a northern Finland birth cohort 1966. *Paediatric and Perinatal Epidemiology, 11,* 298–312.

Jones, P. B., Rantakallio, P., Hartikainen, A.-L., Isohanni, M., & Sipilä, P. (1998). Schizophrenia as a long-term outcome of pregnancy, delivery, and perinatal complications: A 28-year follow-up of the 1966 North Finland general population birth cohort. *American Journal of Psychiatry, 155,* 355–364.

Kaestner, R., & Corman, H. (1995). *The impact of child health and family input on child cognitive development* (NBER Working Paper No. 5257). Cambridge, MA: National Bureau of Economic Research.

Kahn, R. S., Khoury, J., Nichols, W. C., & Lanphear, B. P. (2003). Role of dopamine transporter genotype and maternal prenatal smoking in childhood hyperactive-impulsive, inattentive, and oppositional behaviors. *Journal of Pediatrics, 143,* 104–110.

Kiely, J. L., Brett, K. M., Yu, S., & Rowley, D. L. (1994). Low birth weight and intrauterine growth retardation. In L. S. Wilcox & J. S. Marks (Eds.), *From data to action. CDC's public health surveillance for women, infants, and children* (pp. 185–202). Washington, DC: U.S. Department of Health and Human Services.

Kiernan, K., & Pickett, K. E. (2006). Marital status disparities in maternal smoking during pregnancy, breastfeeding and maternal depression. *Social Science & Medicine, 63,* 335–346.

Korenbrot, C. C., & Moss, N. E. (2000). Preconception, prenatal, perinatal, and postnatal influences on health. In B. D. Smedley & S. L. Syme (Eds.), *Promoting health: Intervention strategies from social and behavioral research* (pp. 125–169). Washington, DC: National Academies Press.

Kramer, M. S., Séguin, L., Lydon, J., & Goulet, L. (2000). Socio-economic disparities in pregnancy outcome: Why do the poor fare so poorly. *Paediatric and Perinatal Epidemiology, 14,* 194–210.

Laitinen, J., Power, C., & Järvelin, M.-R. (2001). Family social class, maternal body mass index, childhood body mass index, and age at menarche as predictors of adult obesity. *American Journal of Clinical Nutrition, 74,* 287–294.

Lambe, M., Hultman, C., Torrång, A., MacCabe, J., & Cnattingius, S. (2006). Maternal smoking during pregnancy and school performance at age 15. *Epidemiology, 17,* 524–530.

Lee, P. A., Chernausek, S. D., Hokken-Koelega, A. C. S., & Czernichow, P. (2003). International small for gestational age advisory board consensus development conference statement: Management of short children born small for gestational age, April 24–October 1, 2001. *Pediatrics, 111,* 1253–1261.

Long, J. S., & Freese, J. (2001). *Regression models for categorical dependent variables using Stata*. College Station, TX: Stata Press.

Lozoff, B., Beard, J., Connor, J., Felt, B., Georgieff, M., & Schallert, T. (2006). Long-lasting neural and behavioral effects of iron deficiency in infancy. *Nutrition Reviews, 64,* S34–S43.

Lundborg, P., & Stenberg, A. (2010). Nature, nurture and socioeconomic policy—What can we learn from molecular genetics? *Economics and Human Biology, 8,* 320–330.

MacArthur, C., Knox, E. G., & Lancashire, R. J. (2001). Effects at age nine of maternal smoking in pregnancy: Experimental and observational findings. *British Journal of Obstetrics and Gynaecology, 108,* 67–73.

Maccari, S., Darnaudery, M., Morley-Fletcher, S., Zuena, A. R., Cinque, C., & Van Reeth, O. (2003). Prenatal stress and long-term consequences: Implications of glucocorticoid hormones. *Neuroscience and Biobehavioral Reviews, 27,* 119–127.

Mäki, P., Riekki, T., Miettunen, J., Isohanni, M., Jones, P. B., Murray, G. K., & Veijola, J. (2010). Schizophrenia in the offspring of antenatally depressed mothers in the northern Finland 1966 birth cohort: Relationship to family history of psychosis. *American Journal of Psychiatry, 167,* 70–77.

Mäki, P., Veijola, J., Räsänen, P., Joukamaa, M., Valonen, P., Jokelainen, J., & Isohanni, M. (2003). Criminality in the offspring of antenatally depressed mothers: A 33-year follow-up of the northern Finland 1966 birth cohort. *Journal of Affective Disorders, 74,* 273–278.

McLaren, L. (2007). Socioeconomic status and obesity. *Epidemiologic Reviews, 29,* 29–48.

Moser, K., Li, L., & Power, C. (2003). Social inequalities in low birth weight in England and Wales: Trends and implications for future population health. *Journal of Epidemiology and Community Health, 57,* 687–691.

Moster, D., Lie, R. T., & Markestad, T. (2008). Long-term medical and social consequences of preterm birth. *New England Journal of Medicine, 359,* 262–273.

Mulder, E. J. H., Robles de Medina, P. G., Huizink, A. C., van den Bergh, B. H., Buitelaar, J. K., & Visser, G. H. A. (2002). Prenatal maternal stress: Effects on pregnancy and the (unborn) child. *Early Human Development, 70,* 3–14.

Nagahawatte, N. T., & Goldenberg, R. L. (2008). Poverty, maternal health, and adverse pregnancy outcomes. *Annals of the New York Academy of Sciences, 1136,* 80–85.
Nowakowski, R. S., & Hayes, N. L. (2008). Radiation, retardation, and the developing brain: Time is the crucial variable. *Acta Pædiatrica, 97,* 527–531.
Olsén, P., Myhrman, A., & Rantakallio, P. (1994). Educational capacity of low birth weight children up to the age of 24. *Early Human Development, 36,* 191–203.
Oreopoulos, P., Stabile, M., Walld, R., & Roos, L. L. (2008). Short-, medium-, and long-term consequences of poor infant health. An analysis using siblings and twins. *Journal of Human Resources, 43,* 88–138.
Orr, S. T., & Miller, C. A. (1995). Maternal depressive symptoms and the risk of poor pregnancy outcome. *Epidemiologic Reviews, 17,* 165–171.
Palloni, A. (2006). Reproducing inequalities: Luck, wallets, and the enduring effect of childhood health. *Demography, 43,* 567–615.
Palloni, A., & Milesi, C. (2006). Economic achievement, inequalities and health disparities: The intervening role of early health status. *Research in Social Stratification and Mobility, 24,* 21–40.
Palloni, A., Milesi, C., White, R. G., & Turner, A. (2008). Triggering inequality and health gradients: Do health selection effects matter? In J. Surkyn, J. van Bavel, & P. Deboosere (Eds.), *Demographic challenges for the 21st century: A state of the art in demography* (pp. 175–209). Brussels, Belgium: Vrije Universiteit Press.
Paneth, N. S. (1995). The problem of low birth weight. *The Future of Children, 5,* 19–34.
Pattenden, S., Dolk, H., & Vrijheld, M. (1999). Inequalities in low birth weight: Parental social class, area deprivation, and "lone mother" status. *Journal of Epidemiology and Community Health, 53,* 355–358.
Pickett, K. E., Luo, Y., & Lauderdale, D. S. (2005). Widening social inequalities in risk for sudden infant death syndrome. *American Journal of Public Health, 95,* 1976–1981.
Pickett, K. E., Wilkinson, R. G., & Wakschlag, L. S. (2009). The psychosocial context of pregnancy smoking and quitting in the Millennium Cohort Study. *Journal of Epidemiology and Community Health, 63,* 474–480.
Pihkala, J., Hakala, T., Voutilainen, P., & Raivio, K. (1989). Uudet suomalaisen sikiön kasvukäyrät [New Finnish fetal growth curves]. *Duodecim, 105,* 1540–1546.
Raatikainen, K., Heiskanen, N., & Heinonen, S. (2005). Marriage still protects pregnancy. *BJOG: An International Journal of Obstetrics and Gynaecology, 112,* 1411–1416.
Raatikainen, K., Läärä, E., Isohanni, M., & Moilanen, I. (1992). Maternal smoking during pregnancy and delinquency of the offspring: An association without causation? *International Journal of Epidemiology, 21,* 1106–1113.
Rantakallio, P. (1969). Groups at risk in low birth weight infants and perinatal mortality. *Acta Paediatrica Scandinavica, 193*(Suppl.), 1–71.
Rantakallio, P. (1978). The effect of maternal smoking on birth weight and the subsequent health of the child. *Early Human Development, 2,* 371–382.
Rantakallio, P. (1988). The longitudinal study of the northern Finland birth cohort of 1966. *Paediatric and Perinatal Epidemiology, 2,* 59–88.
Royston, P. (2004). Multiple imputation of missing values. *The Stata Journal, 4,* 227–241.
Royston, P. (2005). Multiple imputation of missing values: Update. *The Stata Journal, 5,* 1–14.
Rutter, M., & Silberg, J. (2002). Gene-environment interplay in relation to emotional and behavioral disturbance. *Annual Review of Psychology, 53,* 463–490.
Shea, A. K., & Steiner, M. (2008). Cigarette smoking during pregnancy. *Nicotine and Tobacco Research, 10,* 267–278.
Slotkin, T. A. (1998). Fetal nicotine or cocaine exposure: Which one is worse? *Journal of Pharmacology and Experimental Therapeutics, 285,* 931–945.
Smith, J. P. (2009). The impact of childhood health on adult labor market outcomes. *Review of Economics and Statistics, 91,* 478–489.
Stevenson, J., & Fredman, G. (1990). The social environmental correlates of reading ability. *Journal of Child Psychology and Psychiatry, 31,* 681–698.
Szyf, M. (2009). The early life environment and the epigenome. *Biochimica et Biophysica Acta—General Subjects, 1790,* 878–885.
Torche, F. (2011). The effects of maternal stress on birth outcomes: Exploiting a natural experiment. *Demography, 48,* 1473–1491.
Van Buuren, S., Boshuizen, H. C., & Knook, D. L. (1999). Multiple imputation of missing blood pressure covariates in survival analysis. *Statistics in Medicine, 18,* 681–694.
White, R. G., & Palloni, A. (2009). *Child health and the reproduction of inequalities* (Working paper). Madison, WI, and Chicago, IL: University of Wisconsin and Northwestern University.

Winship, C., & Mare, R. D. (1984). Regression models with ordinal variables. *American Sociological Review, 49,* 512–525.

Witlin, A. G., & Sibai, B. M. (1997). Hypertension in pregnancy: Current concepts of preeclampsia. *Annual Review of Medicine, 48,* 115–127.

Wooldridge, J. M. (2002). *Econometric analysis of cross section and panel data*. Cambridge, MA: MIT Press.