IFAU
Institute for Evaluation of Labour
Market and Education Policy

Family, Neighborhood, and Health: Conditions for the Development of Human Capabilities

Evelina Björkegren

DISSERTATION SERIES 2017:4

Presented at the Department of Economics, Uppsala University

The Institute for Evaluation of Labour Market and Education Policy (IFAU) is a research institute under the Swedish Ministry of Employment, situated in Uppsala. IFAU's objective is to promote, support and carry out scientific evaluations. The assignment includes: the effects of labour market and educational policies, studies of the functioning of the labour market and the labour market effects of social insurance policies. IFAU shall also disseminate its results so that they become accessible to different interested parties in Sweden and abroad.

IFAU also provides funding for research projects within its areas of interest. The deadline for applications is October 1 each year. Since the researchers at IFAU are mainly economists, researchers from other disciplines are encouraged to apply for funding.

IFAU is run by a Director-General. The institute has a scientific council, consisting of a chairman, the Director-General and five other members. Among other things, the scientific council proposes a decision for the allocation of research grants. A reference group including representatives for employer organizations and trade unions, as well as the ministries and authorities concerned is also connected to the institute.

Postal address: P O Box 513, 751 20 Uppsala
Visiting address: Kyrkogårdsgatan 6, Uppsala
Phone: +46 18 471 70 70
Fax: +46 18 471 70 71
ifau@ifau.uu.se
www.ifau.se

This doctoral dissertation was defended for the degree of Doctor in Philosophy at the Department of Economics, Uppsala University, September 15, 2017. Essay 3 has been published by IFAU as Working paper 2017:16.

ISSN 1651-4149

Dissertation presented at Uppsala University to be publicly examined in Hörsal 2, Ekonomikum, Kyrkogårdsgatan 10, Uppsala, Friday, 15 September 2017 at 10:15 for the degree of Doctor of Philosophy. The examination will be conducted in English. Faculty examiner: Professor Erik Plug (University of Amsterdam).

Abstract

Björkegren, E. 2017. Family, Neighborhoods, and Health. Conditions for the Development of Human Capabilities. *Economic studies* 169. 230 pp. Uppsala: Department of Economics, Uppsala University. ISBN 978-91-85519-76-7.

Essay 1: We use data from a large sample of adoptees born in Sweden to decompose the intergenerational persistence in health inequality across generations into one pre-birth component, measured by the biological parents' longevity, and one post-birth component, measured by the adopting parents' longevity. We find that most of the health inequality is transmitted via pre-birth factors. In the second part of the paper, we study the background to why children of parents with better educational attainments have better health by decomposing the association into one component attributed to the education of the biological parents and one to the adopting ones. We find that the association can mostly be attributed to the adopting parents, suggesting that parental resources per se, rather than pre-birth (genetic) differences, make up the parental education gradient in child health.

Essay 2: There are large differences in health across neighborhoods in Sweden. To try to answer if there is a causal link between neighborhood conditions in childhood and youth health, I apply two different empirical strategies. First, I use population wide data on families living in different areas in Sweden, and estimate the effects of childhood neighborhood on youth health using data on families that move across the country. Since the choice of moving and where to live is endogenous, I exploit the timing of moves and estimate the effect of siblings' different exposure time to neighborhoods. The second approach utilizes a governmental policy that assigned refugees to their initial neighborhood in Sweden, potentially offering exogenous variation in neighborhoods and allowing me to study the effect of different neighborhoods on youth health. The findings from the two strategies together imply that there are significant neighborhood effects on youth health, but that the effects are contemporaneous and there is no evidence of exposure time effects.

Essay 3: Previous research has shown that birth order affects outcomes such as educational achievements, IQ and earnings. The mechanisms behind these effects are still largely unknown. We examine birth order effects on health, and whether health at young age could be a transmission channel for birth order effects observed later in life. Our results show that firstborn children have worse health at birth. This disadvantage is reversed in early age and later-born siblings are more likely to be hospitalized for injuries and avoidable conditions. In adolescence and as young adults, younger siblings are more likely to be of poor mental health and to be admitted to hospital for alcohol induced health conditions. We also test for reverse causality by estimating fertility responses to the health of existing children. Overall our results suggest that birth order effects are due to differential parental investment because parents' time and resources are limited.

Essay 4: We study the short-, medium- and long-term consequences of health at birth using administrative data from Sweden for individuals born in the years 1973-1979. We contribute to a better understanding of the consequences of early life health by contrasting the effects of birth weight with two other measures of neonatal health: the length and the head circumference of the newborn. Our findings suggest that the use of birth weight alone might lead to an underestimation of the importance of early health. Furthermore, we find that there is a persistent effect of neonatal health on a variety of human capital measures in adolescence and adulthood.

Keywords: Health, Inequality, Mortality, Intergenerational Mobility, Birth-order, Neighborhoods, Birth, Childhood, Youth, Capabilities, Education

Evelina Björkegren, Department of Economics, Box 513, Uppsala University, SE-75120 Uppsala, Sweden.

ISSN 0283-7668
ISBN 978-91-85519-76-7
urn:nbn:se:uu:diva-323695 (http://urn.kb.se/resolve?urn=urn:nbn:se:uu:diva-323695)

Acknowledgements

First of all I wish to thank my supervisors for all the guidance over these years. Helena Svaleryd, thank you for all your support throughout this process. You have shared all my ups and downs, and been the best sounding board. I very much appreciate your honesty as a supervisor and as a researcher. You are a great role model. Hans Grönqvist, thank you for carefully reading all my drafts over and over again, and thank you for your encouragement, it has been crucial these last years. Anders Forslund, thank you for being so kind, supporting the best hockey team, and always willing to discuss interesting topics.

I also want to thank Mårten Palme for being like an informal supervisor to me. I have not only got to learn about health related research but also at lot about academia in general from working together. I am also grateful for having the opportunity to work with and learn from several other experienced researchers, Mikael Lindahl, Emilia Simeonova, Gabriella Conti, Kjell Salvanes, Aline Bütikhofer, and Helena Svaleryd. Also, a special thanks to Richard Blundell for inviting me to stay an inspiring semester at UCL.

My journey to a doctoral degree in Economics was not an obvious one. I started studying Economics without really knowing what kind of subject it was. It was not until I started the master program at Stockholm University and met Nanna, Jenny and Felicia, that I learnt what Economics can be. Thank you for being so inspiring and leading the way. I do not think I would have even thought of pursuing a PhD degree if I have not met you.

I also want to thank IFAU and Anna Sjögren, Eva Mörk, and Helena Svaleryd for having me as a research assistant several years ago. That first experience with Uppsala was decisive for me to send my application to the PhD program in Uppsala a year later.

There are many nice people at the department who have been important to me during the PhD program. First of all, my cohort: Kicki, Sebastian, Jacob, Olof, Gunnar, Josefine, and Daniel. In particular, I want to thank Sebastian for taking care of me the first year when I was lost in Uppsala. And Kicki, you are the most caring person I have ever met and I am lucky that you wanted to share office so I got to know you so well. I also want to thank my fellow PhD students and junior faculty for all the lunches, coffees and inspiring discussions along the way.

There are many other non-economist friends that have been important to me during these years and that have supported me. Thank you for keeping

my mind off my thesis from time to time, reminding me of other important things in life.

Last but not the least I want to thank my family. My parents, Sarah and Curt, you never planned for me to follow an academic track, but you gave me all the tools. Thank you for supporting me and being the best parents one could wish for.

Daniel, my partner in life, and now also my husband, I could not have done this without you. You have kept me on the right track these years, supporting me all the way, and every day reminding me what is important in life.

Stockholm, June 2017
Evelina Björkegren

Contents

Introduction

This thesis consists of four self-contained essays. The four essays all have two common themes which are health and equality of opportunity. Economists are interested in health mainly for two reasons; first health is a key part of our human capital (production input) and secondly health is important in itself as a central measure of wellbeing. From a human capital perspective, early life health is an important predictor for outcomes later in life such as educational attainment, labor market outcomes and adult health (e.g. Currie et al., 2010, and Case et al., 2005). Health is in both cases strongly related to people's capabilities and lifetime opportunities. Or as Angus Deaton writes in his book *The Great Escape (2013)* "Health is the obvious starting point for an enquiry into wellbeing. You need a life to have a *good* life…" (p.24).

The focus of this thesis is primarily on the development of human capabilities in early life. Previous research is pointing towards the importance of early childhood environment for the development of human capabilities (e.g. Currie and Almond, 2011; Cunha and Heckman, 2007). Heckman (2007) summarizes the evidence on the effects of early childhood conditions, and provides a framework for analyzing the origins of human inequality from a developmental perspective. At age *t*, human capability production can be written:

$$\theta_{t+1} = f_t(h, \theta_t, I_t), \quad \text{t=1,2,…,T,}$$

where θ is human capabilities (e.g. health, cognitive, non-cognitive skills), h is parental capabilities (e.g. genes, IQ, education) that are affected by their own parents' investments and genes, and I_t is parental investment in child capabilities. Substituting θ_t repeatedly, the stock of capabilities can be rewritten as a function of all past investments:

$$\theta_{t+1} = m(h, \theta_1, I_1, \dots, I_t),$$

where θ_1 is the genetic and environmental initial conditions received at conception. This model captures some important features of how human capabilities are produced and why investments in early childhood, and even during the fetal period, is key for understanding human inequalities.

In all four essays I utilize Swedish register data to study research questions that concerns equality of opportunity related to early childhood investments. The first essay studies the intergenerational transmission of health and mortality using data on Swedish adoptees to decompose pre-birth and

post-birth family influences. The second essay studies the role of childhood neighborhood conditions for youth health. The third essay concerns the inequality in health within families, across birth order. And the final essay studies the consequences of neonatal health, reflecting the fetal environment.

Intergenerational transmission

The dynamic process suggested by Heckman (2007) involves both the individual's genetic background and parental resources in the formation of health. There are several channels through which parental endowments and investments may affect the health outcomes of their children. Genes inherited from previous generations affect health, but parental investments in their children's health may also have long-term effects. Although such processes will inevitably lead to intergenerational persistence in health inequality, little is known about the intergenerational transmission in the population and about the relative contributions of these two channels. This is the question that Mikael Lindahl, Mårten Palme, Emilia Simeonova, and I study in the first essay.

Previous epidemiological research on mortality using data on Danish adoptees has shown a significant association between biological parents and adopted children, but none, or a weak, association with the adopting parents' mortality (see e.g. Petersen et al., 2005, 2008 and Sørensen et al., 1988). In the economics literature, studies have found that there is a genetic transmission of 20-30 percent for chronic health conditions such as asthma, severe headaches, diabetes and hay fever (Thompson, 2014), and that the adopting family influences health behavior such as drinking and smoking (Sacerdote, 2007) but little evidence is found for a nurture effect on BMI and obesity (Sacerdote, 2007, and Classen and Thompson, 2016).

There is a well-documented relationship between parental educational attainment and child health (see e.g. Case et al., 2002), and a strong intergenerational persistence in educational attainment (e.g. Björklund and Salvanes, 2011; Solon, 1999). This gives rise to the question of the origins of health inequality. Using data on Swedish adoptees, we extend the previous epidemiological literature by specifically studying the role of parental resources measured by educational attainment for long-term health outcomes. We study how both longevity and educational attainments of the biological parents – related to genetic factors and in-utero health – and the corresponding characteristics of the adopting parents – related to health formation and family circumstance during childhood and adolescence – affect the child's health and mortality later in life. We follow the methodology suggested by Björklund et al. (2006), and in their analysis applied to the intergenerational transmission of education and earnings.

Our decomposition results show that the intergenerational association in mortality can be fully attributed to pre-birth factors, because the association

between the life expectancy of the biological parents of the children given up for adoption is as strong as for the children raised by their biological parents. There is no significant association between the longevity of the adopting parents and the mortality risk of the adopted children. Analysis on the association between parental education and child health, show a significant positive effect of the adopting parents' educational attainment on child longevity. We find no such correlation between the biological parents' education and adopted children's mortality, suggesting that parental resources per se, rather than pre-birth (genetic) differences, make up the parental education gradient in child health.

Neighborhoods

The second essay concerns the health inequality across neighborhoods. In Sweden, life expectancy differs by approximately 4 years between areas with the highest and lowest longevity (Statistics Sweden, 2016). There are several reasons why neighborhoods might influence the accumulation of health capital. The seminal work by Jencks and Mayer (1990) identifies four potentially important mechanisms: Peer effects, neighborhood role models, monitoring, and community resources. There is a documented a correlation between places and children's life chances (e.g. Jencks and Mayer, 1990; Brooks-Gunn et al., 1993, and Haveman and Wolfe, 1995) and some evidence showing that neighborhoods are related to child and adolescent health (for reviews, see Leventhal and Brooks-Gunn, 2000, and Sampson et al., 2002).

On average, residents living in poor areas have worse health than residents in more affluent areas. This relationship might not be causal since it is likely that there are factors that impact both families' residential location and children's health, such as family background. In other words, we cannot make any causal claims regarding neighborhood effects by simply comparing children growing up in different areas. Previous experimental research has utilized housing mobility programs in the U.S, primarily the Moving to Opportunity (MTO) program, to study neighborhood effects on child health (e.g. Katz et al., 2001; Kling et al., 2007; and Ludwig et al, 2013). The findings suggest positive effects on female youth's physical and mental health, while the results for males generally show that they did not benefit from moving.

This essay utilizes two different methods to try to handle the problem of selection. The first which uses families that move across areas in Sweden, confirms the association between neighborhoods and health found in previous studies. To estimate causal effects of neighborhoods, I estimate neighborhood exposure time effect between siblings (Chetty and Hendren, 2016). However, no statistically significant effects are found for exposure time to neighborhoods using variation between siblings in time spent in neighborhoods during childhood. To investigate if this result arises because there are

no causal effects of neighborhoods on health, or because neighborhoods affect health instantly through contemporaneous environmental effects rather than through exposure time, I make use of a Swedish governmental policy that placed refugee families in their initial neighborhood. The results from the second empirical strategy confirm the findings in the first part of the paper. Together the results from the two parts imply that there are causal neighborhood effects on youth health, but these effects are instant and do not work through neighborhood exposure time.

Birth order

The third essay is related to inequalities within families. A vast number of studies in various research disciplines have shown that younger siblings have lower educational achievements, IQ and earnings than their older siblings (e.g. Behman and Taubman, 1986; Black et al., 2005; Barclay 2015; and Black et al., 2015). The mechanisms behind these effects are still debated and previous empirical research has struggled to identify the channels. In this third essay Helena Svaleryd and I study how health differences across birth order develops through childhood and, by studying different sorts of health conditions, we try shed some light on the mechanisms giving rise to the negative birth order effect on later life outcomes.

Several hypotheses about the mechanisms through which the birth order effect works have been suggested, including the resource dilution hypothesis (Blake, 1989), strategic parental behavior (Hotz and Pantano, 2015), sibling influences (Zajonc, 1976) and birth endowments. We find that firstborns are disadvantaged at birth. Firstborn children are more likely to be hospitalized for perinatal conditions and congenital malformations in early childhood. We also find that lower birth order children are more likely to die during infancy.

The disadvantage of older siblings is, however, reversed as the child grows older. The causes for hospitalization suggest that later-born siblings are involved in more risky behavior and have a less healthy life style during adolescence. In particular, later-born siblings are more likely to be admitted to hospital for diagnoses related to poor mental health, alcohol consumption, self-harm and injuries. Our results support the hypothesis that birth order effects are due to lower investment in children with a higher birth order. This is in line with the dilution hypothesis presented in Blake (1989) and the finding in Price (2008) that parents spend more time with earlier-born than later-born siblings.

In this essay we also test for reverse causality by estimating fertility responses to the health of existing children. We conclude that the effects on health are not severely biased; however, the large negative birth order effects on infant mortality are partly due to endogenous fertility responses. Parents'

endogenous fertility response to the health and death of previous children lends further support to the hypothesis that parents are resource constrained.

Fetal environment

The forth essay concerns inequality in the very early period, studying the fetal environment. The importance of newborn health for a variety of outcomes throughout the life cycle has been documented in a vast, interdisciplinary literature (see, e.g. Almond et al., 2017, for the most recent review). The main measure of neonatal health used in this literature is birth weight, which has been shown to be associated in a meaningful way with a variety of outcomes ranging from health to education and wages (see e.g. Almond et al., 2005; Black et al., 2007; Figlio et al., 2014).

Birth weight is relatively easy to measure, widely available in several data sources, and contains little measurement error. However, it mainly captures the uterine environment in the last weeks of gestation, at the time when the fetus gains most of his weight. One active area of research in the fetal origins field focuses on searching for more sensitive and predictive measures of health at birth (Torche and Conley, 2016). Differently from birth weight, birth length and head circumference are longer-term cumulative indicators, reflecting the fetal environment since an earlier period, given that the process of formation of bones and neural synapses starts earlier in gestation. Literature in medicine and epidemiology has documented how birth length and head circumference are differentially associated with prenatal investments, such as smoking, alcohol use, and nutritional supplementation (see e.g. Lindley et al., 2000; Ramakrishnan et al., 2010; Shankaran et al., 2004).

In essay four, Aline Bütikofer, Gabriella Conti, Mårten Palme, Kjell Salvanes and I contrast the effects of birth weight with the length and the head circumference of the newborn to gain a better understanding of early life health. We use administrative data for Sweden on a sample of births between 1973 and 1979 to investigate the short, medium and long-term consequences of neonatal health. We employ a decomposition technique recently proposed by Gelbach (2016), which allows us to shed light on the mechanisms through which birth weight impacts later outcomes. Furthermore, by using information on head circumference at birth to distinguish between different types of growth-restricted newborns, we are able to show the relative importance for health and cognitive outcomes of insults differentially affecting the brain. Overall, the findings in the fourth essay emphasize the importance of not focusing exclusively on birth weight when studying neonatal health.

Concluding remarks

This thesis shows that pre-birth factors affect adult health outcomes. Related to the Heckman model introduced in the first section, the first essay shows that the genetic and initial environment θ_1, is important for long-term health outcomes. That the intrauterine environment and investments before birth have a long-term impact is further supported by the analysis in essay four, showing that different measures of health at birth are strong predictors of later outcomes later in life, even within twin pairs. This thesis also shows that investments and upbringing environments in later periods are central for the development of human capabilities. The first essay finds that the adopting mother's educational attainment is a strong predictor of longevity, and the results in the second essay indicate that neighborhoods have a causal effect on youth health. The third essay shows that even within families, there are large differences in health outcomes depending on the order in which the siblings were born and results indicate that these differences might be related to differential parental investments. The findings in this thesis combined show that there is scope for designing policies that enhance children's equality of opportunity.

References

Almond, D., Chay, K. Y., & Lee, D. S. (2005). The costs of low birth weight. The Quarterly Journal of Economics, 120(3), 1031-1083.

Almond, D., Currie, J., & Duque, V. (2017). Childhood Circumstances and Adult Outcomes: Act II (No. w23017). National Bureau of Economic Research.

Barclay, K. J., (2015) "A within-family analysis of birth order and intelligence using population conscription data on Swedish men." *Intelligence*, 49, 134–143.

Behrman, J. R. and P. Taubman (1986) "Birth Order, Schooling, and Earning" *Journal of Labor Economics* 4(3), part 2, 121–145.

Björklund, A., M. Lindahl, and E. Plug (2006) "The Origins of Intergenerational Associations: Lessons from Swedish adoption data" *The Quarterly Journal of Economics*, 999-1028.

Björklund, A., & Salvanes, K. G. (2011). Education and family background: Mechanisms and policies. *Handbook of the Economics of Education*, *3*(3), 201-247.

Black, S. E., Devereux, P. J. and Salvanes, K. G. (2005) "The More the Merrier? The Effects of Family Size and Birth Order on Children's Education", *Quarterly Journal of Economics* 120(2), 669–700.

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. The Quarterly Journal of Economics, 122(1), 409-439.

Black, S. E., P. J. Devereux, and K. G. Salvanes (2015) "Healthy(?), Wealthy and Wise: Birth Order and Adult Health". NBER Working Paper Series 21337.

Blake, Judith (1989) "Family Size and Achievement", Berkeley and Los Angeles, CA: University of California Press.

Brooks-Gunn, J., Duncan, G. J., Klebanov, P. K., & Sealand, N. (1993). Do neighborhoods influence child and adolescent development?. *American journal of sociology*, *99*(2), 353-395.

Case, A., Fertig, A. and Paxson, C. (2005) "The Lasting Impact of Childhood Health and Circumstance", *Journal of Health Economics* 24, 365–389.

Case, A., Lubotsky, D., & Paxson, C. (2002). Economic status and health in childhood: The origins of the gradient. *The American Economic Review*, *92*(5), 1308-1334.

Chetty, R., & Hendren, N. (2016). *The impacts of neighborhoods on intergenerational mobility i: Childhood exposure effects* (No. w23001). National Bureau of Economic Research.

Classen, T. J. and O. Thompson (2016) "Genes and the intergenerational transmission of BMI and obesity" *Economics & Human Biology 23*, 121-133.

Cunha, F., & Heckman, J. (2007). The Technology of Skill Formation. *The American Economic Review*, *97*(2), 31.

Currie, J., & Almond, D. (2011). Human capital development before age five. *Handbook of labor economics*, *4*, 1315-1486.

Currie, J., Stabile, M., Manivong, P. and Roos L. L. (2010) "Child Health and Young Adult Outcomes", *Journal of Human Resources* 45(3), 517–548.

Deaton, A. (2013). *The great escape: health, wealth, and the origins of inequality*. Princeton University Press.

Figlio, D., Guryan, J., Karbownik, K., & Roth, J. (2014). The effects of poor neonatal health on children's cognitive development. The American Economic Review, 104(12), 3921-3955.

Gelbach, J. B. (2016). When do covariates matter? And which ones, and how much?. *Journal of Labor Economics*, *34*(2), 509-543.

Haveman, R., & Wolfe, B. (1995). The determinants of children's attainments: A review of methods and findings. *Journal of economic literature*, *33*(4), 1829-1878.

Heckman, J. J. (2007) "The economics, technology, and neuroscience of human capability formation." *Proceedings of the national Academy of Sciences* 104.33: 13250-13255.

Hotz, V. J. and J. Pantano (2015) "Strategic Parenting, Birth Order, and School Performance" *Journal of Population Economics* 28(4): 911–936.

Jencks, C., & Mayer, S. E. (1990). The social consequences of growing up in a poor neighborhood. *Inner-city poverty in the United States*, *111*, 186.

Katz, L. F., Kling, J. R., & Liebman, J. B. (2001). Moving to opportunity in Boston: Early results of a randomized mobility experiment. *The Quarterly Journal of Economics*, *116*(2), 607-654.

Kling, J. R., Liebman, J. B., & Katz, L. F. (2007). Experimental analysis of neighborhood effects. *Econometrica*, *75*(1), 83-119.

Leventhal, T., & Brooks-Gunn, J. (2000). The neighborhoods they live in: the effects of neighborhood residence on child and adolescent outcomes. *Psychological bulletin*, *126*(2), 309-337.

Lindley, A. A., Becker, S., Gray, R. H., & Herman, A. A. (2000). Effect of continuing or stopping smoking during pregnancy on infant birth weight, crown-heel length, head circumference, ponderal index, and brain: body weight ratio. American Journal of Epidemiology, 152(3), 219-225.

Ludwig, J., Duncan, G. J., Gennetian, L. A., Katz, L. F., Kessler, R. C., Kling, J. R., & Sanbonmatsu, L. (2013). Long-term neighborhood effects on low-income families: Evidence from Moving to Opportunity. *The American Economic Review*, *103*(3), 226-231.

Petersen, L., P. Kragh Andersen and T. Sørensen (2005) "Premature Death of Adult Adoptees: Analyses of a Case-Cohort Sample" *Genetic Epidemiology* 28, 376-382

Petersen, L., P. Kragh Andersen and T. Sørensen (2008) "Genetic and Environmental Effects on Mortality before Age 70 Years" *Epidemiology* 19(3), 472-476.

Price, J. (2008) "Parent-Child Quality Time – Does Birth Order Matter?" *Journal of Human Resources* 43(1), 240–265.

Ramakrishnan, U., Stein, A. D., Parra-Cabrera, S., Wang, M., Imhoff-Kunsch, B., Juárez-Márquez, S., Rivera J. & Martorell, R. (2010). Effects of docosahexaenoic acid supplementation during pregnancy on gestational age and size at birth: randomized, double-blind, placebo-controlled trial in Mexico. Food and nutrition bulletin, 31(2_suppl2), S108-S116.

Sacerdote, B. (2007) "How large are the effects from changes in family environment? A study of Korean American adoptees." *The Quarterly Journal of Economics* 122(1), 119-157

Sampson, R. J., Morenoff, J. D., & Gannon-Rowley, T. (2002). Assessing "neighborhood effects": Social processes and new directions in research. *Annual review of sociology*, *28*(1), 443-478.

Shankaran, S., Das, A., Bauer, C. R., Bada, H. S., Lester, B., Wright, L. L., & Smeriglio, V. (2004). Association between patterns of maternal substance use and infant birth weight, length, and head circumference. Pediatrics, 114(2), e226-e234.

Solon, G. (1999). Intergenerational mobility in the labor market. Handbook of labor economics, 3, 1761-1800.

Sørensen, T., G. Nielsen, P. Kragh Andersen and T. Teasdale (1988) "Genetic and Environmental Influences on Premature Death in Adult Adoptees" *New England Journal of Medicine* 318(12), 727-732.

Statistics Sweden (2016). Life expectancy in Sweden 2011-2015. Demographic reports 2016:4. Örebro: Statistics Sweden

Thompson, O. (2014) "Genetic mechanisms in the intergenerational transmission of health" *Journal of Health Economics* 35, 132-146.

Torche, F., & Conley, D. (2016). A Pound of Flesh. In The Oxford Handbook of Economics and Human Biology.

Zajonc, R. B. (1976) "Family configuration and intelligence: Variations in scholastic aptitude scores parallel trends in family size and the spacing of children", *Science* 192(4236), 227–236.

I. Parental Influences on Health and Longevity: Lessons from a Large Sample of Adoptees

With Mikael Lindahl, Mårten Palme and Emilia Simeonova

1. Introduction

Health inequality – defined as differences between individual health outcomes or differences in health between socioeconomic status (SES) or demographic groups – has recently attracted a renewed interest (Case and Deaton, 2015, or Chetty et al., 2016). It is well understood that the formation of health takes place over a long period of time. Economic models, as in Heckman (2007) and Cunha and Heckman (2007), suggest a dynamic process involving both the individual's genetic background and parental resources. Although such processes will inevitably lead to intergenerational persistence in health inequality, surprisingly little is known about the relative contributions of these two channels. To what extent can health inequality be attributed to genetic differences transmitted between generations - a process arguably outside the purview of the social sciences - relative to differences in parental resources, which can be more directly affected by policy interventions?

In this paper, we use a large sample of Swedish adoptees for which we observe longevity as well as the educational attainment of both biological and adopting parents. We study how the longevity and educational attainments of the biological parents – related to genetic factors and in-utero health – and the corresponding characteristics of the adopting parents – related to health formation and family circumstance during childhood and adolescence – affect the child's health and mortality later in life.

Our dataset is constructed by matching several different administrative registers containing information on health outcomes and educational attainments for biological and adopting parents and their children. We include all adopted children born between 1940 and 1967 in Sweden, in total about 21,000 individuals. For comparison, we also present results on the same outcomes obtained using the population of about 2.8 million children raised with their biological parents and born in the same time-period as the adoptees.

The main outcome of interest is the health status of the children as adults. We use three main measures of this outcome: (i) mortality until April 1 2013; (ii) health indices based on hospitalization data from the Swedish inpatient register; (iii) for females in the sample, birth outcomes of their first-born child obtained from the Swedish birth register. The third measure is motivated by the fact that birth outcomes possibly reflect the health status of the mother giving birth (see e.g. Currie, 2011). Perhaps even more importantly, it allows us to gauge the persistence of health transmission over three generations.

The empirical analysis in this paper is divided into two main parts. In the first part, we study to what extent the intergenerational transmission of health and longevity can be attributed to factors determined before birth – which we measure by the biological parents' longevity – relative to post-birth factors – measured by the adopting parents' longevity. We follow the

methodology suggested by Björklund et al. (2006), and in their analysis applied to the intergenerational transmission of education and earnings.

This part of the empirical analysis relates to individual health inequality in a similar way to how studies on intergenerational earnings mobility relate to our understanding of income inequality. To what extent can we expect the observed inequality to persist across generations? In addition to this, our decomposition analysis sheds light on the question to what extent the observed persistence can be attributed to pre-birth differences and to what extent it reflects differences in living conditions and life habits, formed to a large extent during childhood and adolescence.

Our decomposition results show that the intergenerational association in mortality can be fully attributed to pre-birth factors, because the association between the life expectancy of the biological parents of the children given up for adoption is as strong as for the children raised by their biological parents. There is no significant association between the longevity of the adopting parents and the mortality risk of the adopted children. The decompositions obtained using health measures based on hospitalization data also give more weight to pre-birth influences; the share varies between ⅔ and ¾ depending on the measure and sample used.

In the second part of the paper, we study to what extent the well-documented association between parental educational attainment and child long-term health can be attributed to pre- as opposed to post-birth factors (see e.g. Case et al., 2002). Since there is also a strong intergenerational persistence in educational attainment, this research question is related to the literature on the origins of the health inequality between different SES or education groups, commonly referred to as the education gradient in health (see e.g. Cutler and Lleras-Muney, 2006).

We find a significant positive effect of the adopting parents' educational attainment on child longevity. We find no such correlation between the biological parents' education and adopted children's mortality. We find the result to be robust in a series of sensitivity checks, including an application of the Altonji-Elder-Taber method. When we look at other health outcome measures the significant association between the educational attainments of the adopting parents and child health remains. However, these outcome measures give a more even split between pre-birth and post-birth influences.

Although we do not study the effects of a policy initiative, our results have obvious policy relevance. Notably, we are able to reject the hypothesis that parental resources as measured by educational attainment are not associated with long-term health and longevity of the child. Our results suggest that being adopted and growing up with a mother with college education (15 years of schooling) rather than one with just compulsory schooling (7 years) increases the life expectancy of the child by 2.7 years. This association cannot be interpreted as a causal effect of parental educational attainment per se, since it may be attributed to unobserved parental characteristics correlated

with educational attainment. However, in our setup, it is unrelated to pre-birth factors and such a strong effect is likely to be an important mechanism behind the education gradient in mortality.

The rest of the paper is organized as follows. Section 2 describes how our study is related to the existing literature and outlines the conceptual framework for identifying pre- and post-birth factors in the association between parental education and child long-term health. Section 3 presents the conceptual framework for our econometric models. Section 4 presents the data and descriptive statistics. The main results as well as sensitivity analyses are laid out in Section 5. Section 6 concludes the paper. Finally, the paper contains three Appendices. Appendix A provides a brief historical background and a description of institutions related to the adoption process in Sweden. Appendix B contains results from predictions of life expectancies for the parental generation. Appendix C shows results from various sensitivity analyses.

2. Background and Related Literature

There are several channels through which parental endowments and investments may affect the health outcomes of their children. Genes inherited from previous generations affect health, but parental investments in their children's health may also have long-term effects. Parents can choose to compensate or amplify individual differences among their children. Additive "nature" and "nurture" models are therefore obviously over-simplified, since they ignore such possible interactions between environments and genes and rule out potential epigenetic influences.

Heckman (2007) provides the following framework for analyzing human development from childhood to adulthood:

$$\theta_{t+1} = f_t(h, \theta_t, I_t), \tag{1}$$

where t is an index for time period (t = 1, 2….T), θ represents individual capabilities – such as health, cognitive and non-cognitive skills – at any given time t; h represents pre-birth factors including characteristics of the biological parents inherited by the child (including genes relevant for the development of child capabilities) and I represents investments to promote the development of individual capabilities. These could include very different types of resources, such as formal schooling, vocational training, healthcare or preventive care, and could be provided by the individual's parents in younger ages, the individual him- or herself at an older age or by the public sector. Substituting investments in capabilities into θ_t, θ_{t-1}…, we get

$$\theta_{t+1} = f_t(h, \theta_1, I_1, I_2 \ldots\ldots I_t). \tag{2}$$

Two important cases can be defined in this framework. First, "dynamic complementarity" arises if the stock of child capabilities in period *t-1* amplifies the investments in period *t*, i.e. $\frac{\partial^2 f_t}{\partial \theta_t \partial I_t} > 0$. This would occur if, for example, healthier children have an advantage in obtaining education. Note that we also might think of a form of "dynamic complementarity" due to the stock of pre-birth factors (captured by *h*) leading to higher investments in later periods. Second, "self-productivity" occurs when higher capabilities in one period create higher capabilities in the next period, i.e. $\frac{\partial f_t}{\partial \theta_t} > 0$. An example of such a process would be if educational investments made early on, as suggested by several empirical studies (see e.g. Cutler and Lleras-Muney, 2006, for a review), lead to subsequent investments in health at older ages, ultimately generating diverging health endowments in the population of individuals over time.

This framework gives us at least three insights for how to analyze our data on adoptees.

1. A unique feature of our data is that we are able to separately observe characteristics of the biological parents (*h*) from characteristics of the adopting parents, related to their ability to invest in forming the capabilities of their children (*I*). Potentially, this enables us to separately assess the effect of pre-birth factors from those related to parental investments. However, it requires that we are able to find good enough proxies for these two latent variables.
2. "Dynamic complementarity" and "self-productivity" stress the importance of considering the effect of parental longevity and educational attainments separately to avoid the inclusion of endogenous independent variables in the econometric model. For example, if health status, acquired in part from genetic advantages, affects the parents' educational attainments, this can cause an endogeneity problem.
3. The fact that we are able to observe the educational attainments of the adopted children allows us to examine the underlying mechanisms for the relation between parental education and child health as a form of "dynamic complementarity". Parental investments can both have a direct effect on child health or an indirect effect through the child's own educational attainments, which may in turn have an effect on the child's health status later in life.

The strand of the previous literature closest to our paper is a series of epidemiological studies obtained from data on Danish adoptees (see e.g. Petersen et al., 2005; Petersen et al., 2008; Sørensen et al., 1988). The main research question in this literature is to separate out genetic from environmental factors affecting mortality. The key result is a significant association between the health outcomes of biological parents and adopted children, but none, or a very weak, association with the adopting parents' health.

Reflecting the difference in research focus between medicine and economics, the questions posed in this paper are somewhat different. In addition to health, we study the effect of parental resources, as measured by their educational attainments. We also have a wider set of health outcomes measured throughout the life of the adopted children. This supplementary analysis is important since in the prior epidemiological research on adoptions, as in our study, only a low fraction in the child generation has died at the end of the sample window. Furthermore, we also compare our estimates for adoptees to those obtained on the population of the majority of children raised by their biological parents.[1]

The two papers in the economics literature closest to ours are Sacerdote (2007) and Thompson (2014). Sacerdote (2007) uses data on 1,650 Korean American adoptees placed by the Holt International Children's Services during 1964-1985. Data on a number of health-related and socio-economic outcomes for these children were obtained through a questionnaire collected in 2004-2005. In the part of the empirical analysis that is most similar to ours, Sacerdote estimates the effect of being placed in a highly educated, small family relative to a large family with low education, on education, health-related behavior (such as smoking and drinking), body-mass index (BMI) and the prevalence of asthma among adoptees. The main result is a strong effect on "social" outcomes – such as educational attainments, smoking and alcohol consumption – but no significant effects on the biological outcomes – such as overweight, BMI and prevalence of asthma.

Thompson (2014) uses data from the National Health Interview Survey (NHIS) to study the intergenerational correlation in health conditions for asthma, hay fever, diabetes and chronic headaches. Thompson finds a significant association in the prevalence of medical diagnoses between adopting parents and their adoptive children. Furthermore, he finds that this correlation appears to become stronger as children age. This suggests that examining children's long-term health outcomes could yield a more accurate assessment of the true underlying processes. In another paper, Classen and Thompson (2016) use the same data set and perform a similar analysis on BMI and obesity measures. For these outcomes, they find no association between adoptees and their adoptive parents.

Our study differs from Sacerdote (2007) and the two studies by Thompson (2014) and Classen and Thompson (2016) along several important dimensions. The most important one is that our data include information on the biological parents of the adoptees, which enables us to decompose the pre-

[1] A separate related branch of research examines genetic influences on longevity using samples of twins (see e.g. Herskind et al., 1996, or Hjelmborg et al., 2006). For a discussion about the advantages and disadvantages of the twins- and adoption approaches to inferring "nature" and "nurture" effects, with a focus on economic and social outcomes, see Sacerdote (2011).

and post-birth parental influences on child health.[2] Since we have a much longer follow-up period, we are able to study long-run health outcomes, rather than self-reported health related behavior measures. Finally, our sample size is much larger, potentially allowing us to identify smaller effects due to improved statistical power. Moreover, Thompson (2014) and Classen and Thompson (2016) do not investigate the role of adoptive parents' education.

In addition to the literatures reviewed above and the vast literature that has examined the association between parental education and infant or child health (see Currie, 2008, and Currie and Almond, 2011, for recent reviews), our paper relates to at least four additional strands of the previous literature. First, a number of studies have utilized exogenous changes in parental education to estimate causal effects on child health and have reached different conclusions (Currie and Moretti, 2002; McCrary and Royer, 2011; Lindeboom et al., 2009; Lundborg et al., 2014).[3] Although they are genetically unrelated to their adopted children, the educational attainment of the adopting parents can still be correlated with other parental characteristics that are important in the formation of their children's health. This prevents us from interpreting the association between adopting parents' educational attainments and child health as a causal effect. While the study is clearly related to this literature, our goal is merely to decompose the influences of pre- and post-birth factors in the formation of child health.

Second, our findings relate to the literature on intergenerational mobility in general (see e.g. Solon, 1999, and Black and Devereux, 2011, for overviews) and, in particular, the quite small literature on intergenerational persistence in health outcomes.[4]

Third, because we also study the association between grandparents' longevity and education and grandchildren's birth weight for the adoptive as well as the biological children's samples, this study adds to the research on the intergenerational transmission of birth weight. Currie and Moretti (2007) have demonstrated a strong correlation between maternal health and the birth

[2] Sacerdote (2007) has information on around 100 biological parents. This information is not used in the main analysis.

[3] There is also a literature that estimates the causal effect of other parental resource variables on the health of the next generation. For instance, Cesarini et al. (2016) find no impact on health of the next generation from exogenous positive wealth shocks for the parents through winning large sums on lotteries. However, Akee et al. (2012) and Akee et al. (2016) find positive effects of exogenously increasing parental income on child BMI and child behavioral and emotional health.

[4] Bhalotra and Rawlings (2013) use microdata for 38 developing countries and find that the child's birth weight is negatively related to the mother's health (measured as height) and that this relationship is more negative the more adverse is the social environment. Classen (2010) finds a strong positive association in the Body Mass Index between mothers and children in the US. Johnston et al. (2013) find evidence of intergenerational persistence in mental health across two and three generations using data from the U.K. Trannoy et al. (2010) find parents' longevity and SES to be positively associated with the self-reported health status of the next generation using data for France.

weight of her children, but they also show that family income at the time of the mother's birth is another important factor affecting the transmission mechanisms.

Fourth, because we also study outcomes of the new-born children of the adopted mothers, this study also relates to the literature on multigenerational effects, where several new studies have found grandparents' characteristics to be significant predictors of grandchildren's outcomes (such as education and income), even conditional on parents' outcomes (see Solon, 2015, for a survey). However, none of these studies used outcomes for adoptees in the middle generation, something that would purge the three generational associations from the genetic link between parents and grandparents.[5]

Finally, this study also relates to the growing literature using data on adoptees to study intergenerational persistence in various other outcomes than health and longevity (see e.g. Björklund et al., 2006; Black et al., 2015a; Black et al., 2015b; Hjalmarsson and Lindquist, 2013; Lindquist et al., 2015; Cesarini et al, 2014).

3. Empirical Specifications

We first estimate the following intergenerational model on the population of individuals

$$H_j^{bc} = \beta_0 + \beta_1 Y_j^{bp} + \upsilon_j^{bc}, \tag{3}$$

where H_j^{bc} represents adult health status for the biological child and Y_j^{bp} the biological parents' health status or educational attainment. Subscript *j* indexes the family in which the child is born and raised and superscripts *bc* and *bp* denote the biological child and parent, respectively; υ_j^{bc} is the child-specific error term assumed to be uncorrelated with Y_j^{bp}. The coefficient β_1 measures the strength of the association between adult health of the child and the health or human capital measures of the parents and is a combined effect of many different factors such as genetics, prenatal environment and environment during childhood and adolescence.

As we have data on the characteristics of adoptees and their biological and adoptive parents, we estimate the following model on the population of adoptees:[6]

$$H_i^{ac} = \alpha_0 + \alpha_1 Y_j^{bp} + \alpha_2 Y_i^{ap} + \upsilon_i^{ac}, \tag{4}$$

where *Y* once more measures health or human capital inputs that are transmitted from the biological parent *bp*, or the adoptive parent *ap*, to the adopt-

[5] Plug (2005) estimated the relationship between the schooling of grandchildren, parents and grandparents, where the grandchildren were adopted.

[6] We follow the strategy to separate pre- and post-birth effects from Björklund et al. (2006).

ed child *ac* born in family *j* and adopted and reared in family *i*; v_j^{ac} is a child-specific error term uncorrelated with Y_j^{bp} and Y_i^{ap}.

Before we discuss how we can interpret α_1 and α_2, let us state the following key assumptions of the adoption design:

1) Adoptees are conditionally randomly assigned to adoptive families.

2) The adoption should have taken place close to birth so that it is possible to accurately separate pre and post birth effects.

3) The postnatal pre-adoption environment (e.g., the quality of the nursery homes) is uncorrelated with the genetic background and the post adoption environment (or has no influence on the health of the adopted child).

4) The biological parents have no contact with the adopted child post adoption.

Under these four assumptions, we are able to estimate the association between adult health status and the observable pre- and post-birth characteristics separately by estimating equation (4) using data on adopted children and their biological and adoptive parents. In general, α_2 does not only capture the importance of the adoptive parental characteristic under study, Y_i^{ap}, but also everything else in the adoption family that is correlated with Y_i^{ap}.[7] We interpret the estimates as a measure of the importance of transmission channels stemming from the pre- or post-birth influences, respectively.

The first assumption listed above, that adoptees are conditionally randomly assigned to adoptive families, can be questioned in all empirical studies using data for adoptees (see further the discussion in Section 4.4). We do three sets of sensitivity analyses to check the robustness of our main results with respect to this assumption. First, we look at the robustness with respect to changes in the set of confounding parental characteristics included in the model. We employ the method suggested by Altonji et al. (2005) to get an estimate of how much any unobservable characteristics correlated between the biological and the adopting parents must contribute, relative to the contribution of the observable characteristics, in order to explain away the main results obtained in the study.

Second, we restrict the sample to only include adoptees that moved from their municipality of birth. We cannot directly observe whether relatives or friends of the biological parents adopted some children, but in such cases, children are more likely to stay in the municipality where they were born. Moreover, adopted children who move from their municipality of birth are much less likely to interact with their biological parents post adoption.

In the third sensitivity analysis, we restrict the sample of adoptees to first-borns of their biological mothers. The motivation for this restriction is to exclude adoptees who were given up for adoption because of illness, poverty

[7] For a discussion about the necessary conditions under which α_2 can be interpreted as the causal effect of parents' characteristic on child characteristic, see Holmlund et al. (2011).

or other reasons for inabilities to accommodate a large family of the biological parents, which, in turn, will increase the probability that the adopting parents are related to the biological ones. That is, first-borns are more likely to be given away for adoption simply because they are less likely to have been planned by their biological parents or born in established families.

Note also that equation (4) can easily be extended to account for "nature-nurture-interactions" by adding the product of Y_j^{bp} and Y_i^{ap} to this specification (see Björklund et al., 2006). We investigate the importance of such interactions in Section 5.4.2.

Assuming that adoptees and non-adoptees are drawn from the same distribution, we are also able to decompose an estimate of β_1 into separate entities of pre- and post-birth factors, captured by estimates of α_1 and α_2, which are then interpretable for the population of children. The likelihood of generalizability of the adoption estimates increases if the intergenerational parameter is linear and if the sum of the estimates of α_1 and α_2, using the sample of adoptees, equals an estimate of β_1, obtained in the population of children. We also perform a test of the external validity of the adoption coefficients by estimating these parameters on the sample of families where at least one child has been adopted out from the family and at least one child was not adopted but is instead reared by the biological mother (see Section 5.4.5).

4. Data and Descriptive Statistics

4.1 Sample Definition

We use data from different national registers in Sweden and include all males and females born in Sweden between 1940 and 1967.[8] We use the Multigenerational Register (see Statistics Sweden, 2012) to identify whether a person was adopted as a child. It also contains a personal identifier of the biological mother and father (if known to the authorities) as well as the adopting mother and father.

Table 1 shows the number of observations for the two populations used in this study – adoptees and, as a comparison, non-adoptees – at different stages of the sample selection process. In total, there are 64,889 adoptees who we can identify in our data. About 30,000 of them were adopted by only one parent, in most cases the husband of the child's biological mother. We excluded these individuals from the analysis. We construct two samples from the remaining population. First, a larger sample, including 21,192 individuals for whom we have information on the biological mother as well as the

[8]The lower cohort restriction is motivated by data availability and the upper one by the fact that domestic adoptions in Sweden decreased rapidly in the late 1960s.

adopting mother and father. Second, a smaller sample consisting of 10,831 individuals, for whom we also have information on the biological father.

Table 1. Sample sizes at different stages of the sample selection process.

Born in Sweden 1940-67	Non-adoptees	Adoptees
Non-adopted	3,061,504	
Adopted by at least one parent		64,889
Adopted by both parents		33,312
Not adopted by own parents		33,266
Adopting parents' age is correct *		30,876
Not died or emigrated first year	3,048,981	30,862
Biological mother is identified	3,004,251	22,695
Parents' education is known	2,907,595	21,192
Biological father is identified	2,826,257	10,831

*Adopting mother age 25-47 and adopting father age 25-66 at birth of adopted child.

Figure 1 shows the number of adoptees that we are able to identify in our data by year of birth and different categories. The top curve shows the total number of adoptees with two adopting parents that we are able to identify. The dashed and the thick solid lines below show the observations that we are able to identify, given the different data requirements indicated below the figure. It is evident from the figure that for those born in the first half of the 1940s, we are able to use a small share of the observations, since we are not able to observe data on their biological parents.

Figure 1 also shows that there is an increase in the number of adoptees between 1940 and 1945. This primarily reflects the increase in the overall fertility rate in Sweden. As discussed in Appendix A, there are several reasons for the decline in adoptions between 1945 and 1967.[9] The decrease in domestic adoptions towards the end of our study period was offset by an increase in international adoptions. The number of adopted children for whom we can identify the biological mother increases during the 1940s.

[9] Figure A1 in the Appendix A shows the ratio of adopted children in birth cohorts 1940-1967, which documents the same trends.

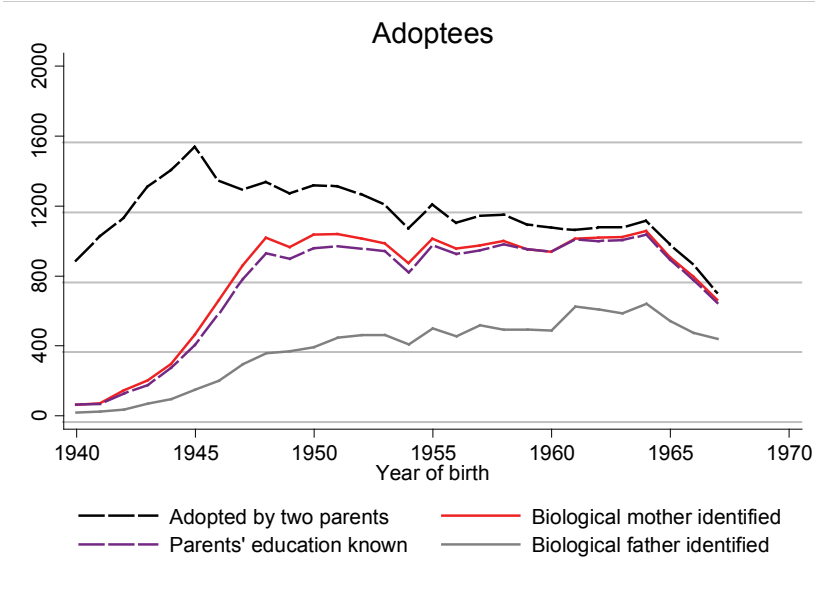


Figure 1. Swedish domestic adoptions by year of birth of the adoptees

4.2 Variable construction

4.2.1 Mortality in the Child Generation

Information on date of death, used for constructing dependent variables that apply to the child generation as well as to the parent generation, is obtained from the national Cause of Death Register (see Socialstyrelsen, 2009a). The Cause of Death Register records dates and International Classification of Diseases, revision 10, (ICD 10) codes for the underlying cause of death for all deaths in Sweden from 1952 and onwards. Our observation period stops on April 1, 2013. This implies that for the child generation, we can observe the oldest person in our sample until age 73 and the youngest until age 45.

In Figure A2 in Appendix A, we show that the share of deaths is quite low for the younger age groups. For the child generation, we therefore use proportional hazard models allowing for right censoring of date of death. The death rates are only somewhat higher among adoptees.

Table A2 in Appendix A shows the distribution of all deaths by the main underlying cause of death observed in the sample of adoptees and the comparison group, respectively. The distributions are fairly similar, although adoptees are somewhat less likely to die from cancer and more likely to die from diseases in the digestive organs and from mental disorders. Panel B in Table A2 shows the shares of the most common causes of hospitalization by main ICD 10 chapter for the groups of adoptees and non-adoptees, respectively. Similarly to the causes of death, the largest differences are in the diagnoses related to problems in the digestive organs and mental disorders.

4.2.2 Longevity in the Parental Generation

A problem with measuring longevity in the parental generation is that a large share of the parents, 41.1 percent for the biological parents and 28.0 percent for the adopting parents in the sample of adoptees, are still alive when we stop observing them in April 2013. To deal with this problem, we impute missing dates of deaths. We do this by first estimating a proportional hazard model based on the Gompertz distribution, which has been shown to provide good predictions of mortality up to age 90 (see Preston et al., 2000, and Chetty el al., 2016). We use the number of hospital stays, time hospitalized as well as indicators for more than 200 diagnosis codes as independent variables. Then, we use the estimated model to predict unobserved ages of death in the sample. Appendix B gives a detailed description of the model, the results from the estimation and an evaluation of the predictions.

A remaining problem is that we do not observe any hospitalization records for 17 percent of the sample that have not died. This implies that we are not able to say anything about their life expectancy other than that they are likely to live longer than those we observe to be hospitalized. To avoid an obvious selection problem, we follow the procedure suggested by Chetty et al. (2014) and use percentile ranks of life expectancy rather than the actual age. To construct these percentile ranks, we divide the sample into three groups. In the first group, which is the largest one with 64 percent of the total sample, we are able to observe age at death. In the second group, corresponding to 19 percent of the population, we use the hospitalization records to predict age of death. Then, it is straightforward to construct the percentile rank based on actual or predicted age of death within each birth cohort for these two groups.

For the third group, where we do not observe any hospitalization records, we are not able to predict differences in the rank *within* the group. Therefore, we assign the same rank, the mid-point of the share of individuals belonging to group three in a particular birth cohort and gender. For example, if the third group corresponds to 10 percent for those born in 1920, we assign percentile rank 95 to all in this group (which is the mid-point between 90 and 100).

4.2.3 Hospitalization

Data for our measures of hospitalization are obtained from the national In-patient Register (see Socialstyrelsen, 2009b). The national In-patient Register includes dates for all hospital stays at Swedish hospitals. This register has a national coverage starting in 1987, and we have access to data for the entire period until 2012. Since the first birth cohort included in our data was born in 1940, we observe all its hospital stays from age 47 and until age 72. The In-patient Register includes ICD codes for the maximum of eight different medical causes of each hospital stay.

We use two measures of health from the hospitalization data. The first, labeled "Hospitalization-based health", is simply the residuals from a linear probability model regression of an indicator variable for whether or not the individual has been in hospital care for each year separately during the observation window on year and year of birth indicators. If the person is dead, we treat him or her as missing. In a second step, we average the residuals for each individual to obtain the measure. This procedure accounts for differences in the probability of hospitalization over the life cycle and we may therefore interpret the resulting variable as a measure of lifetime hospitalization.

The second measure, labeled "Health index", is constructed in three steps.[10] First, for every year, we use a probit model to regress an indicator variable, equal to one if the individual has died within five years and zero otherwise, on the information from the in-patient register for that year (days, visits, and diagnoses) and indicators of year of birth and gender.[11] In a second step, we create a health index ranging between 0 and 1 by predicting the risk of dying within five years. An individual is assigned the value of 1 all years after death occurred; individuals not making any hospital visits are assigned the value of 0. Then, we average over all years. Based on this index, we obtain a percentile rank for each birth cohort and gender separately. The advantage of this measure compared to "Hospitalization-based health" is that it weights the different diagnoses by "severity" based on how likely the person is to die within five years.

4.2.4 Measures Based on Birth Outcomes

Previous research has established that birth outcomes to a large extent reflect the health status of the mother (see e.g. Currie, 2011). This relation enables us to use the birth outcomes of the children of the female adoptees included in our sample as a health measure. Further, weight at birth, and in particular low birth weight (below 2,500 g), is very strongly correlated with health outcomes later in life. Studying health at birth for the third generation enables us to test for multigenerational transmission of health. Our data source is the National Swedish Birth Register. This birth register contains a large amount of information on all births in Sweden from 1973 and onwards. Using the Multigenerational Register, we are able to link births to all children (adopted and biological) included in our sample.

We use four different birth outcome measures: (1) An indicator for low birth weight, i.e. a birth weight below 2,500 grams; (2) Birth weight measured in grams (scaled in percentile ranks); (3) An indicator of the APGAR

[10] The first two follow Cesarini et al. (2016).

[11] We use the first two digits in the ICD10 diagnosis codes (one letter and one number), which constitute about 200 different categories. We do this for the first two diagnoses for each hospital stay. In addition, we include linear variables for the number of hospital stays and the total number of days in hospital care. We control for gender and stratify on birth cohort.

score at five minutes after the birth being below the maximum score of 10; (4) The APGAR score after five minutes.[12]

4.2.5 Educational Attainments

The number of years of schooling in the parental generation is a key independent variable in our empirical analysis. Our main data source for this variable is the 1970 Census. If the information is missing in that Census year, we use data from the 1990 and 2004 waves of the Swedish Education register. As a third option for observations that are still missing, we use the 1960 Census.[13] Overall, we are able to identify educational attainment for 97 percent of the sample. Education in Swedish registries is recorded at seven different levels, which we translate into years of schooling.[14]

4.3 Descriptive Statistics

Table 2 contains sample means and standard deviations (within parentheses) for the main outcome and control variables in the sample of adoptees and non-adoptees. Columns 3 and 5 show descriptive statistics for adoptees that are weighted by the size of the cohorts for non-adoptees.[15] The first panel

[12] The APGAR score is a summary measure recorded by the midwife very shortly after birth and at given times, with the purpose of summarizing the health status of newborn children. It uses five different criteria: Complexion, Pulse rate, Reflex irritability grimace, Activity and Respiratory effort. It is named as a backronym of the included indicators (**A**ppearance, **P**ulse, **G**rimace, **A**ctivity, and **R**espiration) as well as after the anesthesiologist Virginia Apgar, who suggested the score in 1952.

[13] The education measure from 1970 is available for the population of individuals given that the individual was born in 1911 or later and was alive and lived in Sweden in 1970. It is used as the main choice because it measures educational attainment for individuals when they are supposed to have finished their education. Education from 1990-2004 will only be utilized for those parents that were not living in Sweden in 1970, so as not to capture educational investments later in life (which was fairly common in the 1980s and 1990s). Education in 1960, which is less detailed compared to the 1970 information, will only be used for those parents that have died before 1970 and/or that were born before 1911. A problem with the 1960 Census is that the coding of educational attainment is different from our other data sources. Therefore, we use data from individuals that are present in both the 1960 and 1970 census, and are 35-45 years old in 1960, to predict years of schooling from the 1960 census for those missing observations.

[14] Pre-comprehensive school compulsory level = 7 years; 2 comprehensive school or junior secondary school = 9 years; vocational school =10.39 years, secondary school = 12.19 years; secondary school + 1 or 2 years = 14; college or university = 16 years; and PhD = 20 years.

[15] We have chosen non-adopted children and their parents as the reference category when weighting to be able to display the differences among the different categories in a way that is as transparent as possible. Choosing adoptees as the reference would have made the weighting of parents more difficult as we would not be able to compare the biological and adoptive parents of adoptees internally without including another column. In other parts of the paper when we weight the cohorts to compare non-adoptees and adoptees, we weight non-adoptees (adoptees are used as "reference"). The reason is that there are very few adoptees in each year at the beginning of the period and we are reluctant to increase the weight of the cause of death or diagnoses at hospitalization for this sample.

shows information on the children in the two samples. The second panel shows descriptive statistics for the biological parents. On average, the biological parents of adopted children have slightly less education and a shorter life expectancy as compared to those of non-adopted children. The third panel shows descriptive statistics of the adopting parents. Adopting fathers have almost one additional year of education as compared to the biological fathers of the adopted children.[16]

Table 2. Summary statistics of main outcome and control variables

	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Bio father known (weighted)	(4) Adoptees - Large sample	(5) Adoptees - Large sample (weighted)
Panel A: Children					
Female	0.488	0.481	0.477	0.478	0.476
	(0.500)	(0.500)	(0.499)	(0.500)	(0.499)
Dead by April 2013	0.071	0.067	0.086	0.077	0.095
	(0.257)	(0.249)	(0.280)	(0.267)	(0.293)
Hospitalization (rank)	50.103	44.833	45.026	45.191	45.108
	(27.853)	(28.785)	(28.676)	(28.779)	(28.771)
Health index (rank)	50.160	45.052	45.183	45.290	45.220
	(27.662)	(28.479)	(28.371)	(28.493)	(28.501)
Years of schooling	11.718	11.533	11.360	11.537	11.396
	(2.776)	(2.400)	(2.531)	(2.455)	(2.533)
Birth weight first own child (women)	3417.164	3392.612	3395.395	3400.806	3404.492
	(566.278)	(598.281)	(592.944)	(595.242)	(593.646)
Low birth weight own child (women)	0.052	0.066	0.064	0.063	0.061
	(0.223)	(0.248)	(0.245)	(0.243)	(0.240)
APGAR 5 min	9.528	9.508	9.503	9.492	9.500
	(0.954)	(0.980)	(0.999)	(1.016)	(1.015)
Low APGAR 5 min	0.335	0.345	0.346	0.352	0.344
	(0.472)	(0.475)	(0.476)	(0.478)	(0.475)

Cont. next page

[16] In the adoptee sample, biological parents are on average younger than adoptive parents, biological mothers are on average 24 years old at birth, and adoptive mothers are on average 34 years old.

Cont. Table 2

	Non-adoptees	Adoptees - Bio father known	Adoptees - Bio father known (weighted)	Adoptees - Large sample	Adoptees - Large sample (weighted)
Panel B: Biological parents					
Dead by April 2013, mother	0.568	0.495	0.672	0.516	0.668
	(0.495)	(0.500)	(0.470)	(0.500)	(0.471)
Age at death, mother	77.540	69.763	75.022	71.133	75.500
	(12.188)	(13.733)	(12.537)	(13.233)	(12.323)
Rank longevity, mother	49.948	41.155	41.832	41.949	42.805
	(28.394)	(28.671)	(27.853)	(28.566)	(27.897)
Years of schooling, mother	8.235	8.095	7.837	8.128	7.895
	(2.140)	(1.753)	(1.584)	(1.816)	(1.665)
Dead by April 2013, father	0.741	0.682	0.812		
	(0.438)	(0.466)	(0.391)		
Age at death, father	74.512	70.533	73.876		
	(11.820)	(11.563)	(11.060)		
Rank longevity, father	49.908	42.989	45.618		
	(28.642)	(27.728)	(27.286)		
Years of schooling, father	8.780	8.384	8.121		
	(2.773)	(2.172)	(2.045)		
Panel C: Adoptive parents					
Dead by April 2013, mother		0.650	0.596	0.682	0.598
		(0.477)	(0.491)	(0.466)	(0.490)
Age at death, mother		79.068	78.497	79.570	78.424
		(11.231)	(11.569)	(11.164)	(11.684)
Rank longevity, mother		51.060	50.791	51.250	50.706
		(28.153)	(28.180)	(28.194)	(28.258)
Years of schooling, mother		8.527	8.638	8.508	8.694
		(2.426)	(2.491)	(2.413)	(2.517)
Dead by April 2013, father		0.789	0.727	0.811	0.724
		(0.408)	(0.446)	(0.391)	(0.447)
Age at death, father		76.547	76.120	76.991	76.338
		(10.815)	(10.917)	(10.709)	(10.884)
Rank longevity, father		53.477	53.511	53.910	53.914
		(28.112)	(27.992)	(28.048)	(27.826)
Years of schooling, father		9.322	9.394	9.306	9.491
		(3.097)	(3.102)	(3.125)	(3.158)
Observations	2,826,257	10,831	10,831	21,192	21,192

Notes: Standard deviations in parentheses. The summary statistics for non-adoptees and the smaller adoptee sample are weighted by the size of the cohorts for non-adoptees. The weighting is done separately for mothers and fathers, meaning that the biological mothers are comparable to the biological and adopting mothers in the adoptee samples. Since the biological fathers of adopted children are known only for a smaller sample, these are missing in the larger sample of adoptees.

4.4 The Association between Biological and Adopting Parent Characteristics

A possible concern with the interpretation of the coefficient estimates is that of selective placement of adoptees. Table 3 illustrates the correlation in years of education and longevity rank between adopting and biological parents of adoptees.

Table 3. Correlation between biological and adoptive parents for mothers and fathers respectively, standardized by cohort.

	Mothers	Fathers
Years of education	0.1676	0.1757
Rank longevity	0.0290	0.0346

Notes: p-values for significance of all estimates below 0.1 percent.

The correlation for years of schooling is quite similar to that reported by Björklund et al. (2006) for children in cohorts born 1962-1966. The correlation in percentile rank of longevity is positive as well, but much lower. This finding is very important for the purpose of this study, since it suggests that selective placement is relatively less likely to generate biased estimates of the health/mortality correlations using adoption data.[17] However, as these correlations are not zero and because we investigate the importance of educational attainment, we cannot completely disregard this issue.

There are at least two reasons why we would observe a positive correlation for characteristics of biological and adoptive parents. First, this could happen if some adoptions are made by relatives of one of the biological parents. Second, there could be matching on characteristics known to the adoption agency, either because of the demand of parents, or because of a view that an adopted child would be better off in an adoptive family with similar characteristics as the biological parents.

As discussed in Section 4.4, the empirical importance of the first reason – adoptions by relatives – is likely to be very limited since the rule of not allowing people with own biological children to adopt to a large extent precluded parents and siblings of the biological parents from doing that. As further discussed in Appendix A, Nordlöf (2001) estimated these adoptions to be around 1 percent of the total number of adoptions in the Stockholm area. Brandén et al. (2015) confirm this conclusion, although their estimate of the share of adoptions by close relatives is slightly higher at 5.4 percent,

[17] One might be concerned about the fact that the lower rank correlation for longevity is due to this variable being predicted for a sizable share of the parents. However, this does not explain the low correlations. If we use data on the adoptees born in year 1953 or prior (where most parents have died before the end of the sample window), we get that the correlation between mothers' education is 0.1026 and the correlation between mothers' rank longevity is 0.0403. For fathers' education, the correlation is 0.1649 and for fathers' rank longevity, the correlation is 0.0859.

applying to the whole country. They are also able to eliminate those adopted by close relatives from their sample and find that the correlation in years of schooling between (unrelated) adoptive and biological parents of adoptees remains virtually unchanged.

The second reason, matching, is likely to be a more important mechanism. If this matching is made on characteristics observable in the data (such as educational attainment or health characteristics), we are able to control for this in the estimations. In a sensitivity analysis, we will investigate this further by including more detailed health and education data of the biological (adoptive) parents and see what happens to the estimate for the characteristics of adoptive (biological) parents (a similar test was made in Björklund et al. 2006). If we do not see any change, which is what happens (see Section 5.4.1), we can rule out matching on observable characteristics as affecting our conclusions.

The remaining reason would then be matching on characteristics that are unobservable in the data. Björklund et al. (2006) investigate this issue by deriving the magnitude of the bias (modeled as a combination of selective placement and measurement error), finding evidence that the bias accounts for at most 13 percent of the estimated impact of the adoptive and biological parents' characteristics on adoptees' educational attainment. Given the low correlations in the percentile rank of longevity among parents in Table 3, we have no reason to believe that this should be a more severe problem in this setting. Nevertheless, we return to and further examine this issue in Section 5.4.1.

A second potential threat to the random assignment assumption is that adoptees may be non-randomly assigned to adoptive families based on health endowments at birth. This is particularly troubling if e.g. more educated adoptive parents and somehow able to "pick out" healthier children. While we cannot directly test for this because we lack data on health at birth, it is unlikely to happen for several reasons. First, the institutional set up at the time was such that adoptive families were approached as soon as a candidate for adoption became available and there was an excess of candidate adoptive parents relative to available children. Second, unhealthy infants that were given away by their biological mothers were not offered for adoption (see also Appendix A). Finally, Holmlund, Lindahl and Plug (2008) show that there is no significant correlation between adoptive parents' education, the gender of the adoptee and the biological mother's age at birth – the only two pre-existing characteristics that are available in the data and could potentially proxy for infant health at birth.

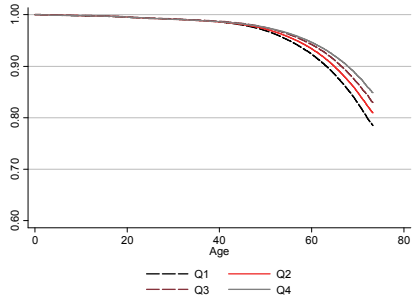
5. Results

We first present results from our study of the intergenerational transmission of health. Then, we show estimates from our separate analysis of the association between parental educational attainment and child health. For each of these analyses, we use three different health outcomes: mortality, hospitalization-based measures and the birth outcomes among second-generation children. Finally, we conduct a number of sensitivity analyses.

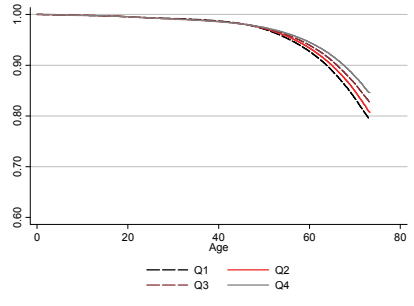
5.1. Intergenerational Transmission of Parental Health

5.1.1 Mortality

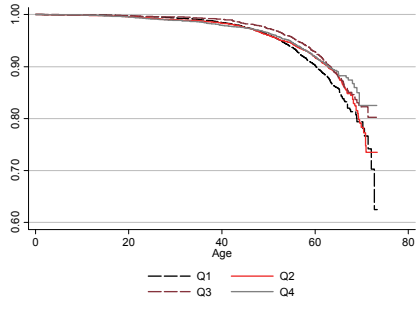
Figure 2 shows Kaplan-Meier estimates of the survival functions of children in four different groups based on quartiles in longevity of their parents. The two upper panels show that there is a stable intergenerational persistence in longevity in the population of children brought up with their biological parents. The two graphs below show that the differences between the groups are very similar among adoptees and their biological parents, although the graphs are noisier due to the smaller sample size. The two lower panels, however, show no clear association between the longevity of the adopting parents and the survival of their adoptive children.



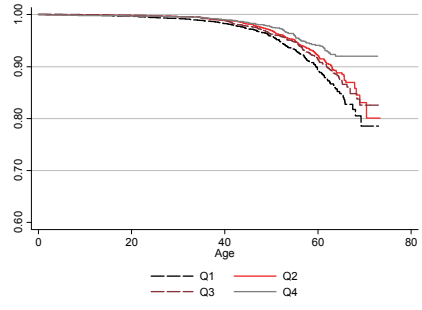
a) Non-Adoptees: Biological Mother



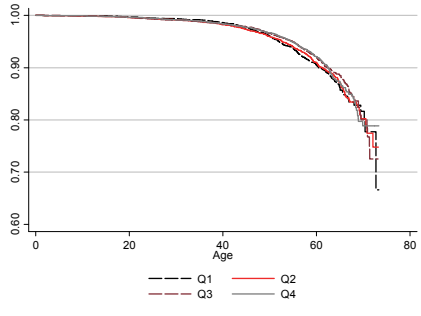
b) Non-Adoptees: Biological Father



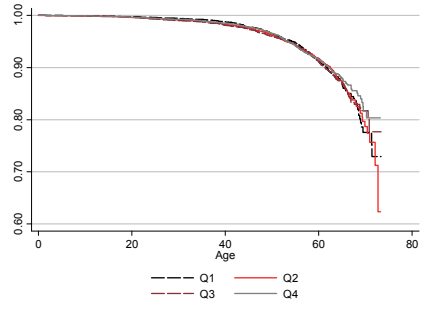
c) Adoptees: Biological Mother



d) Adoptees: Biological Father



e) Adoptees: Adopting Mother



f) Adoptees: Adopting Father

Figure 2. Kaplan-Meier estimates of the relationship between parental longevity in quartiles (Q) and child mortality.

Note: The figures show the relationship between child mortality and parental longevity divided into quartiles for non-adoptees and the large sample of adoptees (the exception is Adoptees – Biological Father that by construction is based on the smaller adoptee sample).

To get a measure of the intergenerational persistence in longevity we estimate Cox proportional hazard model models for associations shown in Figure 2. The reason for using hazard models is to handle the large number of right censored observations on the dependent variable, i.e., people who are still alive by the end of the observation period. The Cox model is relying on the proportional hazard specification, but no particular functional form for the baseline hazard.[18]

Table 4 shows the results. The three columns in Table 4 correspond to the three different Cox proportional hazard model samples: all non-adoptees born in Sweden between 1940 and 1967, adoptees for whom we have data on both biological parents and, finally, the sample of adoptees where we also include those for whom we have no information on the biological father.

The results in Column 1, corresponding to non-adoptees, show that a one percentile move in the distribution of the life expectancy of mothers is associated with a 0.48 percent reduction in mortality of their children. The corresponding estimate for fathers is 0.36 percent, i.e., slightly weaker. Both figures are conditional on the other parent's longevity.

The results in Column 2, corresponding to the smaller sample of adoptees, show significant associations with the biological parents' longevity, but a quite precisely estimated absence of an association with the longevity of the adopting parents. This result is supported by the estimates in the larger sample, presented in Column 3. In the larger sample, we are able to exclude a 0.2 percent reduction in mortality from a one percentile longer life expectancy of the adopting father in the Cox model. The corresponding bound for the adopting mother is 0.3. The magnitudes of the associations with the biological parents are somewhat stronger in the smaller sample of adoptees (Column 2).

[18]As explained in Section 4.2, we handle the problem of a large number of censored observations on the *independent* variable, parental longevity, by using hospitalization data predicting age of death. Finally, for those alive when we stop observing them in April 2013, and for whom we do not observe any hospitalization, we follow e.g. Chetty et al. (2014) and use percentile ranks, rather than longevity measured in time, for the variable where we assign the same rank for this group of individuals.

Table 4. Cox proportional hazard model estimates of the associations between child mortality and parental longevity in percentile ranks

	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Large sample
Longevity, Bio Mother	0.9952***	0.9946***	0.9958***
	(0.0001)	(0.0014)	(0.0009)
Longevity, Bio Father	0.9964***	0.9933***	
	(0.0001)	(0.0014)	
Longevity, Ad Father		1.0000	0.9998
		(0.0013)	(0.0009)
Longevity, Ad Mother		0.9991	0.9988
		(0.0013)	(0.0009)
P-value joint significance			
Biological parents	0.0000	0.0000	0.0000
Adoptive parents		0.8150	0.3568
Observations	2,826,257	10,831	21,192

Notes: Results from Cox proportional hazard models. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort. Column (1) is based on a sample of non-adopted children, column (2) on adoptees for whom we have information on all parents, and in column (3) we add adoptees with unknown biological fathers.

To investigate how these estimates for mortality translate into effects on life expectancies, we need to assign a parametric distribution for the baseline hazard. We use the Gompertz distribution, which has been shown to provide good predictions of mortality up to age 90 (see Preston et al., 2000, and Chetty et al., 2016). The hazard ratio estimates from this model turned out to be very similar to those of the Cox model presented in Table 4, see Table C1 in Appendix C. Using these estimates for non-adoptees, we find that the prediction for one additional year of longevity of the mother on median life expectancy of the child is 0.177 additional years for the child. The corresponding estimate for the father's longevity is 0.122 additional years.[19] This

[19] Using the estimates in Table C1 in Appendix C, we get that the mortality risk decreases by 0.0048 and 0.0036, from one percentile higher rank for mothers and fathers, respectively. This translates into effects for the child that can be interpreted as an increase from the median life expectancy of the child as 0.74 and 0.55 months, respectively (using predictions based on the Gompertz distribution). Since one percentile higher life expectancy on average between the 10th and the 90th percentile is equivalent to 0.349 years (4.18 months) for mothers and 0.375 years (4.50 months) for fathers, we get that the intergenerational association in life is 0.74/4.18=0.177 for mothers and children and 0.55/4.50=0.122 for fathers and children, conditional on the other parent.

is in the range of the estimates of the intergenerational persistence in longevity obtained in previous studies (see Herskind, 1996, for a review).

We conduct two robustness checks for the results presented in Table 4, which concern the measurement of life expectancy in the parent generation. First, we estimate a model where we use two, rather than one, variables for life expectancy in the parental generation: an indicator variable for those we observe die before April 2013 and a variable for age of death for those for whom we are able to observe it. Second, we restrict the sample to older cohorts of parents (with children born prior to 1953), for whom we can observe actual age at death for a much larger fraction of the parents.[20]

The results from these two sensitivity analyses are shown in Tables C2 and C3 in Appendix C, respectively. The results in Table C2 show that both variables measuring the biological parents' longevity turn out statistically significant, but none of the variables measuring the adopting parents' longevity do, i.e., they are qualitatively the same as the main ones presented in Table 4. Moreover, the results shown in Table C3 support the main results since they change very little, and are still precisely estimated, in the sample restricted to the child generation born in 1953 or earlier, with very few censored observations on parent longevity. Hence, we conclude that measurement error bias is unlikely to influence our results in a way that affects our conclusions.

The hazard rate is likely to be different for women and men, as we know that gender is a strong predictor for longevity. In an additional sensitivity analysis, we therefore split the sample by gender and study the associations between child mortality and biological and adoptive parents' longevity. The results, reported in Appendix Table C4, show that the overall associations are robust, but also that there are differences between gender groups in the influence of parental characteristics. For male adoptees, longevity of biological fathers is more important than longevity of biological mothers. In contrast, the association between female adoptees and their biological mothers' longevity is stronger.

Finally, we study to what causes of death we can attribute the association between the biological parents' longevity and child mortality. We expect it to be mostly due to diagnoses with a strong element of biological heritability, rather than related to life styles or quality of health care. We do a competing risk analysis where we differentiate between the six main chapters in

[20] For the cohorts of children born in 1953 or earlier, the fraction of parents that are deceased at the end of our observational window are: For the population of non-adoptees, 81,7%of the mothers and 94.0% of the fathers. For the adopted children, these fractions are 91.4% of the adoptive mothers, 97.3% of the adoptive fathers, 70.0% of the biological mothers and 86.3% of the biological fathers. Hence, the measurement error bias in our estimates is likely to be almost non-existent for adoptive parents for these cohorts. For biological parents, it is still likely to have an impact, meaning that the positive and statistically significant estimates of the association between biological parents' life expectancy and adopted children's mortality risk are underestimated.

ICD 10 coding. In addition, we add a category for "Preventable" diseases and one for diagnoses corresponding to "Treatable" illnesses. The groupings of diagnosis codes are explained in Table C5 in Appendix C. In the competing risk analysis, we simply treat deaths in the diagnosis groups not under study as right censored at the date of death. The assumption ensuring consistency of this model is that the latent risks of death are independent between the different causes of death.

Table C6 shows the results from the competing risk analysis. Panel A shows that all diagnosis groups are significantly associated with the mortality of the parents. The results for the adoptees in Panels B and C show that we can establish a significant relation between the biological parents' mortality and deaths from cancer diseases, circulatory diseases and diseases of the digestive organs, but not for the "preventable" or the "treatable" diseases. These results support the hypothesis that the relation between the biological parents of the adopted children can be attributed primarily to diseases that are not related to lifestyles and healthcare usage.

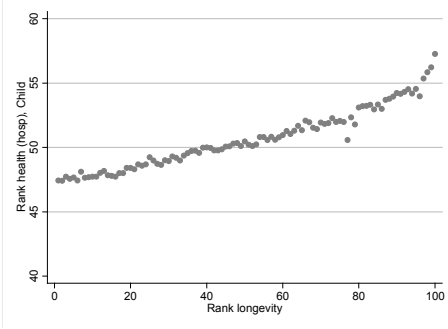
5.1.2 Health Measures Based on Hospitalization Data

Figure 3 shows the relation between percentiles of parental longevity and the hospitalization-based health variable, also in percentile ranks, for the children. The graphs are organized in the same way as in Figure 2. Once more, the graphs for non-adoptees show that there is a very strong intergenerational persistence in health, and it is well approximated by a linear relationship.

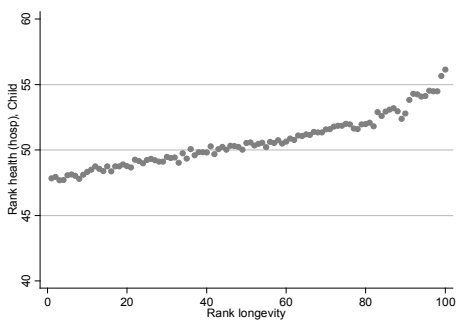
The graphs for the biological mother in the adoptee sample show an almost as strong relation between the biological mother and her children given up for adoption.[21] There is a visible relation also for the biological fathers, although weaker. An interesting feature of the graphs is that those for adoptees are at a lower level than those for the rest of the population, reflecting the fact that, on average, they have inferior health.

Finally, the lower graphs show no apparent relation between the health status of the adopting parents and their children. Importantly, we once more see that the associations for the adoptee samples are approximately linear.[22] This supports the findings from the decompositions of intergenerational associations in the population into the parts that are due to pre- and post-birth factors when we use adoptees.

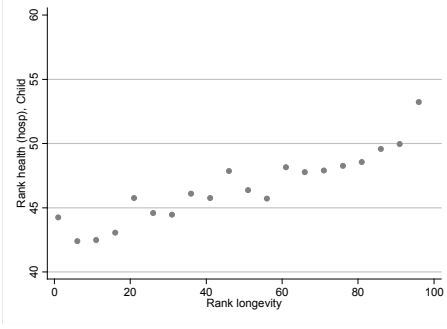
[21] As the sample of adoptees is not large enough to plot percentile ranks, we here instead show child's health rank at each half-decile of parental longevity.
[22] Note that the plot for the biological father of the adoptees is based on yet a smaller sample.



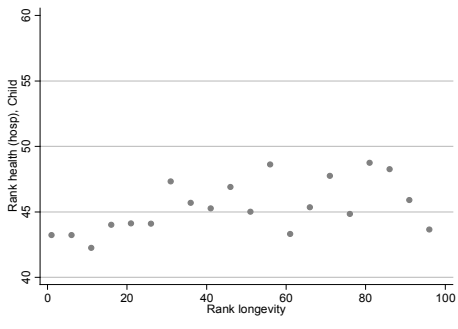
a) Non-adoptees: Biological Mother



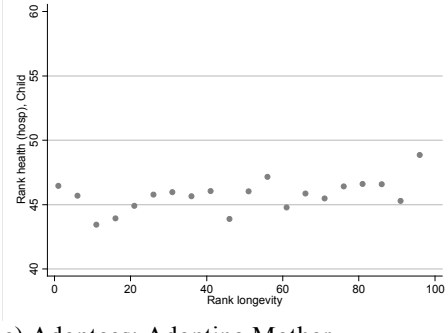
b) Non-adoptees: Biological Father



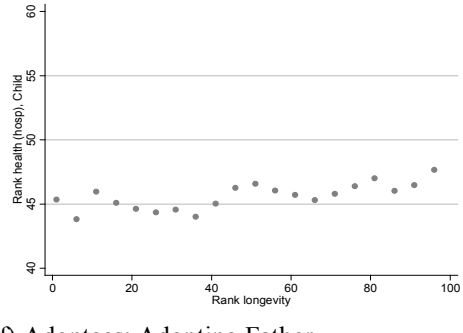
c) Adoptees: Biological Mother



d) Adoptees: Biological Father



e) Adoptees: Adopting Mother



f) Adoptees: Adopting Father

Figure 3. Mean percentile rank of child health (hospitalization based) and percentile rank parental longevity

Notes: The figures plot the relationship between percentile rank child health (hospitalization based) and percentile rank parental longevity for non-adoptees and the large sample of adoptees (the exception is Adoptees – Biological Father that by construction is based on the smaller adoptee sample).

Table 5 reports OLS regression results when we use *Hospitalization-based health* and the *Health index* as health measures for the child generation. As for the specifications presented in Table 4, we use ranks for measuring parental life expectancy and to facilitate the interpretation of the results, we also transform the dependent variables into percentile ranks. Columns 1 and 4 report the results for non-adoptees. The relation between the estimates for mothers and fathers, with the health of the mother being slightly more important for the health outcome of the child, is the same as the results reported for mortality in Table 4. The magnitudes of the estimates for the two indices are remarkably similar suggesting that a one-percentage increase in the mother's relative health is associated with a 0.068 percentile increase in the child's health. The corresponding estimate for the father is about 0.056. Hence, we find that the intergenerational transmission of health in the population is positive but clearly smaller than what is typically found for outcomes such as education and income (see Black and Devereux, 2011).

The results for adoptees – reported in Columns 2, 3, 5 and 6 – are once more very similar to those for non-adoptees for the biological parents. As opposed to the estimates reported for mortality, the measures of the life expectancy of the adopting parents are jointly statistically significantly different from zero for both measures at the 5 percent level. A possible explanation to this finding is that a large share of the individuals has experienced a hospitalization episode, providing us with more power to capture the link between parental and child health than for the mortality outcome.

The fact that the estimates for the adopting parents are jointly statistically significantly different from zero allows us to decompose the intergenerational association in health in pre- and post-births influences, which we attribute to heritability and in-utero experiences (pre-birth) and health habit formation (post-birth) (see e.g. Björklund et al., 2006). For the first hospitalization measure, such decomposition attributes about ¾ of the association to pre- and ¼ to post-births influences in the smaller sample. The corresponding estimates in the larger sample, where we do not observe the biological father, are ⅔ and ⅓, respectively. The decomposition for the other hospitalization index is qualitatively similar, although it places somewhat more weight on the pre-birth influences. [23]

[23] The relatively smaller intergenerational health associations found here are in line with the finding in Mazumder (2011). He finds smaller sibling correlations in health status than for education and family income. This is consistent with a story where pre-birth factors are similarly important for intergenerational mobility of health, education and income, whereas post-birth factors are more important for intergenerational mobility of education and income (Björklund et al., 2006).

Table 5. OLS estimates of associations between percentile rank of parental longevity and child lifetime health measured by indices based on hospitalization data

	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Large sample	(4) Non-adoptees	(5) Adoptees - Bio father known	(6) Adoptees - Large sample
	Hospitalization-based health			Health index		
Longevity, Bio Mother	0.0690***	0.0731***	0.0772***	0.0683***	0.0740***	0.0745***
	(0.0006)	(0.0096)	(0.0069)	(0.0006)	(0.0096)	(0.0069)
Longevity, Bio Father	0.0570***	0.0472***		0.0560***	0.0504***	
	(0.0006)	(0.0100)		(0.0006)	(0.0099)	
Longevity, Ad Mother		0.0115	0.0174**		0.0033	0.0135*
		(0.0099)	(0.0070)		(0.0098)	(0.0070)
Longevity, Ad Father		0.0303***	0.0218***		0.0235**	0.0202***
		(0.0099)	(0.0071)		(0.0098)	(0.0070)
P-value joint significance						
Biological parents	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
Adoptive parents		0.0040	0.0003		0.0506	0.0018
Observations	2,800,885	10,792	21,045	2,800,885	10,792	21,045

Notes: Results from OLS regressions. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort. Columns (1) and (4) are based on a sample of non-adopted children, columns (2) and (5) on adoptees for whom we have information on all parents, and in columns (3) and (6) we add adoptees with unknown biological fathers. The dependent variable in columns (1)-(3) is a measure of hospitalizations, and the dependent variable in columns (4)-(6) is a health index.

5.1.3 Birth Outcomes

The mother's health is likely to be at least partly reflected in the birth outcomes of her children (Currie and Moretti, 2007). This is the first reason why we use birth weight and APGAR scores of the children of female adoptees as a proxy for the health of these women. The second reason is that birth weight is known to correlate strongly with later-life health and thus, it can serve as an additional measure of the intergenerational transmission of health going into the third generation. An important caveat is that selection into giving birth is likely driven by maternal health status, so that healthier women are more likely to conceive and deliver live children. That is why we expect that our estimates are biased downwards and should therefore be interpreted as lower bounds of the true effect.

Panel A in Table 6 shows results from intergenerational regressions where we use two measures of the birth weight of the first-born child as a health measure of the mother: actual birth weight for the first born child transformed into percentile scores to facilitate the interpretation and the probability of low birth weight (<2,500 g). Panel B shows the results when we instead use the actual APGAR score as health measures and an indicator for the APGAR at five minutes below 10.[24] Since we have to restrict the sample to females only for these regressions, the sample sizes are about halved as compared to the regressions shown in the previous tables.

We find highly significant positive effects of the longevity of both biological parents on the birth weight of their grandchildren in the sample of non-adoptees.[25] For adoptees, the significant effect of the longevity of the biological parents remains for the APGAR score measures in the large sample.

[24] We do not scale APGAR into percentile ranks as 66.5% of the children have an APGAR score of 10.

[25] This finding relates to a small but growing literature on multigenerational associations, although estimates of the transmission of health across multiple generations are almost absent in the literature (two exceptions are Johnston et al., 2013, and Piraino et al., 2014). Although birth weight is obviously a non-perfect indicator of adult health, it is known to causally impact many adult outcomes including height (see Black et al., 2007).

Table 6. Associations between percentile rank of parental longevity and firstborn grandchild's health at birth

	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Large sample	(4) Non-adoptees	(5) Adoptees - Bio father known	(6) Adoptees - Large sample
Panel A	*Rank birth weight*			*Low birth weight<2,500 g*		
Longevity, Bio Mother	0.0100***	0.0329*	0.0194	-0.0001***	-0.0001	0.0000
	(0.0012)	(0.0174)	(0.0128)	(0.0000)	(0.0001)	(0.0001)
Longevity, Bio Father	0.0249***	-0.0064		-0.0001***	-0.0003*	
	(0.0011)	(0.0181)		(0.0000)	(0.0001)	
Longevity, Ad Mother		0.0104	0.0212		-0.0000	-0.0001
		(0.0181)	(0.0132)		(0.0002)	(0.0001)
Longevity, Ad Father		-0.0165	-0.0021		0.0002	0.0001
		(0.0181)	(0.0133)		(0.0001)	(0.0001)
P-value joint significance						
Biological parents	0.0000	0.1579	0.1284	0.0000	0.1357	0.9612
Adoptive parents		0.5695	0.2726		0.5088	0.4612
Observations	789,908	3,468	6,396	789,908	3,468	6,396
Panel B	*APGAR 5 min*			*APGAR 5 min<10*		
Longevity, Bio Mother	0.0002***	0.0017***	0.0006	-0.0001***	-0.0006**	-0.0002
	(0.0000)	(0.0006)	(0.0005)	(0.0000)	(0.0002)	(0.0002)
Longevity, Bio Father	0.0002***	-0.0001		-0.0001***	-0.0000	
	(0.0000)	(0.0006)		(0.0000)	(0.0003)	
Longevity, Ad Mother		0.0009	0.0001		-0.0004	0.0001
		(0.0006)	(0.0005)		(0.0003)	(0.0002)
Longevity, Ad Father		-0.0003	-0.0001		0.0001	0.0001
		(0.0006)	(0.0005)		(0.0003)	(0.0002)
P-value joint significance						
Biological parents	0.0000	0.0140	0.2019	0.0000	0.0536	0.2301
Adoptive parents		0.3228	0.9769		0.2496	0.7105
Observations	713,795	3,143	5,745	713,795	3,143	5,745

Notes: Results from OLS regressions. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort. Columns (1) and (4) are based on a sample of non-adopted children, columns (2) and (5) on adoptees for whom we have information on all parents, and in columns (3) and (6) we add adoptees with unknown biological fathers. The dependent variable in columns (1)-(3) in Panel A is birth weight measured in grams and scaled into percentile ranks, and in Panel B, the APGAR measure at five minutes after birth ranges from 0-10. The dependent variable in columns (4)-(6) in Panels A and B is binary.

5.2. Parental Educational Attainments

5.2.1 Mortality

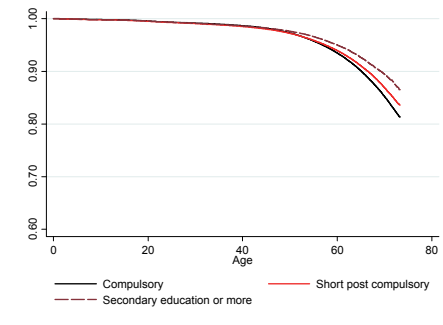
Figure 4 shows the Kaplan-Meier estimates of the survival function for three different groups based on parental educational attainments. The first group only has compulsory education or less; the second group has one or two additional years of education, in most cases vocational schooling, after they finished the compulsory level; finally, the third group has secondary schooling or more. For non-adoptees, there is a clear, visible difference in the survival rates between the groups with different parental education. For adoptees, the graphs are not separable in most cases. However, it is apparent that adoptees with highly educated adopting mothers have a higher survival rate.[26]

Table 7 shows the results for our estimates of the association between parental educational attainments and child mortality. Column 1 shows the results for children raised by their biological parents. The results in this sample suggest that an additional year of maternal and paternal schooling is associated with a decrease in child mortality by 2.3 and 2.5 percent, respectively. The results shown in Columns 2 and 3 reveal that this association can be completely attributed to post-birth influences. An extra year of education for the adopting mother is associated with a 2.9-4.6 percent decrease in mortality, while there is no protective effect associated with the educational attainments of the biological parents.[27]

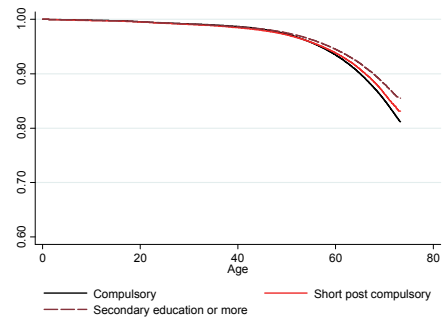
By using a proportional hazard model based on the Gompertz distribution, we can translate the associations between years of schooling and mortality into years of life expectancy. The results are shown in Table C7. For non-adoptees, they predict that one year of additional education for the mother and father is associated with a 0.30 and 0.33 years change in life expectancy, respectively. In the samples of adoptees, the predictions suggest an association between 0.34 and 0.53 years from an additional year of education of the adopting mother, whereas there is no association with the adopting father's educational attainment.

[26] There is a difference in the share with high education among biological and adopting parents. Among the adopted children's biological mothers, only 4 % have secondary education or more, and among adopting mothers 10 % have secondary education.

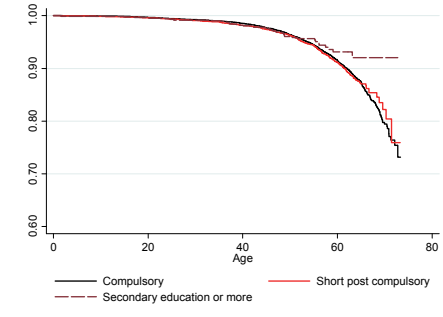
[27] The estimate for years of education for the biological mother is now *positive*, predicting a higher mortality risk. However, there are two reasons why we do not want to emphasize this result. First, when we consider the results from the larger adoption sample in column (3), there is no evidence of such an adverse effect for biological mothers. Second, we find no supporting evidence for this adverse effect in the other health outcomes reported later in the paper.



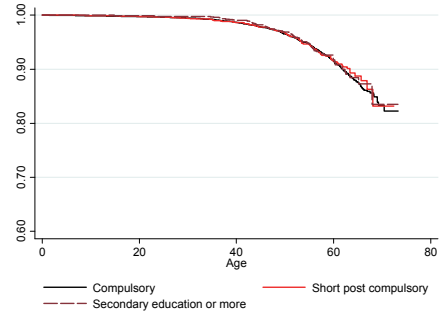
a) Non-Adoptees: Biological Mother



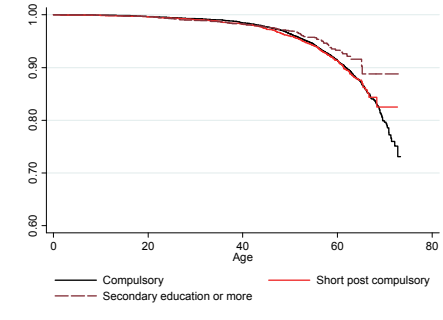
b) Non-Adoptees: Biological Father



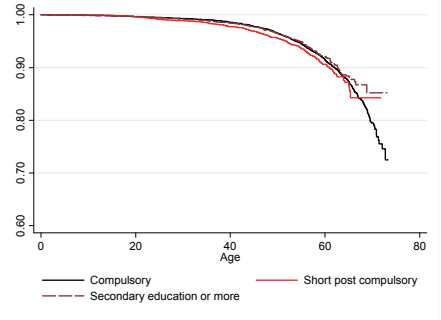
c) Adoptees: Biological Mother



d) Adoptees: Biological Father



e) Adoptees: Adopting Mother



f) Adoptees: Adopting Father

Figure 4. Kaplan-Meier estimates of the relation between parental educational attainments and child mortality

Notes: The figures show the relationship between rank child mortality and parental education for non-adoptees and the large sample of adoptees (the exception is Adoptees – Biological Father which, by construction, is based on the smaller adoptee sample). Parental educational attainment is divided into three groups: Compulsory education=7 years, Short post compulsory>7 years and < 11 years, Secondary education or more>=11 years of schooling.

Table 7.Cox proportional hazard model estimates of the associations between parental years of schooling and child mortality

	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Large sample
Years of education, Bio Mother	0.9769***	1.0445*	0.9895
	(0.0015)	(0.0241)	(0.0150)
Years of education, Bio Father	0.9747***	0.9912	
	(0.0011)	(0.0180)	
Years of education, Ad Father		0.9945	0.9996
		(0.0158)	(0.0102)
Years of education, Ad Mother		0.9527**	0.9700**
		(0.0209)	(0.0138)
P-value joint significance			
Biological parents	0.0000	0.1685	0.4841
Adoptive parents		0.0206	0.0486
Observations	2,826,257	10,831	21,192

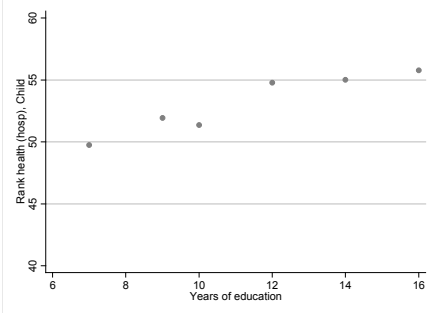
Notes: Results from Cox proportional hazard models. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children, and five-year intervals for parental cohorts. Column (1) is based on a sample of non-adopted children, column (2) on adoptees for whom we have information on all parents, and in columns (3) we add adoptees with unknown biological fathers.

Appendix Table C8 shows results for a flexible model estimating the effect for three different categories of educational attainment. The estimates indicate that the results for the adopting mother are driven by the mother's with secondary education or more. Table C9 shows the results corresponding to those of Table 7 separately for males and females. The results support a stronger association for males in the population. However, the precision is inferior for adoptees and we cannot reject equality of the coefficients between genders. Appendix Table C10 shows the results from a competing risk for parent education corresponding to that of intergenerational transmission of health explained in Section 5.1.1.

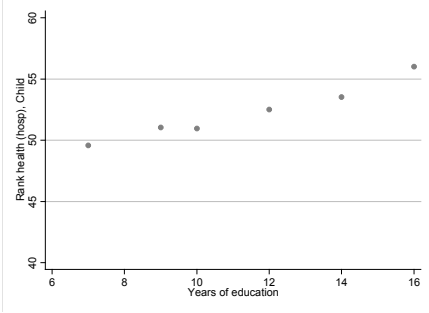
5.2.2 Health Measures Based on Hospitalization Data

Figure 5 shows the relation between parental education, measured in years of schooling, and child health, measured by our first hospitalization index. As expected, there is a visible pattern of increasing health by parental education in the samples of non-adoptees. This pattern also applies to all parental groups in the adoptee sample, except maybe for biological fathers, where the interpretation of the pattern is more ambiguous. The associations are reasonably well approximated by a linear relationship.[28]

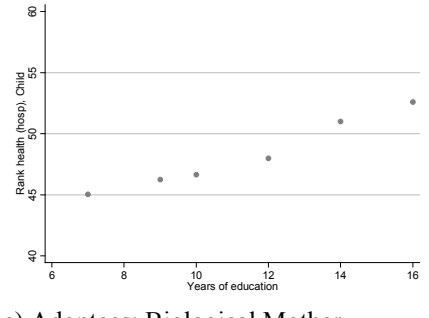
[28] There is a difference in the share with high education among biological and adopting parents. Among the adopted children's biological mothers, 4 % have more than 11 years of edu-



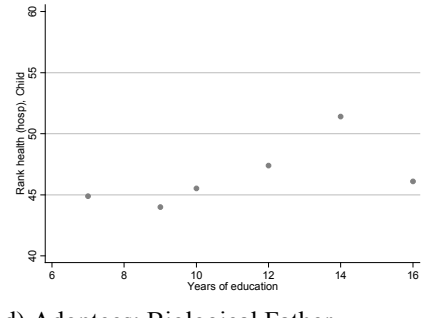
a) Non-Adoptees: Biological Mother



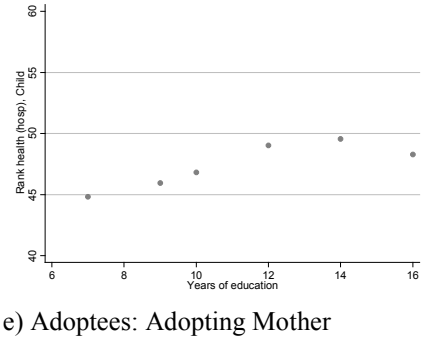
b) Non-Adoptees: Biological Father



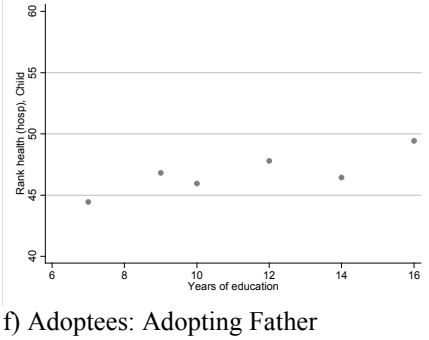
c) Adoptees: Biological Mother



d) Adoptees: Biological Father



e) Adoptees: Adopting Mother



f) Adoptees: Adopting Father

Figure 5. Mean rank child health (hospitalization based) and parental education.

Notes: The figures plot the relationship between rank child health (hospitalization based) and parental education for non-adoptees and the large sample of adoptees (the exception is Adoptees – Biological Father which, by construction, is based on the smaller adoptee sample). Because of small samples in some educational groups, we group these together. 11 years of schooling is plotted together with 12 years, 13 years of schooling is grouped together with 14 years, and 16 years of schooling contains all observations with more than 14 years of schooling.

cation and 2.4 % have 14 years or more. Among adopting mothers 10 % have more than 11 years of education, and 7.9 % have 14 years or more. Thus, the precision of the estimates for this highest educated group of mothers across the biological and adoptive mothers' samples varies significantly.

Table 8 shows the results from our linear regression model for the association between parental educational attainments and the ranks of the two indices based on child hospitalization. The results for non-adoptees, shown in Columns (1) and (4), reveal a highly significant association between the two measures of child health and the educational attainments of the parents. The results for adoptees confirm the significant association between adopting parents years of schooling and child health obtained in Section 5.2.1 using mortality as a measure of child health. Moreover, we now find the education of the biological parents to be jointly significant at the 5 percent level in all four specifications. A decomposition of the relative influence of the biological and adopting parents, respectively, gives 51.5 percent to the biological parents in the first specification using the "Hospitalization-based health" measure and 43.7 percent in the second specification. The corresponding shares for the "Health index" are 47.4 and 43.7 percent, respectively.

5.2.3 Birth Outcomes

Table 9 shows associations between parental educational attainments and grandchildren's birth outcomes. Once more, Panel A shows the results for the two measures based on birth weight and Panel B those on APGAR scores. The educational attainment of the biological parents has a significant effect on the health endowment at birth of grandchildren for all four measures among non-adoptees. An extra year of education among biological mothers is associated with a reduction in the probability of a biological grandchild of low-birth-weight by 2 percent relative to the mean.

For adoptees, the estimates in Table 9 are in general too imprecise to generate any significant results. However, it is worth noting that the educational attainment of the adopting parents is statistically significant for the two measures based on birth weight in the large adoptee sample and marginally significant in the smaller sample.

Table 8. Ordinary least squares estimates of the associations between parental years of schooling and child lifetime health.

	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Large sample	(4) Non-adoptees	(5) Adoptees - Bio father known	(6) Adoptees - Large sample
	Hospitalization-based health			Health index		
Years of education, Bio Mother	0.4064***	0.2205	0.4906***	0.3646***	0.1667	0.4476***
	(0.0092)	(0.1679)	(0.1132)	(0.0092)	(0.1683)	(0.1128)
Years of education, Bio Father	0.4876***	0.3119**		0.4372***	0.2715**	
	(0.0071)	(0.1336)		(0.0071)	(0.1326)	
Years of education, Ad Father		0.2038*	0.3863***		0.1460	0.3092***
		(0.1107)	(0.0783)		(0.1095)	(0.0778)
Years of education, Ad Mother		0.2974**	0.2451**		0.3406**	0.2673***
		(0.1389)	(0.1004)		(0.1374)	(0.0990)
P-value joint significance						
Biological parents	0.0000	0.0128	0.0000	0.0000	0.0457	0.0001
Adoptive parents		0.0003	0.0000		0.0005	0.0000
Observations	2,800,885	10,792	21,045	2,800,885	10,792	21,045

Notes: Results from OLS regressions. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children, and five-year intervals for parental cohorts. Columns (1) and (4) are based on a sample of non-adopted children, columns (2) and (5) on adoptees for whom we have information on all parents, and in columns (3) and (6) we add adoptees with unknown biological fathers. The dependent variable in columns (1)-(3) is a measure of hospitalizations and the dependent variable in columns (4)-(6) is a health index.

Table 9. Ordinary least squares estimates of the associations between parental years of schooling and firstborn grandchild's health at birth.

	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Large sample	(4) Non-adoptees	(5) Adoptees - Bio father known	(6) Adoptees - Large sample
Panel A	*Rank birth weight*			*Low birth weight<2,500 g*		
Years of education, Bio Mother	0.2510***	0.6255**	0.3376	-0.001***	0.0011	0.0012
	(0.0171)	(0.3030)	(0.2054)	(0.0001)	(0.0024)	(0.0017)
Years of education, Bio Father	0.1472***	-0.3452		-0.001***	-0.0009	
	(0.0137)	(0.2489)		(0.0001)	(0.0020)	
Years of education, Ad Father		0.4323**	0.3702***		-0.0024	-0.0022**
		(0.1968)	(0.1416)		(0.0016)	(0.0011)
Years of education, Ad Mother		-0.1598	-0.2889		-0.0007	-0.0011
		(0.2474)	(0.1786)		(0.0020)	(0.0014)
P-value joint significance						
Biological parents	0.0000	0.0685	0.1003	0.0000	0.8425	0.4856
Adoptive parents		0.0776	0.0316		0.1297	0.0120
Observations	789,908	3,468	6,396	789,908	3,468	6,396
Panel B	*APGAR 5 min*			*APGAR 5 min<10*		
Years of education, Bio Mother	0.0049***	0.0034	0.0064	-0.0005**	0.0003	-0.0010
	(0.0006)	(0.0095)	(0.0065)	(0.0002)	(0.0043)	(0.0029)
Years of education, Bio Father	0.0018***	0.0043		-0.001***	-0.0037	
	(0.0005)	(0.0082)		(0.0002)	(0.0036)	
Years of education, Ad Father		0.0112*	0.0064		-0.0017	-0.0001
		(0.0066)	(0.0046)		(0.0028)	(0.0021)
Years of education, Ad Mother		-0.0113	-0.0036		0.0021	-0.0018
		(0.0094)	(0.0063)		(0.0037)	(0.0026)
P-value joint significance						
Biological parents	0.0000	0.7937	0.3210	0.0000	0.5892	0.7292
Adoptive parents		0.2196	0.3755		0.7914	0.6901
Observations	713,795	3,143	5,745	713,795	3,143	5,745

Notes: Results from OLS regressions. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children, and five-year intervals for parental cohorts. Columns (1) and (4) are based on a sample of non-adopted children, columns (2) and (5) on adoptees for whom we have information on all parents, and in columns (3) and (6) we add adoptees with unknown biological fathers. The dependent variable in columns (1)-(3) in Panel A is birth weight measured in grams and scaled into percentile ranks, and in Panel B, the APGAR measure at five minutes after birth ranges from 0-10. The dependent variable in columns (4)-(6) in Panel A and B is binary.

5.3. Including both Longevity and Educational Attainment

So far, we have estimated specifications using *either* parental health or parental education as independent variables. This is, as we explained in Sec-

tions 2 and 3, to avoid bias from mediating factors. However, by extending the model to include measures of both life expectancy *and* educational attainments of biological and adopting parents in the same model, we can potentially learn something about mechanisms underlying the observed associations. Since the model includes endogenous regressors, the results should be interpreted with caution.

The first column in Table 10 shows that, for the sample of non-adoptees, the inverse effect of parental education is robust to the inclusion of controls for parental health. Hence, the longevity of the parents is not a mediating factor in the relationship between educational attainment of parents and children's mortality. This is true also for the results for adoptees shown in Columns 3 and 5. The estimates remain very similar as compared to the specifications with health and educational attainment of parents included separately. Thus, we find that the strong association with the biological parents' health is stable, and that the health of the parents is not a mediating factor in the association between children's mortality and parents' education.

From previous research, we know that there is a strong association between education and health for individuals in the same generation, as well as between parental and child educational attainments. This means that the effect of adopting parents' education on child health can be indirect, going through children's educational attainments,[29] rather than directly through influences during childhood and adolescence. Therefore, we also show results in an extended model where we include children's education in the specification. The estimates for parental health should now be interpreted as the intergenerational association in health that is not driven by the intergenerational association in education.[30] Estimates from this specification are shown in columns 2, 4 and 6 in Table 10.

In Column 2, for the sample of non-adoptees, we see that when we also include a control for the child's educational attainment, the estimates for parents' health are virtually unchanged, showing that the intergenerational association in mortality is not driven by the intergenerational association in education. Turning to the estimates for parental education, we see that the effect of parental education is wiped out and the coefficient even changes signs. In the context of the model suggested by Heckman (2007), this result would be characterized as a dynamic complementarity; the improved educational attainment would help the individual acquire more resources to improve his or her health status.

Columns 4 and 6 show the corresponding results for adoptees. The result in these columns differs somewhat for those obtained from the sample of non-adoptees. The estimates for adopting parents' education are now insig-

[29] This is labeled the "pathway hypothesis" for the relation between income and health (see e.g. Marmot et al., 2001).
[30] See Boserup et al. (2014) for a discussion of the assumptions underlying this interpretation.

nificant. However, this is most likely due to inferior precision in this set of estimates.

Table 10. Cox proportional hazard estimates of the associations between percentile rank of parental longevity, years of schooling and child mortality

	(1)	(2)	(3)	(4)	(5)	(6)
	Non-adoptees		Adoptees - Bio father known		Adoptees - Large sample	
Longevity, Bio Mother	0.9954***	0.9956***	0.9946***	0.9944***	0.9959***	0.9953***
	(0.0001)	(0.0001)	(0.0014)	(0.0015)	(0.0009)	(0.0010)
Longevity, Bio Father	0.9966***	0.9965***	0.9928***	0.9934***		
	(0.0001)	(0.0001)	(0.0014)	(0.0015)		
Longevity, Ad Mother			0.9993	0.9994	0.9990	0.9991
			(0.0014)	(0.0014)	(0.0009)	(0.0009)
Longevity, Ad Father			1.0003	1.0000	0.9998	0.9998
			(0.0013)	(0.0014)	(0.0009)	(0.0009)
Years of education, Bio Mother	0.9814***	1.0055***	1.0558**	1.0741***	0.9943	1.0121
	(0.0015)	(0.0017)	(0.0243)	(0.0263)	(0.0151)	(0.0163)
Years of education, Bio Father	0.9801***	1.0111***	1.0025	1.0187		
	(0.0011)	(0.0012)	(0.0183)	(0.0193)		
Years of education, Ad Mother			0.9577**	0.9631	0.9724**	0.9748
			(0.0210)	(0.0225)	(0.0138)	(0.0151)
Years of education, Ad Father			0.9984	1.0179	1.0018	1.0260**
			(0.0159)	(0.0174)	(0.0103)	(0.0115)
Years of education, Child		0.9003***		0.8590***		0.8752***
		(0.0009)		(0.0155)		(0.0103)
P-value joint significance						
Biological parents	0.0000	0.0000	0.0000	0.0000	0.0001	0.0000
Adoptive parents			0.2130	0.5725	0.1897	0.1597
Observations	2,826,257	2,770,240	10,831	10,693	21,192	20,882

Notes: Results from Cox proportional hazard models. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children, and five-year intervals for parental cohorts. Columns (1)-(2) are based on a sample of non-adopted children, columns (3)-(4) on adoptees for whom we have information on all parents, and in columns (5)-(6) we add adoptees with unknown biological fathers. Adding children's years of schooling in columns (2), (4) and (6) reduces the number of observations slightly because we do not have educational attainment for all children.

In Appendix Tables C11 and C12, we show results from a specification similar to that in Table 10, but with the two hospitalization measures as dependent variables. The results from this exercise are very similar to the corresponding specification for mortality. As for mortality, the conditional estimates are not very different from the unconditional ones. When we also control for children's education, the association between child hospitalization

and parental education disappears, again supporting existence of dynamic complementarities.

5.4. Sensitivity Analyses

5.4.1 Parameter Robustness and the Altonji-Elder-Taber Test

As we described in Section 3, our strategy to identify the influence of pre- and post-birth factors on adult health depends on the assumption that the pre-birth parental characteristics are unrelated to the post-birth parental characteristics. More specifically, conditional on the observed parental characteristics, we assume that unobservable characteristics of the biological parents are uncorrelated with those of the adopting ones. A simple, and informal, way of empirically testing this assumption is to include and exclude the observable parental characteristics to check the stability of the coefficient estimates of main interest.

Table 11 reports results from a robustness check for the two key results obtained in Section 6.1. Panel A shows the results for life expectancy and Panel B those for educational attainments. Column 1 shows the results for the biological mother when we include no other parental controls except indicators for the birth cohort of the biological mother and columns 2 and 3 report the results when we successively add variables for the observable characteristics of the adopting parents. Column 4 shows the results for the adopting parents when we only include indicators for year of birth of the adopting mother in the model. Columns 5 and 6 show the results when we successively add variables measuring the characteristics of the biological mother.

The estimates show that the key results – the estimates for the variables *Longevity, biological mother* in Panel A and *Years of schooling, adopting mother* – are both remarkably robust with respect to different specifications. They also show that the small and statistically insignificant estimates *for Longevity, adopting mother* and the *Years of schooling, biological mother* are very stable with respect to different specifications.

Table 11. Sensitivity analyses of mortality among adoptees

	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Parental life expectancy						
Longevity, Bio Mother	0.9958***	0.9959***	0.9958***		0.9958***	0.9956***
	(0.0009)	(0.0009)	(0.0009)		(0.0009)	(0.0014)
Longevity, Ad Mother		0.9990	0.9985	0.9987	0.9988	0.9989
		(0.0009)	(0.0012)	(0.0009)	(0.0009)	(0.0009)
Longevity, Ad Father		0.9998	0.9999	0.9997	0.9998	0.9997
		(0.0009)	(0.0010)	(0.0009)	(0.0009)	(0.0009)
Years of education, Ad Mother		0.9721**	0.9702**			
		(0.0138)	(0.0139)			
Years of education, Ad Father		1.0014	0.9989			
		(0.0102)	(0.0103)			
Years of education, Bio Mother					0.9900	0.9902
					(0.0147)	(0.0148)
Cohorts, Bio mother	Yes	Yes	Yes	No	Yes	Yes
Cohorts, Ad parents	No	Yes	Yes	Yes	Yes	Yes
Cause of death, Bio mother	No	No	No	No	No	Yes
Region, Bio mother	No	No	No	No	No	Yes
Cause of death, Ad parents	No	No	Yes	No	No	No
Region, Ad parents	No	No	Yes	No	No	No
Panel B: Parental years of schooling						
Years of education, Bio Mother	0.9837	0.9898	0.9882		0.9941	0.9945
	(0.0145)	(0.0150)	(0.0151)		(0.0151)	(0.0152)
Years of education, Ad Mother		0.9707**	0.9689**	0.9696**	0.9718**	0.9732*
		(0.0138)	(0.0139)	(0.0137)	(0.0138)	(0.0139)
Years of education, Ad Father		1.0002	0.9980	0.9985	1.0013	0.9992
		(0.0102)	(0.0104)	(0.0101)	(0.0102)	(0.0103)
Longevity, Ad Mother		0.9988	0.9984			
		(0.0009)	(0.0012)			
Longevity, Ad Father		0.9997	0.9998			
		(0.0009)	(0.0010)			
Longevity, Bio Mother					0.9959***	0.9956***
					(0.0009)	(0.0014)
Cohorts, Bio mother	Yes	Yes	Yes	No	Yes	Yes
Cohorts, Ad parents	No	Yes	Yes	Yes	Yes	Yes
Cause of death, Bio mother	No	No	No	No	No	Yes
Region, Bio mother	No	No	No	No	No	Yes
Cause of death, Ad parents	No	No	Yes	No	No	No
Region, Ad parents	No	No	Yes	No	No	No
Observations	21,192	21,192	21,192	21,192	21,192	21,192

Notes: Results from Cox proportional hazard models using the large sample of adoptees. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children. Each column is adding parental characteristics.

Altonji et al. (2005) suggest a framework for measuring the potential effect of an omitted variable bias relative to that avoided by the included confounders. This framework requires that three assumptions are fulfilled. Most importantly, it requires that the included confounders are "randomly selected" from a larger pool of possible confounders.[31] Building on this framework, Bellows and Miguel (2009) show that the ratio

$$\frac{\hat{\alpha}_{OLS,C}}{\hat{\alpha}_{OLS,NC}-\hat{\alpha}_{OLS,C}},$$

where $\hat{\alpha}_{OLS,C}$ is the coefficient estimate when confounders are included in the specification and $\hat{\alpha}_{OLS,NC}$ is the coefficient estimate when confounders are not included, measures how much the omitted variables must affect the key estimates, relative to the included confounders, in order to "explain away" these results.

To use this measure, we need to obtain OLS estimates corresponding to the Cox proportional hazard model results shown in Table 11. For this purpose, we estimate a linear probability model with an indicator variable that equals one if the individual has died before the end of the period that we are able to observe as the dependent variable in the data. In the model with no confounders, we only include indicators for year of birth for the adopted child along with the variable under study and parental cohort controls (corresponding to columns 1 and 4 in Table 11). In the model with confounders, we include all variables included in the specifications corresponding to Columns 3 and 6 in Table 11, respectively. The results from this linear probability model are presented in Appendix Table C13.

In the model where we estimate the association with the age at death of the biological mother, we get an estimate of -0.00029 (s.e. 0.00006) in the model with no confounders and -0.00029 (s.e. 0.00006) when confounders are included. The corresponding estimates for the adopting mothers' years of schooling are -0.00182 (s.e. 0.00065) and -0.00212 (s.e. 0.00067), respectively. These estimates give a value of 71.5 for the ratio corresponding to the age at death of the biological mother and 7.1 for the years of schooling of the adopting mother.

Given the very high quality of the confounders that we are able to use in the regression, it is very unlikely that the unobservables would be 8.0 times stronger than the included ones. As a comparison, we note that Altonji et al. (2005) rejected the possibility that unobservable characteristics could account for 3.55 times what the included confounders make up for in the context of the effect of Catholic schools on the probability of high school graduation.

[31] "Randomly" should be interpreted as an approximation.

5.4.2 Is there any Evidence of "Nature-Nurture Interactions"?

An advantage of the regression-based approach to decomposing pre- and post-birth associations is that the model can very easily be extended to allow for interactions between pre- and post-birth characteristics ("nature-nurture interactions"). This can be done by adding interaction terms between adoptive and biological parents' characteristics.

The results from such analysis are reported in Appendix Table C14 for child's mortality, hospitalization and health index. In columns 1, 3 and 5, we interact the life expectancy of the adoptive parents with the life expectancy of the biological mother and in columns 2, 4 and 6, we interact the years of schooling of the adoptive parents with years of schooling of the biological mother. All models also include the main effects.

It is evident that interaction effects are mostly non-existent. Only one interaction estimate, out of 12, is statistically significant. In the bottom row, we report the p-value of a test of the interaction effects being jointly zero in each of the models. In column 3 we are close to rejecting no interaction effects, but since both interaction effects have different signs (and none is significant), we could not make any conclusive inference.

We also investigate whether there were "cross-interaction" between parents' life expectancy and schooling, by including 4 additional interaction terms in a model with main effects for both life expectancy and years of schooling for the parents, finding no evidence of such interaction effects (p-value=0.58: not shown in Table C14).

The fact that we cannot reject the absence of interaction effects is at least suggestive of independence of pre-birth and post-birth factors. Hence, the framework in section 2 can be reasonable approximated by an additive model between pre-birth factors h and post-birth factors represented by individual capabilities θ_t and investments I_t. This facilitates the interpretation of our adoption estimates and speaks against an alternative from of dynamic complementarities where later investments depend on pre-birth factors.

5.4.3 Adoptees that Move from their Municipality of Birth

A concern discussed in Section 4 is that the adoptee might still maintain significant contact with the biological parents even after adoption and thus, the characteristics of the biological parents would have effects beyond the in utero period. A related concern is that the biological parents may have pre-adoption contact with the adopting parents and are thereby able to intervene in the adoption process.

One way of limiting the effect of this concern is to restrict the sample to only include those adoptees who move out from their municipality of birth after the adoption. The results from Cox proportional hazard models on a sample restricted to movers are presented in Appendix Table C15. As is

evident from these results, the estimates are very robust to this sample restriction and the key results are still highly significant.

5.4.4 First Born Adoptees

In the final sensitivity analysis, we restrict the sample to include first born adoptees only. As discussed in Section 4, it is more likely that first-born children are adopted away simply because they were not planned by their biological parents and they are less likely to have any contact with their biological parents. The Cox proportional hazard estimates on this sample are reported in Appendix Table C16. Once more the main results are robust.

5.4.5 External Validity

One way of testing for external validity of our sample of adoptees to the overall population is to perform estimations for subsamples of non-adoptees and adoptees who share the same biological mother. These non-adopted and adopted children are likely to share pre-birth unobservables to a much higher degree than our independent samples of non-adoptees and adoptees.[32]

The estimates from this exercise are reported in Appendix Table C17. In the first two columns, we perform estimations using non-adoptees and in the last two columns, we perform estimations for their biological siblings that were adopted away. Reassuringly, we here find a very similar pattern of results as in our main Tables 4 and 7.[33]

6. Conclusions

Two facts about the formation of health and health inequality served as a point of departure for this study. First, that there is intergenerational persistence in mortality, although much weaker than for e.g. labor earnings and educational attainments (Herskind, 1987, reporting estimates of associations in longevity between 0.01 and 0.15 from Pearl, 1931, Cohen, 1964, and Wyshak, 1978). Second, there exists a strong association between parental educational attainments and child long-term health (Smith et al., 1997, and Marmot et al., 2001). Both these facts are confirmed in this study. We have also showed that the Cuhna-Heckman model could be used to understand the potential mechanisms behind these two facts from previous research.

The results obtained on mortality in this study suggest that the intergenerational persistence in mortality can be fully attributed to pre-birth factors, reflected in the longevity of the adopted children's biological parents and not

[32] This type of test was conducted in Björklund et al. (2006), using a very small sample and focusing on income and education of children.

[33] This is perhaps not too surprising given that we only find limited evidence of non-linear effects in our main estimations reported in Table 4 and Table 7.

to transmission of habits or health related behaviors, reflected in the adopting parents' longevity. On the relation between the educational attainment of the parents and child mortality, we found, on the contrary, that the association is mostly attributable to post-birth factors, associated with the adoptive parents' educational attainments.

To analyze the sensitivity of these results we first used alternative measures of longevity in the parent generation; we restricted the sample to the oldest half in the child generation; and we repeated the analysis in the two gender groups separately. To check the validity of the assumption of conditional random placement of the adoptees we used the Altonji-Elder-Taber procedure for assessing parameter stability; we restricted the sample to those who moved out from their municipality of birth after the adoption took place; and, finally, we restricted the sample to the first born of the biological mother. In all sensitivity checks, we find that our main results in the decomposition analysis on mortality, reported above, remain.

Our decomposition analyses using the two health indices and birth outcomes gave, as opposed to the mortality ones, results between pure pre- or post-birth influences on health formation. However, for the intergenerational persistence in health the results still suggest that pre-birth influences are more important for child health outcome: between ⅔ and ¾ is attributable to the biological parents. For the association between parental education and child health the results obtained using the health indices assigns almost half of the total impact to pre-birth influences.

In the framework of the Cuhna-Heckman model, "dynamic complementarity" is a key element in the formation of an individual's health. Such complementarity can take many different forms. Our estimates from the models including "nature-nurture" interactions, which could be interpreted as a form of dynamic complementarities, did not yield any significant results and no empirical support for the existence of this form of dynamic complementarities. However, the results from an extended model suggest that a mechanism behind the association between the adopting parents' schooling and child health, may be that the adopted children themselves obtain more education when placed in a family with higher education. This process can be viewed as a form of dynamic complementarity between human capital formation and health.

References

Akee, R., E. Simeonova, W. Copeland, A. Angold and J. Costello (2013) "Young Adult Obesity and Household Income: Effects of Unconditional Cash Transfers" *American Economic Journal: Applied Economics*, 5(2), 1-28.

Akee, R., E. Simeonova, W. Copeland and J. Costello (2016) "Can Household Income Affect Child Personality Traits and Behaviors?" NBER Working Paper 21562

Allmänna barnhuset (1955) *Adoption. Handbook for social workers and authorities responsible for adoption issues*. Stockholm.

Allmänna barnhuset (1969) *Adoption. Handbook for social workers and authorities responsible for adoption issues*. Stockholm.

Altonji, J. G., T. E. Elder, and C. R. Taber (2005) "Selection on Observed and Unobserved Variables: Assessing the Effectiveness of Catholic Schools" *Journal of Political Economy* 113(1), 151-184.

Bellows, J. and E. Miguel (2009) "War and local collective action in Sierra Leone" *Journal of Public Economics* 93(11), 1144-1157.

Bhalotra, S. and S. Rawlings (2013) "Gradients of the Intergenerational Transmission of Health in Developing Countries" *Review of Economics and Statistics* 95(2), 660-672.

Björklund, A., M.Lindahl, and E. Plug (2004) "Intergenerational Effects in Sweden: What Can We Learn from Adoption Data?" IZA, Discussion Paper 1194.

Björklund, A., M. Lindahl, and E. Plug (2006) "The Origins of Intergenerational Associations: Lessons from Swedish adoption data" *The Quarterly Journal of Economics*, 999-1028.

Black, S. E. and P. J. Devereux (2011) "Recent Developments in the Study of Intergenerational Mobility" in David Card and Orley Ashenfelter (eds.) *Handbook of Labor Economics*, 4, 1487-1541.

Black, S.E., P. J. Devereux, P. Lundborg, and K. Majlesi (2015a) *On the Origins of Risk-Taking* (No. w21332). National Bureau of Economic Research, forthcoming *Journal of Finance*.

Black, S.E., P. J. Devereux, P. Lundborg, and K. Majlesi (2015b) *Poor Little Rich Kids? The Determinants of the Intergenerational Transmission of Wealth* (No. w21409). National Bureau of Economic Research.

Black, S.E., P. J. Devereux and K. G. Salvanes (2007) "From the Cradle to the Job Market? The Effect of Birth Weight on Adult Outcomes of Children" *The Quarterly Journal of Economics* 122(1), 409-439.

Bohman, M.(1970) *Adopted Children and their Families.* Stockholm: Proprius.

Boserup, S. H., W. Kopczuk and C. Thustrup Kreiner (2014). "Intergenerational Wealth Mobility: Evidence from Danish Wealth Records of Three Generations." Working Paper, University of Copenhagen.

Brandén, G., M. Lindahl, and B. Öckert (2017) "The importance of nature-nurture interactions in skill formation: Evidence from a large sample of adoptees", Mimeo, IFAU.

Case, A. and A. Deaton (2015). "Rising morbidity and mortality in midlife among white non-Hispanic Americans in the 21st century" *Proceedings of the National Academy of Sciences*, *112*(49), 15078-15083.

Case, A., Lubotsky, D., & Paxson, C. (2002). Economic status and health in childhood: The origins of the gradient. *The American Economic Review*, *92*(5), 1308-1334.

Cesarini, D., M. Johannesson and S. Oskarsson (2014). "Pre-birth Factors, Post-birth Factors, and Voting: Evidence from Swedish Adoption Data" *American Political Science Review* 108(1), 71–87.

Cesarini, D., E. Lindqvist, R. Östling and B. Wallace (2016) "Wealth, Health, and Child Development: Evidence from Administrative Data on Swedish Lottery Players," *The Quarterly Journal of Economics*, 131(2), 687-738.

Chetty, R., Hendren, N., Kline, P., & Saez, E. (2014). "Where is the land of Opportunity? The Geography of Intergenerational Mobility in the United States". *The Quarterly Journal of Economics*, *129*(4), 1553-1623.

Chetty, R., M. Stepner, S. Abraham, S. Lin, B. Scuderi, N. Turner, A. Bergeron and D. Cutler (2016) "The Association Between Income and Life Expectancy in the United States, 2001-2014" *JAMA* 315(16): 1750-1766.

Cohen, B. H. (1964) "Family patterns of mortality and life span" *Quarterly Review of Biology*, 130-181.

Classen, T. J. (2010) "Measures of the intergenerational transmission of body mass index between mothers and their children in the United States, 1981–2004" *Economics & Human Biology* 8(1), 30-43.

Classen, T. J. and O. Thompson (2016) "Genes and the intergenerational transmission of BMI and obesity" *Economics & Human Biology 23*, 121-133.

Cunha, F., & Heckman, J. (2007). The Technology of Skill Formation. *The American Economic Review*, *97*(2), 31.

Currie, J (2008) "Healthy, Wealthy and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development" Journal of Economic Literature, Vol 47, No 1, pp 87-122

Currie, J. (2011) "Inequality at birth: Some causes and consequences" Working Paper. w16798. National Bureau of Economic Research.

Currie J. and Almond, D. (2011) "Human capital development before age five" in David Card and Orley Ashenfelter (eds.) *Handbook of Labor Economics*, *4*, 1315-1486.

Currie, J. and E. Moretti. (2002) "Mother's education and the intergenerational transmission of human capital: evidence from college openings and longitudinal data" Working Paper w9360. National Bureau of Economic Research.

Currie, J. and E. Moretti (2007) "Biology as Destiny? Short- and Long-Run Determinants of Intergenerational Transmission of Birth Weight" *Journal of Labor Economics* 25, 231-264.

Cutler, D. M. and A. Lleras-Muney (2006) "*Education and health: evaluating theories and evidence"* National Bureau of Economic Research, Working Paper w12352.

Herskind, A. M., M. McGue, N. V. Holm, T. I. A. Sørensen, B. Harvald and J. W. Vaupel (1996) "The heritability of human longevity: a population-based study of 2,872 Danish twin pairs born 1870-1900" *Human Genetics*, 97, 319-323.

Heckman, J. J. (2007) "The economics, technology, and neuroscience of human capability formation." *Proceedings of the national Academy of Sciences* 104.33: 13250-13255.

Hjalmarsson, R. and M. J. Lindquist (2013) "The origins of intergenerational associations in crime: lessons from Swedish adoption data" *Labour Economics*, *20*, 68-81.

Hjelmborg, J., Iachine, I., Skytthe, A., Vaupel, J. W., McGue, M., Koskenvuo, M., Kaprio, J., N. Pedersen and Christensen, K. (2006) "Genetic influence on human lifespan and longevity" *Human Genetics*, *119*(3), 312-321.

Holmlund, H., M. Lindahl, and E. Plug (2008) "The causal effect of parents' schooling on children's schooling: A comparison of estimation methods" *IZA Working Paper 3630, August* 2008

Holmlund, H., M. Lindahl, and E. Plug (2011) "The causal effect of parents' schooling on children's schooling: A comparison of estimation methods" *Journal of Economic Literature* 49(3), 615-651.

Johnston, DW, S Schurer, MA Shields (2013) "Exploring the intergenerational persistence of mental health: Evidence from three generations" *Journal of Health Economics* 32(6), 1077-1089.

Lindeboom, M., A. Llena-Nozal, and B. van Der Klaauw. (2009) "Parental education and child health: Evidence from a schooling reform." *Journal of Health Economics* 28(1), 109-131.

Lindquist, M., J. Sol and M. van Praag (2015) "Why Do Entrepreneurial Parents Have Entrepreneurial Children?" *Journal of Labor Economics* 33(2), 269-296.

Lundborg, P., A. Nilsson and D. O. Rooth (2014) "Parental education and offspring outcomes: evidence from the Swedish compulsory School Reform" *American Economic Journal: Applied Economics 6*(1), 253-278.

Marmot, M., M. Shipley, E. Brunner, and H. Hemingway (2001) "Relative contribution of early life and adult socioeconomic factors to adult morbidity in the Whitehall II study" *Journal of Epidemiology and Community Health 55*(5), 301-307.

Mazumder, B. (2011) "Family and Community Influences on Health and Socioeconomic Status: Sibling Correlations Over the Life Course," *The B.E. Journal of Economic Analysis & Policy* 11(3).

McCrary, J. and H. Royer (2011) "The Effect of Female Education on Fertility and Infant Health: Evidence from School Entry Policies Using Exact Date of Birth." *The American Economic Review* 101(1), 158-195.

Miller, B. C., Fan, X., Grotevant, H. D., Christensen, M., Coyl, D., & van Dulmen, M. (2000). "Adopted adolescents' overrepresentation in mental health counseling: Adoptees' problems or parents' lower threshold for referral?" *Journal of the American Academy of Child & Adolescent Psychiatry*, *39*(12), 1504-1511.

Nordlöf, B. (2001) "Svenska adoptioner i Stockholm 1918–1973 (Swedish adoptions in Stockholm 1918–1973)" FOU-rapport 2001:8 Socialtjänstförvaltningen, Stockholm Stad.

Oskarsson, S., K.-O. Lindgren and C. Dawes (2015) "It Runs in the Family". Mimeo. Department of Political Science, Uppsala University.

Pearl, R. (1931) "Studies on human longevity. IV. The inheritance of longevity". Preliminary report. *Human Biology*, *3*(2), 245.

Petersen, L., P. Kragh Andersen and T. Sørensen (2005) "Premature Death of Adult Adoptees: Analyses of a Case-Cohort Sample" *Genetic Epidemiology* 28, 376-382

Petersen, L., P. Kragh Andersen and T. Sørensen (2008) "Genetic and Environmental Effects on Mortality before Age 70 Years" *Epidemiology* 19(3), 472-476.

Piraino, P., S. Muller, J. Cilliers, and J. Fourie (2014) "The transmission of longevity across generations: The case of the settler Cape Colony" *Research in Social Stratification and Mobility* 35, 105–119.

Plug, E. (2005) "Does Family Income Matter for Schooling Outcomes? Using Adoptees as a Natural Experiment" *Economic Journal* 115, 879–906.
Preston, S., P. Heuveline, and M. Guillot (2000) *Demography: measuring and modeling population processes*. Blackwell Publisher Inc., Malden, USA.
Sacerdote, B. (2007) "How large are the effects from changes in family environment? A study of Korean American adoptees." *The Quarterly Journal of Economics* 122(1), 119-157.
Sacerdote, B. (2011), "Nature and Nurture Effects On Children's Outcomes : What Have We Learned From Studies of Twins and Adoptees?" in J. Benhabib, A. Bisin and M. O. Jackson, eds., *Handbook of Social Economics* (Amsterdam: Elsivier) Chapter 1.
Smith, G. D., Hart, C., Blane, D., Gillis, C., & Hawthorne, V. (1997) "Lifetime Socioeconomic Position and Mortality: Prospective Observational Study" *British Medical Journal*, *314*(7080), 547.
Solon, G. (1999) "Intergenerational Mobility in the Labor Market" in Orley Ashenfelter and David Card, eds., *Handbook of Labor Economics,* Volume III (Amsterdam: Elsevier) Chapter 29.
Solon, G. (2015). "What Do We Know So Far about Multigenerational Mobility?", Mimeo Michigan State University, January 2015
Sørensen, T., G. Nielsen, P. Kragh Andersen and T. Teasdale (1988) "Genetic and Environmental Influences on Premature Death in Adult Adoptees" *New England Journal of Medicine* 318(12), 727-732.
Socialstyrelsen (2009a) *The Swedish Cause of Death Registry.* Sweden, Stockholm. 2009.
Socialstyrelsen (2009b) *The Swedish National Patient Register. Inpatient diseases in Sweden 1987-2007.* Sweden, Stockholm. 2009.
Statistics Sweden (2012). "The Multi-generation register 2012. A description of contents and quality" Report BE96BR1301. Örebro: Statistics Sweden.
Thompson, O. (2014) "Genetic mechanisms in the intergenerational transmission of health" *Journal of Health Economics* 35, 132-146.
Trannoy, A., S. Tubeuf, F. Jusot, and M. Devaux (2010) "Inequality of Opportunity in health in France: a first pass" *Health Economics* 19(8), 921-938.
Wyshak, G. (1978) "Fertility and longevity in twins, sibs, and parents of twins" *Social Biology*, *25*(4), 315-330.

Appendix A: Adoptions in Sweden 1940-1967

A.1 The History and Institutions of Adoptions

Adoptions in the period when the children we study in this paper were born were very different from what they are today, in Sweden and in most other Western industrialized countries. At that time, adoptions dominated by children born in Sweden, and their biological parents were in most cases young and lacked economic resources, or were stigmatized by having an unplanned child, which prevented them from taking care of the baby. International adoptions, although started already in the 1950s, did not overtake domestic adoptions until in the late 1960s. We show the number of Swedish domestic adoptions by year in Figure 1 and as a share of all Swedish-born children in Figure A1.

Domestic adoptions in Sweden have been described in several previous academic works and government documents. Two studies, Bohman (1970) and Nordlöf (2001), use primary sources. Bohman (1970) gives a broad overview and presents results from different empirical comparisons between adoptees and non-adoptees. Nordlöf (2001) focuses on adoptions in the city of Stockholm between 1919 and 1973. She uses archival records from the Stockholm child welfare office (*Barnavårdsnämnden*), which administrated adoptions, to give a description of the adopted children and their families. Several empirical studies using adoptee data, e.g. Björklund et al. (2004) and Oskarsson et al. (2015), also give comprehensive overviews of adoptions in Sweden.

Sweden had its first law regulating adoptions in 1917. This law was changed on several occasions since it was first implemented. However, the original law prescribed several principles that are still in use. One such principle is that the adoption should be "in the best interest of the child", both regarding whether or not the adoption should take place at all and the choice of adopting parents. Another principle was that no payments were allowed between the adopting and the biological parents. Finally, the adopted child should have all the rights regarding inheritance from the adopting parents that their biological children would have had.[34]

[34] The main principle was that the adopted child's rights to inherit his or her biological parents were lost. However, until 1959, some legal connection was kept between the biological parents and the adopted child. These adoptions are sometimes called weak adoptions and entailed that the child was still the heir of her/his biological parents and they were responsible for supporting the child economically if the new adopting parents could not. These legal responsibilities did not imply any further contact between the child and the biological parents. From 1959 onwards, these kinds of weak adoptions do no longer occur and in 1971, all weak adoptions were retroactively made strong, i.e., all legal ties were also cut between the biological parents and the child.

The law also prescribed that the adoption should be finalized in a court decision. All administrative work preparing for the adoption, including all contacts with the biological and the adopting parents, was carried out by the child welfare offices (*Barnavårdsnämnderna*). An adoption could be cancelled if both the adopting parents and the child agreed on it when the child had reached the age of majority, or as the result of misbehavior of either party. The latter category included different kinds of abuse of the child as well as general criminal behavior. In 1944, the law was extended to also include major health problems and defects of the adopted child. However, Nordlöf (2001) concludes that cancellations of adoptions were extremely rare in the Stockholm area in the period 1918-1973.

A.2 The Biological Parents

Bohman (1970) and Nordlöf (2001) give a fairly consistent description of the mothers who gave up their children for adoption:[35] they were on average substantially younger than mothers who kept their children; they were, except for a few rare cases, unmarried or divorced; and they did, on average, have a lower socio-economic status as compared to the rest of the population, although the differences were quite small. The largest occupational category of these mothers in Nordlöf's study was maids (26 percent), followed by office workers (18 percent) and restaurant workers (15 percent). In most cases, the child was voluntarily given up for adoption with the predominant reason being lack of housing and economic resources for supporting the child. In some very rare cases, it was because the mother died when giving birth or because she suffered from severe health problems.[36]

Bohman (1970) has a description of the biological fathers. Similar to the mothers they were on average younger than those who did not give up their children for adoption; they had a slightly lower average education level, although the difference was quite small; and they had a higher rate of registered alcohol abuse and crime rate.

Nordlöf attributes the rapid decline in domestic adoptions by the end of the 1960s to changes in social policy – including the introduction of housing allowances, the improvement of general housing conditions, increased child allowances and the introduction of childcare. Other important changes in society were the reduced social stigma of having children without being

[35] In Section 4, Descriptive Statistics, we return to comparisons between the characteristics of the biological mothers who gave up their children for adoptions and those who did not.

[36] In our sample, this is very rare because of the sample restrictions we have made. The restrictions require that parents are present in the Census in the year 1960. Most of our adoptees were born in the period prior to that. However, in the later period, we have about 70 children who have mothers that died close to birth (own birth or adopted sibling's birth). Excluding these children does not affect our results.

married or being in a steady relationship, the increased availability and usage of contraceptives, and the liberalization of the legislation for abortion.[37]

A.3. The Adopted Child

Most adoptions took place when the child was an infant. The mother had to wait until she had recovered from delivery before she could make the final decision to give the child up for adoption. The child was therefore initially placed in a nursery home and thereafter placed in a prospective adoptive family. The recommendation was that the placement be made before the child was six months old and that the trial period should be between three to six months. If the trial period went well, the adoptive parents would apply to the court for a legal adoption decision.

The children underwent a medical examination before they were adopted. The recommendations for this procedure were described in the *Handbook for Social Workers* (see e.g. Allmänna barnhuset, 1955). Nordlöf (2001) writes that children with physical or mental defects were in general not adopted, but stayed in foster care homes. This was also true for children whose mothers were prostitutes or who were conceived after a rape.

Bohman (1970) finds no significant difference in health at age 10-11 between his sample of adoptees and a control group of non-adoptees of the same age. Oskarsson et al. (2015) interpret this lack of difference as a net effect of two counteracting forces. Adoptees are to a larger extent than non-adoptees born by low SES mothers, which would indicate that they have inferior health. However, as a result of the medical testing before the adoptions took place, children who were eventually adopted are positively selected from this group.[38] In addition, the adoptive parents do often represent higher SES households, which could also have a positive impact on adopted children's health.

We here use our data to show some results where we compare cause of death and health characteristics between adoptees and non-adoptees. Figure A2 shows the number of deaths by year of birth and broken down by the most common causes of death in our sample, which are circulatory diseases, cancer and all other causes of death.[39] The left-hand panel corresponds to the sample of adoptees and the right-hand panel to the comparison group of non-

[37] A law allowing abortion without any particular reason until the end of week 18 in the pregnancy was passed in 1974. However, it was preceded by a gradual increase in the number of abortions over the previous decade, as the necessary conditions for obtaining a legal abortion were relaxed.

[38] In Section 5 we compare the health status in our sample of adoptees to non-adoptees in the same age group.

[39] Note that the graphs with the share of deaths among adoptees are less smooth than the corresponding graphs for non-adoptees. This is because of the much smaller sample size among adoptees, especially for the early cohorts (for the number of adoptees by birth cohort, see Figure 1).

adoptees. Comparing the death rates in the two panels, it can be seen that it is somewhat higher among adoptees and that the graphs for adoptees are quite noisy as a result of small sample sizes. The share of deaths is quite low for the younger age groups. For the child generation, we therefore use proportional hazard models allowing for right censoring of date of death.

The top panel in Table A1 shows the distribution of all deaths by the main underlying cause of death observed in the sample of adoptees and the comparison group, respectively. The six most common causes of death according to the main chapter in the ICD 10 code are included together with a seventh category, "Other", corresponding to all causes not included in the six most common ones. The last column in Panel A of Table A1 shows the *p*-values for a test of equality between the shares of deaths in the two samples that can be attributed to each of the causes considered. The distributions are quite similar, although adoptees are somewhat less likely to die from cancer and more likely to die from diseases in the digestive organs and from mental disorders.[40]

Panel B in Table A1 shows the shares of the most common causes of hospitalization by main ICD 10 chapter for the groups of adoptees and non-adoptees, respectively. Similarly to the causes of death, the largest differences are in the diagnoses related to problems in the digestive organs and mental disorders.

A.4. The Adopting Parents

The legal requirements for adopting were few; adoptive parents had to be free of tuberculosis, sexually transmitted diseases and be at least 25 years old. In practice, local social authorities followed the recommendation that the adopting family should be young enough to be able to be the biological parents, they had to have adequate housing and they should be married. Furthermore, the father should have a steady income, also implying that the mother should be able to stay at home. The adopting family's suitability for taking care of a child was evaluated by the child welfare offices (*Barnavårdsnämnderna*).

Until 1944, families with own biological children were not allowed to adopt. Nordlöf (2001) documents that it was rare that these families were admitted to adopt also after 1944, since there was always a shortage of children available for adoption and the child welfare offices considered it a dis-

[40] The results in Table A1 are not adjusted for educational attainment or other measure of SES, which could explain the differences. We also know from previous research that adoptees have worse mental health than non-adoptees (see e.g. Miller et al., 2000). Included in digestive causes are K70 (ICD 10), which is an alcohol-related liver disease. The mean of K70 is 0.027 among adoptees, and 0.016 among non-adoptees. This implies that about half of the adoptees' digestive death is alcohol related, and the figure is slightly lower for non-adoptees. This does not explain the entire difference, however.

advantage to have own biological children. This convention did, to a large extent, rule out adoptions in the immediate families of the biological mother or father, such as their mothers or siblings. Nordlöf (2001) estimates such adoptions by close relatives to be only around 1 percent of all adoptions in her sample.

A.5. Matching of Children and Adopting Parents

The social workers were instructed to find adopting parents that fit the child given up for adoption (see e.g. Allmänna barnhuset, 1955). Characteristics such as height and eye color were mentioned in the instructions. However, as pointed out by Björklund et al. (2004), the information available to the social worker about the biological mothers was likely to be quite limited. This was also acknowledged in the instructions, which is reflected in the following quote: "The social worker's ambition to find an adoptive home that fits a specific child particularly well is often unrealistic. The important task is to find good adoptive parents who can be expected to give children in general good conditions."[41]

The prospective adopting parents were able to post requests for characteristics of the child based on heredity. According to Nordlöf (2001), very few used that opportunity in other ways than just stating that they wanted a "healthy child". In very rare cases there were requests for children of mothers with good grades in school. The biological mothers were also able to post requests concerning the prospective adopting parents. Again, very few used that opportunity. Nordlöf (2001) found one request for an "intellectual" and one for an "artistic" family in her material.

From the instructions to the social workers, there are no indications that direct matching on health status between the prospective adopting parents and the child took place.

[41] This quotation is originally from Allmänna barnhuset (1969) and was obtained by us from Björklund et al. (2004).

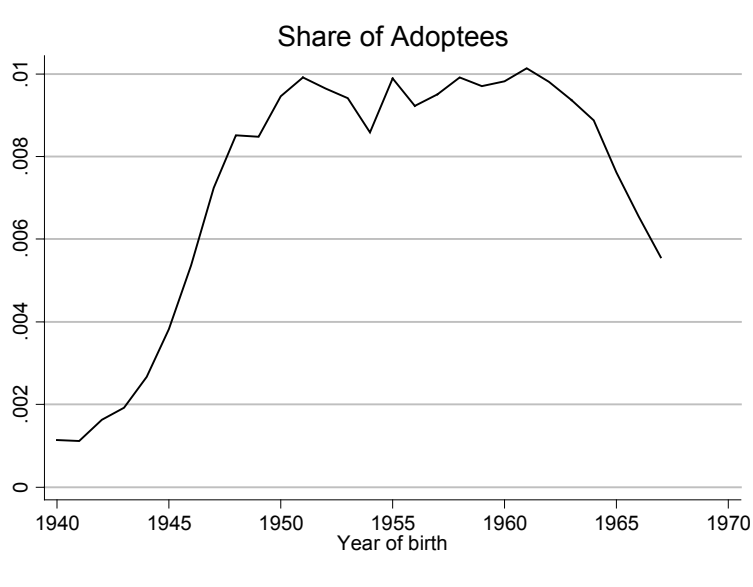


Figure A1. Share of adoptees of total number of children by year of birth.

Notes: The figure shows the share of children who were adopted by two parents, relative to non-adoptees, born in year 1940-1967 in Sweden.

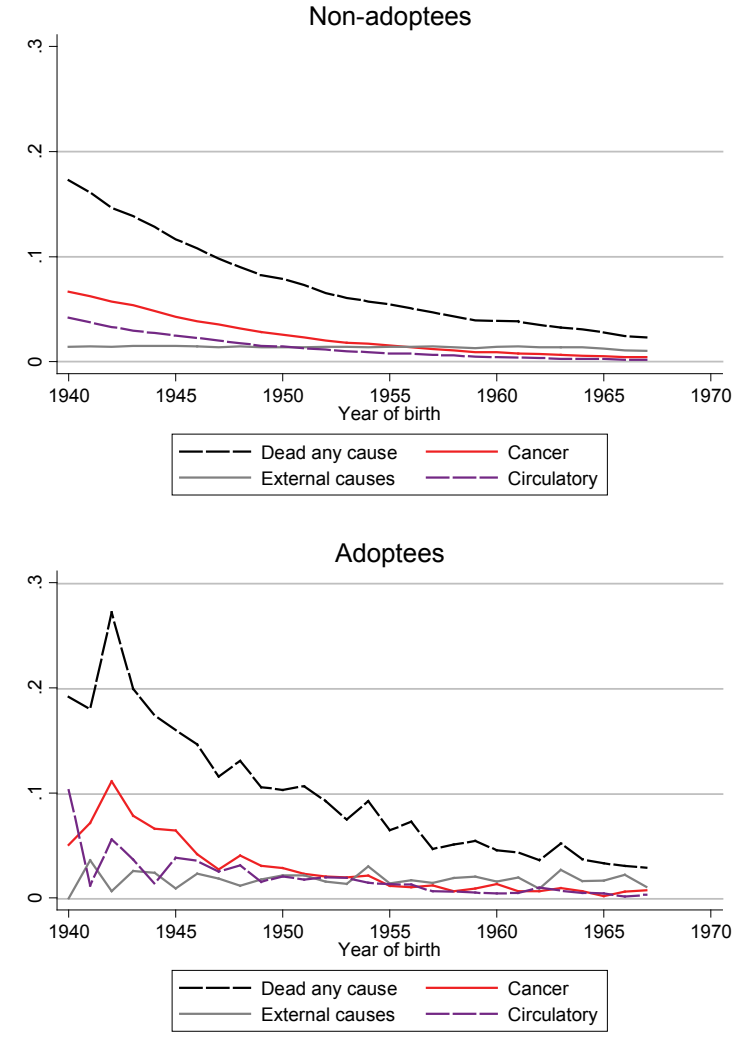


Figure A2. Share of individuals in the child-generation sample who died before April 1, 2013. Non-adoptees in the upper panel and adoptees in the lower panel.

Table A1. Share of deaths and hospitalizations by cause

	Non-adoptees (weighted)	Adoptees (large sample)	*p*-values mean diff
Panel A: Causes of death			
Cancer	0.301	0.254	0.0000
External causes	0.233	0.229	0.7064
Circulatory	0.165	0.181	0.0936
Digestive	0.039	0.059	0.0007
Mental	0.022	0.035	0.0048
Respiratory	0.030	0.032	0.6156
Other	0.210	0.209	0.9772
Share of deaths	0.058	0.077	0.0000
Tot # of deaths	200,350	1,634	
Panel B: Causes of hospitalization			
Cancer	0.095	0.071	0.0000
External causes	0.097	0.099	0.1464
Circulatory	0.092	0.082	0.0000
Digestive	0.097	0.089	0.0000
Mental	0.142	0.202	0.0000
Musculoskeletal	0.065	0.060	0.0000
Genitourinary	0.066	0.057	0.0000
Other	0.346	0.341	0.0017
Mean # hospitalizations/person	0.6037	0.6619	0.0000
Tot # hospitalizations	7,536,949	75,389	

Notes: In the third column *p*-values of test for equal share in the group of adopted and non-adopted children are shown. Non-adoptees are weighted by cohort size to be comparable with adoptees.

Appendix B: Predicted Age at Death

A problem with measuring longevity in the parental generation is that quite a large share of the parents, 41.1 percent for the biological parents and 28.0 percent for the adopting parents in the sample of adoptees, are still alive when we stop observing them in April 2013. To deal with this problem, we impute missing dates of deaths for those with a hospitalization record. This is done by first estimating a proportional hazard model based on the Gompertz distribution, which has turned out to provide good predictions of mortality up to the age of 90 (see Preston et al., 2000, and Chetty el al., 2016). We use the following specification of the index function:

$$Mortality_i = \alpha + \beta H(days)_{ai} + \gamma H(visits)_{ai} + \delta D_{ai} + \theta C_i \,, \quad (5)$$

where $H(days)_{ai}$ is the number of days individual i has been hospitalized at age a, $H(visits)_{ai}$ is the number of hospital visits during the same period, D_{ai} is a set of indicators capturing the conditions for which the individual has been hospitalized and C_i is a set of indicators for birth cohort. We use the first letter and the first number in the ICD10 to group diagnoses, giving us 200 indicators for various causes of hospitalization. We run this regression separately for women and men as their health care consumption and longevity develop differently. We also divide the sample into four different age groups (a), which are displayed below in table B1 (giving us eight different samples in total). The reason for splitting the sample by age is that hospitalization data are available in the years 1987-2011, giving us a window in which we observe individuals' hospital stays at different ages. An obvious problem when using hospitalization data to measure health is that we do not observe visits when an individual is deceased. Therefore, we limit the sample used in the estimation to those alive in all years. For example, the first sample is women born 1911-1921 and we observe their hospital records at ages 76-86. Then, we only use women still alive at age 86 in the regression. In the next step, we use the estimated model to predict unobserved age of death in the sample.

Table B1 displays the different samples and evaluations of our predictions. The first forecast evaluation measure is the mean absolute deviation in years, and the second is the correlation between predicted and observed age at death. We do well in predicting age at death for older cohorts and worse for younger cohorts. This might be because hospital visits and diagnoses are more informative about health and mortality in older ages. Note also that our main results in this paper remain very similar if we restrict the sample to those children born before 1953 (see Table C3). In this subsample, most parents have died and for those who have not, the error in predicting age of death is much smaller.

Table B1. Description of sample used to predict age at death among parents using hospitalization data, and evaluation of the prediction

Year of birth	1911-1921	1922-1931	1932-1946	1947-1953
Hospitalization age	76-86	65-75	55-65	48-58
Panel A: Women				
Mean absolute deviation (years)	2.481	4.662	8.537	8.053
Correlation predicted and observed age at death	0.359	0.405	0.554	0.471
Panel B: Men				
Mean absolute deviation (years)	2.244	3.998	7.226	7.412
Correlation predicted and observed age at death	0.342	0.382	0.529	0.447

Notes: The table displays the eight different samples used to predict age at death for parents still alive at the end of the observed period (ends in April 2013). In the regression, we use days in hospital, number of hospitalizations, indicators of diagnoses and birth cohorts. The regression is run separately by gender and age group. Only individuals alive at all ages (each group separately) are included in the regression. In a second step, the estimated model is used to predict age at death. Panel A shows the evaluation of the prediction among women, and Panel B the corresponding measures for men.

Appendix C: Additional Results

Table C1. Estimates from a proportional hazard model based on the Gompertz distribution of the associations between percentile rank of parental longevity and child mortality.

	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Large sample
Longevity, Bio Mother	0.9952***	0.9946***	0.9958***
	(0.0001)	(0.0014)	(0.0009)
Longevity, Bio Father	0.9964***	0.9932***	
	(0.0001)	(0.0014)	
Longevity, Ad Father		1.0000	0.9997
		(0.0013)	(0.0009)
Longevity, Ad Mother		0.9991	0.9987
		(0.0014)	(0.0009)
P-value joint significance			
Biological parents	0.0000	0.0000	0.0000
Adoptive parents		0.8148	0.3542
Observations	2,826,257	10,831	21,192

Notes: Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for birth. Column (1) is based on a sample of non-adopted children, column (2) on adoptees for whom we have information on all parents, and in column (3) we add adoptees with unknown biological fathers.

Table C2. Cox proportional hazard model estimates of the associations between parental longevity and child mortality. Parental longevity is measured by two variables; actual age at death among deceased, and a variable indicating who are still alive.

	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Large sample
Age at death, Bio Mother	0.9916***	0.9834***	0.9865***
	(0.0002)	(0.0040)	(0.0028)
Age at death, Bio Father	0.9928***	0.9868***	
	(0.0002)	(0.0039)	
Alive 2013, Bio Mother	0.7743***	0.8864	0.8795**
	(0.0050)	(0.0751)	(0.0503)
Alive 2013, Bio Father	0.7939***	0.7883**	
	(0.0066)	(0.0823)	
Age at death, Ad Mother		1.0001	0.9992
		(0.0043)	(0.0027)
Age at death, Ad Father		1.0009	1.0010
		(0.0039)	(0.0025)
Alive 2013, Ad Mother		0.8637	0.9407
		(0.0952)	(0.0722)
Alive 2013, Ad Father		1.0700	0.9250
		(0.1442)	(0.0891)
P-value joint significance			
Biological parents	0.0000	0.0000	0.0000
Adoptive parents		0.7407	0.7839
Observations	2,826,257	10,831	21,192

Notes: Results from Cox proportional hazard models. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for birth cohort of children, and five-year intervals for parental cohorts. Age at death is actual age at death among parents that have deceased (demeaned) and Alive is an indicator for being alive at the end of the observed period (April 2013). Column (1) is based on a sample of non-adopted children, column (2) on adoptees for whom we have information on all parents, and in column (3) we add adoptees with unknown biological fathers.

Table C3. Cox proportional hazard model estimates of the associations between percentile rank of parental longevity and child mortality. Child generation born before 1953 v/s after 1953.

	(1)	(2)	(3)	(4)	(5)	(6)
	Non-adoptees		Adoptees: Bio father known		Adoptees: Large sample	
	<53	>=53	<53	>=53	<53	>=53
Longevity, Bio Mother	0.9953***	0.9947***	0.9947**	0.9946***	0.9953***	0.9964***
	(0.0001)	(0.0001)	(0.0021)	(0.0019)	(0.0013)	(0.0013)
Longevity, Bio Father	0.9966***	0.9959***	0.9922***	0.9942***		
	(0.0001)	(0.0001)	(0.0020)	(0.0019)		
Longevity, Ad Father			0.9999	1.0004	0.9997	0.9999
			(0.0019)	(0.0018)	(0.0012)	(0.0013)
Longevity, Ad Mother			1.0008	0.9975	0.9993	0.9980
			(0.0020)	(0.0018)	(0.0012)	(0.0013)
P-value joint significance						
Biological parents	0.0000	0.0000	0.0000	0.0001	0.0003	0.0067
Adoptive parents			0.9123	0.3959	0.8317	0.3165
Observations	1,270,597	1,555,660	3,043	7,788	7,291	13,901

Notes: Results from Cox proportional hazard models. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for birth cohort. Columns (1), (3) and (5) consist of adoptees born before January 1st 1953, and (2), (4) and (6) consist of adoptees born January 1st 1953 or later. Columns (1)-(2) are based on a sample of non-adopted children, columns (3)-(4) on adoptees for whom we have information on all parents, and in columns (5)-(6) we add adoptees with unknown biological fathers.

Table C4. Cox proportional hazard model estimates of the associations between percentile rank of parental longevity and child mortality by gender.

	(1)	(2)	(3)	(4)	(5)	(6)
	Non-adoptees		Adoptees: Bio father known		Adoptees: Large sample	
	Women	Men	Women	Men	Women	Men
Longevity, Bio Mother	0.9950***	0.9952***	0.9941**	0.9948***	0.9966**	0.9953***
	(0.0001)	(0.0001)	(0.0023)	(0.0018)	(0.0016)	(0.0012)
Longevity, Bio Father	0.9969***	0.9961***	0.9965	0.9912***		
	(0.0001)	(0.0001)	(0.0022)	(0.0018)		
Longevity, Ad Father			1.0000	1.0001	1.0015	0.9989
			(0.0022)	(0.0017)	(0.0015)	(0.0011)
Longevity, Ad Mother			0.9964	1.0009	0.9986	0.9990
			(0.0022)	(0.0017)	(0.0015)	(0.0011)
P-value joint significance						
Biological parents	0.0000	0.0000	0.0105	0.0000	0.0286	0.0001
Adoptive parents			0.2843	0.8490	0.3947	0.3673
Observations	1,379,831	1,446,426	5,214	5,617	10,127	11,065

Notes: Results from Cox proportional hazard models. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for birth cohort. Columns (1), (3) and (5) consist of women and (2), (4) and (6) consist of men. Columns (1)-(2) are based on a sample of non-adopted children, columns (3)-(4) on adoptees for whom we have information on all parents, and in columns (5)-(6) we add adoptees with unknown biological fathers.

Table C5. Diagnosis codes for different categories

Diagnosis	ICD10
Cancer	C00-D48
Circulatory	I00-I99
Respiratory	J00-J99
External	S00-T98, V01-Y98
Mental	F00-F99
Digestive	K00-K93
Preventable	C33-C34, K70, K74.3-K74.6
Treatable	A15-A19, B90, C53, I05-I09, J00-J99, J45, J46, K35-K38, K40-K46, I10-I15, I60-I69, K80-K81

Table C6. Competing risk Cox proportional hazard model estimates of the associations between percentile rank of parental longevity and child mortality by cause of death.

	(1) Cancer	(2) Circ	(3) External	(4) Digestive	(5) Mental	(6) Resp	(7) Prev	(8) Treat
Panel A: Non-adoptees								
Longevity, Bio	0.9971***	0.9916***	0.9967***	0.9939***	0.9925***	0.9929***	0.9944***	0.9919***
Mother	(0.0001)	(0.0002)	(0.0002)	(0.0004)	(0.0005)	(0.0004)	(0.0004)	(0.0003)
Longevity, Bio	0.9983***	0.9926***	0.9983***	0.9941***	0.9938***	0.9943***	0.9938***	0.9930***
Father	(0.0001)	(0.0002)	(0.0002)	(0.0004)	(0.0005)	(0.0004)	(0.0004)	(0.0003)
P-value joint significance								
Biological parents	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
Observations	2,826,257	2,826,257	2,826,257	2,826,257	2,826,257	2,826,257	2,826,257	2,826,257
Panel B: Adoptees Bio father known								
Longevity, Bio	0.9951*	0.9886***	0.9970	0.9840***	0.9943	0.9903	0.9888	0.9914
Mother	(0.0029)	(0.0038)	(0.0028)	(0.0059)	(0.0068)	(0.0071)	(0.0071)	(0.0054)
Longevity, Bio	0.9945**	0.9913**	0.9913***	0.9979	1.0034	0.9982	0.9908	0.9919
Father	(0.0027)	(0.0035)	(0.0030)	(0.0049)	(0.0071)	(0.0072)	(0.0064)	(0.0055)
Longevity, Ad	1.0021	0.9996	0.9980	0.9975	0.9940	1.0029	1.0038	1.0002
Mother	(0.0027)	(0.0034)	(0.0027)	(0.0044)	(0.0059)	(0.0069)	(0.0054)	(0.0053)
Longevity, Ad Father	0.9994	1.0068**	1.0002	0.9983	1.0003	0.9964	1.0017	1.0025
	(0.0025)	(0.0031)	(0.0028)	(0.0046)	(0.0062)	(0.0071)	(0.0053)	(0.0052)
P-value joint significance								
Biological parents	0.0268	0.0002	0.0092	0.0238	0.6698	0.3586	0.0696	0.0963
Adoptive parents	0.7224	0.0852	0.7703	0.7291	0.5586	0.7537	0.7502	0.8930
Observations	10,831	10,831	10,831	10,831	10,831	10,831	10,831	10,831

Cont. next page

Cont. Table C6

	(1) Cancer	(2) Circ	(3) External	(4) Digestive	(5) Mental	(6) Resp	(7) Prev	(8) Treat
Panel C: Adoptees Large sample								
Longevity, Bio	0.9974	0.9914***	0.9999	0.9877***	0.9948	0.9893**	0.9991	0.9935*
Mother	(0.0019)	(0.0023)	(0.0019)	(0.0042)	(0.0043)	(0.0049)	(0.0043)	(0.0037)
Longevity, Ad	0.9995	0.9977	0.9984	0.9998	0.9975	1.0012	0.9952	0.9984
Mother	(0.0018)	(0.0022)	(0.0018)	(0.0033)	(0.0039)	(0.0043)	(0.0037)	(0.0036)
Longevity, Ad Father	0.9992	1.0025	1.0015	0.9969	1.0007	0.9993	0.9938*	1.0016
	(0.0017)	(0.0021)	(0.0019)	(0.0037)	(0.0040)	(0.0046)	(0.0036)	(0.0036)
P-value joint significance								
Biological parents	0.1721	0.0002	0.9784	0.0032	0.2284	0.0306	0.8283	0.0785
Adoptive parents	0.8673	0.3339	0.5003	0.7100	0.7786	0.9356	0.1023	0.8357
Observations	21,192	21,192	21,192	21,192	21,192	21,192	21,192	21,192

Notes: Results from Cox proportional hazard models. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort. Panel A is based on a sample of non-adopted children, Panel B on adoptees for whom we have information on all parents, and in Panel C we add adoptees with unknown biological fathers. The grouping of the different diagnoses is displayed in Table C5.

Table C7. Estimates from a proportional hazard model based on the Gompertz distribution of the associations between parental years of schooling and child mortality.

	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Large sample
Years of education, Bio Mother	0.9769***	1.0446*	0.9894
	(0.0015)	(0.0243)	(0.0150)
Years of education, Bio Father	0.9747***	0.9913	
	(0.0011)	(0.0182)	
Years of education, Ad Father		0.9948	0.9996
		(0.0159)	(0.0102)
Years of education, Ad Mother		0.9522**	0.9699**
		(0.0209)	(0.0138)
P-value joint significance			
Biological parents	0.0000	0.1706	0.4817
Adoptive parents		0.0202	0.0488
Observations	2,826,257	10,831	21,192

Notes: Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for birth for children, and five-year intervals for parental cohorts. Column (1) is based on a sample of non-adopted children, column (2) on adoptees for whom we have information on all parents, and in column (3) we add adoptees with unknown biological fathers.

Table C8. Cox proportional hazard model estimates of the associations between parental educational attainments measured in three education levels and child mortality. Compulsory education (7 years) is the excluded category.

	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Large sample
Edu >=9 & <11 yrs, Bio mother	0.9242***	1.1397	0.9905
	(0.0060)	(0.1014)	(0.0585)
Edu >=11 yrs, Bio mother	0.8135***	1.2085	0.8418
	(0.0103)	(0.2841)	(0.1374)
Edu >=9 & <11 yrs, Bio father	0.9210***	0.9767	
	(0.0066)	(0.0967)	
Edu>=11 yrs, Bio father	0.8479***	0.9774	
	(0.0066)	(0.1244)	
Edu >=9 & <11 yrs, Ad mother		0.9778	0.9759
		(0.0974)	(0.0659)
Edu >=11 yrs, Ad mother		0.6430**	0.7664**
		(0.1256)	(0.0919)
Edu >=9 & <11 yrs, Ad father		1.0062	1.1361*
		(0.1156)	(0.0871)
Edu >=11 yrs, Ad father		0.9356	0.9669
		(0.1079)	(0.0738)
P-value joint significance			
Biological parents	0.0000	0.6424	0.5729
Adoptive parents		0.1000	0.0308
Observations	2,826,257	10,831	21,192

Notes: Results from Cox proportional hazard models. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children, and five-year intervals for parental cohorts. Column (1) is based on a sample of non-adopted children, column (2) on adoptees for whom we have information on all parents, and in column (3) we add adoptees with unknown biological fathers.

Table C9. Cox proportional hazard model estimates of the associations between parental years of schooling and child mortality by gender.

	(1)	(2)	(3)	(4)	(5)	(6)
	Non-adoptees		Adoptees - Bio father known		Adoptees - Large sample	
	Women	Men	Women	Men	Women	Men
Years of education, Bio Mother	0.9819***	0.9738***	1.0756**	1.0224	1.0117	0.9752
	(0.0024)	(0.0019)	(0.0379)	(0.0311)	(0.0238)	(0.0192)
Years of education, Bio Father	0.9840***	0.9689***	0.9990	0.9839		
	(0.0017)	(0.0013)	(0.0300)	(0.0224)		
Years of education, Ad Father			1.0195	0.9803	1.0006	0.9995
			(0.0243)	(0.0208)	(0.0161)	(0.0131)
Years of education, Ad Mother			0.9388*	0.9595	0.9619*	0.9743
			(0.0317)	(0.0277)	(0.0215)	(0.0178)
P-value joint significance						
Biological parents	0.0000	0.0000	0.0924	0.6259	0.6224	0.2009
Adoptive parents			0.1729	0.0480	0.1709	0.2489
Observations	1,379,831	1,446,426	5,214	5,617	10,127	11,065

Notes: Results from Cox proportional hazard model estimates. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children, and five-year intervals for parental cohorts. Columns (1), (3) and (5) consist of women and (2), (4) and (6) consist of men. Columns (1)-(2) are based on a sample of non-adopted children, columns (3)-(4) on adoptees for whom we have information on all parents, and in columns (5)-(6) we add adoptees with unknown biological fathers.

Table C10. Competing risk Cox proportional hazard model estimates of the associations between parental years of schooling and child mortality by cause of death.

	(1) Cancer	(2) Circ	(3) External	(4) Digestive	(5) Mental	(6) Resp	(7) Prev	(8) Treat
Panel A: Non-adoptees								
Years of education, Bio Mother	0.9816***	0.9650***	0.9816***	0.9503***	0.9716***	0.9712***	0.9639***	0.9706***
	(0.0026)	(0.0038)	(0.0030)	(0.0077)	(0.0098)	(0.0086)	(0.0074)	(0.0064)
Years of education, Bio Father	0.9849***	0.9530***	0.9799***	0.9650***	0.9505***	0.9547***	0.9639***	0.9597***
	(0.0018)	(0.0026)	(0.0023)	(0.0054)	(0.0070)	(0.0062)	(0.0050)	(0.0045)
P-value joint significance								
Biological parents	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
Observations	2,826,257	2,826,257	2,826,257	2,826,257	2,826,257	2,826,257	2,826,257	2,826,257
Panel B: Adoptees – Bio father known								
Years of education, Bio Mother	1.0492	1.0218	1.0583	0.8966	1.0554	1.2210	1.1758*	1.0912
	(0.0474)	(0.0630)	(0.0425)	(0.0926)	(0.1209)	(0.1650)	(0.1148)	(0.1001)
Years of education, Bio Father	1.0135	1.0145	0.9731	0.9580	1.0724	0.8594	0.9874	1.0001
	(0.0344)	(0.0434)	(0.0350)	(0.0718)	(0.0816)	(0.0971)	(0.0841)	(0.0635)
Years of education, Ad Mother	0.9201*	1.0309	0.9246**	1.0125	0.9248	0.9489	0.9375	0.9409
	(0.0437)	(0.0530)	(0.0349)	(0.0715)	(0.1180)	(0.0864)	(0.1143)	(0.0809)
Years of education, Ad Father	0.9859	0.9533	1.0386	1.0305	0.9607	1.0017	0.8684*	0.9763
	(0.0307)	(0.0406)	(0.0317)	(0.0431)	(0.1049)	(0.0697)	(0.0735)	(0.0674)
P-value joint significance								
Biological parents	0.4876	0.8667	0.2976	0.3049	0.5151	0.2664	0.2434	0.5554
Adoptive parents	0.0895	0.5307	0.1157	0.7004	0.6202	0.8072	0.1123	0.5072
Observations	10,831	10,831	10,831	10,831	10,831	10,831	10,831	10,831

Cont. next page

Cont. Table C10.

	(1) Cancer	(2) Circ	(3) External	(4) Digestive	(5) Mental	(6) Resp	(7) Prev	(8) Treat
Panel C: Adoptees – Large sample								
Years of education, Bio Mother	0.9838	0.9957	1.0255	0.8455**	0.9968	1.0673	1.0321	1.0609
	(0.0300)	(0.0368)	(0.0281)	(0.0618)	(0.0749)	(0.0844)	(0.0712)	(0.0582)
Years of education, Ad Mother	0.9753	0.9840	0.9666	0.9684	1.0038	0.9930	0.9426	0.9763
	(0.0290)	(0.0322)	(0.0247)	(0.0516)	(0.0796)	(0.0633)	(0.0660)	(0.0523)
Years of education, Ad Father	0.9762	1.0092	1.0103	1.0424	0.9305	0.9730	0.9751	0.9751
	(0.0210)	(0.0247)	(0.0202)	(0.0327)	(0.0556)	(0.0427)	(0.0461)	(0.0429)
P-value joint significance								
Biological parents	0.5920	0.9083	0.3592	0.0216	0.9663	0.4102	0.6471	0.2811
Adoptive parents	0.1648	0.8774	0.4076	0.3995	0.4160	0.7417	0.4241	0.6087
Observations	21,192	21,192	21,192	21,192	21,192	21,192	21,192	21,192

Notes: Results from Cox proportional hazard models. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort for children, and five-year intervals for parental birth cohorts. Panel A is based on a sample of non-adopted children, Panel B on adoptees for whom we have information on all parents, and in Panel C we add adoptees with unknown biological fathers. The grouping of the different diagnoses is displayed in Table C5.

Table C11. Ordinary least squares estimates of the associations between parental longevity in percentile ranks, years of schooling and hospitalization based health in percentile ranks among children

	(1)	(2)	(3)	(4)	(5)	(6)
	Non-adoptees		Adoptees - Bio father known		Adoptees - Large sample	
Longevity, Bio Mother	0.0638***	0.0549***	0.0679***	0.0630***	0.0709***	0.0653***
	(0.0006)	(0.0006)	(0.0098)	(0.0098)	(0.0070)	(0.0070)
Longevity, Bio Father	0.0512***	0.0439***	0.0439***	0.0433***		
	(0.0006)	(0.0006)	(0.0101)	(0.0101)		
Longevity, Ad Mother			0.0080	0.0013	0.0121*	0.0045
			(0.0099)	(0.0100)	(0.0071)	(0.0071)
Longevity, Ad Father			0.0267***	0.0233**	0.0182**	0.0134*
			(0.0099)	(0.0099)	(0.0071)	(0.0071)
Years of education, Bio Mother	0.3217***	0.1420***	0.1067	-0.0505	0.3988***	0.2015*
	(0.0092)	(0.0094)	(0.1677)	(0.1682)	(0.1130)	(0.1136)
Years of education, Bio Father	0.3993***	0.1466***	0.2138	0.0704		
	(0.0071)	(0.0074)	(0.1346)	(0.1349)		
Years of education, Ad Mother			0.2670*	0.1840	0.2054**	0.1287
			(0.1390)	(0.1390)	(0.1005)	(0.1004)
Years of education, Ad Father			0.1419	0.0067	0.3402***	0.1824**
			(0.1108)	(0.1111)	(0.0784)	(0.0790)
Years of education, Child		0.8956***		1.1773***		1.1802***
		(0.0065)		(0.1190)		(0.0836)
P-value joint significance						
Biological parents	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
Adoptive parents			0.0005	0.0816	0.0000	0.0009
Observations	2,800,885	2,763,958	10,792	10,688	21,045	20,846

Notes: Results from OLS regressions. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children, and five-year intervals for parental cohorts. Columns (1)-(2) are based on a sample of non-adopted children, columns (3)-(4) on adoptees for whom we have information on all parents, and in columns (5)-(6) we add adoptees with unknown biological fathers. Adding children's years of schooling in columns (2), (4) and (6) reduces the number of observations slightly because we do not have educational attainment for all children.

Table C12. Ordinary least squares estimates of the associations between parental longevity in percentile ranks, years of schooling and health index in percentile ranks among children

	(1)	(2)	(3)	(4)	(5)	(6)
	Non-adoptees		Adoptees - Bio father known		Adoptees - Large sample	
Longevity, Bio Mother	0.0637***	0.0547***	0.0689***	0.0645***	0.0688***	0.0636***
	(0.0006)	(0.0006)	(0.0097)	(0.0097)	(0.0070)	(0.0069)
Longevity, Bio Father	0.0509***	0.0437***	0.0483***	0.0472***		
	(0.0006)	(0.0006)	(0.0100)	(0.0100)		
Longevity, Ad Mother			-0.0001	-0.0065	0.0086	0.0010
			(0.0098)	(0.0098)	(0.0070)	(0.0070)
Longevity, Ad Father			0.0200**	0.0168*	0.0172**	0.0119*
			(0.0098)	(0.0098)	(0.0070)	(0.0070)
Years of education, Bio Mother	0.2848***	0.0937***	0.0515	-0.1176	0.3602***	0.1562
	(0.0092)	(0.0094)	(0.1683)	(0.1688)	(0.1127)	(0.1132)
Years of education, Bio Father	0.3506***	0.0895***	0.1767	0.0464		
	(0.0071)	(0.0074)	(0.1334)	(0.1334)		
Years of education, Ad Mother			0.3112**	0.2236	0.2306**	0.1518
			(0.1377)	(0.1377)	(0.0992)	(0.0990)
Years of education, Ad Father			0.0874	-0.0586	0.2624***	0.0835
			(0.1097)	(0.1098)	(0.0779)	(0.0783)
Years of education, Child		0.9357***		1.2122***		1.2380***
		(0.0065)		(0.1178)		(0.0829)
P-value joint significance						
Biological parents	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
Adoptive parents			0.0045	0.1996	0.0000	0.0358
Observations	2,800,885	2,763,958	10,792	10,688	21,045	20,846

Notes: Results from OLS regressions. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children, and five-year intervals for parental cohorts. Columns (1)-(2) are based on a sample of non-adopted children, columns (3)-(4) on adoptees for whom we have information on all parents, and in columns (5)-(6) we add adoptees with unknown biological fathers. Adding children's years of schooling in column (2), (4) and (6) reduces the number of observations slightly because we do not have educational attainment for all children.

Table C13. Ordinary least squares estimates of the associations between parental longevity in percentile ranks, years of schooling and child mortality.

	(1)	(2)	(3)	(4)
Longevity, Bio Mother	-0.00029***	-0.00029***		-0.00025***
	(0.00006)	(0.00006)		(0.00009)
Years of education, Ad Mother		-0.001636**	-0.00182***	-0.00212***
		(0.000787)	(0.00065)	(0.00067)
Years of education, Ad Father		-0.000207		
		(0.000674)		
Longevity, Ad Mother		-0.000137		
		(0.000097)		
Longevity, Ad Father		-0.000015		
		(0.000084)		
Years of education, Bio Mother				-0.000494
				(0.000958)
Cohorts, Bio mother	Yes	Yes	No	Yes
Cohorts, Ad mother	No	Yes	Yes	Yes
Cohort, Ad father	No	Yes	No	No
Cause of death, Bio mother	No	No	No	Yes
Municipality, Bio mother	No	No	No	Yes
Cause of death, Ad mother	No	Yes	No	No
Cause of death, Ad father	No	Yes	No	No
Municipality, Ad parents	No	Yes	No	No
Observations	21,192	21,192	21,192	21,192

Notes: Results from a linear probability model using the large sample of adoptees. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children. Each column adds parental characteristics.

Table C14. Interaction effects. The associations between parental longevity in ranks and child health

	(1)	(2)	(3)	(4)	(5)	(6)
	Mortality (Cox)		*Rank Hospitalization health(OLS)*		*Rank Health index (OLS)*	
Longevity, Bio Mother	1.0000		0.0764***		0.0669***	
	(0.0026)		(0.0191)		(0.0190)	
Longevity, Ad Mother	0.9992		0.0294**		0.0194	
	(0.0016)		(0.0126)		(0.0125)	
Longevity, Ad Father	1.0024		0.0098		0.0086	
	(0.0016)		(0.0126)		(0.0125)	
Longevity, AdMother *BioMother	1.0000		-0.0003		-0.0001	
	(0.0000)		(0.0002)		(0.0002)	
Longevity, BioMother *AdFather	0.9999**		0.0003		0.0003	
	(0.0000)		(0.0002)		(0.0002)	
Education, Bio Mother		1.0199		0.2081		0.2222
		(0.0566)		(0.3802)		(0.3774)
Education, Ad Mother		0.9952		-0.1149		0.0275
		(0.0558)		(0.4050)		(0.4022)
Education, Ad Father		1.0030		0.4766		0.3358
		(0.0469)		(0.3278)		(0.3269)
Education, Ad Mother *BioMother		0.9970		0.0414		0.0274
		(0.0063)		(0.0454)		(0.0452)
Education, BioMother *AdFather		0.9996		-0.0100		-0.0029
		(0.0055)		(0.0374)		(0.0374)
P-value joint significance						
Interactions	0.1078	0.8354	0.2738	0.6147	0.4655	0.7758
Observations	21,192	21,192	21,045	21,045	21,045	21,045

Notes: Results from Cox proportional hazard models in columns (1)-(2) and OLS regressions in columns (3)-(6), using the large sample of adoptees. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children, and five-year intervals for parental cohorts. Columns (1), (3) and (5) display results for associations between parental longevity in percentile ranks and child mortality (1) and health (3 and 5), including interactions between biological mother and adopting parents' characteristics. Columns (2), (4), and (6) display associations between parental education and child mortality (2) and health (4 and 6), including interactions between biological mother and adopting parents' characteristics.

Table C15. Cox proportional hazard model estimates of the associations between parental longevity in ranks and child mortality. Sample restricted to those who moved out from the municipality of birth

	(1)	(2)	(3)	(4)
	Bio father known		Large sample	
Longevity, Bio Mother	0.9946***		0.9959***	
	(0.0015)		(0.0010)	
Longevity, Bio Father	0.9941***			
	(0.0015)			
Longevity, Ad Mother	0.9982		0.9986	
	(0.0014)		(0.0010)	
Longevity, Ad Father	0.9999		0.9999	
	(0.0014)		(0.0009)	
Years of education, Bio Mother		1.0284		0.9824
		(0.0262)		(0.0161)
Years of education, Bio Father		0.9837		
		(0.0194)		
Years of education, Ad Mother		0.9453**		0.9682**
		(0.0225)		(0.0149)
Years of education, Ad Father		1.0006		1.0029
		(0.0174)		(0.0111)
P-value joint significance				
Biological parents	0.0000	0.2722	0.0000	0.2776
Adoptive parents	0.4618	0.0248	0.3224	0.0760
Observations	9,302	9,302	18,225	18,225

Notes: Results from Cox proportional hazard models. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. The samples consist of adoptees with biological mothers living in a different municipality than their adopting mothers in the 1960 Census. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children, and five-year intervals for parental cohorts. Columns (1)-(2) are based on adoptees for which we have information on all parents, and in columns (3)-(4) we add adoptees with unknown biological fathers.

Table C16. Cox proportional hazard model estimates of the associations between parental longevity in ranks and adopted child mortality. Sample restricted to first-born children.

	(1)	(2)	(3)	(4)
	Bio father known		Large sample	
Longevity, Bio Mother	0.9963*		0.9955***	
	(0.0020)		(0.0012)	
Longevity, Bio Father	0.9925***			
	(0.0020)			
Longevity, Ad Mother	0.9978		0.9986	
	(0.0020)		(0.0012)	
Longevity, Ad Father	0.9986		0.9985	
	(0.0020)		(0.0012)	
Years of education, Bio Mother		1.0520*		0.9846
		(0.0311)		(0.0187)
Years of education, Bio Father		1.0220		
		(0.0235)		
Years of education, Ad Mother		0.9097***		0.9446***
		(0.0283)		(0.0179)
Years of education, Ad Father		1.0153		1.0043
		(0.0223)		(0.0135)
P-value joint significance				
Biological parents	0.0001	0.0861	0.0002	0.4140
Adoptive parents	0.4384	0.0073	0.2368	0.0060
Observations	4,990	4,990	11,302	11,302

Notes: Results from Cox proportional hazard models. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. The samples consist of firstborn children that were given up for adoption. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children, and five-year intervals for parental cohorts. Columns (1)-(2) are based on adoptees for which we have information on all parents, and in columns (3)-(4) we add adoptees with unknown biological fathers.

Table C17. The association between outcomes for non-adopted and adopted children and their biological parents, using the sample of siblings with biological mothers who have given up at least one child for adoption and who raised at least one biological child

	(1)	(2)	(3)	(4)
	Non-adoptees		Adoptees	
Longevity, Bio Mother	0.9946***		0.9957***	
	(0.0008)		(0.0011)	
Years of education, Bio Mother		0.9796		1.0053
		(0.0153)		(0.0182)
Longevity, Ad Father			1.0001	
			(0.0010)	
Longevity, Ad Mother			0.9982*	
			(0.0011)	
Years of education, Ad Father				1.0046
				(0.0122)
Years of education, Ad Mother				0.9721*
				(0.0165)
P-value joint significance				
Biological parents	0.0000	0.0017	0.0001	0.7689
Adoptive parents			0.2389	0.2205
Observations	27,989	27,989	14,851	14,851

Notes: Results from Cox proportional hazard models. Standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include indicators for gender and birth cohort of children, and five-year intervals for parental cohorts. Samples in columns (1)-(2) are based on a sample of non-adopted children who have a biological sibling that has been given up for adoption. Columns (3)-(4) are based on the corresponding adoptees, i.e. those that have biological siblings that were not adopted.

II. Neighborhoods and Youth Health: Everybody Needs Good Neighbors?

1. Introduction

There are large differences in health status across countries, but health also differs within countries and across groups of people (World Health Organization, 2008). This is also true in Sweden where life expectancy differs by approximately 4 years between areas with the highest and lowest longevity (Statistics Sweden, 2016). Is there a causal link between neighborhoods and health, or do people sort across areas in a way that produces these health disparities? There is plenty of evidence suggesting that the early years in life are important in forming later health outcomes (e.g. Heckman, 2007), therefore it is particularly relevant to study to what extent neighborhoods shape the accumulation of health capital among children and adolescents. This is also the aim of this paper.

There are several reasons why neighborhoods might influence the accumulation of health capital. The seminal work by Jencks and Mayer (1990) identifies four potentially important mechanisms: Peer effects, neighborhood role models, monitoring, and community resources. Peer effects related to health outcomes among adolescents could operate through learning risky behavior from friends such as drinking, smoking or having unsafe sex (see Card and Giuliano, 2013, on peer effects and sexual activity; Kremer and Levy, 2008, on alcohol; and Damm and Dustmann, 2014, on social interaction and criminal behavior). Peer effects could also be positive for health outcomes if it increases, for example physical activities, such as sports. Adult influences could work very much in the same way as peer effects by providing good or bad role models. The quality of local institutions, such as schools and health care, is potentially also important (Aizer and Currie, 2004). Other types of neighborhood characteristics could also matter for health outcomes such as proximity of gyms, parks and roads. The possibility of exercising nearby in a park or in a gym is probably beneficial for health, while living next to a highway is likely to be detrimental for health due to air pollution.

On average, residents living in poor areas have worse health than residents in more affluent areas. This relationship might not be causal since it is likely that there are factors that impact both families' residential location and children's health, such as family background. In other words, we cannot make any causal claims regarding neighborhood effects by simply comparing children growing up in different areas. This paper utilizes two different methods to try to handle the problem of selection. In the first part of the paper I use population wide data and estimate the effects of neighborhoods on youth health using data on families that move across the country. More specifically, I study whether children moving to areas where children have worse health outcomes when growing up, will experience deteriorated health in adolescence themselves. Since the choice of moving and where to live is endogenous, I exploit the timing of moves and compare children of different

ages that move into a neighborhood.[42] My data allow me to identify families which make it possible to control for family fixed effects. This way I am effectively utilizing the variation in siblings' different exposure time to an area to identify neighborhood effects. The importance of parental income varies across areas in Sweden; therefore I will also study neighborhood effects on youth health allowing the effect to vary with family income.

In the second part of the paper I utilize a governmental policy that assigned refugees to their initial neighborhood in Sweden. This policy was in place during the period 1985-1994, and meant that authorities placed refugees in suitable neighborhoods in a way that in practice offered potentially exogenous variation in neighborhoods, and allows me to study the effect of different areas on youth health.[43]

Studying movers and exposure time for areas has the advantage of estimating neighborhood effects from the entire distribution of Swedish families and places. This arguably offers high generalizability of the results. The more selective sample in the second part might of course limit the scope for generalization of the results, but the quasi-experimental design of the placement policy should on the other hand increase internal validity. Refugees are also a very interesting to study as they in most societies represent one of the most socioeconomically disadvantaged groups (e.g. OECD, 2017), and hence are potentially more susceptible to the neighborhood.

This paper documents large difference in the rate of hospitalized youth across areas in Sweden. Family income is also important for youth health, and the effect varies over the country. The first part of this paper, which uses movers across areas, confirms the association between neighborhoods and health found in previous studies. However, no statistically significant effects are found for exposure time to neighborhoods using variation between siblings in time spent in neighborhoods during childhood. To investigate if this result arises because there are no causal effects of neighborhoods on health, or because neighborhoods affect health instantly through contemporaneous environmental effects rather than through exposure time, I make use of the governmental policy that placed refugee families in their initial neighborhood. The results from the second empirical strategy confirm the findings in the first part of the paper. Together the results from the two parts imply that there are causal neighborhood effects on youth health, but these effects are instant and do not work through neighborhood exposure time.

[42] The first part of the paper follow quite closely the empirical method developed in Chetty and Hendren (2016). They estimate neighborhood effects on earnings, college attendance rates, and fertility and marriage rates by studying movers across commuting zones in the U.S. They find that the outcomes of children whose families move converge to those of permanent residents in the destination at a rate of approximately 4% per year.

[43] Several previous studies have used the placement policy to study the effects of living in different areas (e.g. Edin et al., 2003, on labor market outcomes; Åslund et al., 2011, on school performance; and Grönqvist et al., 2012, on income inequality and adult health).

Heckman (2007) provides a framework for thinking of how childhood environment effects the development of human capabilities. In the model, health is a function of parental capabilities (e.g. IQ, genes, education), previous periods' health and investments. A key feature is that the process is dynamic, e.g. previous periods capabilities affect the ability to attain higher capabilities in the next periods. This is often referred to as developmental effects, which stand in contrast to contemporaneous, or situational, effects (Sampson, 2012). In this paper, health outcomes are measured using data on hospitalizations in adolescence. The main outcome is hospitalization related to any condition, but I also study three specific conditions, mental illness, accidents, and risky behavior, which has been shown to be relevant in the previous neighborhood effects literature. I find an effect on all these health measures from moving to an area with worse health outcomes, however I do not find any support for exposure time effects. The risk of accidents might be related to the neighborhood through the physical local environment, or through local cultural behavior. Along the same lines, risky behavior might also be affected by the immediate presence of peers. Hence, accidents and risky behavior among youth are likely to be more closely related to contemporaneous relations and immediate surroundings rather than previous exposure. For example, moving to a new neighborhood in which the new classmates drink alcohol more frequently, there is potentially an immediate effect on the likelihood on drinking alcohol that is unrelated to the time spent in the new neighborhood. Alcohol consumption and health are related, and it is possible that there are developmental effects, or neighborhood exposure time effects, but that these are long-term and hence not captured studying health among adolescents. Apart from acute conditions related to heavy drinking, severe damages from alcohol consumption take some time to develop and hence maybe we should not expect to find any neighborhood exposure time effects on health outcomes related to alcohol consumption among adolescents, but possibly we need to measure health outcomes 30-40 years later.

This paper relates to a large literature, primarily in sociology, that has documented a correlation between places and children's life chances (e.g. Jencks and Mayer, 1990; Brooks-Gunn et al., 1993; and Haveman and Wolfe, 1995). Fewer studies have examined how neighborhoods are related to child and adolescent health (for reviews, see Leventhal and Brooks-Gunn, 2000, and Sampson et al., 2002). However, the main part of the existing literature cannot claim to estimate causal effects of neighborhoods. What we know about causal effects of neighborhoods on youth health is mainly based on data from housing mobility programs in the U.S, primarily the Moving to Opportunity (MTO) program.[44]

[44] There are a couple of studies on neighborhoods and mortality among youth. Votruba and Kling (2009) find substantial reductions in mortality among young black males from taking part in the Gautreaux housing program in Chicago. The effects were mainly driven by large

MTO operated during the 1990s in five cities in the U.S: Baltimore, Boston, Chicago, Los Angeles and New York. Interested families from high poverty census tracts were randomly assigned to an experimental group, a comparison group, or a control group. The experimental group received a voucher to live in a low poverty area and obtained counseling assistance. The comparison group received an unrestricted voucher and the control group did not get any additional assistance.[45] Katz et al. (2001) examine the short-run effects of MTO and they find lower prevalence of injuries and asthma attacks among children and fewer behavioral problems among boys, however no statistically significant effects for girls.[46] The interim study by Kling et al. (2007) documents positive effects on female youths, who experienced improved mental health and less risky behavior. However, in contrast with Katz et al. (2001), they find that the intervention had negative effects on boys, who experienced increased physical health problems and more risky behavior. Ludwig et al. (2013) study the long-term effects among the MTO participants 10-15 years after the intervention, and in line with the interim work they found positive effects on female youth's physical and mental health, while the results for males show that they did not benefit from moving.[47]

The experimental features of the MTO program have offered valuable insights of the causal effects of neighborhoods on health. However, by construction, the MTO studies are based on rather small and selective samples.[48] The first contribution of this paper is the use of population data; in the first part the total sample consists of almost 900,000 children of which 140,463 move once during childhood. In the MTO studies all families are initially living in very distressed areas and move to significantly better neighbor-

---

reductions in homicides. Jacob et al. (2013) study a housing voucher system in Chicago to which families were randomly assigned from a waiting list. Receiving a voucher decreased mortality rates for female children and youths, while the program did not have the same protective effect for males.

[45] Families were eligible for the MTO program if they had children and lived in public housing or assisted housing in a census tract with a poverty rate of 40 percent or more. Interested families with a complete application were selected from a waiting list and randomly assigned to an experimental group, a comparison group, or a control group (Katz et al., 2001).

[46] Katz et al. (2001) did not find any effects on earnings and employment from MTO. Neither did Kling et al. (2007) or Ludwig et al. (2013). The first to find effects in economic outcomes in the MTO program were Chetty et al. (2016) who focused on neighborhood exposure time and that found positive effects on children's income and college attendance among those moving before age 13. They did not have any data on health outcomes.

[47] Ludwig et al. (2013) also found positive effects of moving on adult health: lower BMI, psychological distress, and diabetes. This is in line with the findings in Katz et al. (2001), showing that families in the program experienced increased safety when moving, and adults' general and mental health improved, and Kling et al. (2007) showing that the MTO program had positive effects on adult mental health.

[48] Katz et al. (2001) have a sample of 612 children, Kling et al. (2007) have a sample of 749 (experimental) + 510 (Section 8) treated children, and Ludwig et al. (2013) have sample of 1,437 (experimental) + 1,031 (Section 8) treated children.

hoods, which raise the question of generalizability (Sampson, 2008). An advantage of the first part of this paper is that I estimate the effect of neighborhoods from movers across all areas in Sweden. Hence, the estimated neighborhood effects come from variation of all types of neighborhoods and families. Furthermore, estimating neighborhood effects from all types of families allow me to take into account that neighborhoods might differentially affect children's health depending on parental income. This might potentially be important as previous research has shown a strong relationship between parental income and children's health (e.g. Case et al., 2002, and Mörk et al., 2014).

The second main contribution of this paper is that I study the convergence in health outcomes. Previous research has focused on the effect of moving to more affluent areas, while in both parts of this paper I study how health outcomes in neighborhoods affects health of those moving in. By studying the convergence of health status I am able to more directly test the hypothesis of neighborhood effects without a priori taking a stand on what characteristics in a neighborhood that is important. The third main contribution of this paper to the literature on neighborhoods and health is the use of register data on hospitalizations to measure youth health.[49] The Swedish setting is particularly suitable because health care is free for all children, which likely limits the problem of different health care seeking behavior across groups. Furthermore, I make use of In-patient data which further limit the problem of different health seeking behavior, as it only records over-night stays that requires relatively poor health.

The rest of the paper is structured as follows: The data is described in Section 2, Section 3 presents the empirical specification and results for the first empirical strategy using families that move across the country, in Section 4, the placement policy, empirical specification and results for the second part of the paper is presented. Lastly, Section 5 concludes.

2. Data

The data used for the analysis come from merging several national administrative registers for children born 1984-1992 and their parents. Family links are identified through the Multigenerational Register (see Statistics Sweden, 2013), which contains a personal identifier of children and parents. Neighborhoods are defined as municipalities; a municipality is the smallest administrative unit in Sweden holding elections and collecting taxes. Schools and

[49] The studies of the MTO program use self-reported health status. The main concern with self-reported health is that it depends on social experience, i.e. so called reporting heterogeneity that could cause bias (Sen, 2002). This could be particularly problematic in studies of neighborhood effects if reporting bias is correlated with residential area. Another concern is attrition, which also might introduce bias.

kindergartens are also administered at municipal level making it suitable for studying children's development. There are 290 Swedish municipalities, which on average had 32,500 inhabitants year 2010.[50]

Data on place of residence is available from year 1985 onwards. Children's location cannot be directly observed in the data, but parental residential location is observed and it is reasonable to assume that the children in this sample live with their parents. If parents are living in different areas I assume that the child lives with her mother. If information on mother is missing (very few are) because of death or because she has emigrated, I use the father's location. Family income is defined as the sum of parents earnings averaged over age 2-15 of the child, and then ranked by child cohort in the national distribution.[51]

The Swedish setting offers high qualitative health measures by administrative hospitalizations records. Health outcomes are measured for ages 16-19 and data come from the In-patient register. Health care is free for all children in Sweden, which likely limits the problem of different health care seeking behavior across groups. Furthermore, the use of In-patient data further limits the problem of different health seeking behavior, as it only records over-night stays which requires relatively poor health. The limitation using administrative data for measuring health is that I will not be able to differentiate between health outcomes below the threshold of seeking health care. The main measure of overall health is hospitalization for any cause. Following the previous literature I look closer at hospitalizations due to mental problems, accidents, and risky behavior, which is defined as any hospitalization related to alcohol consumption, addiction, self-harm or teenage pregnancy.[52] Since only women are hospitalized for pregnancies, boys that become teenage fathers are identified from the Multi-generational register. Health outcomes are coded as dummy variables that equals 1 if hospitalized at least once during ages 16-19, and 0 otherwise. Table A1, in Appendix, relates the different diagnoses to specific ICD-codes.

There are significant differences in share of hospitalized youth across the country: the mean hospitalization rates ranges from 0.216 in Orsa (Dalarna

[50] It is possible that by defining a neighborhood to be this relatively large geographical unit I am not capturing the variation that exits within municipalities. However, in terms of estimation it is of importance that there are enough observations in each cell to be able to measure the quality of each neighborhood without too much noise. In relation to previous studies on neighborhood effects Swedish municipalities are relatively narrow, for example Chetty and Hendren (2016) use commuting zones (CZ) as their primary measure (average population about 430,000), and Bertrand et al. (2000) are using Public Use MicrodataArea (PUMA) (average population about 160,000) and the Metropolitan Statistical Area (MSA) (average population about 1,000,000)

[51] Mothers' age at birth is 28 on average and fathers is 31 for children in this sample. Measuring parental income over a 13-year period when the child is age 2-15 is therefore likely to be a good proxy for lifetime earnings (Haider and Solon, 2006).

[52] Previous studies also look at asthma and mortality. I do not study these health outcomes since hospitalizations related to asthma and youth mortality are very are among in Sweden.

county) to 0.93 in Olofström (Blekinge county). Figure 1, left panel, shows that the general pattern is that hospitalization rates are higher in the northern part of the country and also in an area in the south east. The right panel in Figure 1 shows the gradient in hospitalization rates, that is the relationship between parents' percentile income rank and child hospitalization. Darker areas in the figure represent a steeper gradient. The slope of the gradient varies substantially across the country, ranging from 0.20 to negative numbers; in 12 percent of the municipalities children in richer families have higher risk of being hospitalized than children in poorer families. The steepest slope is found in Bjurholm (Västerbotten county); a move from the 25th percentile to the 75th percentile income rank is associated with 8.5 percent decrease in hospitalization rate. A comparison between the left and right panel in Figure 1 reveals that there is not a strong correspondence between areas with high levels of hospitalizations and areas with a steep gradient. Thus, a neighborhood might treat children growing up there very differently depending on parental income. For example, a neighborhood where children on average fare well might be a bad place for poor children to grow up if all health-promoting activities are expensive, or if all housing available to low income families are located next to a highway.

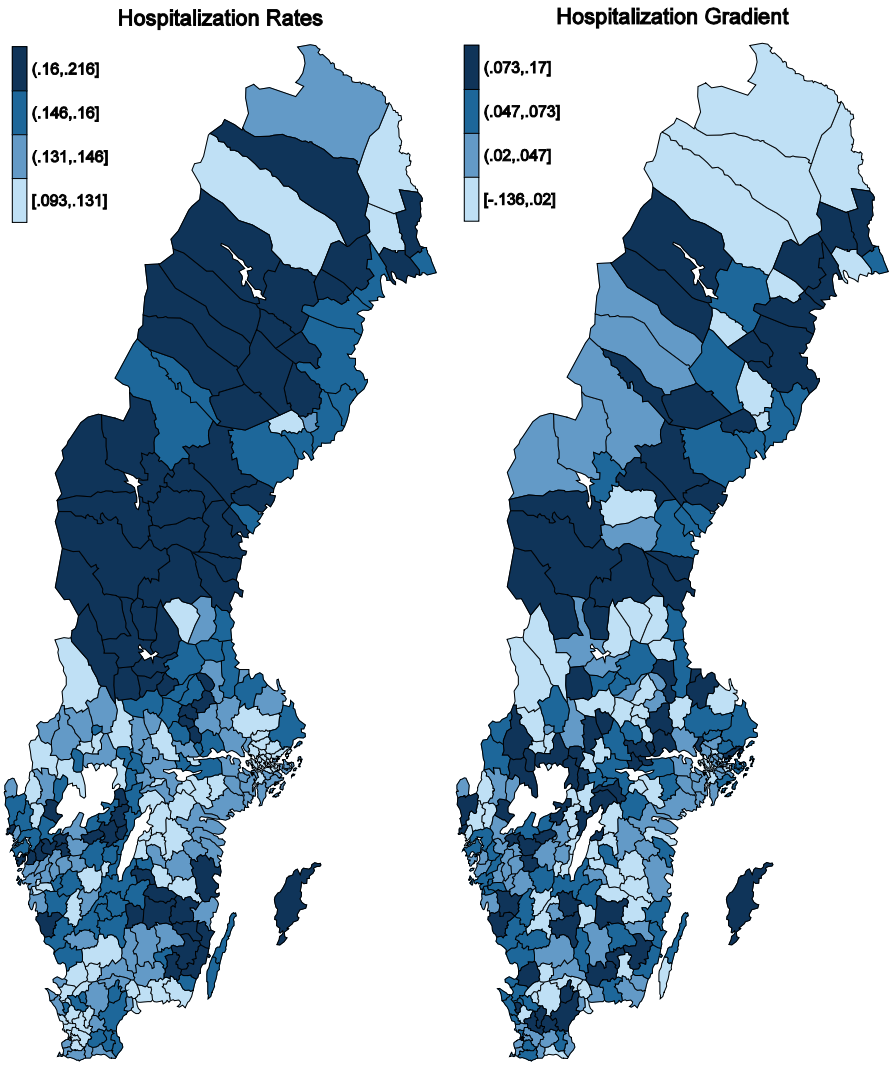


Figure 1. Hospitalization rates and health gradients across municipalities in Sweden

Notes: The left panel displays the share of hospitalized youth in each municipality among permanent residents. Darker areas represent higher share of hospitalized children. The right panel shows the slope of the gradient. Children in families with low income ranks generally have higher rates of hospitalizations than children in families with higher income rank. Darker areas are areas with steeper slope, i.e. the difference in hospitalization rates among poor children and rich children is larger. Both figures include controls for gender and birth cohort.

Figure A1 in Appendix, shows the relationship between parental income and youth health by child gender. The graphs show that the gradient for any hospitalization is rather linear, youth in the top decile has a hospitalization rate of 0.12, while youth in the bottom decile has a hospitalization rate of 0.18. The gradient is also pronounced for mental conditions and risky behavior in

the bottom half of the income distribution, but flattens out in the top half. Females have higher overall hospitalization rates than males, and they are more likely to be hospitalized for mental conditions and risky behavior. Males on the other hand have a higher hospitalization rate related to accidents. This pattern confirms that it is important to consider gender differences when studying neighborhood effects on health. Figure A2 in Appendix, shows that there is no strong time trend in hospitalizations; mean hospitalization rate is constant at 0.15 across birth cohorts.

3. Movers across Neighborhoods

The main research question in this paper is whether children moving to neighborhoods where children have worse health outcomes in adolescence, will experience deteriorated health themselves. In this section I will describe movers within Sweden, the empirical method applied to estimate the effect of neighborhoods on health using movers, and lastly the results.

3.1 Movers

Table 1 summarizes the main variables used in the analysis by moving status. Movers are evenly distributed across birth cohorts, and there is no clear difference in mean health outcomes of children. If anything, movers have slightly higher risk of being hospitalized for mental illness and risky behavior. Movers have parents that on average have lower income rank but also somewhat higher education. This could be explained by the difference in parental birth cohorts, movers' parents are on average almost a year younger. Children that move also have higher risk of having parents that have separated or being non-employed at some point in during childhood.[53] This could very well reflect the reasons for moving and could have an independent effect on the health outcomes of children. Hence, it might be important to include yearly controls for parental separation, employment and income in the analysis. [54]

[53] These are some of the strongest predictors for moving (see Mincer, 1978, on families moving decisions, and Heidrich, 2016, for a later discussion on the Swedish case).

[54] An individual is defined as employed if he performed at least one hour of paid work per week in November, otherwise he is defined as non-employed in that year. From 1990 and onwards there exist a variable that identifies families living together in the same housing property. Unmarried couples living together with a common child is also defined as a family. I define parents as separated in a year if they do not have the same family identifying number, for years 1985-1989 I use 1990's status. Family income is defined as the sum of biological parents earnings averaged over age 2-15 of the child, and then ranked by child cohort in the national distribution. Earnings are deflated by the Swedish Consumer Price Index, base year 2007.

Table 1. Summary statistics: Individual characteristics of permanent residents and movers

	Permanent		Movers	
	Mean	S.D.	Mean	S.D.
Female	0.48	(0.50)	0.49	(0.50)
Year of birth, child	1988.25	(2.55)	1988.24	(2.56)
Percentile rank income, parents	52.56	(27.65)	48.80	(30.80)
Years of schooling, father	11.67	(2.28)	12.08	(2.49)
Years of schooling, mother	12.17	(2.20)	12.39	(2.32)
Separated, parents	0.33	(0.47)	0.54	(0.50)
Unemployed, father	0.35	(0.48)	0.47	(0.50)
Year of birth, mother	1959.40	(5.60)	1960.18	(5.64)
Year of birth, father	1956.53	(6.41)	1957.22	(6.56)
Hospitalized	0.14	(0.35)	0.15	(0.36)
Mental illness	0.02	(0.15)	0.03	(0.17)
Accidents	0.03	(0.17)	0.03	(0.18)
Risky behavior	0.03	(0.16)	0.04	(0.19)
Observations	729,748		140,463	

Who are the families moving across neighborhoods in Sweden? Overall, about 23 percent of the children move at least once during ages 1-15.[55] Children in the lower part of the income distribution are overrepresented among movers, but children in the top of income distribution are also relatively frequent movers. Among movers, the large share only move once, however children in the lowest part of income distribution are overrepresented among more frequent movers. Table A2a in Appendix characterizes moves by parent income quintile. Table A2b displays the moving pattern among one time movers that are used in the analyses. A child is identified as a mover at age 2 if her parents moved between the year of the child's first birth day and the forthcoming year. The share of moves is rather evenly distributed across childhood, however most children move in early ages. This is likely due to the fact that parents with young children are on average younger and hence more inclined to move. The relatively large share of movers, and their representative characteristics, ensures high external validity of the results. Figure 1 shows that there is significant variation in health outcomes across the country. Figure 2 displays the distribution of change in neighborhood health outcomes when moving. For 40.6 percent of the children, health outcomes in destination area differ by more than one standard deviation from health outcomes in origin area.

[55] Chetty and Hendren (2016) show that 21 percent of children move at least once across commuting zones in their population wide data over the sample period (1996-2012).

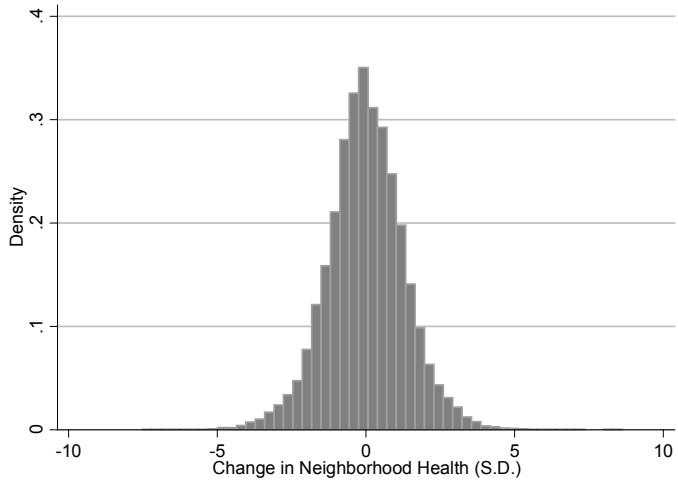


Figure 2. The distribution of change in neighborhood health (S.D.) when moving

3.2 Empirical Method[56]

The quality of a neighborhood is defined by the health outcomes at ages 16-19 of the permanent residents, which are defined as those not moving during childhood (ages 1-15). I am estimating the following model on the sample of movers:

$$h_i = \alpha_{oc} + \beta \overline{h}_{dc} + \gamma \boldsymbol{X}_i + \varepsilon_i,$$

where h_i is health outcome for individual i, α_{oc} is a fixed effect for the origin municipality by birth cohort c, $\overline{h}_{dc}$ is the health outcomes among permanent residents in the destination area d, among children born in cohort c (standardized with mean 0 and standard deviation 1). $\boldsymbol{X_i}$ includes indicators for child gender and birth order since we know from previous research that these factors are strong predictors of child health (Mörk et al., 2014, and Björkegren and Svaleryd, 2017). The health care system is organized at the county level in Sweden. Therefore, $\boldsymbol{X_i}$ also includes county fixed effects to be able to hold constant any differences in hospitalization rates due to organizational differences across areas.[57] For this model to capture causal effects of neighborhoods on health there cannot be any selection on destination area linked to individual health given origin, child cohort and gender. This is a strong assumption and the estimates from this model should therefore be interpreted with caution.

[56] This part follows quite closely the work by Chetty and Hendren (2016).

[57] A drawback of including county fixed effects is that they might also absorb some of the potential neighborhood mechanisms. For example, access to sports activities or closeness to emitting industries might be correlated within counties. However, to be able to accurately compare hospitalization rates across areas, county fixed effects are needed.

As previous studies have shown, parental income is an important predictor of child health. The gradient also has different slopes across areas in Sweden (see Figure 1). Therefore, I test whether children converge to their peers in the same part of the parental income distribution. Another reason for considering parental income is that income might capture housing area and hence a more narrow neighborhood. Family income is measured as the sum of biological parents' earnings and then averaged over childhood, ages 2-15.[58] The estimated model can now be written as:

$$h_i = \alpha_{qoc} + \beta \overline{h}_{qdc} + \gamma \boldsymbol{X}_i + \varepsilon_i,$$

where α_{qoc} is a fixed effect for origin o, by birth cohort c, by parental income quintile q, and $\overline{h}_{qdc}$ is the health outcomes among permanent residents in the destination area d, among children born in cohort c, with parents in income quintile q.

Previous studies have found that neighborhood effects increase linearly with exposure time for adult outcomes such as collage attendance, earnings, and marriage (Chetty and Hendren, 2016). To test if neighborhood exposure time matter for youth health, neighborhood quality is interacted with age at move, M_i[59]:

$$h_i = \alpha_{qocm} + \beta_1 \overline{h}_{qdc} + \beta_2 M_i \overline{h}_{qdc} + \gamma \boldsymbol{X}_i + \varepsilon_i,$$

where the fixed effect α_{qocm} now captures origin o, by birth cohort c, by parental income quintile q, by moving age m. This model estimates causal effects of neighborhoods under the assumption that selection effects do not vary with the child's age when moving. This assumption might be invalidated if families moving late are different from families moving with young children; parents moving with children in different ages might for example invest differently in their children and that could have an independent effect on youth health outcomes. Family fixed effects are added in the model to control for unobserved difference between families. The model then uses the variation between siblings in the exposure time for different neighborhoods. Adding family fixed effects does not solve the problem of other time-varying factors such as changes in family income that could change when moving and have an independent effect on youth health outcomes. Therefore, I add controls for yearly family income during childhood, as well as father employment status and an indicator of parental separation for each year.

The models presented above are computationally burdensome to estimate due to the large number of fixed effects. Therefore, as a baseline model I

[58] Income is deflated to be comparable over time (2007 years level).

[59] Age at move is linearly interacted with health in destination and is defined as 16 minus age at move. Effectively the interaction will estimate exposure time to new neighborhood. Exposure time is also tested in a more flexible specification using indicators of grouped age at move.

estimate a model that control parametrically for the main part of the fixed effects.[60] The estimated neighborhood effects come from estimating the difference in the quality of the origin and destination area, controlling for the outcomes in the origin area, in the following model:

$$h_i = \sum_{s=1984}^{1992} I(c_i = c)(\alpha_c^1 + \alpha_c^2 \bar{h}_{oc}) + \beta \Delta_{odc} + \gamma \boldsymbol{X}_i + \varepsilon_i,$$

where $I(c_i = c)$ is an indicator function that is equal to one when $c_i = c$ and 0 otherwise, α_c^1 is a cohort fixed effect and $\bar{h}_{oc}$ captures health outcomes among permanent residents in origin neighborhood and is allowed to vary over cohorts as it is interacted with cohort indicators. The second term, Δ_{odc}, is the neighborhood effect of interest, and the third term, $\boldsymbol{X}_i$, controls for gender, birth order, and county as before. As previously, the effect is also allowed to vary with parental income. This gives us the following specification:

$$h_i = \sum_{c=1984}^{1992} I(c_i = c)(\alpha_c^1 + \alpha_c^2 \bar{h}_{qoc}) + \alpha^3 p_i + \beta \Delta_{qodc} + \gamma \boldsymbol{X}_i + \varepsilon_i,$$

where the second term captures parental income rank, p_i and health in both origin and destination area are income quintile rank specific.[61] As in previous model I want to test whether being exposed longer to a neighborhood matters. Therefore I add exposure time to the model:

$$h_i = \sum_{c=1984}^{1992} I(c_i = c)\left(\alpha_c^1 + \alpha_c^2 \bar{h}_{qoc}\right) + \sum_{m=2}^{15} I(m_i = m)(\alpha_m^1 + \alpha_m^2 p_i) + \beta_1 \Delta_{qodc} + \beta_2 M_i \Delta_{qodc} + \gamma \boldsymbol{X}_i + \varepsilon_i,$$

where the first term controls for cohort and origin, the second term now contains indicators of moving age and allows this effect to vary with parental income rank, p_i. The third term capture the main effect of neighborhood health and the fourth term captures the exposure time effect of interest.[62] To the exposure time models I also add family fixed effects and time varying controls for parental income, separation and unemployment.

[60] As later shown, the results are not sensitive to the choice of model.
[61] p_i is percentile income rank. Ideally I would like to control for percentile rank also in the fixed effects models, however this yields too many fixed effects when interacted with neighborhood, cohort and moving age.
[62] Here as before, M_i is exposure time defined as 16 minus age at move.

3.3 Baseline Results

Children moving to a neighborhood where permanent residents have worse health outcomes will also experience deteriorated health themselves. Table 2 shows the baseline regression results for any hospitalization in column (1), hospitalizations related to mental conditions in column (2), accidents in column (3), and risky behavior in column (4). Holding constant health outcomes in origin, the results reveal that children moving to areas where children in their own birth cohort do worse, have an increased risk of being hospitalized. Moving to an area with one standard deviation higher hospitalization rates (any condition) increases the probability of being hospitalized in adolescence with 0.77 percentage points, which relative to the mean of the dependent variable corresponds to 5.0 percent. The analogous figure for hospitalizations related to mental health is 9.0 percent, for accidents 6.6 percent, and for risky behavior 9.2 percent relative to the mean.

Table 2. Association between neighborhood health (S.D.) and probability of being hospitalized

	(1) Hosp	(2) Mental	(3) Accident	(4) Risky
Δ Health	0.0077***	0.0028***	0.0021***	0.0034***
	(0.001)	(0.001)	(0.001)	(0.001)
Mean	0.153	0.031	0.032	0.037
Observations	140,463	140,463	140,463	140,463
N clusters	53,961	53,961	53,961	53,961

Notes: Results from linear probability models. Each column represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, indicators for cohort, birth order, county and gender. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

To test whether the convergence in health increases with time spent in the neighborhood as a child, exposure time is included in the model. Table 3 presents results from the model where health in destination area is interacted with exposure time, defined as the number of years the child spent in the destination area up until age 16. Column (1) includes all children moving once, in column (2) the sample is restricted to children in families for which I observe at least two siblings, column (3) adds family fixed effects to control for unobserved heterogeneity between families, and column (4) adds time-varying controls for family income, parental separation and father unemployment. Overall, the results show no evidence of convergence in health related to time spent in a neighborhood during upbringing.

Table 3. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Δ Hosp*Exposure	-0.0001	0.0001	-0.0002	-0.0002
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.153]	[0.150]	[0.150]	[0.150]
Δ Mental*Exposure	0.0000	-0.0000	-0.0002	-0.0002
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.031]	[0.031]	[0.031]	[0.031]
Δ Accident*Exposure	0.0001	0.0002	-0.0001	-0.0001
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.032]	[0.032]	[0.032]	[0.032]
Δ Risky*Exposure	-0.0000	-0.0002	-0.0003	-0.0003
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.037]	[0.037]	[0.037]	[0.037]
Time-varying controls	No	No	No	Yes
Observations	140,463	59,496	59,496	59,496
N clusters	53,961	29,483	29,483	29,483

Notes: Results from linear probability models. Each cell represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, differences in health between destination and origin, indicators for age at move, cohort, birth order, county and gender. Mean of dependent variables are shown in square brackets. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Given that family income is a strong predictor of child health, and because we know that there is a difference in the slope of the gradient between municipalities, it is potentially important to allow the effect to vary with parental income rank. The results are presented in the Appendix, Tables A3a-A3b. The estimate for hospitalization, any cause, is slightly weaker than the effects presented in Table 2, but still sizeable. The effect of moving to an area with one standard deviation higher hospitalization rates increases the risk of being hospitalized with 0.4 percentage points, which is 2.9 percent relative to the mean. For hospitalizations related to mental health and risky behavior, the estimates are very close to the estimates in Table 2. There are no statistically significant effects for accidents. Including exposure time effects, the results are in line with the results presented in Table 3, showing no evidence of exposure time effects.

3.4 Robustness

Tables A4-A5 in the Appendix present the results for the first model with all fixed effects. The results confirm that the parametric baseline model works very well and produces results that are close to the fixed effects models. Both the estimates for the overall association between places and health, and the exposure time effects, are almost identical to the baseline results showing sizeable correlations between neighborhoods and health, but no evidence of exposure time effects.

The exposure time effects models presented so far all are based on a linear model, assuming that there is a constant effect of neighborhood exposure in childhood on youth health outcomes. This might be too restrictive if for example there are certain ages during childhood when a child is particularly susceptible to the neighborhood. Therefore, I create indicators for moving age and interact these with neighborhood health. The results are presented in Tables A6a-A6d for all outcomes, and these results confirm previous findings that there is no evidence of any neighborhood exposure time effects on health in adolescence.

3.5 Heterogeneous Effects

Gender: Previous research has shown that neighborhoods often have differential effects on female and male health (e.g. Kling et al., 2007, and Ludwig et al., 2013). Therefore, I estimate the baseline models separately by gender. Results are presented in the Appendix, Tables A7a-A7b show effects for females, and Tables A8a-A8b present results for males. The association between places and health is very similar across gender, taking the difference in sample mean of the dependent variable into account. Again, there is no evidence of any exposure time effects.

Quality of neighborhoods: It might be the case that neighborhoods do not matter for children's health outcomes as long as neighborhoods are good enough. To test this I look closer at children in families that move to the neighborhoods with the poorest health outcomes. Places are ranked by the average health outcomes in respective category over all cohorts and income groups. The results in Tables A9a-A9b show small and insignificant neighborhood effects and no exposure time effects for children moving to the 50 worst places.

Parental income: I split the sample by parental income rank to test whether lower income children are more vulnerable to neighborhood conditions. Low income families are defined as families with parental income below the 20^{th} percentile. Tables A10a-A10b show estimates that are generally in line with the baseline results for all children, which imply that in this sample of Swe-

dish children, there is no support for the hypothesis that children born in families with lower income are more susceptible to the neighborhood.

Foreign background: We know from previous research that individuals with a foreign background on average have a lower socioeconomic status and hence might be more susceptible to the neighborhood influences. I define a child as having foreign background if both parents are born outside the Nordic countries. Tables A11a-A11b show no support for this hypothesis, however the sample size is very small.

3.6 Placebo Tests

In the first model, without exposure time effects, identification hinges on the assumption that there is no selection on destination area linked to individual health given origin, child cohort and gender. One way of testing this is if outcomes before moving are affecting later moving decisions. The rich data allow me to test this assumption directly. Health a birth, measured as hospitalizations related to perinatal and congenital malformations in the early period in life, ages 0-1, can be observed for cohorts born 1987-1992. Estimating the baseline specification without exposure time, where treatment is hospitalization (for any condition, mental illness, accidents, and risky behavior) ages 16-19 among permanent residents just as before, but individual outcomes in adolescence are replaced with health at birth, shows that there is little evidence of such selection. These results are shown in Table 4.

Table 4. Placebo test: Association between neighborhood health (S.D.) and health at birth

	(1) Hosp	(2) Mental	(3) Accident	(4) Risky
Δ Health	-0.0019*	-0.0017	-0.0006	-0.0001
	(0.001)	(0.001)	(0.001)	(0.001)
Mean	0.092	0.092	0.092	0.092
Observations	99,083	99,083	99,083	99,083
N clusters	36,826	36,826	36,826	36,826

Notes: Results from linear probability models. Each column represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, indicators for cohort, birth order, county and gender. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

The identifying assumption in the second model that explores neighborhood exposure time is that selection effects do not vary with the child's age when moving. To test this assumption I run the baseline specification with health at birth on exposure time. If the assumption is valid, there should be no effect of future moving pattern on previous health. The results, shown in Table 5, show

no evidence of selection that vary with child's age when moving (exposure time).

Table 5. Placebo test: Exposure time effects of neighborhood health (S.D.) and health at birth

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Δ Hosp*Exposure	-0.0000	-0.0001	-0.0001	-0.0002
	(0.000)	(0.000)	(0.000)	(0.000)
Δ Mental*Exposure	-0.0001	0.0003	0.0005	0.0005
	(0.000)	(0.000)	(0.000)	(0.000)
Δ Accident*Exposure	-0.0000	-0.0001	-0.0006	-0.0006
	(0.000)	(0.000)	(0.001)	(0.001)
Δ Risky*Exposure	0.0000	0.0002	0.0001	0.0001
	(0.000)	(0.000)	(0.000)	(0.000)
Time-varying controls	No	No	No	Yes
Mean	0.092	0.089	0.089	0.089
Observations	99,083	44,084	44,084	44,084
N clusters	36,826	21,154	21,154	21,154

Notes: Results from linear probability models. Each cell represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, differences in health between destination and origin, indicators for age at move, cohort, birth order, county and gender. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

3.7 Discussion

The analysis in this section shows that there is a correlation between places and adolescent health. Children in families that move to places where permanent residents have worse health outcomes, have worse health in adolescence themselves. However, the analysis do not show any evidence of exposure time effects. This result could arise if there are causal effects of neighborhoods on youth health but these are contemporaneous and independent of exposure time. In the entire sample, only 1.7 percent move across municipalities at age 15, which suggests that almost all children stayed in their destination neighborhood during late adolescence when health outcomes are measured. The other plausible explanation is that the association between places and health is entirely driven by selection. In the next section of I will try to determine whether there are causal effects of neighborhoods on adolescent health, and if any potential effects are contemporaneous or if exposure time matter. To this end, I make use of a governmental policy that placed refugees in their initial neighborhood of residence. This policy provides potentially exogenous variation in neighborhood for refugee children arriving to Sweden in the late 1980's and early 1990's.

4. Quasi-experiment: Refugee Placement Policy

In this section I utilize a government policy that placed refugees in their initial neighborhood of residence to study neighborhood effects on child health. First I present the refugee placement policy, then the data and empirical method is discussed, and lastly the results.

4.1 Refugee Placement Policy[63]

Sweden has a relatively large share of immigrant population, in 2015, 17 percent of the population of 9.9 million was foreign-born. During the 1970's, the previous labor immigrants were replaced with refugees and family reunification migrants. In the mid 1980's some municipalities were dissatisfied with the rise in the number of immigrants as they perceived this as a burden on the local budget. The government then gave the Immigration Board the task of assigning asylum seekers to suitable municipalities with the aim to speed up the integration process. Family reunification immigrants were exempted from the policy. In late 1980's the number of receiving municipalities increased from 60 to 277 of Sweden's then 284 municipalities.

When first arriving to Sweden, asylum seekers were placed in refugee centers while waiting for the residence permit decision. The refugee centers were placed all over Sweden and there was no correlation between port of entry and which center the asylum seeker were placed in. On average, the asylum seeker waited three to twelve months for residence permit. Thereafter, the refugee was assigned to the municipality where they had been given residence. Families were treated as a single unit, hence children moved with their parents to the new location. The aim of the policy was originally to place immigrants in neighborhoods where opportunities for finding a job and education were good. However, the Swedish housing market was booming at the same period which severely limited the possibility to perform this task. In practice this implied that refugees were placed where housing could be found.

The placement officers never met the refugees in person, but the officers had some information on the refugees they were placing; they knew their age, education, gender, marital status, family size and country of origin. Hence, it is crucial to control for these individual characteristics in the regression.[64] It was possible for refugees to state their preferred municipality. In practice few did this and for those who did the possibility to fulfill their

[63] This part of the paper summarizes what is known about the refugee placement policy and is based primarily on the previous work by Edin et al. (2003) and Åslund et al. (2011).

[64] Unfortunately, there is no good way of checking for balance, i.e. to study whether the placement was random given the observed characteristics. This is because it does not exist any information in the administrative data available that was not available for the placement officers.

preference was very limited. This was because most of those who stated a location preference wanted to be placed in the largest urban areas in Sweden: Stockholm, Gothenburg or Malmö. However, the explicit goal of the policy was to reduce the inflow to these areas, and the booming housing market made it very difficult to find vacant housing in these areas. Furthermore, since placement was made soon after receiving a residence permit, the joint probability of finding a vacant housing in the preferred neighborhood and receiving a residence permit at the same time was very low. The refugees were allowed to move after the placement, but they were still required to take part in an 18-month introduction program in their assigned municipality in order to qualify for social assistance during the initial period. Figure A3 in Appendix shows the share of children still living in their assigned municipality by year. The figure shows that eight years after arrival, about 50 percent were still living in their assigned municipality.

Table 6 shows that the refugee children arriving in this time period have much poorer parents on average but they have on average only slightly lower education than permanent residents in Sweden as expected. Almost all fathers have been non-employed/unemployed at least once during the observed period. The refugee children have somewhat lower risk of being hospitalized for any condition, even though differences are small.

Table 6. Summary statistics: Individual characteristics of refugees and permanent residents

	Refugees		Permanent	
	Mean	S.D.	Mean	S.D.
Female	0.48	(0.50)	0.49	(0.50)
Year of birth, child	1987.11	(2.36)	1988.24	(2.56)
Percentile rank income, parents	13.86	(13.92)	48.80	(30.80)
Years of schooling, father	11.12	(2.71)	12.08	(2.49)
Years of schooling, mother	10.56	(2.56)	12.39	(2.32)
Separated, parents	0.48	(0.50)	0.54	(0.50)
Non-employed/Unemployed, father	0.99	(0.09)	0.47	(0.50)
Year of birth, mother	1961.18	(5.51)	1960.18	(5.64)
Year of birth, father	1956.89	(6.29)	1957.22	(6.56)
Hospitalized	0.12	(0.33)	0.15	(0.36)
Mental illness	0.02	(0.13)	0.03	(0.17)
Accidents	0.02	(0.15)	0.03	(0.18)
Risky behavior	0.03	(0.17)	0.04	(0.19)
Observations	35,754		752,367	

4.2 Empirical Method

The setting in which the policy took place is the main arguments why the policy provide plausibly exogenous variation in the initial location (see previous applications in e.g. Edin et al., 2003; Åslund et al., 2001; Grönqvist et al., 2012). However, as pointed out in Nekby and Pettersson-Lidbom (2017),

the aggregated inflow of refugees is potentially correlated with unobserved municipality trends. However, for this study design I do not need to make any assumption about the correlation between the rate of inflow of refugees and potentially unobserved local characteristics (e.g. local political preferences).[65] The crucial assumption for a causal interpretation of the estimates is that families could not influence the placement, given the set of family characteristics known by the placement officer. Thus, it is important to control for all information given to the placement officers, luckily these variables are available in my administrate data. Unfortunately, it does not exist any published individual data on which were placed in the program. As previous studies, I have to use an indirect approach of identifying which individuals were placed through the program by combining information of year of arrival and region of origin.[66]

Following closely the baseline empirical model in part one, I am estimating the following model on the sample of refugee children:

$$h_i = \alpha + \beta \overline{h}_{dc} + \delta \boldsymbol{X}_i + \gamma \boldsymbol{Z}_f + \varepsilon_i,$$

where h_i is health outcome of individual i, $\overline{h}_{dc}$ is the health outcomes among permanent residents in destination area d, born in cohort c, $\boldsymbol{X}_i$ is a vector of individual characteristics including indicators of region of origin,[67] destination county[68], child cohort, gender, birth order and immigration year. $\boldsymbol{Z}_f$ is a vector of family characteristics including number of children, parental marital status, age at immigration and educational attainment.[69]

[65] This issue is of greater importance when the policy is used for studying the effect on people already living in the designated municipalities, e.g. Dahlberg, Edmark and Lundqvist (2012) study the effect on voter preferences for redistribution of refugee inflow. In contrast, in this study I examine the effect of being placed in a neighborhood on the outcomes of the refugee children.

[66] Region of origins are specified in Appendix, Table A12. Unfortunately exact country of origin is missing in the data for some regions. However, exact country of origin exists for immigrants arriving year 1985-1989. In Appendix, Tables A16a-A16b, results using the sample for which I observe exact country of origin is are presented. The results with country of origin fixed effects give very similar results as the model controlling for region of origin in the same sample.

[67] The region of origin fixed effects are likely to control for differential inclination to seek medical care as well as potential discriminatory behavior of the medial staff depending on ethnicity.

[68] Adding county fixed effects to the model controls for differences in the health care system across regions in Sweden. County fixed effects might also potentially solve a problem of the placement policy. As described, some refugees stated a preferred municipality. This wish was rarely met, however when it was met, these county fixed effects are likely to capture some of this effect as it controls for differences in preferences across places.

[69] Adopting the model from part one where neighborhood effects are allowed to vary with income, is more complicated in this context as the policy by definition placed children in specific neighborhoods. Hence, family income cannot be argued to be the deciding factor for residential location. Another problem is that newly arrived families have on average very low earnings and income might thus be a poor predictor of child health for these individuals. The refugees were in principle not allowed to work during the introductory program.

Furthermore, I utilize age at immigration among children to estimate a model with neighborhood exposure time.[70] I will also add family fixed effects in which the variation in exposure time between siblings is used to identify neighborhood effects.

4.3 Results

Table 7 shows that refugee children that were placed in neighborhoods where permanent residents, children in their own birth cohort, had one standard deviation worse health outcomes, will have a 0.38 percentage point increased risk of being hospitalized in adolescence. This corresponds to 3 percent relative to the sample mean. The corresponding figures for mental health is 7.3 percent, for accidents 7.0 percent, and for risky behavior 5.7 percent relative to the sample mean. The estimated magnitudes are in line with what was found in Section 3 for accidents, but 20 percent smaller for mental health, and about 40 percent smaller for hospitalizations for any condition and risky behavior.

Table 7. The effect of being placed in a neighborhood with one S.D. worse health on the probability of being hospitalized

	(1) Hosp	(2) Mental	(3) Accidents	(4) Risky
Health	0.0038**	0.0013*	0.0016*	0.0017*
	(0.002)	(0.001)	(0.001)	(0.001)
Mean	0.121	0.018	0.023	0.030
Observations	35,754	35,754	35,754	35,754
N clusters	2,427	2,427	2,427	2,427

Notes: Results from linear probability models. Each column represents results from a separate regression. All specifications include indicators for region of origin, child cohort, child gender, birth order, year of immigration, parents' civil status, destination county, and controls for parental years of schooling, number of children, and parents' age at immigration. Standard errors are presented in parenthesis and clustered on destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

As in Section 3, exposure time is added to the model to test whether longer exposure time to a neighborhood matters for health outcomes. Following the same structure, I also add family fixed effects that utilize differences in exposure time between siblings. Family fixed effects controls for exact place of origin, but also local neighborhood and preferences and could hence be helpful in estimating neighborhood effects also in this setting. Table 8 displays the results from these regressions, which show no statistically significant effects of exposure time to neighborhood.

[70] Given the set of restrictions that is put on the data, i.e. that children should be born year 1984-1992 and immigrated year 1985-1994, children will be age 0-10 when arriving to Sweden. Outcomes are still measured age 16-19, and exposure time is defined as age 16 minus age at immigration.

Table 8. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Hosp.*Exposure	0.0004	0.0006	-0.0008	-0.0007
	(0.001)	(0.001)	(0.001)	(0.001)
	[0.121]	[0.125]	[0.125]	[0.125]
Mental*Exposure	0.0001	0.0002	0.0008	0.0009
	(0.000)	(0.000)	(0.001)	(0.001)
	[0.018]	[0.018]	[0.018]	[0.018]
Accident*Exposure	-0.0001	0.0000	0.0001	0.0001
	(0.000)	(0.000)	(0.001)	(0.001)
	[0.023]	[0.024]	[0.024]	[0.024]
Risky*Exposure	0.0002	-0.0002	-0.0003	-0.0002
	(0.000)	(0.000)	(0.001)	(0.001)
	[0.030]	[0.033]	[0.033]	[0.033]
Time-varying controls	No	No	No	Yes
Observations	35,754	18,937	18,937	18,937
N clusters	2,427	2,275	2,275	2,275

Notes: Results from linear probability models. Each cell represents results from a separate regression. All specifications include indicators for region of origin, child cohort, child gender, birth order, parents' civil status, age at immigration, destination county, and controls for health in destination by cohort, parental years of schooling, number of children, and parents' age at immigration. Mean of dependent variables are shown in square brackets. Standard errors are clustered on destination by child cohort. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

4.4 Robustness

In Table 8, exposure time to neighborhood is introduced linearly. It is possible that the effect is non-linear and that the estimated model is too restrictive. The results from a more flexible specification using indicators for moving age are presented in Appendix, Table A13a-A13d. The results show that there is no significant difference in convergence in health outcomes between children immigrating before age 6, and children immigrating later (ages 6-10).

The placement policy was most strongly applied during years 1987-1991 (Edin et al., 2003). As a robustness check I limit the sample to those arriving to Sweden during that period. The results are found in Appendix, Table A14, and are very similar to the results for the entire period.

One limitation of the data is that country of origin is missing for some countries. Instead controls for region of origin are used in the main analysis for these places. However, data on exact country of birth exist for years 1985-1989. To check if the use of regions affects the findings, I compare results using controls for exact country of birth with the results using region for refugees arriving 1985-1989. The results are displayed in Table A15a-

A15b. The two tables show almost identical estimates, which strongly suggest that using region of birth for some countries is not affecting the results.

4.5 Discussion

The results presented in this section show that refugee children that were initially placed in neighborhoods where permanent residents had worse health outcomes, were more likely to be hospitalized in adolescence. The estimated magnitudes are in line with what was found using movers across areas in Sweden for accidents, but 20 percent smaller for mental health, and about 40 percent smaller for hospitalizations for any condition and risky behavior. Furthermore, also in line with the previous results, there is no evidence of any exposure time effects.

5. Conclusions

The aim of this paper has been to estimate neighborhood effects on youth health. To answer the question of whether neighborhoods affect health outcomes I have applied two different empirical methods. The first method uses variation in neighborhood conditions from families that move across areas. The results from this part confirm the correlation between neighborhoods and youth health found in previous observational studies. The effects are ranging from 5-9 percent from moving to a neighborhood with one standard deviation higher hospitalization rates, depending on cause of hospitalization. To pin down any causal effects, differences in exposure time to neighborhoods are compared between siblings. The results from this exercise show no evidence of exposure time effects of neighborhoods. These conflicting results can arise because of two reasons: The first potential explanation is that there are no causal neighborhood effects on adolescent health and that the association between places and health is mainly driven by selection. A second reason could be that there are causal effects of neighborhoods on health, but these effects are contemporaneous, hence exposure time to neighborhoods in childhood does not matter.

To try to determine whether there are any causal effects of neighborhoods on adolescent health, the second part of the paper uses a placement policy that offers potential exogenous variation in initial neighborhood for refugees. The results confirm the results from the first part that there are sizeable neighborhood effects on health outcomes, but there is no significant effect of exposure to neighborhood. The effects of being placed in a neighborhood with a one standard deviation worse health on the risk of being hospitalized are ranging from 3-7 percent depending on condition, on average the estimates are 30 percent smaller than what was found in the first part using movers across neighborhoods.

The findings from the two parts together imply that neighborhoods affect health in adolescence, but that there are no exposure time effects. The main health outcome is hospitalization related to any condition in adolescence, but I also study three specific conditions, mental illness, accidents, and risky behavior. The results could depend on the age of the individuals when outcomes are studied, but could also depend on the conditions analyzed. The risk of accidents might be related to the neighborhood through the physical local environment, or through local cultural behavior. Along the same lines, risky behavior might also be affected by the immediate presence of peers. Hence, accidents and risky behavior among youth are likely to be more closely related to contemporaneous relations and immediate surroundings rather than previous exposure. It is possible that there exist exposure time effects on health for this group, but that the effects are not detectable already in adolescence.

The results are in line with the findings in Ludwig et al. (2013) that find smaller effects in a longer term follow up from MTO than the interim evaluation did. The neighborhood conditions between treatment and control groups in MTO decreased over time, and if contemporaneous conditions are more important for youth health than the developmental effect, this could explain the results. Further research is needed to gain more understanding on the developmental effects of neighborhoods and long run health outcomes.

References

Aizer, A., & Currie, J. (2004). Networks or neighborhoods? Correlations in the use of publicly-funded maternity care in California. *Journal of public Economics*, *88*(12), 2573-2585.

Bertrand, M., Luttmer, E. F., & Mullainathan, S. (2000). Network effects and welfare cultures. *The Quarterly Journal of Economics*, *115*(3), 1019-1055.

Björkegren E. & Svaleryd H. (2017) Birth order and child health. (No 2017:3) Working paper series, Department of Economics, Uppsala University.

Brooks-Gunn, J., Duncan, G. J., Klebanov, P. K., & Sealand, N. (1993). Do neighborhoods influence child and adolescent development?. *American journal of sociology*, *99*(2), 353-395.

Card, D., & Giuliano, L. (2013). Peer effects and multiple equilibria in the risky behavior of friends. *Review of Economics and Statistics*, *95*(4), 1130-1149.

Case, A., Lubotsky, D., & Paxson, C. (2002). Economic status and health in childhood: The origins of the gradient. *The American Economic Review*, *92*(5), 1308-1334.

Chetty, R., & Hendren, N. (2016). *The impacts of neighborhoods on intergenerational mobility i: Childhood exposure effects* (No. w23001). National Bureau of Economic Research.

Chetty, R., Hendren, N., & Katz, L. F. (2016). The effects of exposure to better neighborhoods on children: New evidence from the Moving to Opportunity experiment. *The American Economic Review*, *106*(4), 855-902.

Dahlberg, M., Edmark, K., & Lundqvist, H. (2012). Ethnic diversity and preferences for redistribution. *Journal of Political Economy*, *120*(1), 41-76.

Damm, A. P., & Dustmann, C. (2014). Does growing up in a high crime neighborhood affect youth criminal behavior?. *The American Economic Review*, *104*(6), 1806-1832.

Edin, P. A., Fredriksson, P., & Åslund, O. (2003). Ethnic enclaves and the economic success of immigrants—Evidence from a natural experiment. *The quarterly journal of economics*, *118*(1), 329-357.

Grönqvist, H., Johansson, P., & Niknami, S. (2012). Income inequality and health: Lessons from a refugee residential assignment program. *Journal of health economics*, *31*(4), 617-629.

Haider, S., & Solon, G. (2006). Life-cycle variation in the association between current and lifetime earnings. *The American Economic Review*, *96*(4), 1308-1320.

Haveman, R., & Wolfe, B. (1995). The determinants of children's attainments: A review of methods and findings. *Journal of economic literature*, *33*(4), 1829-1878.

Heckman, J. J. (2007). The economics, technology, and neuroscience of human capability formation. Proceedings of the national Academy of Sciences, 104(33), 13250-13255.

Heidrich, S. (2016). The Effect of Moving during Childhood on Long Run Income: Evidence from Swedish Register Data, Working paper

Jacob, B. A., Ludwig, J., & Miller, D. L. (2013). The effects of housing and neighborhood conditions on child mortality. *Journal of health economics*, *32*(1), 195-206.

Jencks, C., & Mayer, S. E. (1990). The social consequences of growing up in a poor neighborhood. *Inner-city poverty in the United States*, *111*, 186.

Katz, L. F., Kling, J. R., & Liebman, J. B. (2001). Moving to opportunity in Boston: Early results of a randomized mobility experiment. *The Quarterly Journal of Economics*, *116*(2), 607-654.

Kling, J. R., Liebman, J. B., & Katz, L. F. (2007). Experimental analysis of neighborhood effects. *Econometrica*, *75*(1), 83-119.

Kremer, M., & Levy, D. (2008). Peer effects and alcohol use among college students. *The Journal of Economic Perspectives*, *22*(3), 189-189.

Leventhal, T., & Brooks-Gunn, J. (2000). The neighborhoods they live in: the effects of neighborhood residence on child and adolescent outcomes. *Psychological bulletin*, *126*(2), 309-337.

Ludwig, J., Duncan, G. J., Gennetian, L. A., Katz, L. F., Kessler, R. C., Kling, J. R., & Sanbonmatsu, L. (2013). Long-term neighborhood effects on low-income families: Evidence from Moving to Opportunity. *The American Economic Review*, *103*(3), 226-231.

Mincer, J. (1978). Family migration decisions. *Journal of Political Economy*, *86*(5), 749-773.

Mörk, E., Sjögren, A., & Svaleryd, H. (2014). Hellre rik och frisk. *Om familjebakgrund och barns hälsa. Stockholm: SNS Förlag*.

Nekby, L., & Pettersson Lidbom, P. (2016). Revisiting the relationship between ethnic diversity and preferences for redistribution. *The Scandinavian Journal of Economics*.

OECD (2017). Understanding the socio-economic divide in Europe. *Paris: OECD.*

Sampson, R. J. (2008). Moving to Inequality: Neighborhood Effects and Experiments Meet Social Structure 1. *American Journal of Sociology*, *114*(1), 189-231.

Sampson, R. J. (2012). *Great American city: Chicago and the enduring neighborhood effect*. University of Chicago Press.

Sampson, R. J., Morenoff, J. D., & Gannon-Rowley, T. (2002). Assessing "neighborhood effects": Social processes and new directions in research. *Annual review of sociology*, *28*(1), 443-478.

Sen A. (2002). Health: perception versus observation. *BMJ*, 324, 860-861.

Statistics Sweden (2013). The Multi-generation register 2012. A description of contents and quality, Population and Welfare Statistics 2013:1. Örebro: Statistics Sweden.

Statistics Sweden (2016). Life expectancy in Sweden 2011-2015. Demographic reports 2016:4. Örebro: Statistics Sweden

Votruba, M. E., & Kling, J. R. (2009). Effects of neighborhood characteristics on the mortality of black male youth: Evidence from Gautreaux, Chicago. *Social Science & Medicine*, *68*(5), 814-823.

World Health Organization. (2008). Closing the gap in a generation. *Geneva: WHO*.

Åslund, O., Edin, P. A., & Fredriksson, P. (2011). Peers, neighborhoods, and immigrant student achievement: Evidence from a placement policy. *American Economic Journal: Applied Economics*, *3*(2), 67-95.

Appendix

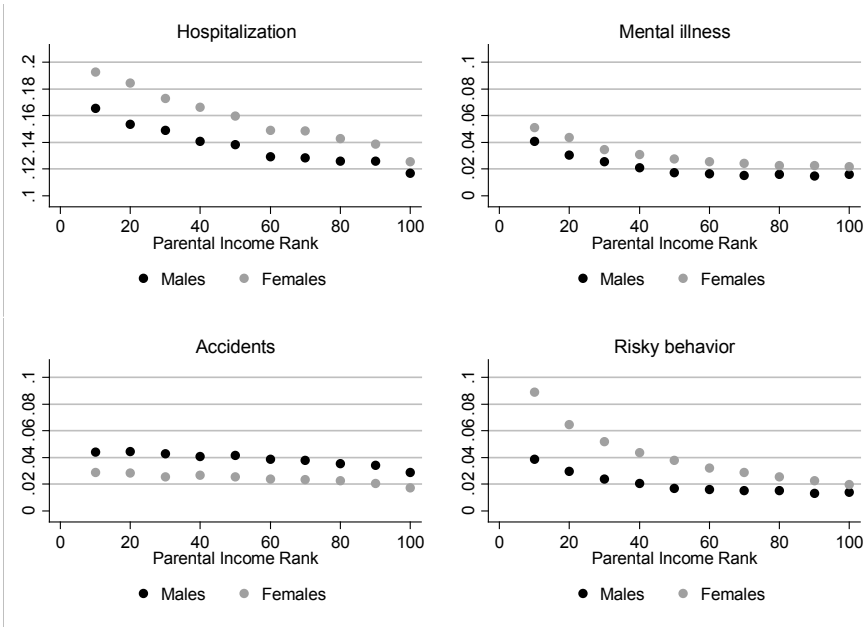


Figure A1. Hospitalization and causes of hospitalization by gender and parental income deciles

Notes: The four figures show share of youth that has been hospitalized at least once ages 16-19 by gender and parental income decile. Note that hospitalizations (any cause) have a different level on the y-axis because mental illness, accidents and risky behavior are subsets of all hospitalizations.

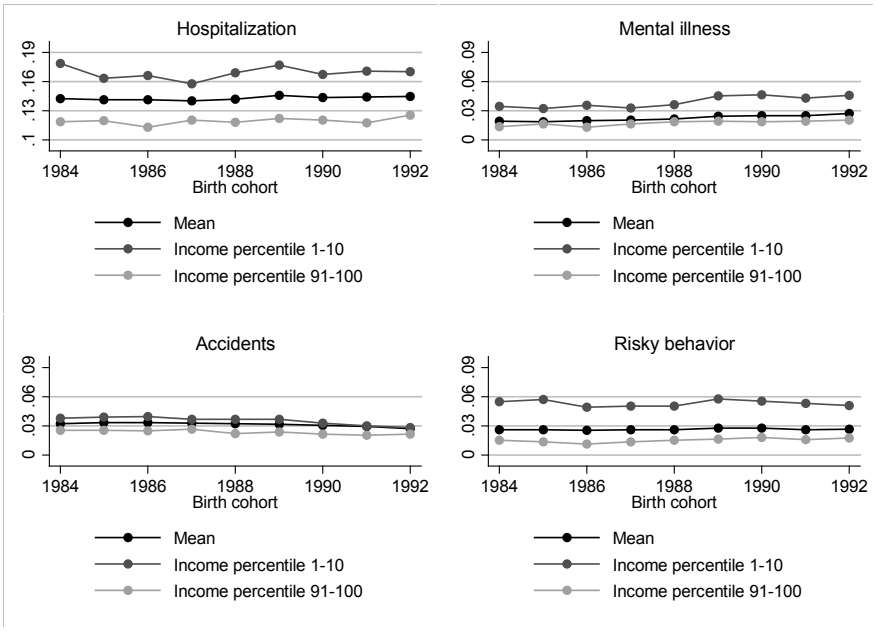


Figure A2. Hospitalization and causes of hospitalization by birth cohort and parental income

Notes: The four figures show share of youth that has been hospitalized at least once ages 16-19 by birth cohort and parental income decile. Note that hospitalizations (any cause) have a different level on the y-axis because mental illness, accidents and risky behavior are subsets of all hospitalizations.

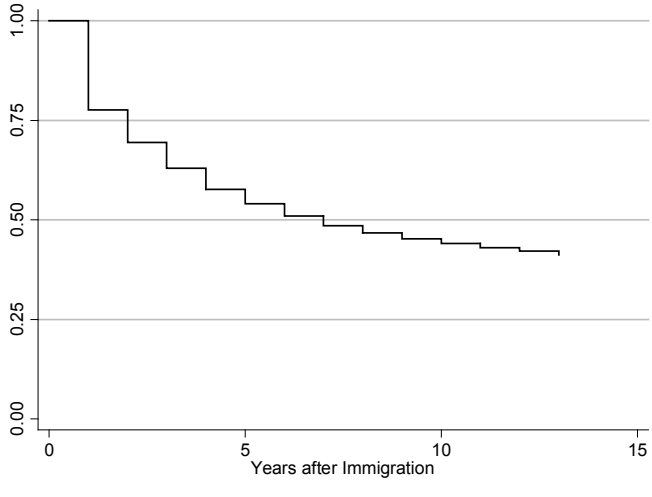


Figure A3. Survival analysis: Share of refugee children in the initial neighborhood

Notes: The figure shows the share of refugee children that remained in the initial neighborhood years after immigration.

Table A1. Diagnoses and ICD codes

Variable		**Definition**
Hospitalization (any cause)		=1 if admitted to hospital with any condition
Mental health problems		=1 if admitted to hospital with diagnosis codes F00-F99
Accidents		=1 if admitted to hospital with diagnosis codes V01-Y59
Risky behavior	Alcohol abuse	=1 if admitted to hospital with diagnosis codes T51, X45, X65, Y15, F10, K70, K85, K86.0–1 E24.4, G31.2, G62.1, G72.1, I42.6, K29.2, 035.4,
	Addiction	=1 if admitted to hospital with diagnosis codes T36-T49
	Self-harm	=1 if admitted to hospital with diagnosis codes Intentional self-harm X60-X84, event of undetermined intent Y10-Y34
	Pregnancy	= if admitted to hospital with diagnosis codes O00-O99
Perinatal conditions and congenital malformations		=1 if admitted to hospital with diagnosis codes P00-P96 and Q00-Q99

Table A2a. Share of children moving (percent) by number of moves by parent income quintile

Moves	q1	q2	q3	q4	q5	q1-q5
0	64.34	76.25	82.70	83.93	77.98	77.13
1	18.82	14.94	12.17	12.00	16.53	14.86
2	9.74	5.91	3.74	3.05	4.21	5.30
3	3.96	1.87	0.98	0.75	0.99	1.70
4	1.78	0.70	0.30	0.19	0.22	0.63
5	0.77	0.21	0.08	0.06	0.05	0.23
≥6	0.59	0.12	0.04	0.01	0.02	0.15
Total	100.00	100.00	100.00	100.00	100.00	100.00

Notes: The table shows the share of children moving ages 1-15, by number of moves by parental income quintile. The last column summarizes the share of moves independent of parental income.

Table A2b. Age at move (among one time movers)

Age at move	Frequency	Percent
2	22,513	16.03
3	17,909	12.75
4	15,079	10.74
5	12,422	8.84
6	10,922	7.78
7	9,178	6.53
8	7,069	5.03
9	6,840	4.87
10	6,811	4.85
11	6,297	4.48
12	6,288	4.48
13	6,428	4.58
14	6,080	4.33
15	6,627	4.72
Total	140,463	100.00

Notes: The table shows the share of children moving by age among one time movers.

Table A3a. Association between neighborhood health (S.D.) and probability of being hospitalized, allowing for differences over parental income

	(1) Hosp	(2) Mental	(3) Accident	(4) Risky
Δ Health	0.0045***	0.0026***	0.0004	0.0031***
	(0.001)	(0.001)	(0.001)	(0.001)
Mean	0.153	0.031	0.032	0.037
Observations	140,463	140,463	140,463	140,463
N clusters	82,502	82,502	82,502	82,502

Notes: Results from linear probability models. Each column represents one regression. All specifications include controls for health in origin by child cohort by parental income quintile interacted with cohort indicators, indicators for parental income percentile, cohort, birth order, county and gender. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort by parental income quintile.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A3b. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized, allowing for differences over parental income

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Δ Hosp*Exposure	-0.0000	-0.0000	-0.0002	-0.0002
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.153]	[0.150]	[0.150]	[0.150]
Δ Mental*Exposure	-0.0000	-0.0001	-0.0003	-0.0003
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.031]	[0.031]	[0.031]	[0.031]
Δ Accidents*Exposure	0.0001	0.0001	-0.0001	-0.0001
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.032]	[0.032]	[0.032]	[0.032]
Δ Risky*Exposure	-0.0000	-0.0001	-0.0002	-0.0002
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.037]	[0.037]	[0.037]	[0.037]
Time-varying controls	No	No	No	Yes
Observations	140,463	59,496	59,496	59,496
N clusters	82,502	41,376	41,376	41,376

Notes: Results from linear probability models. Each cell represents one regression. All specifications include controls for health in origin by child cohort by parental income quintile interacted with cohort indicators, difference in health between destination and origin, indicators for parental income percentile interacted with age at move, indicators for age at move, child cohort, birth order, county and gender. Mean of dependent variables are shown in square brackets. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort by parental income quintile. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A4a. Fixed effects models: Association between neighborhood health (S.D.) and probability of being hospitalized

	(1) Hosp	(2) Mental	(3) Accident	(4) Risky
Health	0.0069***	0.0028***	0.0018***	0.0029***
	(0.001)	(0.001)	(0.001)	(0.001)
Mean	0.153	0.031	0.032	0.037
Observations	140,463	140,463	140,463	140,463
N clusters	53,961	53,961	53,961	53,961

Notes: Results from linear probability models. Each column represents one regression. All specifications include origin by child cohort fixed effects, indicators of child gender, birth order, and county. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A4b. Fixed effects models: Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Hosp*Exposure	-0.0001	-0.0003	-0.0006	-0.0007
	(0.000)	(0.001)	(0.001)	(0.001)
	[0.153]	[0.150]	[0.150]	[0.150]
Mental*Exposure	0.0000	-0.0005	0.0000	0.0000
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.031]	[0.031]	[0.031]	[0.031]
Accident*Exposure	0.0004**	0.0002	-0.0003	-0.0003
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.032]	[0.032]	[0.032]	[0.032]
Risky*Exposure	-0.0000	-0.0002	-0.0001	-0.0002
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.037]	[0.037]	[0.037]	[0.037]
Time-varying controls	No	No	No	Yes
Observations	140,463	59,496	59,496	59,496
N clusters	53,961	29,483	29,483	29,483

Notes: Results from linear probability models. Each cell represents results from one regression. All specifications include origin by child cohort by child moving age fixed effects, indicators of child gender, birth order, and county, and controls for health in destination by child cohort. Mean of dependent variables are shown in square brackets. Standard errors are clustered on origin by destination by child cohort. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A5a. Fixed effects models: Association between neighborhood health (S.D.) and probability of being hospitalized, allowing for differences over parental income

	(1) Hosp	(2) Mental	(3) Accident	(4) Risky
Health	0.0041***	0.0022***	-0.0000	0.0026***
	(0.001)	(0.001)	(0.001)	(0.001)
Mean	0.153	0.031	0.032	0.037
Observations	140,463	140,463	140,463	140,463
N clusters	82,502	82,502	82,502	82,502

Notes: Results from linear probability models. Each column represents one regression. All specifications include origin by child cohort by parental quintile fixed effects, indicators of child gender, birth order, and county. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort by parental income quintile.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A5b. Fixed effects models: Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized, allowing for differences over parental income

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Hosp*Exposure	0.0001	0.0013	0.0002	0.0002
	(0.000)	(0.001)	(0.001)	(0.001)
	[0.153]	[0.150]	[0.150]	[0.150]
Mental*Exposure	0.0001	-0.0003	-0.0002	-0.0002
	(0.000)	(0.001)	(0.001)	(0.001)
	[0.031]	[0.031]	[0.031]	[0.031]
Accident*Exposure	0.0005**	0.0001	-0.0002	-0.0002
	(0.000)	(0.000)	(0.001)	(0.001)
	[0.032]	[0.032]	[0.032]	[0.032]
Risky*Exposure	0.0001	0.0007	-0.0004	-0.0004
	(0.000)	(0.001)	(0.001)	(0.001)
	[0.037]	[0.037]	[0.037]	[0.037]
Time-varying controls	No	No	No	Yes
Observations	140,463	59,496	59,496	59,496
N clusters	82,502	41,376	41,376	41,376

Notes: Results from linear probability models. Each cell represents results from one regression. All specifications include origin by child cohort by parental income quintile by child moving age fixed effects, indicators of child gender, birth order, and county, and controls for health in destination by child cohort by parental income quintile. Mean of dependent variables are shown in square brackets. Standard errors are clustered on origin by destination by child cohort by parental quintile. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A6a. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized (any condition)

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Δ Health	0.0088***	0.0103***	0.0071*	0.0070*
	(0.002)	(0.003)	(0.004)	(0.004)
Δ Health* Move age <6	-0.0015	-0.0020	-0.0044	-0.0043
	(0.002)	(0.003)	(0.004)	(0.004)
Δ Health* Move age 6-10	-0.0018	-0.0031	-0.0066	-0.0065
	(0.002)	(0.003)	(0.004)	(0.004)
Move age <6	-0.0155***	-0.0122***	-0.0129	-0.0152*
	(0.003)	(0.004)	(0.009)	(0.009)
Move age 6-10	-0.0059**	-0.0075*	-0.0051	-0.0070
	(0.003)	(0.004)	(0.007)	(0.007)
Time-varying controls	No	No	No	Yes
Mean	0.153	0.150	0.150	0.150
Observations	140,463	59,496	59,496	59,496
N clusters	53,961	29,483	29,483	29,483

Notes: Results from linear probability models. Each column represents one regression. Omitted category is moving age 11-15. All specifications include controls for health in origin by child cohort interacted with cohort indicators, indicators for cohort, birth order, county and gender. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A6b. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized (mental conditions)

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Δ Health	0.0023**	0.0025*	0.0033	0.0032
	(0.001)	(0.001)	(0.002)	(0.002)
Δ Health* Move age <6	0.0003	-0.0006	-0.0020	-0.0020
	(0.001)	(0.002)	(0.002)	(0.002)
Δ Health* Move age 6-10	0.0015	0.0012	-0.0010	-0.0008
	(0.001)	(0.002)	(0.002)	(0.002)
Move age <6	-0.0093***	-0.0079***	-0.0122***	-0.0130***
	(0.001)	(0.002)	(0.004)	(0.004)
Move age 6-10	-0.0048***	-0.0046**	-0.0061*	-0.0066*
	(0.001)	(0.002)	(0.003)	(0.003)
Time-varying controls	No	No	No	Yes
Mean	0.031	0.031	0.031	0.031
Observations	140,463	59,496	59,496	59,496
N clusters	53,961	29,483	29,483	29,483

Notes: Results from linear probability models. Each column represents one regression. Omitted category is moving age 11-15. All specifications include controls for health in origin by child cohort interacted with cohort indicators, indicators for cohort, birth order, county and gender. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A6c. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized (accidents)

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Δ Health	0.0013	-0.0005	-0.0020	-0.0022
	(0.001)	(0.001)	(0.002)	(0.002)
Δ Health *Move age <6	0.0012	0.0020	-0.0002	-0.0000
	(0.001)	(0.002)	(0.002)	(0.002)
Δ Health *Move age 6-10	0.0007	0.0020	-0.0000	0.0001
	(0.001)	(0.002)	(0.002)	(0.002)
Move age <6	-0.0021*	-0.0013	-0.0053	-0.0074*
	(0.001)	(0.002)	(0.004)	(0.004)
Move age 6-10	-0.0007	-0.0023	-0.0054	-0.0067**
	(0.001)	(0.002)	(0.003)	(0.003)
Time-varying controls	No	No	No	Yes
Mean	0.032	0.032	0.032	0.032
Observations	140,463	59,496	59,496	59,496
N clusters	53,961	29,483	29,483	29,483

Notes: Results from linear probability models. Each column represents one regression. Omitted category is moving age 11-15. All specifications include controls for health in origin by child cohort interacted with cohort indicators, indicators for cohort, birth order, county and gender. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A6d. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized (risky behavior)

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Δ Health	0.0037***	0.0043***	0.0041*	0.0041*
	(0.001)	(0.001)	(0.002)	(0.002)
Δ Health* Move age <6	-0.0003	-0.0015	-0.0026	-0.0027
	(0.001)	(0.002)	(0.002)	(0.002)
Δ Health* Move age 6-10	-0.0006	-0.0004	-0.0022	-0.0023
	(0.001)	(0.002)	(0.002)	(0.002)
Move age <6	-0.0108***	-0.0110***	-0.0144***	-0.0148***
	(0.001)	(0.002)	(0.005)	(0.005)
Move age 6-10	-0.0056***	-0.0060***	-0.0101***	-0.0104***
	(0.002)	(0.002)	(0.004)	(0.004)
Time-varying controls	No	No	No	Yes
Mean	0.037	0.037	0.037	0.037
Observations	140,463	59,496	59,496	59,496
N clusters	53,961	29,483	29,483	29,483

Notes: Results from linear probability models. Each column represents one regression. Omitted category is moving age 11-15. All specifications include controls for health in origin by child cohort interacted with cohort indicators, indicators for cohort, birth order, county and gender. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A7a. Association between neighborhood health (S.D.) and probability of being hospitalized among females

	(1) Hosp	(2) Mental	(3) Accident	(4) Risky
Δ Health	0.0099***	0.0035***	0.0018**	0.0047***
	(0.002)	(0.001)	(0.001)	(0.001)
Mean	0.165	0.036	0.026	0.050
Observations	68,801	68,801	68,801	68,801
N clusters	33,017	33,017	33,017	33,017

Notes: Results from linear probability models. Each column represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, indicators for cohort, birth order, county and gender. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A7b. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized among females

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Δ Hosp*Exposure	-0.0001	-0.0007	-0.0014	-0.0013
	(0.000)	(0.001)	(0.001)	(0.001)
	[0.165]	[0.158]	[0.158]	[0.158]
Δ Mental*Exposure	0.0002	0.0004	0.0002	0.0002
	(0.000)	(0.000)	(0.000)	(0.001)
	[0.036]	[0.035]	[0.035]	[0.035]
Δ Accident*Exposure	0.0000	-0.0004	-0.0006	-0.0005
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.026]	[0.025]	[0.025]	[0.025]
Δ Risky*Exposure	0.0001	-0.0001	-0.0002	-0.0002
	(0.000)	(0.000)	(0.001)	(0.001)
	[0.050]	[0.051]	[0.051]	[0.051]
Time-varying controls	No	No	No	Yes
Observations	68,801	15,572	15,572	15,572
N clusters	33,017	10,446	10,446	10,446

Notes: Results from linear probability models. Each cell represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, differences in health between destination and origin, indicators for age at move, cohort, birth order, county and gender. Mean of dependent variables are shown in square brackets. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A8a. Association between neighborhood health (S.D.) and probability of being hospitalized among males

	(1) Hosp	(2) Mental	(3) Accident	(4) Risky
Δ Health	0.0055***	0.0022***	0.0025***	0.0022***
	(0.001)	(0.001)	(0.001)	(0.001)
Mean	0.141	0.026	0.038	0.024
Observations	71,662	71,662	71,662	71,662
N clusters	33,905	33,905	33,905	33,905

Notes: Results from linear probability models. Each column represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, indicators for cohort, birth order, county and gender. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort.
* p < 0.10, ** p < 0.05, *** p < 0.01

Table A8b. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized among males

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Δ Hosp*Exposure	0.0000	0.0001	0.0001	0.0002
	(0.000)	(0.001)	(0.001)	(0.001)
	[0.141]	[0.137]	[0.137]	[0.137]
Δ Mental*Exposure	-0.0001	-0.0002	-0.0004	-0.0005
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.026]	[0.024]	[0.024]	[0.024]
Δ Accident*Exposure	0.0001	0.0004	-0.0001	-0.0001
	(0.000)	(0.000)	(0.001)	(0.001)
	[0.038]	[0.037]	[0.037]	[0.037]
Δ Risky*Exposure	-0.0002	-0.0003	-0.0001	-0.0001
	(0.000)	(0.000)	(0.000)	(0.000)
	[0.024]	[0.024]	[0.024]	[0.024]
Time-varying controls	No	No	No	Yes
Observations	71,662	17,040	17,040	17,040
N clusters	33,905	11,310	11,310	11,310

Notes: Results from linear probability models. Each cell represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, differences in health between destination and origin, indicators for age at move, cohort, birth order, county and gender. Mean of dependent variables are shown in square brackets. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A9a. Association between neighborhood health (S.D.) and probability of being hospitalized among children moving to the 50 neighborhoods with poorest health outcomes

	(1) Hosp	(2) Mental	(3) Accident	(4) Risky
Δ Health	0.0024	0.0009	0.0003	0.0006
	(0.003)	(0.002)	(0.001)	(0.002)
Mean	0.131	0.023	0.025	0.029
Observations	23,681	16,424	37,690	19,777
N clusters	8,467	7,141	11,331	7,723

Notes: Results from linear probability models. Each column represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, indicators for cohort, birth order, county and gender. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A9b. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized among children moving to the 50 neighborhoods with poorest health outcomes

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Δ Hosp*Exposure	-0.0005	-0.0007	-0.0011	-0.0012
	(0.000)	(0.001)	(0.001)	(0.001)
Mean	[0.131]	[0.128]	[0.128]	[0.128]
Observations	23,681	10,020	10,020	10,020
N clusters	8,467	4,689	4,689	4,689
Δ Mental*Exposure	-0.0003	-0.0004	-0.0001	-0.0001
	(0.000)	(0.000)	(0.001)	(0.001)
Mean	[0.023]	[0.024]	[0.024]	[0.024]
Observations	16,424	7,094	7,094	7,094
N clusters	7,141	3,906	3,906	3,906
Δ Accident*Exposure	0.0001	0.0004	-0.0002	-0.0002
	(0.000)	(0.000)	(0.001)	(0.001)
Mean	[0.025]	[0.024]	[0.024]	[0.024]
Observations	37,690	15,326	15,326	15,326
N clusters	11,331	6,260	6,260	6,260
Δ Risky*Exposure	0.0003	0.0002	0.0006	0.0004
	(0.000)	(0.000)	(0.001)	(0.001)
Mean	[0.029]	[0.030]	[0.030]	[0.030]
Observations	19,777	8,372	8,372	8,372
N clusters	7,723	4,200	4,200	4,200
Time-varying controls	No	No	No	Yes

Notes: Results from linear probability models. Each cell represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, differences in health between destination and origin, indicators for age at move, cohort, birth order, county and gender. Mean of dependent variables are shown in square brackets. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A10a. Association between neighborhood health (S.D.) and probability of being hospitalized among children with parental income <20 percentile

	(1) Hosp	(2) Mental	(3) Accident	(4) Risky
Δ Health	0.0108***	0.0040***	0.0042***	0.0040***
	(0.002)	(0.001)	(0.001)	(0.001)
Mean	0.179	0.045	0.037	0.060
Observations	33,068	33,068	33,068	33,068
N clusters	20,328	20,328	20,328	20,328

Notes: Results from linear probability models. Each column represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, indicators for cohort, birth order, county and gender. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A10b. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized among children with parental income <20 percentile

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Δ Hosp*Exposure	-0.0001	0.0000	-0.0000	-0.0000
	(0.000)	(0.001)	(0.001)	(0.001)
	[0.179]	[0.178]	[0.178]	[0.178]
Δ Mental*Exposure	0.0000	0.0000	0.0005	0.0004
	(0.000)	(0.000)	(0.001)	(0.001)
	[0.045]	[0.043]	[0.043]	[0.043]
Δ Accident*Exposure	-0.0000	0.0004	0.0005	0.0005
	(0.000)	(0.000)	(0.001)	(0.001)
	[0.037]	[0.036]	[0.036]	[0.036]
Δ Risky*Exposure	-0.0004	-0.0004	-0.0005	-0.0006
	(0.000)	(0.000)	(0.001)	(0.001)
	[0.060]	[0.061]	[0.061]	[0.061]
Time-varying controls	No	No	No	Yes
Observations	33,068	13,589	13,589	13,589
N clusters	20,328	9,962	9,962	9,962

Notes: Results from linear probability models. Each cell represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, differences in health between destination and origin, indicators for age at move, cohort, birth order, county and gender. Mean of dependent variables are shown in square brackets. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A11a. Association between neighborhood health (S.D.) and probability of being hospitalized among children with parents' with foreign background

	(1) Hosp	(2) Mental	(3) Accident	(4) Risky
Δ Health	0.0069	0.0027	0.0007	0.0035
	(0.006)	(0.003)	(0.003)	(0.002)
Mean	0.132	0.023	0.019	0.030
Observations	8,733	8,733	8,733	8,733
N clusters	5,439	5,439	5,439	5,439

Notes: Results from linear probability models. Each column represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, indicators for cohort, birth order, county and gender. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A11b. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized among children with parents with foreign background

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Δ Hosp*Exposure	0.0011	0.0003	-0.0015	-0.0017
	(0.001)	(0.001)	(0.003)	(0.003)
	[0.132]	[0.122]	[0.122]	[0.122]
Δ Mental*Exposure	-0.0002	-0.0017**	-0.0008	-0.0011
	(0.000)	(0.001)	(0.001)	(0.001)
	[0.023]	[0.019]	[0.019]	[0.019]
Δ Accident*Exposure	-0.0002	-0.0001	0.0001	0.0001
	(0.000)	(0.000)	(0.001)	(0.001)
	[0.019]	[0.015]	[0.015]	[0.015]
Δ Risky*Exposure	0.0001	-0.0017**	-0.0014	-0.0017*
	(0.000)	(0.001)	(0.001)	(0.001)
	[0.030]	[0.028]	[0.028]	[0.028]
Time-varying controls	No	No	No	Yes
Observations	8,733	3,263	3,263	3,263
N clusters	5,439	2,228	2,228	2,228

Notes: Results from linear probability models. Each cell represents one regression. All specifications include controls for health in origin by child cohort interacted with cohort indicators, differences in health between destination and origin, indicators for age at move, cohort, birth order, county and gender. Mean of dependent variables are shown in square brackets. Standard errors are presented in parenthesis and clustered on origin by destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A12. Region of birth, refugees

Region of Birth	Percent of sample
1. Former Yugoslavia	40.61
2. Poland	2.15
3. The Baltic states	0.46
4. Eastern Europe 1 (Rumania, The former USSR, Bulgaria, Albania)	4.46
5. Eastern Europe 2 (Hungary, The former Czechoslovakia)	0.94
6. Mexico and Central America	1.09
7. Chile	3.26
8. Other South America	1.67
9. African Horn (e.g., Ethiopia and Somalia)	7.21
10. North Africa (Arabic countries: e.g., Morocco and Tunisia), Arabian Peninsula, and Middle East (e.g., Lebanon, Syria)	10.51
11. Other Africa	1.97
12. Iran	10.01
13. Iraq	7.30
14. Turkey	2.99
15. South East Asia (e.g., Vietnam and Thailand)	3.31
16. Other Asia (e.g., Sri Lanka, Bangladesh, and Afghanistan)	2.07
Total	100

Table A13a. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized (any condition) among refugees

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Health	0.0019	0.0019	0.0077	0.0079
	(0.003)	(0.004)	(0.005)	(0.005)
Health*Immigration age<6	0.0028	0.0035	-0.0003	-0.0000
	(0.003)	(0.005)	(0.006)	(0.006)
Immigration age<6	-0.0090*	-0.0076	-0.0049	-0.0052
	(0.005)	(0.007)	(0.011)	(0.012)
Time-varying controls	No	No	No	Yes
Mean	0.121	0.125	0.125	0.125
Observations	35,754	18,937	18,937	18,937
N clusters	2,427	2,275	2,275	2,275

Notes: Results from linear probability models. Each column represents results from a separate regression. Omitted category is immigration age >5. All specifications include indicators for region of origin, child cohort, child gender, birth order, parents' civil status, destination county, and controls for parental years of schooling, number of children, and parents' age at immigration. Standard errors are clustered on destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A13b. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized (mental conditions) among refugees

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Health	0.0011	-0.0009	-0.0018	-0.0019
	(0.001)	(0.002)	(0.002)	(0.002)
Health*Immigration age<6	0.0004	0.0019	0.0038	0.0039
	(0.002)	(0.002)	(0.003)	(0.003)
Immigration age<6	-0.0037*	-0.0085***	-0.0063	-0.0073
	(0.002)	(0.003)	(0.005)	(0.005)
Time-varying controls	No	No	No	Yes
Mean	0.018	0.018	0.018	0.018
Observations	35,754	18,937	18,937	18,937
N clusters	2,427	2,275	2,275	2,275

Notes: Results from linear probability models. Each column represents results from a separate regression. Omitted category is immigration age >5. All specifications include indicators for region of origin, child cohort, child gender, birth order, parents' civil status, destination county, and controls for parental years of schooling, number of children, and parents' age at immigration. Standard errors are clustered on destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A13c. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized (accidents) among refugees

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Health	0.0018	0.0007	0.0020	0.0020
	(0.001)	(0.002)	(0.002)	(0.002)
Health*Immigration age<6	-0.0004	0.0007	0.0005	0.0006
	(0.002)	(0.002)	(0.003)	(0.003)
Immigration age<6	-0.0003	-0.0036	-0.0056	-0.0052
	(0.002)	(0.003)	(0.005)	(0.005)
Time-varying controls	No	No	No	Yes
Mean	0.023	0.024	0.024	0.024
Observations	35,754	18,937	18,937	18,937
N clusters	2,427	2,275	2,275	2,275

Notes: Results from linear probability models. Each column represents results from a separate regression. Omitted category is immigration age >5. All specifications include indicators for region of origin, child cohort, child gender, birth order, parents' civil status, destination county, and controls for parental years of schooling, number of children, and parents' age at immigration. Standard errors are clustered on destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A13d. Exposure time effects of neighborhood health (S.D.) and probability of being hospitalized (risky behavior) among refugees

	(1) All	(2) Siblings	(3) Fam FE	(4) Fam FE
Health	0.0023	0.0009	0.0019	0.0017
	(0.001)	(0.002)	(0.003)	(0.003)
Health*Immigration age<6	-0.0010	-0.0014	-0.0016	-0.0014
	(0.002)	(0.002)	(0.003)	(0.003)
Immigration age<6	-0.0082***	-0.0110***	-0.0079	-0.0089
	(0.003)	(0.004)	(0.007)	(0.007)
Time-varying controls	No	No	No	Yes
Mean	0.030	0.033	0.033	0.033
Observations	35,754	18,937	18,937	18,937
N clusters	2,427	2,275	2,275	2,275

Notes: Results from linear probability models. Each column represents results from a separate regression. Omitted category is immigration age >5. All specifications include indicators for region of origin, child cohort, child gender, birth order, parents' civil status, destination county, and controls for parental years of schooling, number of children, and parents' age at immigration. Standard errors are clustered on destination by child cohort.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A14. The effect of being placed in a neighborhood with one S.D. worse health on the probability of being hospitalized among refugees, immigration year 1987-1991

	(1) Hosp	(2) Mental	(3) Accidents	(4) Risky
Health	0.0048	0.0003	0.0014	0.0014
	(0.003)	(0.001)	(0.001)	(0.001)
Observations	12,400	12,400	12,400	12,400
Mean	0.119	0.019	0.023	0.030
N clusters	1,714	1,714	1,714	1,714

Notes: Results from linear probability models. Each column represents results from a separate regression. Sample restricted to immigrants arriving year 1987-1991. All specifications include indicators for region of origin, child cohort, child gender, birth order, year of immigration, parents' civil status, destination county, and controls for parental years of schooling, number of children, and parents' age at immigration. Standard errors are presented in parenthesis and clustered on destination by child cohort.
$p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A15a**.** The effect of being placed in a neighborhood with one S.D. worse health on the probability of being hospitalized among refugees, country of origin fixed effects

	(1) Hosp	(2) Mental	(3) Accidents	(4) Risky
Health	0.0084*	0.0017	0.0004	0.0019
	(0.005)	(0.002)	(0.002)	(0.002)
Observations	5,932	5,932	5,932	5,932
Mean	0.118	0.019	0.024	0.031
N clusters	1,117	1,117	1,117	1,117

Notes: Results from linear probability models. Each column represents results from a separate regression. Sample restricted to immigrants arriving year 1985-1989. All specifications include indicators for country of origin, child cohort, child gender, year of immigration, parents' civil status, destination county and controls for parental years of schooling, number of children and parents' age at immigration. Standard errors are clustered on destination by child cohort. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A15b. The effect of being placed in a neighborhood with one S.D. worse health on the probability of being hospitalized among refugees, region of origin fixed effects

	(1) Hosp	(2) Mental	(3) Accidents	(4) Risky
Health	0.0079	0.0017	0.0004	0.0017
	(0.005)	(0.002)	(0.002)	(0.002)
Observations	5,932	5,932	5,932	5,932
Mean	0.118	0.019	0.024	0.031
N clusters	1,117	1,117	1,117	1,117

Notes: Results from linear probability models. Each column represents results from a separate regression. Sample restricted to immigrants arriving year 1985-1989. All specifications include indicators for region of origin, child cohort, child gender, year of immigration, parents' civil status, destination county and controls for parental years of schooling, number of children and parents' age at immigration. Standard errors are clustered on destination by child cohort. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

III. Birth Order and Child Health

With Helena Svaleryd

1. Introduction

Health status during childhood is an important predictor for outcomes later in life such as educational attainment, labor market outcomes and adult health.[71] Poor health is strongly correlated with socioeconomic background and is transmitted across generations, which may be due to persistent factors such as genetics, family investments or institutions.[72] However, long-term outcomes do not only differ systematically between families but also within families, holding many of these persistent factors constant. A vast number of studies in various research disciplines have shown that younger siblings have lower educational achievements, IQ and earnings than their older siblings.[73] The mechanisms behind these effects are still debated and previous empirical research has struggled to identify the channels. Our objective is to study how health develops through childhood and, by studying different sorts of health conditions, to shed some light on the mechanisms giving rise to the negative birth order effect on later life outcomes.

What can we learn from studying birth order effects? It can be difficult to think of policy implications of birth order since it is impossible to alter, and is not in the hands of policy makers. However, there is a random assignment of elementary abilities since, at conception, a child gets a half of each parent's genes. This gene setup does not differ systematically between siblings and birth order and thus we can interpret the effects of birth order causally. In other words, differences by birth order should depend on pre- and postnatal influences rather than pre-determined conditions, which also opens up for policy interventions.[74] Learning about what is important in the family environment for children's long-term outcomes is crucial beyond our understanding of birth order effects.

---

[71] See, for example, Currie et al. (2010) and Case et al. (2005). Currie et al. (2010) compare Canadian siblings and find that the physical health status in early childhood is a strong predictor for young adult outcomes, mainly because it is a strong predictor for later health. Mental health problems, however, have an independent effect on future outcomes. Case et al. (2005) also find negative effects of poor childhood health on educational attainment, health and social status as an adult. For a review article on socioeconomic status and child health, see Currie (2009).

[72] See Smith (1999) for an overview of the health gradient and, for example, Lindahl et al. (2015) on the nature and nurture decomposition of mortality and health, and Mörk et al. (2014) on family background and child health.

[73] For example, Behrman and Taubman (1986) find birth-order effects on schooling and earnings among young US adults; Black et al. (2005) find birth-order effects on education, adult earnings, and teenage childbearing using a rich data set on the Norwegian population. Barclay (2015b) uses conscription data from Sweden and find birth-order effects on IQ and Black et al. (2015) find birth-order effects on personality traits.

[74] The policy implications will depend on the findings. If the results show that it is investments and time alone with parents as young that are important, this could, for example, indicate that day care for older siblings is important while parents are on parental leave with the youngest child.

Previous evidence on child health and birth order shows that firstborn children are disadvantaged at birth with lower birth weight and worse health (see, for example, Brenoe and Molitor, 2015; Modin, 2002; Swamy et al., 2012). However, the health disadvantage of firstborn children seems to be reversed in adulthood. Later-born siblings have a higher mortality risk both in working age and older age (Modin, 2002; Barclay and Kolk, 2015). The research on birth order effects on childhood health after birth is limited. Moreover, the existing studies use small samples and are unable to control for unobserved differences across families. Using data from the National Longitudinal Survey of Youth 1997, Argys et al. (2006) find that later-born siblings are more likely to engage in risky behavior such as smoking, drinking alcohol and marijuana usage. There is some evidence that later-born children in large families run a greater risk of experiencing accidents in early childhood (Nixon and Pearn, 1978; Bijur, Golding and Kurzon, 1988). A weakness with the studies of birth order effects on experiences of accidents is that they do not control for family size and may thus suffer from selection problems since large families may be inherently different from smaller families. To avoid this issue, we use a large register dataset from Sweden and estimate the effect of birth order using a family-fixed effects specification. Thus, we identify the birth order effects by comparing siblings within the same family, thereby controlling for family-level unobserved characteristics and observable characteristics such as family size.

Several hypotheses about the mechanisms through which the birth order effect works have been suggested, including the resource dilution hypothesis (Blake, 1989), strategic parental behavior (Hotz and Pantano, 2015), sibling influences (Zajonc, 1976) and birth endowments. However, there is limited empirical evidence on which underlying mechanisms are most important. By making use of our comprehensive data, which includes detailed information on medical diagnoses, we shed some light on the mechanisms behind the observed birth order effects.

Our results lend support to the idea that firstborns are disadvantaged at birth for biological reasons. Firstborn children are more likely to be hospitalized for perinatal conditions and congenital malformations in early childhood. We also find that lower birth order children are more likely to die during infancy. One possible explanation is that the womb becomes more effective at nurturing the fetus for each new pregnancy, in particular between the first and second pregnancy (Khong et al. 2003). The disadvantage of older siblings is, however, reversed as the child grows older. In adolescence, the second sibling is 14 percent more likely to be hospitalized and the third sibling is 20 percent more likely to be admitted to hospital, as compared to the firstborn child. The causes for hospitalization suggest that later-born siblings are involved in more risky behavior and have a less healthy life style during adolescence. In particular, later-born siblings are more likely to be admitted to hospital for diagnoses related to poor mental health, alcohol consumption,

self-harm and injuries. Our results suggest that part of the explanation is that parents do not look after younger siblings to the same extent, perhaps due to time and other resource constraints since there are positive birth order effects on injuries and avoidable conditions, which are conditions that should not be the cause for hospitalization if taken care of properly, for example diarrhea, anemia and asthma.

The gene-set up at conception across siblings is random, implying that by comparing siblings within the same family, we can estimate causal effects of birth order on health. However, if parents base subsequent fertility decisions on the health of already born children, the estimates may be biased. Negative associations between children's outcomes and birth order could be an effect of endogenous fertility decisions if parents refrain from having more children when a particularly demanding child is born. This response is often referred to as optimal stopping. Studies on birth order effects generally ignore this problem of possible reverse causality since it is difficult to identify random variation in the 'quality' of children that is observable by parents at an early age when fertility decisions are generally made. We test for this directly by studying whether early ill-health or death of born children affects the probability of having another child. Our results show that having an unhealthy child decreases the probability of having another child and if the family has another child, the spacing between the children increases. In contrast, if the child dies, it increases the probability that the parents have another child and decreases the spacing between pregnancies. This would imply that the sibling order of the last child born into the family is related to the health of already born children. To remedy this endogeneity problem, we remove the last born child in all families and re-estimate the effect of birth order on health and mortality. Although the sample size is significantly smaller, the estimated effects of birth order on health remain very similar. Re-estimating the birth order effects on mortality on this sample reduces the original estimates on infant mortality by 30-40 percent. However, there is still a clear birth order effect on infant mortality suggesting that lower birth order children are disadvantaged at birth as compared to higher birth order children.

Our results support the hypothesis that birth order effects are due to lower investment in children with a higher birth order. Younger siblings are more likely to be hospitalized for avoidable conditions, injuries and risky behavior such as excess alcohol consumption. This is in line with the dilution hypothesis presented in Blake (1989) and the finding in Price (2008) that parents spend more time with earlier-born than later-born siblings. It could also be that the family environment changes with older siblings in the family and more time and attention is needed to achieve the same 'investment' in the child. The parents' endogenous fertility response to the health and death of previous children lends further support to the hypothesis that parents are resource constrained.

The rest of the paper is organized as follows. Section 2 reviews previous empirical research and suggested hypotheses explaining the differential outcomes of children with different birth order. In Section 3 we describe the empirical strategy and in Section 4 the data used in the study. We present the results on birth order and health in Section 5 and our findings on optimal stopping in Section 6. Section 7 investigates potential heterogeneity, and finally Section 8 concludes the paper.

2. Related Literature and Mechanisms

2.1 Health and Birth order

Previous research has shown that firstborn children have worse health at birth than their later-born siblings. The causes of the better health status of later-born siblings at birth are investigated by Brenoe and Molitor (2015) using Danish registry data. They find that firstborns are disadvantaged at birth, measured by a number of different birth outcomes, as compared to later-born siblings and that this is unlikely to depend on the behavior of the mother. For example, they find that women are less likely to go to check-ups etc. for later-born siblings, which suggests that mothers take greater care during pregnancies with the firstborn child. Hence, the observed birth order effects are not driven by the behavior of the mother and they conclude that there are biological differences depending on birth order, which could be caused by changes in the womb, as found by Khong et al. (2003).[75][76] However, these changes cannot explain the reverse birth order pattern that is found on educational outcomes later in life. Rather, controlling for endowments at birth increases the birth order effects on outcomes later in adulthood; this is also noted in Black et al. (2011).

Modin (2002) studies the mortality risk over the life cycle for a sample of individuals born in Sweden in 1915-1929. She shows that the mortality risk is u-shaped at infancy; it is highest for firstborn children and children with birth order five and higher. At all other ages, she documents a positive correlation between birth order and mortality risk. However, Modin is not able to control for family size and to the extent that parents who have larger families are different, the correlation between birth order and health without controlling for family size may falsely attribute these differences to birth order. Barclay and Kolk (2015) find an increased risk of death and poor health in

[75] Their results suggest that pregnancy results in permanent changes in the spiral arteries which play a vital role in supplying nutrients to the placenta and fetus. This could explain why the birth weight increases with parity, particularly between the first and second born.

[76] Studies of different mammals have shown that primiparous females are less successful in rearing a calf than females with earlier births. However, it is not clear whether there are biological reasons for this pattern or whether it is due to lack of rearing experience (see e.g Ibanze et al. 2013)

adulthood for higher birth order siblings also when controlling for family size. Using Swedish registry data, they document a higher mortality risk between the ages 30 and 69 for individuals with a higher birth order, in particular for mortality due to cancers of the respiratory system and to external causes. Using Norwegian data, Black et al. (2015) study self-reported health and find birth order effects in different directions depending on the type of health problem. They find that later-born siblings are more likely to smoke and have poorer self-reported physical and mental health in their 40's. Firstborns are, on the other hand, more likely to be overweight, obese and have high blood pressure. In contrast to the last result, Barclay and Myrskylä (2014) find, when studying the physical fitness among 18 year old men in Sweden, a monotonic negative effect of birth order which could suggest that later-born siblings take less care of their health.

As discussed in the introduction, less is known about birth order and health in childhood and adolescence. Previous studies tend to support the idea that higher birth order siblings engage in more risky behavior such as smoking and that this behavior begins in early age. Argys et al. (2006) use data from the US (NLSY79) and study risky behavior such as smoking, drinking alcohol and marijuana usage at age 12-16. They find a positive correlation between this type of risky behavior and having an older sibling. Another study finds that birth order affects delinquency behavior both among individuals in Florida and Denmark; Breining et al. (2017) show that second-born siblings have more disciplinary problems at school and are more likely to enter the criminal system than firstborns. Two small sample studies, which could not control for family size, have found that younger siblings are more likely to experience accidents (Bijur et al., 1988, Nixon and Pearn, 1978).

2.2 Mechanisms

Our study is also closely related to the literature studying the mechanisms behind the documented pattern that higher birth order children have lower cognitive and non-cognitive skills, lower educational attainment and lower earnings. Theoretically, birth order effects could emerge through several different channels. Broadly, we could divide these different channels into two categories: biological differences, and differences in the environment where the children grow up. The first category, which is related to health at birth, does not receive any support in the previous literature. As discussed earlier, firstborn children are more likely to have worse health at birth than their younger siblings, not better. The finding that the explanation is not biological is also supported by the evidence found in Barclay (2015a). He finds that the effects of the sibling order of adopted children are associated with differences in educational attainment. Compared to results from families with biological children, he finds that the birth order effects are slightly

stronger in families with adopted children. This strongly indicates that the birth order effects are driven by intra-family social dynamics rather than by biological differences.

The post-birth differences in family environment could be due to many factors such as, for example, parental time and investment and changes in the family environment due to the presence of children of different ages. The dilution hypothesis (Blake, 1989), which could be traced back to Becker and Tomes' (1976) influential article on the quantity and quality of children, argues that birth order effects could be explained by parental time and financial constraints. The firstborn child will not have to share parental time with any siblings, at least not during the first period in life. Since parental time is limited, eventual consecutive children will get less parental quality time during the first years. However, related to this, parents might become better parents over time which could possibly mitigate the parental dilution effect or even reverse the total effect. Using US data on time usage, Price (2008) finds that parents do, on average, spend an equal amount of time with each child at every point in time. Thus, aggregating over the whole childhood, parents spend less time with each additional child. The differences are especially large between first and later-born siblings in the time spent with their parents in early childhood.[77]

A recent study by Black et al. (2016) estimates the effect of parental resources by studying the effect of having a disabled sibling and concludes that the negative sibling spillover is partly due to lower parental time exposure and financial resources. A couple of studies have tried to test whether earlier birth order differences in investments can explain later outcomes. Monfardini and See (2012) find that the relationship between birth order and education remains significant and negative even when controlling for maternity time with the child. Lehmann et al. (2016) explore in utero and early childhood investments in health, education and maternal emotion/verbal responsiveness during the child's first year. However, controlling for variations in early childhood factors, the birth order effects are robust. Using data from the National Longitudinal Survey of Youth, Children and Young Adults (NLSY-C) 1979 in a structural framework, Pavan (2015) finds that the differences in parental investments account for more than one-half of the gap in cognitive skills among siblings. A somewhat different mechanism is explored in Hotz and Pantano (2015). In a model of strategic parenting, they find that it may be optimal for parents to be stricter with earlier-born children. Using the NLSY-C, they find some support for their model as earlier-born children are subject to more rules and monitoring by parents than later-born children. That first-borns are supervised more than their siblings is also

[77] Price does not have siblings in his data, instead he uses a matching strategy to compare a firstborn child to a second-born child. He does not have any information on completed fertility and the time use data is only for one parent.

found by Avrett et al. (2011). In an evolutionary perspective, it may be beneficial to invest in a child with higher potential returns. Stanton et al. (2014) find when studying maternal investment among chimpanzees that primiparous mothers invest more in their infant than multiparous mothers. However, since firstborns have worse health at birth, the investment in firstborns appears to be compensatory since the probability of survival did not differ by birth order.

Related to the dilution hypothesis is the confluence model with the idea that the intellectual development of a child depends on the average intellectual environment which can be considered as the average of all members in the family (Zajonc, 1976). When the first child is born, the intellectual environment is relatively high, but it will decrease quickly as the family grows since intellectual growth is a function of age. Zajonc (1976) also finds support for the no one to teach hypothesis, stating that the youngest child (and an only child) will not get the chance to teach younger siblings, which could be important for learning. The idea that older siblings in the family change the family environment, which has detrimental effects on later-born children, may also be applicable to the health outcomes studied in this paper. Older siblings may create a more hazardous family environment by introducing toys or activities which are suitable for older children. Another plausible mechanism is that later-born children are, on average, more exposed to family disruptions such as divorces, or experience the loss of a parent at younger ages. Family disruptions could have a negative effect on educational attainment. Björklund et al. (2007) observe this negative relationship between parental separation and children's educational attainment using both Swedish and US data. However, performing a sibling-difference estimation, this relationship is no longer significant, indicating that the negative relationship is due to selection rather than causation.

As discussed in the introduction, the birth order pattern may also be explained by parents' fertility decisions; if parents have a child who is difficult to rear, this might influence their decision not to have another child and give rise to a non-causal correlation between birth order and child outcomes. Pavan (2015) uses a structural approach and estimates an achievement production function which accounts for selection bias due to endogenous fertility decisions of mothers. Using US data from the National Longitudinal Survey of Youth, Children and Young Adults, he finds that optimal stopping, where parents stop having children after getting a difficult child, cannot explain the birth order effects.

3. Empirical Strategy

To estimate causal effects of birth order on health, we would like to have a random assignment of birth order. This is in fact the case within families, since a child receives a random half of each parent's genes at conception. Thus, by controlling for family fixed effects and, thereby, exploiting only the variation in health between siblings, we will capture the prenatal and postnatal birth order effects on health.[78] However, since there are trends in our health measures over time, we also need to control for birth cohorts. This creates an unbalance in family background by birth order because higher birth order children in a cohort do, on average, have older mothers and mothers with larger families have their first child at a younger age. This may bias the estimated effect of birth order. To reduce the bias, we control for the mother's age at birth. As a consequence, we are identifying the effect of birth order from unequal spacing of children. If unequal spacing is due to some other family characteristics, the estimate may still be biased.[79] More specifically, we will estimate the effect of birth order on children's health using the following model:

$$H_{if} = \alpha + \sum_{b=2}^{k} \beta_b \, (Birth\ Order)_{if} + \gamma X_i + \delta_f + \varepsilon_{if},$$

where H is health status, i denotes the individual child and f denotes family. β_b captures the birth order effect (where k = 2, 3, 4 or >4 is the birth order) relative to the firstborn child. We control for other individual-specific characteristics in X_i, including mother's age at birth (third-order polynomial), father's characteristics and indicators for the child's sex and birth cohort. δ_f are family fixed effects capturing all time-invariant characteristics of the family.[80] The child's birth order is set by the number of births of the mother.[81]

By including a fixed family effect, we are identifying the effect of birth order on families where at least one child has been sick or, in the case when we are studying mortality, on families where at least one child has died. A concern may be that families with a sick or dead child are different from

[78] This is true for siblings with the same mother and father. For siblings with different fathers there will could be birth order effects in gene-composition if there the fathers of later born siblings are systematically different from fathers of later born siblings.

[79] See Black et al. 2015 for a further discussion of the empirical challenges when estimating birth-order effects.

[80] Tables A1 and A4 in Appendix display the results from estimations of the model with and without family fixed effects and separately for each parity. In specifications without family-fixed effects, we add control variables for: mother's age at first birth, family size, birth cohort of the mother, and the mother's educational attainment.

[81] In our definition, siblings have the same mother but may have different fathers.

other families, implying that the results are not generalizable to the whole population. This may be a problem, especially for very rare events such as child mortality. We will discuss this issue when presenting the results on mortality and investigate the question of heterogenous effects in Section 7.

A potential threat to the identification when studying birth order effects is that the effects may be due to reverse causality, i.e., in our setting, this implies that the child's health affects parents' fertility decision. A negative (positive) health effect of birth order may arise if parents stop having children after a particularly unhealthy (healthy) child. For example, suppose that an unhealthy child requires more time from time constrained parents. In that case, families with an unhealthy child will postpone or perhaps even refrain from having another child, thus giving rise to negative birth order effects on health. In the extreme case, the child may be of such poor health that it dies. In that case, parents are not time constrained and may decide to have another child which may give rise to a pattern where higher birth order children are less likely to die. Van den Berg et al. (2016) study the impact of child deaths due to unintentional accidents on parental outcomes and find an increased probability that mothers have another child two to four years after the death of the child. Thus, endogenous fertility decisions may give rise to spurious negative birth order effects if the child is unhealthy and positive birth order effects if the child dies.

These hypotheses are difficult to test since the health status of a child is to large extent associated with parental characteristics. Although there is some randomness to the health status of the child, it is difficult to think of any exogenous factor – unrelated to parental characteristics and other factors determining preferences for family size – which affects the child's health that we can use to estimate the causal effects of the child health of previous children on family size. Instead, we make use of our rich data with detailed information on parental background characteristics and study whether the probability of having another child is affected by the initial health status of previous children when controlling for a battery of parental characteristics and characteristics of already born children. Thus, we estimate the following model:

$$P(another\ child)_f = \alpha + \sum_{b=1}^{3} \beta_b Health_{if} + \sum_{b=1}^{3} \mu_b \mathbf{Z}_{if} + \gamma \mathbf{X}_f + \varepsilon_f,$$

where *f* denotes family and *i* individual child. $Health_{if}$ is the health status of already born children, β_b captures the effect of the health of the firstborn, second-born and third-born child. **Z** includes indicators of sex and cohort year of the born children, **X** includes family-specific factors which might affect the probability of having another child (educational attainment, mother's age at first birth (third-order polynomial), both parents birth cohorts, the residential region of the parents the year before first birth, whether any of

them have a foreign background and their incomes before the first birth) and ε_f is the error term. The model is estimated separately for the decision to have one or more children, two or more children, three or more children and four or more children. When studying the decision to have one or more children, we include all families with one or more children and estimate the effect of the health status of the first child at age 0–2 on having another child. For the decision to have two or more children, we estimate the effect of the health status of the first child at age 0–4 and the second child at age 0–2 on the probability of having another child in the population of families with two or more children. Finally, we estimate the effect of the health status of the first child at age 0–6, the second born at age 0–4 and the health status of the third child at age 0–2 on the probability of having four or more children on the population of families with three or more children. The idea is to analyze whether parents base their decision to have another child on whether they had a previous "bad draw" or a previous "good draw". To capture the health status, which should be of relevance for subsequent fertility decisions, we measure the health of the youngest child at age 0–2, the health of the second youngest at age 0–4 and the health of the oldest child at age 0–6, assuming there to be about two years between each sibling.[82] If there is a correlation between the error term and the health of the previous child, the estimated β will be biased. Thus, the identification of the effect hinges on whether we manage to control for all factors which affect both the probability of having another child and the health of previous children.

4. Data

Our data set merges information from several administrative registers covering the universe of all children born in Sweden 1968-2005. Children and parents are linked through the *Multigenerational Register* which includes information on family relations starting in 1932. To this, we add information from different administrative registers to follow children from birth to age 24 and their parents. Health status will be measured with administrative register data on hospitalization and mortality. Data on hospitalization comes from the *Swedish National Inpatient Register* which contains information on hospital admissions 1987–2011. It includes administrative information on the date of admission, the number of days in hospital, and discharge diagnosis classified according to the International Classification of Diseases (ICD). Information on mortality comes from the *National Cause of Death Register* which contains the date of death and the main underlying cause of death coded according to ICD. Information on parental characteristics comes from the *Longitu-*

[82] In our sample, the median spacing between the first and the second child is 29 months, between the second and the third 39 months and between the third and the fourth 34 months.

dinal Integration Database for Health Insurance and Labour Market Studies (LISA) which integrates data from population, tax, and social insurance registers.

4.1 Health Measures

Health is measured by hospital admissions and mortality observed in the registry data. A benefit of using register data is that it covers the whole population and thus, does not suffer from non-representativeness, which is often a problem in surveys when participation is voluntary. The first measure that we use is an indicator of whether the child has been admitted to hospital. The potential problem with using hospitalizations as a measure of health is that it might capture health consumption rather than the underlying health status. This should be a minor problem in our setting for three reasons. First, as all individuals in Sweden are covered by the public health care insurance and healthcare is free of charge for children, family financial resources do not directly affect the usage of the health care system.[83] Second, it is unlikely that admittance to hospital is determined by parental preferences since patients are only allowed to stay overnight in hospital if the medical staff consider it necessary. As shown by Table A4 in Appendix, the birth order effects that we observed are confirmed when studying longer hospital stays; thus, it is unlikely that our estimates are influenced by the parents' preferences for hospital care. Third, since we compare siblings within a family, we are controlling for all in-variant family factors that affect the health care consumption of all children in the family, such as the parents' inclination to consume healthcare and the average health status among the children. Our second health measure is mortality. The benefit of studying mortality as a health outcome is that it is an objective and unambiguous measure. However, studying mortality among young individuals might be less informative, since death is a very rare event in childhood (especially after infancy) and therefore captures very little health disparities. Here, we study the association between birth order and mortality in infancy and up to age 24.[84]

Figure 1 shows infant mortality rates and Figure 2 hospitalization rates for different age categories. The figures show a downward trend in adverse

[83] In-patient care is free for children up to age 18. At ages 19─24, the fee varies across counties. In some counties, in-patient care is free up to the age of 24 but most counties charge a fee ranging from 80 to 100 SEK (8─10 Euros) per night after the age of 20.

[84] The regulations regarding how to categorize children that die during pregnancy or at birth have not changed over the time period covered in this study. A child should be registered at the Swedish Tax Agency if he or she was born in Sweden or has a mother that is registered as a Swedish resident. All live births, and in utero deaths beyond week 28 of gestation, are defined as children. If gestation is unknown, the child should be at least 35 centimeters. In utero deaths decreased over the years 1973–1985, but have since then been constant at 3–4 deaths per 1,000 births. The regulations changed in 2008 (cohorts born after 2005 are not included in our study) to 22 weeks of gestation (Socialstyrelsen, 2009).

health events among young children. Infant mortality has decreased during the whole period as well as hospitalizations among the younger age categories. For the older age categorizes, hospitalization rates have been rather stable over the period 1987–2011. It is clear from these pictures that there are strong time trends in our measures of health and it is important to take the trends into account. There are also large differences between age categories. The youngest (age 0–6) and the oldest (age 19–24) are most likely to be admitted to hospital.[85] The least likely to end up in hospital are children 7–12 years old.

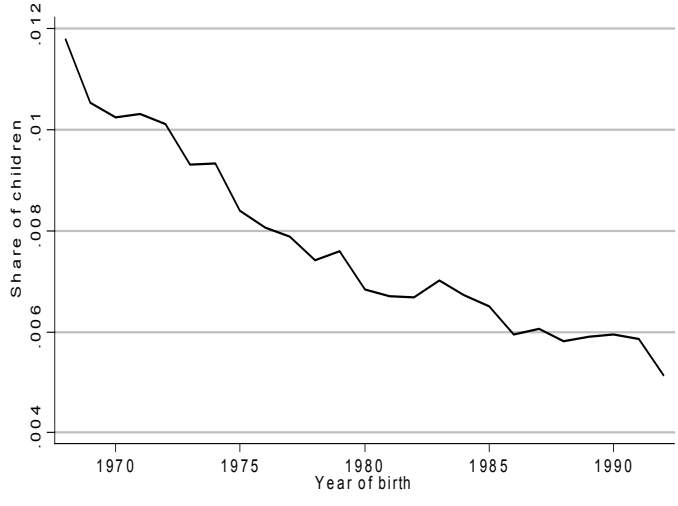


Figure 1. Infant mortality, years 1968-1992[86]

[85] The time series for age 0─6 stops in 2009 because the cohort born 2009 is the youngest cohort in the data.

[86] We lack data on those individuals that died in the same year as they were born after year 1992.

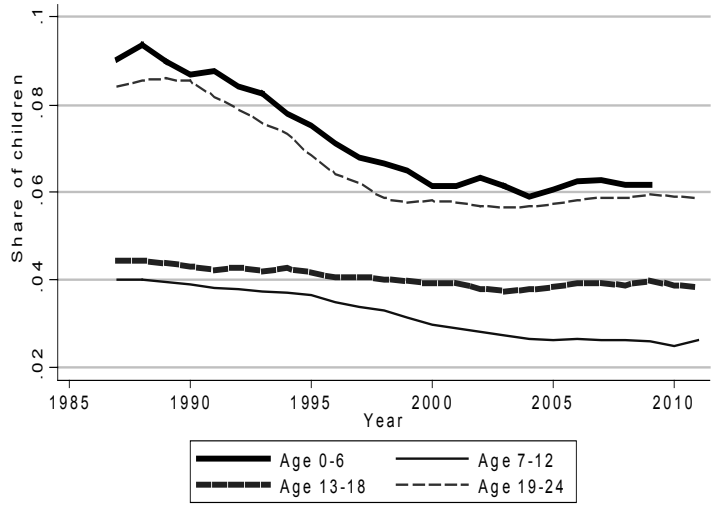


Figure 2. Hospitalization by age category, years 1987–2011

Our objective is to study how health develops through childhood and, by studying different sorts of health problems, to shed some light on the mechanisms leading to the negative birth order effect on later life outcomes. As general measures of health we study whether a child has been admitted to in-patient care. To measure health at birth, we study in-patient care in early childhood (age 0–6) for diagnoses related to congenital malformations and perinatal conditions which originate from conditions in utero or at birth. It is not straightforward to define causes for hospitalization due to parental behavior during childhood. We will primarily focus on two measures: Our first measure is in-patient care due to injuries and being poisoned. The motivation is that hospitalization for injuries and poison in early childhood should be related to how closely the child is looked after. Our second measure is in-patient care for 'avoidable' conditions. These are conditions that would not have been a cause for hospitalization if the child had had access to timely and effective primary care.[87] This measure is commonly used as an indicator

[87] Avoidable conditions, or as also called in the literature, ambulatory care-sensitive conditions, include conditions where appropriate primary care may prevent the onset of, control an acute episode of, or manage a chronic condition or illness. Avoidable conditions can be divided into three categories: conditions that can be prevented through vaccination; selected chronic conditions that can be managed by pharmaceuticals, patient education and lifestyle; acute conditions for which hospitalization is commonly avoidable with antibiotics or other medical intervention. The concept is frequently used when evaluating the quality of primary care as well as in research. For example, Billings et al. (1993) study the association between socioeconomic status and hospitalization rates due to avoidable conditions among communities in the US. We use the definition for children suggested by the Public Health Information Development Unit in Australia which is based on a comprehensive review of the literature (Page et al. 2007). A complete list of the diagnoses can be found in Appendix Table A1.

of the quality of, or access to, primary care, but a higher incidence of hospitalization due to avoidable health conditions could also be due to parents not seeking care in time.[88] Avoidable hospitalization includes conditions such as anemia, asthma, diabetes and chronic obstructive pulmonary disease.

We also study in-patient care related to diseases of the respiratory system since previous research has shown that diseases of the respiratory system are related to later in life outcomes such as school performance and labor market success (Case et al., 2005), and it is also the most common cause for hospitalization in early childhood. As the child grows older, both the family environment and the child's own behavior will affect the causes for being admitted to hospital. To investigate when potential health differences between siblings appear, we study the age intervals 0–6, 7–12, 13–18 and 19–24. For adolescents and young adults, we study in-patient care for injuries and poisoning, self-harm, mental health conditions and conditions caused by excess consumption of alcohol. As a test that there is nothing inherently different between younger and older siblings, we study in-patient care due to cancer since cancer among children and adolescents can be considered to be random and not affected by parental or child behavior.[89] A shortcoming with using cancer as a test of our identification strategy is that it is a rare condition among children.[90]

4.2 Sample Restrictions

Our main sample consists of children who were born in 1968–2005 and who have parents that we observe in the data. Since our outcome measures are limited to certain years, we cannot observe all children at every age. Hospitalization measures are observed for 1987–2011 and information about which particular cohorts are included when studying health at certain ages is displayed in Table 2.[91] We exclude families with only one child since we cannot estimate birth order effects within families for these children. We also exclude families with multiple births (twins, triplets etc.), since their circumstances differ compared to siblings born as singleton births. For example, the pre-natal circumstances are likely to differ and earlier born siblings do not have any time alone with the parents. Furthermore, we make the

[88] The last point being closest to what we study as we are using family fixed effects. Access to health care (in terms of distance, family connections etc.) and quality of primary care should be the same for all siblings within a family.

[89] In contrast to cancer among adults, research has shown that most childhood cancers do not have any outside causes. There is a genetic component for some types of childhood cancer but as the genetic set-up among siblings is random, this should not give rise to any birth-order effects. See the discussion of the causes of childhood cancer on the American Cancer Society' webpage. http://www.cancer.org.

[90] See Appendix Table A1 for a complete list of all ICD-codes included in each condition category.

[91] Grades at 9th grade (age 16) are observed for cohorts born 1972–1994.

restriction that all children must have been born in Sweden to limit the risk that the children have experienced very different circumstances in the first years of life. For the same reason, we exclude families that have adopted a child, or given a child up for adoption. Lastly, in our main sample, we have complete families, meaning that we restrict our sample to families with completed fertility, imposing the restriction that mothers are at least 45 years old in 2009.[92]

4.3 Summary Statistics

The demographic characteristics of our sample are displayed in Table 1. Families in our sample, which consist of families with two, three, four or more children, have 2.8 children on average. On average, children were born in 1982, their mothers were born in 1953 and their fathers were almost three years older than their mothers.

Table 2 shows that hospitalization is most common among the youngest children (aged 0–6): about 37 percent have been admitted to hospital at least once. The lowest admission rates are found among children aged 7–12, thereafter the rates are increasing with age. Table 2 also displays which diagnoses that are most common by age category and will guide us in deciding which outcomes to study at what age. Hospitalizations related to perinatal and congenital malformations are by far most common among the youngest children. Almost all cases occur within the first year of life, 7.9 percent of all 0–1 year olds are admitted to hospital with this diagnosis. In contrast, hospitalization for mental health conditions and conditions related to self-harm and alcohol are most common in adolescence and among young adults.

[92] 2009 is the year in which our sample is drawn from the Multigenerational register. The Multigenerational register is continuously updated by Statistic Sweden and the variables birth order and number of children are collected, and created, within the register. Since the register has a very good coverage from 1932 onwards, we can be confident that we are capturing all siblings at the beginning of our sample period but additional restrictions have to be made regarding mother's age to be confident that we have complete families also at the end of the period.

Table 1. Demographic variables

	Mean	SD
Number of children	2.810	(1.031)
Birth order 1	0.373	(0.484)
Birth order 2	0.396	(0.489)
Birth order 3	0.166	(0.372)
Birth order 4	0.046	(0.209)
Birth order >4	0.019	(0.137)
Female	0.485	(0.500)
Year of birth, child	1981.833	(8.204)
Month of birth, child	6.234	(3.366)
Year of birth, mother	1953.331	(6.878)
Year of birth, father	1950.727	(7.753)
Years of education, mother	12.069	(2.408)
Years of education, father	11.731	(2.586)
Observations	2,106,531	

Table 2. Hospitalizations and medical conditions by age

	Age 0–6	Age 7–12	Age 13–18	Age 19–24
Hospitalization	0.368	0.164	0.188	0.199
	(0.482)	(0.370)	(0.391)	(0.400)
Perinatal & Congenital malformation	0.087	0.011	0.007	0.005
	(0.282)	(0.104)	(0.084)	(0.069)
Respiratory & Eye/Ear	0.150	0.037	0.032	0.029
	(0.357)	(0.190)	(0.176)	(0.168)
Injury and poison	0.058	0.049	0.065	0.062
	(0.233)	(0.216)	(0.247)	(0.240)
Avoidable conditions	0.072	0.015	0.015	0.015
	(0.258)	(0.121)	(0.121)	(0.121)
Mental health	0.006	0.004	0.020	0.024
	(0.078)	(0.063)	(0.141)	(0.153)
Self-harm	0.000	0.000	0.006	0.008
	(0.021)	(0.015)	(0.080)	(0.090)
Alcohol	0.000	0.000	0.011	0.008
	(0.020)	(0.014)	(0.105)	(0.091)
Cancer	0.004	0.003	0.004	0.005
	(0.062)	(0.054)	(0.062)	(0.069)
Observations	644,589	1,155,264	1,474,603	1,463,458

Notes: Means and standard deviations (in parenthesis).

5. The Effect of Birth order on Health

In this section, we present the results from the first model where birth order is modeled to affect health. First, we present the results on the probability of being admitted to hospital and for the different diagnoses discussed in Section 4.

5.1 Hospitalization

Table 3 and Table 4 present the results of regressing health measured as hospitalization for different diagnoses on birth order, family-fixed effects, and a set of additional controls, which are discussed in Section 3. Table 3 contains the results for children aged 0–6. Column (1) displays the results for ever being hospitalized for any condition. The risk decreases by 1.3 percentage points (3.5 percent) for the second-born child and by 1.5 percentage points (4.1 percent) for the third-born child relative to the firstborn child. No statistically significant difference is found for children with a higher birth order. Columns (2) – (5) report the results for the diagnoses discussed in the previous section and a clearer pattern emerges across diagnoses. A strong negative effect of being firstborn is found on perinatal conditions and the risk of being born with congenital malformations. Second-born children are 4.1 percentage points less likely to be hospitalized, which corresponds to a 47 percent reduction given the mean of 8.7 percent. For the remaining conditions, there is a positive relationship between birth order and being admitted to hospital. These effects are also increasing over birth order. For conditions related to the respiratory system and eyes and ears, which is the most common diagnoses category, 15 percent of all children aged 0–6 in our sample have been hospitalized for any of these conditions and the risk is 2.4 percentage points (16 percent) higher among second-born children than among their older sibling. The effect is twice as large for a sibling order higher than 4. Second-born children are 1 percentage point more likely to end up in hospital for conditions related to injuries and siblings with birth order 5 or higher are 2.5 percentage points more likely, which corresponds to 17 and 43 percent, respectively. For avoidable conditions, the effects range from 0.9 to 2.3 percentage points (16 and 40 percent). For cancer, which is rare in this age category, we find no birth order effects.

To see how the effects on different conditions develop, we estimate the birth order effects as infants (age 0–1) and at age 0–3. The results presented in Appendix Table A3 show that the effect on perinatal conditions is apparent among infants whereas the birth order effect on admittance to hospital for injuries appears later. This pattern is expected as perinatal conditions are due to conditions before or at birth and injuries are due to factors in the family environment after birth.

In Panel B, the results for children aged 7–12 are displayed. No birth order effect is present for all-cause hospitalization or for perinatal conditions and congenital malformations. However, an interesting pattern is present in column (3), showing that children with a higher birth order have a lower probability of being hospitalized than their older siblings for conditions related to the respiratory system and eyes and ears. These conditions might be caused by infections transmitted from younger siblings since they are most prevalent among young children, a child aged 0–6 is almost five times as likely to be hospitalized for these conditions as compared to children aged 7–12. Thus, lower-parity siblings with younger siblings are more likely to be exposed to infections when they are 7–12 years old than later-born siblings who will not have any small children in the household when they are in the same ages. The birth order effect for injuries is positive and only slightly smaller than what is found at age 0–6, second-born children are 12 percent more likely to be injured and fourth-born children have a 29 percent higher risk as compared to their oldest sibling. The results for avoidable conditions reveal a weak negative and mainly statistically insignificant relation, and the birth order effects on cancer are zero also in this age category.

In sum, the overall risk of being admitted to hospital across birth order is somewhat lower for second- and third-born children than for firstborns in the youngest age category. However, the overall admission rates conceal underlying systematic differences across birth order in health. Inspecting the effects on the probability of receiving different conditions, our results show that younger siblings have better health at birth compared to firstborn children. Later-born siblings are, however, more likely to be hospitalized for other conditions that could be related to parental investments and the family environment.

Table 3. Birth order effects on hospitalization and diagnoses ages 0–6 and 7–12

	(1) Hospital-ization	(2) Perinatal & congen-ital mal.	(3) Respiratory & eye/ear	(4) Injury & poisoning	(5) Avoidable diagnoses	(6) Cancer
Panel A: Age 0–6						
Birth order 2	-0.013***	-0.041***	0.024***	0.010***	0.009***	-0.001
	(0.004)	(0.002)	(0.003)	(0.002)	(0.002)	(0.000)
Birth order 3	-0.015**	-0.047***	0.034***	0.016***	0.016***	-0.001
	(0.007)	(0.004)	(0.005)	(0.004)	(0.004)	(0.001)
Birth order 4	-0.007	-0.047***	0.043***	0.018***	0.023***	-0.002
	(0.010)	(0.006)	(0.008)	(0.005)	(0.006)	(0.001)
Birth order >4	0.003	-0.043***	0.052***	0.025***	0.023***	-0.001
	(0.015)	(0.009)	(0.011)	(0.008)	(0.008)	(0.002)
Observations	644,589	644,589	644,589	644,589	644,589	644,589
R-square	0.618	0.608	0.615	0.572	0.597	0.573
Mean	0.368	0.087	0.150	0.058	0.072	0.004
N clusters	360,806	360,806	360,806	360,806	360,806	360,806
Panel B: Age 7–12						
Birth order 2	0.001	-0.001	-0.005***	0.006***	-0.001**	-0.000
	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)
Birth order 3	0.001	-0.000	-0.008***	0.010***	-0.002	-0.000
	(0.003)	(0.001)	(0.002)	(0.002)	(0.001)	(0.001)
Birth order 4	0.001	-0.000	-0.009***	0.014***	-0.002	-0.000
	(0.005)	(0.002)	(0.003)	(0.003)	(0.002)	(0.001)
Birth order >4	-0.002	0.000	-0.011***	0.012**	-0.003	0.001
	(0.008)	(0.002)	(0.004)	(0.005)	(0.003)	(0.001)
Observations	1,155,264	1,155,264	1,155,264	1,155,264	1,155,264	1,155,264
R-square	0.534	0.522	0.530	0.509	0.526	0.509
Mean	0.164	0.011	0.037	0.049	0.015	0.003
N clusters	578,318	578,318	578,318	578,318	578,318	578,318

Notes: Results from linear probability models with family fixed effects. The omitted category is firstborn child. Standard errors are clustered by family. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include controls for mother's age at birth, and indicators for child's birth cohort and gender. For siblings with different fathers, indicators for father's cohort and educational attainment are included.

Table 4, Panel A, shows the effect of sibling order on hospitalization and diagnoses for children aged 13–18. Across all outcomes, we find strong, positive birth order effects. The risk of being hospitalized for any condition is 9 percent higher for second-born children as compared to firstborn, and the risk increases over birth order and is 21 percent higher for fifth- or higher order born siblings. Focusing on mental ill-health, and conditions related to self-harm and alcohol consumption, we find monotonically increasing ef-

fects of birth order. The size of these effects ranges from 15 percent for mental health for second-born children to 77 percent for diagnoses related to self-harm for fifth or higher birth order born children. If we relate these effects to the socioeconomic gradient in hospitalization, we find that the effects are sizeable; Mörk et al. (2014) show that children with parents with incomes in the lowest percentile are 40 percent more likely to end up in hospital than children from families with the highest incomes, for injuries the gradient is 33 percent and for mental health conditions about 70 percent. The effect on cancer is again zero in adolescence and young adulthood, indicating that this condition affects children of different birth order with equal likelihood.

A very similar pattern is found in Panel B which displays the results for young adults aged 19–24; however, the birth order pattern for mental health and conditions related to self-harm is significantly less pronounced. Since there are strong effects on alcohol related conditions, it is possible that some of the other outcomes are related to alcohol consumption. In particular injuries, poor mental health and self-harm might be correlated with conditions related to alcohol. In the Appendix, Table A5, we test this by deducting any hospital stay related to these conditions if the same individual has also been hospitalized for conditions related to alcohol in the same age span. We find a lower effect on hospitalization for mental health conditions suggesting a connection between mental health and alcohol problems and self-harm and alcohol problems for the older age category. The other results remain the same.

The results by family size, with and without family fixed effects, are reported in the Appendix, Table A2. Overall, the findings that we report are rather robust across specifications. The birth order effects, from the estimation with fixed family effects, do not seem to vary with parity, implying that we can pool all families regardless of size. In specifications without family fixed effects, there is a clear negative birth order effect on hospitalization for the youngest ages which is not robust to the inclusion of a family fixed effect (see the results in Table A2, panel A and B). The reference category is always the firstborn child.

Table 4. Birth order effects on hospitalization and diagnoses ages 13–18 and 19–21

	(1) Hospital-ization	(2) Resp eye/ear	(3) Injury & poisoning	(4) Avoidable	(5) Mental health	(6) Self-harm	(7) Alcohol	(8) Cancer
Panel A: Age 13–18								
Birth order 2	0.017***	0.005***	0.007***	0.002***	0.003***	0.002***	0.004***	0.000
	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.000)	(0.000)
Birth order 3	0.025***	0.008***	0.010***	0.004***	0.005***	0.002***	0.005***	0.000
	(0.003)	(0.002)	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Birth order 4	0.035***	0.011***	0.017***	0.005***	0.007***	0.004***	0.008***	0.000
	(0.005)	(0.002)	(0.003)	(0.002)	(0.002)	(0.001)	(0.002)	(0.001)
Birth order >4	0.040***	0.008**	0.012**	0.006**	0.011***	0.005**	0.008***	0.001
	(0.008)	(0.004)	(0.005)	(0.002)	(0.003)	(0.002)	(0.002)	(0.001)
Obs.	1474603	1474603	1474603	1474603	1474603	1474603	1474603	1474603
R-square	0.525	0.512	0.508	0.519	0.500	0.485	0.487	0.510
Mean	0.188	0.032	0.065	0.015	0.020	0.006	0.011	0.004
N clusters	737,256	737,256	737,256	737,256	737,256	737,256	737,256	737,256
Panel B: Age 19–21								
Birth order 2	0.017***	0.003***	0.007***	0.002***	0.001	0.001**	0.001***	0.000
	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.000)	(0.000)
Birth order 3	0.026***	0.005***	0.009***	0.003***	0.000	0.001	0.002**	0.000
	(0.003)	(0.001)	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Birth order 4	0.031***	0.006**	0.011***	0.004**	0.003	0.002	0.003*	0.001
	(0.006)	(0.002)	(0.003)	(0.002)	(0.002)	(0.001)	(0.001)	(0.001)
Birth order >4	0.028***	0.007*	0.012**	0.006**	0.000	-0.000	0.003	0.001
	(0.009)	(0.004)	(0.005)	(0.003)	(0.004)	(0.002)	(0.002)	(0.001)
Obs.	1463458	1463458	1463458	1463458	1463458	1463458	1463458	1463458
R-square	.5054218	.4896683	.493885	.4827051	.5132974	.4930366	.4950307	.4850051
Mean	.1993791	.0289868	.0615337	.0147753	.023903	.0082305	.0083829	.0047907
N clusters	709,654	709,654	709,654	709,654	709,654	709,654	709,654	709,654

Notes: Results from linear probability models with family fixed effects. The omitted category is firstborn child. Standard errors are clustered by family. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include controls for mother's age at birth, and indicators for child's birth cohort and gender. For siblings with different fathers, indicators for father's cohort and educational attainment are included.

5.2. Mortality

Next, we study the association between birth order and mortality at different ages. The results presented in Table 3 and Table 4 showed that firstborn children are more likely to be hospitalized for conditions originating in utero or at birth, whereas later-born children are more likely to be hospitalized in adolescence and as young adults. The results for mortality in Table 5 show a similar pattern. The results in the first column show that firstborn children are more likely to die before the age of one than later-born siblings and the effect is large relative to the average mortality rate: the second-born child has a 0.11 percentage point lower probability of dying and the third child a 0.33 percentage point lower probability as compared to the firstborn child. In contrast to the previous results on hospitalization due to perinatal conditions and congenital malformations, the mortality risk decreases monotonically with birth order.

Compared to the mean mortality rates in the population, the effects of birth order are huge. The large effect is partly due to the low incidence since a small number of deaths constitute a large change in the share of dead. The number of observations in the analytical sample, with a dead child in the family, when estimating the effect of birth order on infant mortality is only 32,000. The mean mortality rate in this sample is, of course, much higher, 0.33, than in the total population. If we instead post the question, how much lower is the likelihood of a second born dying as an infant as compared to a firstborn in the population of families with at least one dead child, the effect is 3.3 percent. In Section 7, we will have a closer look at whether families with at least one dead child are different in terms of observable characteristics.

Columns 2–5 in Table 5 show the effect of birth order on mortality in each age category. At age 1–6, later-born children still have a significantly lower mortality risk than their firstborn sibling. At ages 7–18, there is no birth order effect on mortality. For the oldest age group, the results indicate a reversed pattern; later-born siblings have an increased mortality risk as compared to their firstborn sibling. The overall findings in this section confirm our results on hospitalizations: lower birth order children have worse health at birth, but this change during their upbringing and firstborn children have better health than their younger siblings at older ages.

Table 5. Birth order effects on mortality

	(1) Infant mortality	(2) Age 1–6	(3) Age 7–12	(4) Age 13–18	(5) Age 19–24
Birth order 2	-0.011***	-0.001***	0.000	0.000	0.001***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Birth order 3	-0.033***	-0.003***	0.000	0.000	0.001*
	(0.001)	(0.000)	(0.000)	(0.000)	(0.000)
Birth order 4	-0.052***	-0.006***	0.000	0.001	0.001*
	(0.001)	(0.001)	(0.000)	(0.001)	(0.001)
Birth order >4	-0.068***	-0.007***	0.001	0.001	0.001
	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)
Observations	1,608,555	1,608,555	1,608,555	1,608,555	1,608,555
R-square	0.392	0.414	0.441	0.452	0.458
Mean	0.007	0.002	0.001	0.001	0.003
N clusters	739518	739518	739518	739518	739518

Notes: Results from linear probability models with family fixed effects. The omitted category is firstborn child. Standard errors are clustered by family. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include controls for mother's age at birth, and indicators for child's birth cohort and gender. For siblings with different fathers, indicators for father's cohort and educational attainment are included.

The results by family size, with and without family fixed effects, can be found in the Appendix, Table A6. The effects on mortality are several times larger in the family fixed effects models, suggesting that they are picking up some additional variation that we cannot control for with our rich set of other background characteristics. We will discuss this further in the next section where we investigate endogenous fertility decisions.

6. Optimal Stopping

As discussed in the empirical strategy section, if families stop having children when they have a child with poor health, children with a higher birth order will be less healthy, given family size. Thus, an endogenous fertility response could explain the birth order effects on health. Likewise, if parents respond to the death of a child by having another child, this will have the effect that a higher birth order will be correlated with lower mortality rates. To investigate whether families base their fertility decision on the health of previous children, we study if the health of previous children affects the probability of having another child, controlling for a range of factors which could affect family size (e.g., parental education and birth cohorts, income before first birth, parental age at first birth and residential region before first birth).

The first two columns of Table 6 show the effects of the firstborn child's health on the probability of having a second child. The result in the first column indicates that the early health status of the firstborn child, measured as in-patient care in the first two years of life, reduces the probability of the family having another child by 3.2 percentage points or, relative to the mean probability, by 3.9 percent. Hospitalizations for perinatal conditions decrease the probability by 4.0 percentage points. The results displayed in Columns 4 to 5 show the effect of first- and second-born children's health, on the probability of having a third child. As for the decision to have one or two children, the health of the last child affects whether the family chooses to have another child. However, the effect is smaller in magnitude; the probability that a two-child family decides to have a third child is 1.5 percent lower if the second child has been receiving in-patient care during its first year of life. In contrast, admittance to hospital of previous children does not seem to affect the probability that families with three children decide to have a fourth child.

Next we study whether a child's death affects the probability of having another child. The third column in Table 6 shows that when a mother has lost a child, the probability that she has another child increases by 0.1 percentage points, or 12 percent. The effect of a child's death in infancy is larger for the probability of having a third or a fourth child. The result in Column 6 shows that the probability of having a third child increases by 0.385 percentage points if the first child dies and 0.483 percentage points if the second child dies, which implies an increase of 89 and 115 percent, respectively. The probability of having a fourth child, if the third child has died in infancy, increases by over 300 percent. Thus, the results strongly indicate that the endogenous fertility response of a child's death could give rise to negative birth order effects on mortality, i.e. lower birth order children are more likely to die.

If parents respond to the health or death of previous children by changing their subsequent fertility decisions, the spacing between siblings may also be affected by the health or death of earlier-born siblings. As we can see in Table 7, the spacing of siblings is correlated with the health and death of earlier-born children. The results presented in Column 1 show that if the firstborn child is admitted to hospital during its first year of life, which increases the spacing between the first and the second child by 1.2 months. If the child is admitted to hospital with congenital malfunction or perinatal conditions, the spacing increases by 1 month (Column 2). In contrast, if the first child dies as an infant, the spacing to the next child decreases by 7.6 months (Column 3). The spacing between higher-order siblings is not correlated with the health of earlier-born children (Columns 4, 5, 7 and 8). However, as seen in Columns 6 and 9, the death of an earlier-born child reduces the spacing between later-born siblings. The spacing between the second and third birth is reduced by 19.3 months if the second child dies.

Table 6. Probability of having another child, given the health of older sibling(s)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Probability to have >1 child			Probability to have >2 children			Probability to have >3 children		
Firstborn child									
Hospitalization	-0.032***			-0.004*			0.000		
	(0.002)			(0.002)			(0.002)		
Cong. mal &		-0.040***			-0.004			0.001	
perinatal		(0.003)			(0.003)			(0.003)	
Dead			0.102***			0.375***			-0.006
			(0.011)			(0.015)			(0.010)
Second born child									
Hospitalization				-0.006**			-0.003		
				(0.002)			(0.002)		
Cong. mal &					-0.013***			0.002	
perinatal					(0.004)			(0.004)	
Dead						0.483***			0.031**
						(0.016)			(0.016)
Third born child									
Hospitalization							0.000		
							(0.002)		
Cong. mal &								-0.001	
perinatal								(0.004)	
Dead									0.348***
									(0.071)
Observations	212,549	212,549	250,358	154,878	154,878	187,217	34,682	34,682	45,369
R-square	0.245	0.244	0.242	0.121	0.121	0.138	0.117	0.117	0.122
Mean	0.840	0.840	0.840	0.421	0.421	0.421	0.100	0.100	0.100

Notes: Results from linear probability models. Robust standard errors. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression. Columns (1) – (3) include all family sizes, Columns (4) – (6) include all families with 2 or more children, and Columns (7) – (9) include all families with more than 3 children. The sample consists of cohorts born 1987-2005. All regressions include controls for mother's age at first birth, parental birth cohorts, educational attainments, incomes and region before first birth, and indicators for foreign background and previous children's birth cohort, mother's age at birth and gender.

Table 7. Spacing (months) between children, given the health of older sibling(s)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Spacing between 1st and 2nd child			Spacing between 2nd and 3rd child			Spacing between 3rd and 4th child		
Firstborn child									
Hospitalization	1.199***			0.445			-1.000		
	(0.116)			(0.302)			(0.949)		
Cong. mal & perinatal		1.022***			-0.130			0.689	
		(0.173)			(0.468)			(1.497)	
Dead			-7.623***			-12.498***			0.561
			(0.612)			(0.914)			(2.600)
Second born child									
Hospitalization				0.316			1.324		
				(0.327)			(1.044)		
Cong. mal & perinatal					0.223			3.406*	
					(0.596)			(1.970)	
Dead						-19.337***			-9.510***
						(0.884)			(2.181)
Third born child									
Hospitalization							0.529		
							(1.095)		
Cong. mal & perinatal								-2.429	
								(1.893)	
Dead									-15.696***
									(3.092)
Observations	156,065	156,065	187,807	39,936	39,936	52,771	5,575	5,575	8,139
R-square	0.062	0.061	0.066	0.110	0.110	0.108	0.283	0.284	0.236
Mean	35.338	35.338	43.464	47.771	47.771	46.843	44.089	44.089	44.537

Notes: Results from linear probability models. Robust standard errors. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression. Columns (1) – (3) include all family sizes, Columns (4) – (6) include all families with 2 or more children, and Columns (7) – (9) include all families with more than 3 children. The sample consists of cohorts born 1987–2005. All regressions include controls for mother's age at first birth, parental birth cohorts, educational attainments, incomes and region before first birth, and indicators for foreign background and previous children's birth cohort, mother's age at birth and gender.

Admittedly, the models estimated in this section may suffer from bias due to selection, since the identification strategy hinges on the assumption that we are able to control for all factors that determine both fertility and the health of the child. However, given the large battery of control variables and the consistency of the results, the analysis provides suggestive evidence that the health of born children affects subsequent fertility decisions.

The results suggest that parents are resource constrained and having a child with poor health, which may require more time from the parents, reduces the probability of having another child for a given family size preference. If parents have a firstborn with poor health, but decide to have another child, they are more likely to postpone that birth. On the other hand, if the child dies, resources are freed and parents are more likely to have another child. The spacing between the births is then shorter than average spacing. Assuming that parents who have a child with poor health have fewer children than planned and parents who experience the death of a child have more children than planned, the sibling order of the last child is not independent of the health or death of already born children and the estimated birth order effects are biased. To remedy this problem, we remove the last born child in every family and re-estimate the effects of sibling order on child health and mortality. If the effects are much smaller, it would be an indication that the effects of birth order found in Section 5 are largely due to endogenous fertility responses.

Table 8 presents the estimated birth order effects on infant mortality and health at age 0–6 and age 13–18. The results in panel A, Column 1, show large birth order effects on infant mortality also in the restricted sample. However, the estimates are smaller than the results, for the full sample, presented in Table 5. The second child has a 118 percent lower probability of dying and the third born a 300 percent lower probability as compared to the firstborn. The estimated effects using the whole sample presented in Table 5 was a 167 percent lower risk for the second born and a 501 percent lower risk for the third born. The reduced effect indicates that endogenous fertility responses can explain at least part of the birth order effect on infant mortality.

Panel A, columns 3 to 6, presents the estimated effects of birth order on the probability of all-cause and cause-specific hospitalization. The estimates are remarkably similar to the birth order effects estimated on the full sample. Restricting the sample by removing all last born children reduced the number of observations from over a million to 167,876, implying that we lose precision. For less common conditions, such as injuries and avoidable conditions, the estimates are no longer statistically significant although the estimates are of a similar magnitude to those estimated on the full sample. The lower panel displays the results from estimating the effect of birth order on different causes of hospitalization at the age of 13–18. These estimated effects are virtually exactly the same as those estimated on the full sample presented in Table 4. The results for the categories 7–12 and 19–24 are dis-

played in Table A7 in the Appendix. For age category 7–12, the results remain the same, while for age category 19–24, the effects on the rare conditions such as mental health, self-harm and alcohol-related conditions lose statistical significance.

Table 8. Birth order effects on infant mortality and health ages 0–6 and 13–18, restricted sample

	(1) Infant mortality	(2) Hospital-ization	(3) Perinatal & cong. mal	(4) Resp eye/ear	(5) Injury	(6) Avoidable	(7)
Panel A: Infant mortality and hospitalization different causes age 0–6							
Birth order 2	-0.015***	-0.004	-0.039***	0.026***	0.011***	0.006	
	(0.001)	(0.008)	(0.005)	(0.006)	(0.004)	(0.004)	
Birth order 3	-0.038***	-0.001	-0.044***	0.044***	0.014*	0.012	
	(0.002)	(0.016)	(0.009)	(0.012)	(0.008)	(0.009)	
Birth order 4	-0.056***	0.002	-0.044***	0.050***	0.015	0.016	
	(0.003)	(0.024)	(0.014)	(0.018)	(0.013)	(0.014)	
Birth order >4	-0.074***	0.022	-0.053***	0.058**	0.023	0.014	
	(0.004)	(0.034)	(0.020)	(0.026)	(0.018)	(0.019)	
Obs	593,322	167,876	167,876	167,876	167,876	167,876	
R-square	0.440	0.664	0.646	0.664	0.626	0.650	
Mean	0.013	0.379	0.082	0.157	0.062	0.072	
N clusters	278469	102,215	102,215	102,215	102,215	102,215	
Panel B: Hospitalization different causes age 13–18							
	Hospital-ization	Resp eye/ear	Injury	Avoidable	Mental	Self-harm	Alcohol
Birth order 2	0.016***	0.005***	0.007***	0.003***	0.003***	0.002**	0.003***
	(0.003)	(0.001)	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)
Birth order 3	0.025***	0.007**	0.010**	0.004**	0.006***	0.002	0.005**
	(0.006)	(0.003)	(0.004)	(0.002)	(0.002)	(0.001)	(0.002)
Birth order 4	0.038***	0.013***	0.019***	0.008***	0.009**	0.005**	0.008***
	(0.009)	(0.004)	(0.006)	(0.003)	(0.004)	(0.002)	(0.003)
Birth order >4	0.035***	0.010	0.009	0.007	0.006	0.004	0.004
	(0.014)	(0.006)	(0.009)	(0.004)	(0.006)	(0.003)	(0.004)
Obs	518,861	518,861	518,861	518,861	518,861	518,861	518,861
R-square	0.530	0.517	0.513	0.521	0.502	0.490	0.490
Mean	0.197	0.034	0.069	0.016	0.022	0.007	0.013
N clusters	260,991	260,991	260,991	260,991	260,991	260,991	260,991

Notes: Results from linear probability models with family fixed effects. The omitted category is firstborn child. Standard errors are clustered by family. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include controls for mother's age at birth, and indicators for child's birth cohort and gender. For siblings with different fathers, indicators for father's cohort and educational attainment are included.

In this section, we have made a novel attempt to answer the long standing question on optimal stopping and reverse causality. We have estimated the effects on fertility, given the health status of previous children. The results are in line with the hypothesis that family resources are important, not only

as a direct explanation to birth order effects, but also indirectly by affecting fertility decisions. The care of a sick child is likely to be demanding, financially and emotionally, but also in terms of time. Mortality is considered the most severe health outcome. However, considering families' resource constraint, and preference for children, the early loss of a child will free resources and hence, the fertility response will be different. The endogenous fertility response is important, not only for our study but also for the interpretation of birth order effects found in other studies.

7. Heterogeneity

We have shown results suggesting that firstborns are at disadvantage at birth but as the child grows older, later-born children run a greater risk of being admitted to hospital. Our results suggest that this may be due to different access to parental resources. To further investigate the mechanisms, we study whether the effects differ depending on family resources in the form of parental educational attainment.[93] We also investigate whether the effects differ depending on the gender of the child.

Another reason why it is interesting to study heterogeneous effects with respect to family background is that, as discussed in Section 3, if families on which we estimate the birth order effect are different from the population at large, the estimated effects may not be externally valid. Since we use a fixed effects approach the effects are estimated on families which at least one child has been admitted to hospital, or in the estimations of mortality, at least one child died. The concern is that families with a sick or a dead child is different from other families. Table 9 displays characteristics of families which are included, and not included, in the analytical sample for the estimation on a particular outcome. The first row shows that couples' who have lost a child are somewhat more likely to have a lower education level, to be born in another country, and to have more children.[94] As expected, the probability of having an unhealthy child, or having lost a child, is larger if you have many children, as is evident from the last two columns. However, the difference between the groups is larger for infant mortality, which is also in line with the results that families that experience the death of a child are likely to have another child. The education level is lower among families that have a child who has been admitted to hospital; a pattern which is visual for all conditions. Foreign-born parents are underrepresented among children

[93] Another potential measure of access to parental resources is spacing; short spacing may imply less own time with the parents. Since we find that spacing is affected by the health and death of earlier-born children, we abstain from studying this since an analysis of the effect of spacing would suffer from endogeneity problems

[94] Families are defined as highly educated if the mother has more than 12 years of schooling. In the Swedish setting, this implies that she has continued to study after high school.

admitted to hospital for any cause, but are more likely to have a child admitted to hospital with conditions related to mental health, self-harm, and alcohol consumption. Overall, the differences in family background factors between the analytical sample and the full population are small. Nevertheless, we will now study whether there are any heterogeneous effects with respect to parental education. Regarding whether the effects vary across family size, the results presented in the Appendix, Table A2 and Table A6, show that the birth order effects are similar.

7.1 Educational Attainment

It is possible that the birth order effects could vary across families depending on parental educational attainment, as parents with a higher education are likely to have more resources, which they could potentially use to mitigate investment deficits in younger children. We test if family background is important in a simple model where we interact birth order with educational attainment. To save space, only the main results are reported in Table 10, which strongly indicate that there does not seem to be any heterogeneity in terms of the mother's educational attainment. If anything, the results in Column (2) show a small negative effect on perinatal conditions and congenital malformations, implying that a higher education among mothers might exacerbate the negative birth order effect.[95]

[95] Our results are in line with the findings in previous studies. Black et al. (2005) split the sample by mother's education (12 years used as the cut-off) finding small differences. If anything, they find slightly stronger effects among mothers with high education on children's education. Bjerkedal et al. (2007) find stronger negative birth-order effects on IQ between first- and second-born children in families with highly educated mothers, but no difference between second- and third-born children.

Table 9. Descriptive statistics in families with and without a sick or dead child, respectively

	High education	High education	Foreign born	Foreign born	Family size	Family size
Dead/sick child in the family	Yes	No	Yes	No	Yes	No
Infant mortality	0.405	0.464	0.152	0.148	3.818	2.792
	(0.491)	(0.499)	(0.359)	(0.355)	(1.305)	(1.016)
Hospitalized 0–6	0.545	0.572	0.158	0.164	2.914	2.695
	(0.498)	(0.495)	(0.365)	(0.370)	(1.132)	(0.918)
Hospitalized 7–12	0.485	0.524	0.140	0.158	3.049	2.724
	(0.500)	(0.499)	(0.347)	(0.365)	(1.168)	(0.935)
Hospitalized 13–18	0.443	0.487	0.135	0.149	3.031	2.673
	(0.497)	(0.500)	(0.341)	(0.356)	(1.153)	(0.893)
Hospitalized 19–24	0.377	0.447	0.132	0.143	3.007	2.672
	(0.485)	(0.497)	(0.339)	(0.350)	(1.153)	(0.898)
Perinatal 0–6	0.540	0.560	0.167	0.159	2.956	2.793
	(0.498)	(0.496)	(0.373)	(0.366)	(1.219)	(1.012)
Injury 0–6	0.552	0.557	0.152	0.162	3.088	2.784
	(0.497)	(0.497)	(0.359)	(0.368)	(1.246)	(1.017)
Injury 7–12	0.491	0.514	0.138	0.154	3.119	2.793
	(0.500)	(0.500)	(0.344)	(0.361)	(1.218)	(0.996)
Injury 13–18	0.433	0.477	0.133	0.145	3.116	2.752
	(0.496)	(0.499)	(0.340)	(0.352)	(1.221)	(0.961)
Mental 13–18	0.412	0.474	0.172	0.142	3.273	2.781
	(0.492)	(0.499)	(0.377)	(0.349)	(1.335)	(0.986)
Self-harm 13–18	0.384	0.472	0.187	0.143	3.371	2.795
	(0.486)	(0.499)	(0.390)	(0.350)	(1.358)	(1.002)
Alcohol 13–18	0.398	0.473	0.169	0.143	3.273	2.791
	(0.490)	(0.499)	(0.375)	(0.350)	(1.318)	(0.998)

Table10. Birth order effects by mother's education

	(1) Hosp	(2) Perinatal & cong. mal	(3) Hosp	(4) Hosp	(5) Mental	(6) Hosp
Age	0–6	0–6	7–12	13–18	13–18	19–24
Birth order 2	-0.014***	-0.040***	-0.001	0.017***	0.004***	0.017***
	(0.004)	(0.003)	(0.002)	(0.002)	(0.001)	(0.002)
Birth order 3	-0.012	-0.042***	-0.000	0.025***	0.006***	0.024***
	(0.008)	(0.005)	(0.004)	(0.004)	(0.001)	(0.004)
Birth order 4	-0.009	-0.043***	-0.002	0.034***	0.009***	0.029***
	(0.012)	(0.007)	(0.006)	(0.006)	(0.002)	(0.006)
Birth order >4	0.001	-0.039***	-0.003	0.039***	0.010***	0.026***
	(0.016)	(0.010)	(0.008)	(0.009)	(0.004)	(0.009)
High edu*	0.002	-0.002	0.004	0.000	-0.001	0.001
Birth order 2	(0.005)	(0.003)	(0.002)	(0.002)	(0.001)	(0.002)
High edu	-0.006	-0.008*	0.002	-0.001	-0.002	0.006
*Birth order 3	(0.007)	(0.004)	(0.004)	(0.003)	(0.001)	(0.004)
High edu	0.007	-0.010	0.009	0.004	-0.003	0.004
*Birth order 4	(0.012)	(0.007)	(0.006)	(0.007)	(0.003)	(0.008)
High edu*	0.008	-0.009	0.001	0.005	0.003	0.005
Birth order >4	(0.021)	(0.012)	(0.012)	(0.013)	(0.006)	(0.016)
Observations	644,589	644,589	1155264	1474603	1474603	1463458
R-square	0.618	0.608	0.534	0.525	0.500	0.505
Mean	0.368	0.087	0.164	0.188	0.020	0.199
N clusters	360,806	360,806	578,318	737,256	737,256	709,654

Notes: Results from linear probability models with family fixed effects. The omitted category is firstborn child. Standard errors are clustered by family. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include controls for mother's age at birth, and indicators for the child's birth cohort and gender. For siblings with different fathers, indicators for father's cohort and educational attainment are included.

7.2. Gender

Next we study whether the birth order effects differ between boys and girls. It is important to study heterogeneity across gender for several reasons. To start with, it is known from the previous literature (e.g. Mörk et al., 2014) that boys and girls have different probabilities of being hospitalized for certain conditions. For example, boys are more likely to be injured and girls have a higher risk of being hospitalized for mental conditions in adolescence. Birth order effects on educational attainment and earnings have also been shown to be larger for girls (Black et al., 2005). We control for gender in all our regressions, but that will not help us understand whether the effects

that we observe are driven by one gender. Once more, we study potential heterogeneity by setting up a simple model where we interact birth order with gender.

Table 11 shows the results for children aged 0–6 and 7–12. The result that stands out is that girls are healthier than boys, in terms of all diagnoses, at these young ages. Concerning the differential birth order effects across gender, the results are not conclusive. In the youngest age category, gender differences in health are small, and the interactions are only statistically significant for perinatal conditions and congenital malformations. However, these effects depend on the differences in means between boys and girls. Correcting for this, the difference in health between a later-born boy and his firstborn brother is as large as the difference between a later-born girl and her firstborn sister. The difference in hospitalization over birth order is, however, higher for girls 7–12 years old (ranging from 5 to 9 percent over birth order) than for boys. Concerning perinatal conditions and congenital malformations at age 7–12, second-born boys have a marginally lower risk of being hospitalized. This negative effect disappears for girls and, if anything, it increases marginally over birth order.

In Table 12, we look more closely at gender differences in the older age groups. At age 13–18, the birth order effects are marginally stronger for girls than for boys for hospitalization; the effect ranges from 10–26 percent for girls over birth order, to be compared with 8–17 percent for boys. The largest differences are found for hospitalizations related to mental conditions and alcohol-related hospitalizations. A third-born girl is 48 percent more likely to be hospitalized for mental conditions as compared to a firstborn girl. This gap is 18 percent between a firstborn boy and a third-born boy. For alcohol related conditions, third-born girls are 61 percent more likely to be admitted to hospital as compared to firstborn girls, whereas the difference between third-born and firstborn boys is 27 percent.

In the oldest age group, 19–24, the heterogeneous effects are once again small. Since females are less likely to end up in hospital for injuries, the birth order effect is somewhat larger for females. A second-born male is 9 percent more likely to be admitted to hospital for injuries as compared to a firstborn male, whereas the difference is 15 percent for females. For self-harm, the effect is only statistically significant for females: a fourth born female is 28 percent more likely to be admitted for self-harm compared to firstborn female. However, self-harm is rare among males in this age span.

Table 11. Birth order effects, by gender at age 0-12

	(1) Hosp	(2) Perinatal cong.mal	(3) Resp eye/ear	(4) Injury	(5) Avoidable	(6) Cancer
Panel A: Age 0–6						
Birth order 2	-0.019***	-0.047***	0.020***	0.011***	0.008***	-0.001
	(0.005)	(0.003)	(0.004)	(0.002)	(0.003)	(0.001)
Birth order 3	-0.018**	-0.052***	0.032***	0.019***	0.016***	-0.001
	(0.008)	(0.005)	(0.006)	(0.004)	(0.004)	(0.001)
Birth order 4	-0.011	-0.050***	0.041***	0.020***	0.024***	-0.002
	(0.012)	(0.007)	(0.009)	(0.006)	(0.007)	(0.002)
Birth order >4	-0.012	-0.053***	0.045***	0.022**	0.023**	-0.002
	(0.017)	(0.010)	(0.013)	(0.009)	(0.009)	(0.002)
Female	-0.086***	-0.037***	-0.055***	-0.014***	-0.023***	-0.000
	(0.005)	(0.003)	(0.003)	(0.002)	(0.002)	(0.001)
Birth order 2*Female	0.012*	0.012***	0.006	-0.002	0.004	0.000
	(0.006)	(0.004)	(0.005)	(0.003)	(0.003)	(0.001)
Birth order 3*Female	0.007	0.010**	0.004	-0.008**	-0.001	0.001
	(0.007)	(0.004)	(0.005)	(0.004)	(0.004)	(0.001)
Birth order 4*Female	0.008	0.006	0.006	-0.005	-0.002	0.001
	(0.011)	(0.006)	(0.008)	(0.006)	(0.006)	(0.001)
Birth order>4*Female	0.032**	0.020**	0.013	0.005	-0.000	0.001
	(0.014)	(0.008)	(0.010)	(0.007)	(0.008)	(0.002)
Observations	644,589	644,589	644,589	644,589	644,589	644,589
R-square	0.618	0.608	0.615	0.572	0.597	0.573
Mean female	0.329	0.072	0.124	0.050	0.061	0.004
Mean male	0.405	0.101	0.174	0.065	0.082	0.004
N clusters	360,806	360,806	360,806	360,806	360,806	360,806

Cont. next page

Cont. Table 11

	Hosp	Perinatal cong.mal	Resp eye/ear	Injury	Avoidable	Cancer
Panel B: Age 7–12						
Birth order 2	-0.003	-0.002**	-0.005***	0.005***	-0.002**	-0.000
	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)
Birth order 3	-0.004	-0.002	-0.009***	0.009***	-0.002*	-0.001
	(0.004)	(0.001)	(0.002)	(0.002)	(0.001)	(0.001)
Birth order 4	-0.004	-0.002	-0.010***	0.013***	-0.002	-0.000
	(0.006)	(0.002)	(0.003)	(0.004)	(0.002)	(0.001)
Birth order >4	-0.008	-0.002	-0.012***	0.011**	-0.001	0.001
	(0.009)	(0.003)	(0.004)	(0.005)	(0.003)	(0.001)
Female	-0.042***	-0.008***	-0.007***	-0.019***	-0.003***	-0.000
	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)
Birth order 2*Female	0.009***	0.002***	0.001	0.002	0.001	0.000
	(0.003)	(0.001)	(0.002)	(0.002)	(0.001)	(0.000)
Birth order 3*Female	0.010***	0.002**	0.002	0.002	0.001	0.001
	(0.004)	(0.001)	(0.002)	(0.002)	(0.001)	(0.001)
Birth order 4*Female	0.011*	0.003**	0.001	0.001	-0.001	0.000
	(0.006)	(0.002)	(0.003)	(0.004)	(0.002)	(0.001)
Birth order>4*Female	0.013	0.005**	0.002	0.001	-0.004	-0.000
	(0.008)	(0.002)	(0.004)	(0.005)	(0.003)	(0.001)
Observations	1,155,264	1,155,264	1,155,264	1,155,264	1,155,264	1,155,264
R-square	0.534	0.522	0.530	0.509	0.526	0.509
Mean female	0.145	0.007	0.034	0.040	0.014	0.003
Mean male	0.182	0.014	0.040	0.058	0.016	0.003
N clusters	578,318	578,318	578,318	578,318	578,318	578,318

Notes: Results from linear probability models with family fixed effects. The omitted category is firstborn child. Standard errors are clustered by family. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include controls for mother's age at birth, and indicators for child's birth cohort. For siblings with different fathers, indicators for father's cohort and educational attainment are included.

Table 12. Birth order effects, by gender at age 13–24

	(1) Hosp	(2) Resp & eye/ear	(3) Injury	(4) Avoidable	(5) Mental	(6) Self-harm	(7) Alcohol
Panel A: Age 13–18							
Birth order 2	0.015***	0.004***	0.007***	0.003***	0.002***	0.001***	0.003***
	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.001)
Birth order 3	0.019***	0.008***	0.009***	0.005***	0.003*	0.001	0.003***
	(0.004)	(0.002)	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)
Birth order 4	0.027***	0.009***	0.016***	0.005***	0.004*	0.002*	0.006***
	(0.006)	(0.003)	(0.004)	(0.002)	(0.002)	(0.001)	(0.002)
Birth order >4	0.031***	0.007*	0.010	0.007**	0.005	0.001	0.006**
	(0.009)	(0.004)	(0.006)	(0.003)	(0.004)	(0.002)	(0.003)
Female	0.001	0.008***	-0.023***	0.005***	0.005***	0.006***	-0.001*
	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.001)
Birth order 2*Female	0.005*	0.002	0.001	-0.001	0.002*	0.001	0.001
	(0.003)	(0.001)	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)
Birth order 3*Female	0.012***	0.001	0.003	-0.002**	0.005***	0.003***	0.004***
	(0.004)	(0.002)	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)
Birth order 4*Female	0.017***	0.004	0.002	0.000	0.006***	0.005***	0.004*
	(0.006)	(0.003)	(0.004)	(0.002)	(0.002)	(0.001)	(0.002)
Birth order>4*Female	0.019**	0.002	0.004	-0.003	0.011**	0.008***	0.005
	(0.009)	(0.004)	(0.006)	(0.003)	(0.004)	(0.003)	(0.003)
Observations	1,474,603	1,474,603	1,474,603	1,474,603	1,474,603	1,474,603	1,474,603
R-square	0.525	0.512	0.508	0.519	0.500	0.485	0.487
Mean female	0.192	0.037	0.054	0.017	0.024	0.010	0.012
Mean male	.0184	0.027	0.076	0.013	0.017	0.003	0.011
N clusters	737,256	737,256	737,256	737,256	737,256	737,256	737,256

Cont. next page

Cont. Table 12.

	Hosp	Resp & eye/ear	Injury	Avoidable	Mental	Self-harm	Alcohol
Panel B: Age 19-24							
Birth order 2	0.018***	0.003***	0.007***	0.002**	0.000	0.001	0.001*
	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.001)
Birth order 3	0.027***	0.004**	0.009***	0.004***	-0.002	-0.000	0.002
	(0.004)	(0.002)	(0.002)	(0.001)	(0.002)	(0.001)	(0.001)
Birth order 4	0.027***	0.007***	0.010**	0.005***	0.001	0.000	0.002
	(0.006)	(0.003)	(0.004)	(0.002)	(0.003)	(0.002)	(0.002)
Birth order >4	0.023**	0.007*	0.013**	0.004	-0.002	-0.003	0.002
	(0.010)	(0.004)	(0.006)	(0.003)	(0.004)	(0.002)	(0.003)
Female	0.016***	0.001	-0.033***	0.004***	0.001*	0.004***	-0.003***
	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.000)
Birth order 2*Female	-0.002	0.001	-0.000	-0.000	0.002	0.001*	0.001
	(0.003)	(0.001)	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)
Birth order 3*Female	-0.003	0.003	0.001	-0.001	0.004***	0.002*	0.001
	(0.004)	(0.002)	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)
Birth order 4*Female	0.007	-0.003	0.002	-0.002	0.004	0.003*	0.001
	(0.007)	(0.003)	(0.004)	(0.002)	(0.003)	(0.002)	(0.002)
Birth order>4*Female	0.010	-0.001	-0.003	0.004	0.005	0.005*	0.002
	(0.010)	(0.004)	(0.006)	(0.003)	(0.005)	(0.003)	(0.003)
Observations	1,463,458	1,463,458	1,463,458	1,463,458	1,463,458	1,463,458	1,463,458
R-square	0.505	0.490	0.494	0.483	0.513	0.491	0.495
Mean female	0.207	0.030	0.045	0.016	0.026	0.011	0.007
Mean male	0.192	0.028	0.077	0.013	0.022	0.006	0.009
N clusters	709,654	709,654	709,654	709,654	709,654	709,654	709,654

Notes: Results from linear probability models with family fixed effects. The omitted category is firstborn child. Standard errors are clustered by family. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include controls for mother's age at birth, and indicators for child's birth cohort. For siblings with different fathers, indicators for father's cohort and educational attainment are included.

8. Conclusions

In this paper, we examine the relationship between birth order and child health. We find that firstborns are more likely to be hospitalized due to congenital malformations and perinatal conditions in early childhood. However, the disadvantage of firstborn children at birth is reversed in older age when younger siblings are more likely to be hospitalized for injuries and avoidable conditions. Our results indicate that the dilution hypothesis, which emphasizes the importance of constrained parental resources, is crucial for our understanding of birth order effects. In adolescence, we find positive birth order effects on hospitalizations, including hospitalizations related to poor mental health and alcohol-related conditions. The causes for hospitalization suggest that later-born siblings are involved in more risky behavior, have a less healthy life style and worse mental health in older age.

Birth order effects may arise as a result of endogenous fertility decisions. We show that a large part of the negative birth order effects on infant mortality are non-causal, and instead related to parents' fertility response to the loss of a child. Families, of all sizes, who lose a child, are more likely to have another child, giving rise to a non-causal negative effect of birth order on infant mortality. Taking some of the endogenous responses into account by removing the last born child, we show that there is still a negative effect of birth order on infant mortality.

We also find that hospitalization at an early age affects subsequent fertility decisions, but in the opposite direction. Parents with an unhealthy child are less likely to have another child. This effect is, however, much smaller, especially for higher parities, and is less likely to explain the birth order effects on health. The endogenous fertility responses are also in line with the dilution hypothesis; caring for a sick child is likely to require considerable resources both in terms of time, but also financially as well as emotionally. In contrast, the early loss of a child will free resources and given families' preference for children, the fertility response will be the opposite. Hence, we conclude that endogenous fertility responses are important to take into consideration when studying birth order effects and possibly other questions related to the family environment.

That family environment is important for health outcomes is informative for policies which aim at improving child outcomes. The clear birth order effects on conditions such as injuries and avoidable conditions in early ages suggest that later-born children get less parental attention.

References

Argys, L., D. I. Rees, S.L. Averett, and B. Witoonchart (2006) "Birth order and risky adolescence behavior", *Economic Inquiry* 44(2), 215–233.

Avrett, S., Arggys, L. and D. Rees (2011) "Older siblings and adolescent risky behavior: does parenting play a role?", *Journal of Population Economic* 24, 957-978.

Barclay, K.J. (2015a) "Birth order and educational attainment: evidence from fully adopted sibling groups", *Intelligence* 48, 109–122.

Barclay, K. J., (2015b) "A within-family analysis of birth order and intelligence using population conscription data on Swedish men." *Intelligence*, 49, 134–143.

Barclay K. and M. Kolk (2015) "Birth Order and Mortality: A Population-Based Cohort Study", *Demography* 52, 613–639.

Barclay K. and M. Myrskylä (2014) "Birth order and physical fitness in early adulthood: Evidence from Swedish military conscription data", *Social Science & Medicine* 123, 141–148.

Becker G. S. and N. Tomes (1976) "Child Endowments and the Quantity and Quality of Children", *Journal of Political Economy* 84(4), Part 2: Essays in Labor Economics in Honor of H. Gregg Lewis, 143–162.

Behrman, J. R. and P. Taubman (1986) "Birth Order, Schooling, and Earning" *Journal of Labor Economics* 4(3), part 2, 121–145.

van den Berg, G. J., P. Lundborg and J. Vikström (2016) "Economics of grief", *Economic Journal* doi:10.1111/ecoj.12399.

Bijur, P. E., J. Golding, and M. Kurzon (1988) "Childhood accidents, family size and birth order", *Social Science & Medicine* 26(8), 839–843.

Bjerkedal, T., P. Kristensen, G. A. Skjeret, and J. I. Brevik (2007) "Intelligence test scores and birth order among young Norwegian men (conscripts) analyzed within and between families" *Intelligence* 35, 503–514.

Björklund, A., D. K. Ginther, M. Sundström (2007) "Family structure and child outcomes in the USA and Sweden" *Journal of Population Economics* 20, 183–201.

Blake, Judith (1989) "Family Size and Achievement", Berkeley and Los Angeles, CA: University of California Press.

Black, S. E., Devereux, P. J. and Salvanes, K. G. (2005) "The More the Merrier? The Effects of Family Size and Birth Order on Children's Education", *Quarterly Journal of Economics* 120(2), 669–700.

Black, S. E., P. J. Devereux, and K. G. Salvanes (2011) "Older and Wiser? Birth Order and IQ of Young Men". *CESifo Economic Studies* 57(1), 103–120.

Black, S. E., P. J. Devereux, and K. G. Salvanes (2015) "Healthy(?), Wealthy and Wise: Birth Order and Adult Health". NBER Working Paper Series 21337.

Black, S.E., Breining, S., Figlio, D., Guryan J., Karbownik, K., Skyt Nielsen H., Roth J. and M. Simonsen (2016) "Sibling Spillovers" mimio

Black, S.E.,Grönqvist, E and B. Öckert."Born to Lead? The effect of Birth Order on Non-Cognitive Abilities." *Review of Economics and Statistics*, forthcoming.

Billings, J., Zeital, J., Lukomnik, J., Carey, TS., Blank, AE. and L. Newman (1993) "Impact of socioeconomic status on hospital use in New York City" *Health Affairs* 12(1), 162–173.

Brenoe, A. A. and R. Molitor (2015) "Birth order and Health of Newborns: What can we learn from Danish Registry data", CINCH working paper 13.

Breining, S., Doyle, J., Figlio, D., Karbownik, K. and J. Roth (2017) "Birth Order and Delinquency: Evidence from Denmark and Florida. NBER WP 23038.

Case, A., Fertig, A. and Paxson, C. (2005) "The Lasting Impact of Childhood Health and Circumstance", *Journal of Health Economics* 24, 365–389.

Currie, J. (2009) "Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development", *Journal of Economic Literature* 47(1), 87–122.
Currie, J., Stabile, M., Manivong, P. and Roos L. L. (2010) "Child Health and Young Adult Outcomes", *Journal of Human Resources* 45(3), 517–548.
Hotz, V. J. and J. Pantano (2015) "Strategic Parenting, Birth Order, and School Performance" *Journal of Population Economics* 28(4): 911–936.
Ibánze, B, E. Moreno and A. Barbosa (2013) "Parity, but not inbreeding, affects juvenile mortality in two captive endangered gazelles", *Animal Conservation* 16, 108–117.
Khong, T. Y., E. D. Adema, and J. J. H. M. Erwich (2003) "On an Anatomical Basis for The Increase in Birth Weight in Second and Subsequent Born Children", *Placenta* 24(4) 348–353.
Lehmann, J.-Y. K., A. Nuevo-Chiquero, and M. Vidal-Fernandez (2016) "The Early Origins of Birth Order Differences in Children's Outcomes and Parental Behavior", *Journal of Human Resources* doi:10.3368/jhr.53.1.0816-8177
Lindahl, M., Lundberg, E., Palme, M. and Simeonova, E. (2015) "Pre- versus Post-Birth Parental Influences in the Formation of Health: Lessons from a Large Sample of Adoptees" *Unpublished manuscript.*
Modin, B. (2002) "Birth Order and Mortality: a Life-long Follow-up of 14,200 Boys and Girls Born in Early 20th Century Sweden", *Social Science & Medicine* 54, 1051–1064.
Monfardini, C. and S. G. See (2012) "Birth Order and Child Outcomes: Does Maternal Quality Time Matter?", *IZA Working Paper 6825*
Mörk, E., Sjögren, A., and Svaleryd, H. (2014) "Heller rik och frisk – om familjebakgrund och barns hälsa", SNS
Nixon J. and J. Pearn (1978) "An investigation of socio-demographic factors surrounding childhood drowning accidents", *Social Science & Medicine*, 12, 387–390.
Page, A., Ambrose, S., Glover, J. and D. Hetzel (2007) "Atlas of Avoidable Hospitalisations in Australia: ambulatory care-sensitive conditions" Public Health Information Development Unit, The University of Adelaide .
Pavan (2015) "On the production of skills and the birth order effect" *Journal of Human Resources* November doi:10.3368/jhr.51.3.0913-5920R.
Price, J. (2008) "Parent-Child Quality Time – Does Birth Order Matter?" *Journal of Human Resources* 43(1), 240–265.
Smith, J. (1999) "Healthy Bodies and Thick Wallets: The Dual Relationship between Health and Economic Status" *Journal of Economic Perspectives* 13, 145–166.
Socialstyrelsen (2009) "Graviditeter, förlossningar och nyfödda barn"
Stanton, M., Lonsdorf, E., Pusey, A., Goodall, J. and C. Murray (2014) **"**Maternal Behavior by Birth Order in Wild Chimpanzees (*Pan troglodytes*) Increased Investment by First-Time Mothers" Current Anthropology 55, 483–489.
Swamy G.K., Edwards, S., Gelfand, A., James, S.A. and M. L. Miranda (2012) "Maternal age, birth order, and race: differential effects on Birthweight" *Journal of Epidemiology and Community Health* 66, 136–142.
Zajonc, R. B. (1976) "Family configuration and intelligence: Variations in scholastic aptitude scores parallel trends in family size and the spacing of children", *Science* 192(4236), 227–236.

Appendix

Table A1. Diagnoses codes (ICD 10)

Variable	Definition
Hospitalization	=1 if admitted to hospital that year with any medical condition
Hospitalization for diagnoses code indicating alcohol abuse	=1 if admitted to hospital with diagnosis codes T51, X45, X65, Y15, F10, K70, K85, K86.0–1 E24.4, G31.2, G62.1, G72.1, I42.6, K29.2, 035.4,
Hospitalization for diagnoses code avoidable conditions	=1 if admitted to hospital with diagnosis codes D50, E10–E11, E13–E14, E86 G40–G41, H66–H67, H66–H67, I11, I20, I29, I50, J02–J03, J06,J43–J47, K24, K26–K28, K52, N10–N12, N70, N73–N74, O15, R56
Hospitalization for injury or poisoning	=1 if admitted to hospital with diagnosis codes S00–T98
Hospitalization for diseases of the respiratory system and conditions related to ears and eyes	=1 if admitted to hospital with diagnosis codes J00–J99 or H00–H95
Hospitalization for diagnoses code indicating self-harm behavior	=1 if admitted to hospital with diagnosis codes Intentional self-harm X60–X84, event of undetermined intent Y10–Y34
Hospitalization for diagnoses code indicating mental health problems	=1 if admitted to hospital with diagnosis codes F00–F99
Hospitalizations for cancer/tumors	=1 if admitted to hospital with diagnosis codes C00–D48
Hospitalizations for perinatal conditions and congenital malformations	=1 if admitted to hospital with diagnosis codes P00–P96 and Q00–Q99

Table A2. Hospitalization by family size, with and without family fixed effects

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	2-Child families		3-Child families		4-Child families		>4Child families		All	
Panel A. Hospitalization age 0–6										
Birth order 2	-0.028***	-0.006	-0.017***	-0.010*	-0.012	-0.016	-0.019	-0.026	-0.023***	-0.013***
	(0.002)	(0.006)	(0.003)	(0.006)	(0.008)	(0.012)	(0.017)	(0.022)	(0.002)	(0.004)
Birth order 3			-0.033***	-0.012	-0.015*	-0.024	-0.022	-0.039*	-0.035***	-0.015**
			(0.004)	(0.010)	(0.008)	(0.016)	(0.017)	(0.023)	(0.003)	(0.007)
Birth order 4					-0.010	-0.015	-0.033*	-0.049*	-0.039***	-0.007
					(0.010)	(0.021)	(0.018)	(0.026)	(0.004)	(0.010)
Birth order>4							-0.026	-0.042	-0.045***	0.003
							(0.019)	(0.030)	(0.007)	(0.015)
Fam FE	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Observations	303,083	303,083	222,729	222,729	78,662	78,662	40,115	40,115	644,589	644,589
R-square	0.014	0.664	0.017	0.602	0.021	0.563	0.024	0.481	0.015	0.618
Mean	0.367	0.367	0.363	0.363	0.378	0.378	0.384	0.384	0.368	0.368
N clusters	185,978	185,978	120,727	120,727	38,482	38,482	15,619	15,619	360,806	360,806
Panel B. Hospitalization age 7–12										
Birth order 2	-0.006***	0.004	0.001	0.002	0.004	0.005	-0.000	-0.002	-0.003***	0.001
	(0.001)	(0.003)	(0.002)	(0.003)	(0.004)	(0.005)	(0.007)	(0.008)	(0.001)	(0.002)
Birth order 3			-0.009***	-0.005	0.005	0.010	-0.001	-0.004	-0.009***	0.001
			(0.002)	(0.005)	(0.004)	(0.007)	(0.008)	(0.010)	(0.002)	(0.003)
Birth order 4					-0.003	0.008	-0.005	-0.008	-0.011***	0.001
					(0.005)	(0.010)	(0.008)	(0.012)	(0.003)	(0.005)
Birth order>4							-0.013	-0.015	-0.015***	-0.002
							(0.009)	(0.015)	(0.004)	(0.008)
Fam FE	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Observations	527,716	527,716	411,693	411,693	146,076	146,076	69,779	69,779	1,155,264	1,155,264
R-square	0.009	0.621	0.009	0.499	0.011	0.429	0.014	0.355	0.009	0.534
Mean	0.159	0.159	0.165	0.165	0.173	0.173	0.177	0.177	0.164	0.164
N clusters	310,833	310,833	190,466	190,466	56,052	56,052	20,967	20,967	578,318	578,318

Cont. next page

Cont. Table A2

Panel C. Hospitalization age 13–18										
Birth order 2	0.010***	0.021***	0.010***	0.015***	0.014***	0.013***	0.008	0.007	0.011***	0.017***
	(0.001)	(0.003)	(0.002)	(0.003)	(0.003)	(0.004)	(0.006)	(0.007)	(0.001)	(0.002)
Birth order 3			0.009***	0.025***	0.021***	0.024***	0.015**	0.014	0.012***	0.025***
			(0.002)	(0.005)	(0.004)	(0.007)	(0.007)	(0.009)	(0.002)	(0.003)
Birth order 4					0.025***	0.036***	0.022***	0.026**	0.017***	0.035***
					(0.005)	(0.010)	(0.008)	(0.012)	(0.003)	(0.005)
Birth order>4							0.027***	0.030**	0.021***	0.040***
							(0.009)	(0.015)	(0.004)	(0.008)
Fam FE	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Observations	690,063	690,063	519,880	519,880	180,611	180,611	84,049	84,049	1,474,603	1,474,603
R-square	0.004	0.623	0.005	0.479	0.007	0.401	0.010	0.346	0.005	0.525
Mean	0.180	0.180	0.189	0.189	0.204	0.204	0.219	0.219	0.188	0.188
N clusters	413,331	413,331	233,082	233,082	66,005	66,005	24,838	24,838	737,256	737,256
Panel D. Hospitalization age 19–24										
Birth order 2	0.010***	0.020***	0.018***	0.015***	0.021***	0.016***	0.029***	0.017**	0.019***	0.017***
	(0.002)	(0.003)	(0.002)	(0.003)	(0.003)	(0.005)	(0.005)	(0.007)	(0.001)	(0.002)
Birth order 3			0.026***	0.025***	0.033***	0.023***	0.041***	0.015	0.028***	0.026***
			(0.003)	(0.005)	(0.004)	(0.007)	(0.007)	(0.010)	(0.002)	(0.003)
Birth order 4					0.046***	0.040***	0.059***	0.024*	0.037***	0.031***
					(0.006)	(0.011)	(0.008)	(0.014)	(0.003)	(0.006)
Birth order>4							0.074***	0.029*	0.041***	0.028***
							(0.009)	(0.017)	(0.004)	(0.009)
Fam FE	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Observations	691,001	691,001	511,186	511,186	177,433	177,433	83,838	83,838	1,463,458	1,463,458
R-square	0.007	0.583	0.008	0.469	0.011	0.411	0.015	0.355	0.009	0.505
Mean	0.188	0.188	0.201	0.201	0.219	0.219	0.242	0.242	0.199	0.199
N clusters	389,624	389,624	226,426	226,426	67,110	67,110	26,494	26,494	709,654	709,654

Notes: Results from linear probability models. The omitted category is firstborn child. Standard errors are clustered by family. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression. In regressions with family fixed effects we include controls for mother's age at birth, and indicators for the child's birth cohort and gender. For siblings with different fathers, indicators for father's cohort and educational attainment are included. In regressions without family FE, we add father's characteristics and controls for mother's age at first birth, and indicators for mother's educational attainment and cohort. In (7) we add indicators for family size.

Table A3. Birth order effects, hospitalization and diagnoses, ages 0–1 and 0–3

	(1) Hospitali-zation	(2) Perinatal & con-genital mal.	(3) Respiratory & eye/ear	(4) Injury	(5) Avoidable	(6) Cancer
Panel A: Age 0–1						
Birth order 2	-0.004	-0.040***	0.036***	0.003***	0.014***	-0.000
	(0.003)	(0.002)	(0.002)	(0.001)	(0.001)	(0.000)
Birth order 3	0.008	-0.046***	0.054***	0.003	0.022***	-0.000
	(0.006)	(0.004)	(0.004)	(0.002)	(0.003)	(0.001)
Birth order 4	0.021**	-0.046***	0.070***	0.002	0.030***	-0.001
	(0.009)	(0.006)	(0.006)	(0.003)	(0.004)	(0.001)
Birth order >4	0.041***	-0.042***	0.079***	0.004	0.035***	-0.000
	(0.013)	(0.009)	(0.009)	(0.004)	(0.006)	(0.001)
Obs.	645,554	645,554	645,554	645,554	645,554	645,554
R-sq.	0.611	0.609	0.596	0.561	0.585	0.568
Mean	0.228	0.079	0.078	0.014	0.038	0.002
N clusters	360,944	360,944	360,944	360,944	360,944	360,944
Panel B: Age 0–3						
Birth order 2	-0.009***	-0.041***	0.032***	0.005***	0.011***	-0.001
	(0.003)	(0.002)	(0.002)	(0.001)	(0.002)	(0.000)
Birth order 3	-0.004	-0.046***	0.047***	0.009***	0.018***	-0.001
	(0.007)	(0.004)	(0.005)	(0.003)	(0.004)	(0.001)
Birth order 4	0.003	-0.046***	0.060***	0.008*	0.025***	-0.002*
	(0.010)	(0.006)	(0.007)	(0.004)	(0.005)	(0.001)
Birth order >4	0.014	-0.041***	0.066***	0.013**	0.027***	-0.002
	(0.014)	(0.009)	(0.010)	(0.006)	(0.008)	(0.002)
Observations	644,893	644,893	644,893	644,893	644,893	644,893
R-square	0.616	0.608	0.609	0.567	0.594	0.560
Mean	0.307	0.083	0.120	0.034	0.061	0.003
N clusters	360,860	360,860	360,860	360,860	360,860	360,860

Notes: Results from linear probability models with family fixed effects. The omitted category is firstborn child. Standard errors are clustered by family. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include controls for mother's age at birth, and indicators for child's birth cohort and gender. For siblings with different fathers, indicators for father's cohort and educational attainment are included.

Table A4. Birth order effects on longer hospital stays

	More than 1 day				More than 7 days			
	(1) Age 0–6	(2) Age 7–12	(3) Age 13–18	(4) Age 19–24	(5) Age 0–6	(6) Age 7–12	(7) Age 13–18	(8) Age 19–24
Birth order 2	-0.010***	0.000	0.010***	0.013***	-0.007***	0.001	0.002***	0.005***
	(0.003)	(0.001)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.001)
Birth order 3	-0.004	-0.000	0.014***	0.020***	-0.002	0.002	0.004***	0.009***
	(0.006)	(0.003)	(0.003)	(0.003)	(0.004)	(0.001)	(0.002)	(0.002)
Birth order 4	0.002	-0.002	0.019***	0.023***	0.004	0.000	0.003	0.010***
	(0.009)	(0.004)	(0.004)	(0.005)	(0.006)	(0.002)	(0.002)	(0.003)
Birth order>4	0.014	-0.008	0.017***	0.022***	0.015*	-0.001	0.004	0.005
	(0.013)	(0.006)	(0.006)	(0.007)	(0.009)	(0.003)	(0.004)	(0.004)
Observations	644,589	1,155,264	1,474,603	1,463,458	644,589	1,155,264	1,474,603	1,463,458
R-square	0.617	0.535	0.527	0.499	0.608	0.530	0.516	0.492
Mean	0.242	0.091	0.110	0.123	0.082	0.022	0.032	0.038
N clusters	360806	578318	737256	709654	360806	578318	737256	709654

Notes: Results from linear probability models with family fixed effects. Omitted category is firstborn child. Standard errors are clustered by family. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include controls for mother's age at birth, and indicators for child's birth cohort and gender. For siblings with different fathers, indicators for father's cohort and educational attainment are included.

Table A5 Birth order effects on hospitalizations not related to alcohol

	Age 13–18			Age 19–24		
	(1)	(2)	(3)	(4)	(5)	(6)
	Mental - alc	Self harm - alc	Injury - alc	Mental - alc	Self harm - alc	Injury - alc
Birth order 2	0.001	0.001***	0.006***	-0.000	0.001	0.007***
	(0.000)	(0.000)	(0.001)	(0.001)	(0.000)	(0.001)
Birth order 3	0.001	0.002***	0.008***	-0.001	0.000	0.008***
	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)
Birth order 4	0.001	0.003***	0.013***	0.001	0.001	0.010***
	(0.002)	(0.001)	(0.003)	(0.002)	(0.001)	(0.003)
Birth order >4	0.003	0.003*	0.009*	-0.001	-0.002	0.009*
	(0.002)	(0.002)	(0.005)	(0.003)	(0.002)	(0.005)
Observations	1,474,603	1,474,603	1,474,603	1,463,458	1,463,458	1,463,458
R-square	0.501	0.488	0.507	0.509	0.491	0.492
Mean	0.012	0.005	0.061	0.017	0.006	0.058
N clusters	737,256	737,256	737,256	709,654	709,654	709,654

Notes: Results from linear probability models with family fixed effects. The omitted category is firstborn child. Hospitalizations related to mental conditions, self-harm and injuries, where the same individual has not been hospitalized for alcohol related conditions in the same age category, are considered. Standard errors are clustered by family. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include controls for mother's age at birth, and indicators for child's birth cohort and gender. For siblings with different fathers, indicators for father's cohort and educational attainment are included.

Table A6 Birth order effects on infant mortality by family size, with and without family fixed effects

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	2-Child families		3-Child families		4-Child families		>4-Child families		All	
Birth order 2	-0.001***	-0.004***	-0.002***	-0.014***	-0.001	-0.015***	0.001	-0.007***	-0.001***	-0.011***
	(0.000)	(0.000)	(0.000)	(0.001)	(0.001)	(0.001)	(0.002)	(0.002)	(0.000)	(0.000)
Birth order 3			-0.013***	-0.044***	-0.010***	-0.043***	0.000	-0.020***	-0.008***	-0.033***
			(0.001)	(0.001)	(0.001)	(0.002)	(0.002)	(0.003)	(0.000)	(0.001)
Birth order 4					-0.023***	-0.081***	-0.002	-0.035***	-0.015***	-0.052***
					(0.002)	(0.003)	(0.002)	(0.004)	(0.001)	(0.001)
Birth order >4							-0.011***	-0.058***	-0.019***	-0.068***
							(0.003)	(0.005)	(0.001)	(0.002)
Fam FE	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Observations	753,493	753,493	564,567	564,567	197,231	197,231	93,264	93,264	1,608,555	1,608,555
R-square	0.002	0.551	0.005	0.406	0.007	0.344	0.007	0.287	0.007	0.392
Mean	0.002	0.002	0.008	0.008	0.015	0.015	0.015	0.015	0.007	0.007
N clusters	409111	409111	234823	234823	68753	68753	26831	26831	739518	739518

Notes: Results from linear probability models. Omitted category is firstborn child. Standard errors are clustered by family. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression. In regressions with family fixed effects we include controls for mother's age at birth, and indicators for child's birth cohort and gender. For siblings with different fathers, indicators for father's cohort and educational attainment are included. In regressions without family FE, we add father's characteristics and controls for mother's age at first birth, and indicators for mother's educational attainment and cohort.

Table A7 Birth order effects on health ages 7–12 and 19–24, restricted sample

	(1) Hospitalization	(2) Perinatal cong. mal	(3) Resp eye/ear	(4) Injury	(5) Avoidable
Panel A: Hospitalization different causes age 7-12					
Birth order 2	-0.002*	-0.004**	0.009***	-0.002	0.000
	(0.001)	(0.002)	(0.002)	(0.001)	(0.001)
Birth order 3	-0.003	-0.006*	0.012***	-0.002	-0.001
	(0.002)	(0.004)	(0.004)	(0.002)	(0.001)
Birth order 4	-0.004	-0.009*	0.021***	-0.003	-0.001
	(0.003)	(0.005)	(0.006)	(0.003)	(0.002)
Birth order >4	-0.007*	-0.014*	0.018**	-0.008	-0.001
	(0.004)	(0.008)	(0.009)	(0.005)	(0.002)
Observations	375,947	375,947	375,947	375,947	375,947
R-square	0.535	0.550	0.530	0.544	0.519
Mean	0.011	0.043	0.051	0.017	0.003
N clusters	194,684	194,684	194,684	194,684	194,684

Cont. next page

Cont. Table A7

Panel B: Hospitalization different causes age 19–24

	Hospitalization	Resp eye/ear	Injury	Avoidable	Mental	Self-harm	Alcohol
Birth order 2	0.018***	0.003**	0.009***	0.001	0.001	0.001	0.002**
	(0.003)	(0.001)	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)
Birth order 3	0.022***	0.005*	0.013***	0.003*	0.000	0.000	0.002
	(0.006)	(0.003)	(0.004)	(0.002)	(0.003)	(0.002)	(0.002)
Birth order 4	0.025**	0.007	0.016**	0.004	0.006	0.002	0.003
	(0.010)	(0.004)	(0.006)	(0.003)	(0.005)	(0.003)	(0.003)
Birth order >4	0.037**	0.004	0.025**	0.005	0.002	0.001	0.004
	(0.015)	(0.006)	(0.010)	(0.005)	(0.007)	(0.004)	(0.004)
Observations	536,064	536,064	536,064	536,064	536,064	536,064	536,064
R-square	0.521	0.504	0.509	0.497	0.522	0.501	0.502
Mean	0.209	0.029	0.067	0.015	0.028	0.010	0.010
N clusters	267,896	267,896	267,896	267,896	267,896	267,896	267,896

Notes: Results from linear probability models with family fixed effects. The omitted category is firstborn child. Standard errors are clustered by family. *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression and all regressions include controls for mother's age at birth, and indicators for child's birth cohort and gender. For siblings with different fathers, indicators for father's cohort and educational attainment are included. Table A 1 Diagnoses and ICD10 codes

IV. Consequences of Health at Birth

With Aline Bütikofer, Gabriella Conti, Mårten Palme, and Kjell Salvanes

1. Introduction

The importance of newborn health for a variety of outcomes throughout the life cycle has been documented in a vast, interdisciplinary literature, to which economics has significantly contributed in the recent years (see e.g. Almond et al., 2017, for the most recent review). The main measure of neonatal health used in this literature is birth weight, which has been shown to be associated in a meaningful way with a variety of outcomes ranging from health to education and wages (see e.g. Almond et al., 2005; Black et al., 2007; Figlio et al., 2014).

Birth weight is relatively easy to measure, hence widely available in several data sources, and contains little measurement error, especially when obtained from vital records. However, birth weight might mainly capture the uterine environment in the last weeks of gestation, at the time when the fetus gains most weight. Additionally, the fact that the newborn has achieved a certain weight provides no information about the ranges of environmental factors experienced during pregnancy, since the same weight at birth can be obtained by following different trajectories in utero. For these reasons, one active area of research in the fetal origins field focuses on searching for more sensitive and predictive measures of health at birth (Torche and Conley, 2016).

In this paper, we contribute to a better understanding of the consequences of early life health by contrasting the effects of birth weight with those of two other measures of neonatal health: the length and the head circumference of the newborn. Birth length and head circumference are long-term cumulative indicators, reflecting most of the fetal period since the process of formation of bones and neural synapses starts early in gestation. While the use of these other two birth measures has been limited in economics, a literature in medicine and epidemiology has documented how they are differentially associated with prenatal investments, such as smoking, alcohol use, and nutritional supplementation (see e.g. Lindley et al., 2000; Ramakrishnan et al., 2010; Shankaran et al., 2004).

We use high-quality administrative data for Sweden on a sample of births between 1973 and 1979 to investigate the short, medium and long-term consequences of neonatal health. We provide within-twin pair evidence to control for unobserved differences between children. Our work provides key advances to the existing literature by investigating the value of using additional birth measures. On the one hand, by exploiting a decomposition technique recently proposed by Gelbach (2016), we are able to shed light on the mechanisms through which birth weight impacts later outcomes. On the other hand, by using information on birth head circumference to distinguish different types of growth-restricted newborns, we are able to show the relative importance for health and cognitive outcomes of insults differentially

affecting the brain. Overall, our contribution emphasizes the importance of not focusing exclusively on birth weight when studying neonatal health.

The remainder of the paper is structured as follows. Section 2 provides an overview of the recent literature. Section 3 outlines the empirical strategy. We discuss the data and provide descriptive statistics in Section 4. We discuss our results in Section 5. Section 6 provides a brief conclusion.

2. Literature and Mechanisms

The literature on the "fetal origin hypothesis" goes back to Barker et al. (1989) and has grown rapidly in recent years (see e.g. Almond et al., 2017, for an overview). Barker showed that birth weight is correlated with health in adulthood, and argued that adverse fetal conditions during the prenatal period have persistent consequences. Currie and Hyson (1999) took the fetal origins hypothesis to economics, showing that low birth weight children not only have worse health, but also have lower test scores and are less likely to be employed as adults.

The earliest literature relied on cross-sectional variation, showing an association between, primarily, birth weight and medium- and long-term outcomes such as health and economic outcomes. Concerned with the causal interpretation of these results, the literature developed and employed within-sibling and -twin variation to control for unobserved differences between children in family background, and even genetic set-up, that can bias the estimates. These studies find meaningful causal effects of birth weight on later outcomes such as health, IQ, earnings and education.[96] Another strand of the literature has used historical events that affect the uterine environment to overcome the problem of potential confounders.[97]

[96] For example, Behrman and Rosenzweig (2004) study the effect of birth weight on adult health and earnings using U.S. data. Black et al. (2007) study the effect of birth weight on both short-term health outcomes, and longer-run outcomes such as height and IQ age 18, earnings and education, using Norwegian data. Oreopoulos et al. (2008) study the effect of birth weight, Apgar scores and gestational length on health, education and labor force attachment using Canadian data. Royer (2009) study the effect of birth weight on education, later pregnancy complications and next generation birth weight using U.S. data. Rosenzweig and Zhang (2013) study educational outcomes, wages and health using Chinese data. Figlio et al. (2014) use data on children born in Florida, USA, and study the effect of birth weight on children's test scores.

[97] For example, Lumey and Stein (1997) find that the Dutch famine in 1944 affected next generation children's birth weight; Almond (2006) find that infected mothers in the 1918 Influenza epidemic had children that were more likely to be disabled and had lower educational attainments as well as lower wages; Almond et al. (2009) find that radioactive fallout from Chernobyl had negative effects on children's cognitive ability in Sweden; and Almond et al. (2007) find that fetal exposure to acute maternal malnutrition in the Chinese famine 1959-1961, had negative effects on literacy, labor market status, wealth, and marriage market outcomes.

In spite of extensive research, the mechanisms behind the fetal origin hypothesis are still much of a black box (Almond and Currie, 2011). One hypothesis is that the prenatal period is particularly sensitive because it is important for determining which parts of the genome are expressed (Petronis, 2010). We will add to the previous literature by studying the consequences of early life health by contrasting the effects of birth weight with those of two other measures of neonatal health: the length and the head circumference of the newborn.

It is well known that the fetus puts on most of the weight in the third trimester, while the head circumference and length of the fetus develops throughout the fetal period. Hence, the focus on birth weight in the previous literature might mainly capture variation in the in-utero environment late in pregnancy and possible forego the importance of earlier developmental periods. In the first trimester, months 1-3, cell division takes place initially and the embryo develops into a fetus. Already in week 3 the embryo begins to develop a brain, spinal cord and heart. Week 6-10 of the developmental period is particularly sensitive since this is when the fetus develops most organs and systems.

In week 11, the head is half the size of the fetus. From this period onwards, the head grows at a slower pace than the rest of the fetus and will at birth be about one fourth of the full birth length. In the second trimester, month 4-6, the organs systems already works quite well and the fetal growth is rapid. From conception, up until week 20, the fetus has a rapid and increasing growth in length. After week 20, the growth rate in length is decreasing. In week 24-26 the brain develops fast. In the third trimester, the fetus is storing fat on the body, and at week 37 the fetus is full length. The last weeks the fetus mainly puts on more fat (Martini et al., 2009).

Although the economics literature has focused mainly on birth weight, there is epidemiological research on the association between head circumference, birth length and a variety of outcomes.[98] The focus in the epidemiological literature has been on correlations and focusing on health outcomes, finding strong associations between birth weight and BMI, as well as birth length and height.[99] We extend the existing epidemiological literature by

[98] Birth length and head circumference have been used as outcomes in some economic studies; Persson and Rossin-Slater (forthcoming) find that infants that were prenatally exposed to maternal stress from family ruptures 0.18 percent shorter, and have 0.1 percent smaller head circumference. These results on child health are in line with results from maternal stress in Black et al. (2016).

[99] For example, Sorensen et al. (1999) find a strong correlation between birth weight, birth length and adult height among Danish men. Controlling for birth weight and length simultaneously, the effect for birth length persisted while the effect of birth weight almost disappeared. Rasmussen and Johansson (1998) study the correlation between BMI and three measures of health at birth: weight, length and ponderal index (weight/length^3) using data on Swedish men. They find strong associations between birth weight and BMI, as well as ponderal index and BMI. In a Finish study on twins using survey data, Pietiläinen et al. (2001) find that height in adolescence was predicted by birth weight and length as well as parents'

also studying human capital outcomes: GPA, cognitive skills, and educational attainment. While the existing literature has been focusing on correlations, our emphasis is on twin fixed effects models that limits the problem of confounding factors, such as genetics. Furthermore, in contrast to the existing literature, we apply a decomposition procedure (Gelbach, 2016) that takes into account in which order the measures are added when studying the effects of the different measures simultaneously. The aim of the decomposition analysis is to shed light on the mechanisms through which birth weight matter.

There is evidence that it is not only the gross size of the infant that is important, but also the relative proportions. One way of studying body proportionality is by decomposing low birth weight infants into two types, symmetric and asymmetric. Intrauterine growth restriction (IUGR) is the result of some circumstance during pregnancy that reduces the functioning of the placenta.[100] An early insult will impair the cellular reproduction and this will reduce the gross size of the fetus, although the fetus might continue to grow normally throughout the remaining part of the pregnancy. Asymmetric growth restriction is typically related to some shock later in pregnancy and characterized by the preservation of blood flow to essential organs like the brain. The gross size of asymmetrically IUGR neonates is also reduced due to the insult, although the fetal brain continues to get sufficient nutrition and oxygen. This is often referred to as the brain sparing effect in the medical literature (see Robinson, 2013, and references therein).

There is little research on the long-term effects related to the brain-sparing hypothesis. Robinson (2013) studies the effect of different types of growth restricted infants on IQ tests ages 4 and 7 using U.S. data. Robinson finds that children that are born symmetrically growth restricted perform worse compared with children born with asymmetrically growth restricted. Using Swedish register data we study a variety of short- and long-term outcomes using variation within twin pairs in different types of growth restriction.

height. They also find that that birth weight and parental BMI was the strongest predictors for BMI in adolescence. Morris et al. (1998) study children born in Brazil, and find that short birth length was associated with developmental delay at 12 months, but that only children born with short birth length and low birth weight had an increased risk of infant mortality and hospitalization.

[100] For example, the placenta functioning could be reduced from multiple gestations, tumors, and infections. Maternal characteristics such as body size, nutrition, and other types of life style related behaviors could has also been shown to affect fetal growth. There are also environmental factors such as toxic chemicals that could affect the fetal growth through the placenta (Robinson, 2013, and references therein).

3. Empirical Specification

We estimate the relation between different measures for health at birth and the various individual outcomes.[101] The first specification is used for the sample of singleton children and is defined as:

$$Outcome_i = \alpha + \beta \ln(Health\ at\ Birth_i) + \gamma' \boldsymbol{X}_i + \varepsilon_i, \quad (1)$$

where i indexes individual child, and the vector of controls, $\boldsymbol{X}$ includes an indicator for child gender, indicators for mother's age when giving birth and mother's years of schooling, indicators for birth order, and indicators for year by month of birth. Our specification also includes indicators for gestational age (measured in weeks), so that our object of interest is really fetal growth in the three dimensions we study, i.e., birth weight, birth length and head circumference at birth. The interpretation of the policy parameter in this model, β, is the difference in the outcome variable associated with a one percent change in the respective health at birth measure. Since the log specification gives relatively more weight to the bottom of the distribution, we also show results where we standardize the health measures by gestational week and gender, so to have mean 0 and standard deviation 1.[102]

To study how much of the variation in the different outcomes each of the three indicators of health at birth account for, we first measure how much the coefficient estimate of the variable under study moves when we add the two other indicators to the specification, i.e.,

$$\hat{\beta}^{uc} - \hat{\beta}^{c} = \delta, \quad (2)$$

where $\hat{\beta}^{uc}$ is the coefficient estimate from the unconditional specification where we only have included the one birth outcome measure under study and excluded the other two from the specification; $\hat{\beta}^{c}$ is the corresponding estimate from the conditional specification, where we have included the other two measures.

We next ask two to what extent each of the two initially excluded health at birth measures contribute to the overall change in the estimate. To do this, we use a decomposition technique proposed by Gelbach (2016). The Gelbach decomposition uses the well-known expression for omitted variable bias and calculates the share each of the additional health at birth measures contributes to in moving the initial coefficient estimate of the policy variable when going from the unconditional to the conditional model. A main ad-

[101] We follow the previous literature using the natural log of the different birth measures for our main specification. Non-parametric estimates are presented in Figure 3. These show a concave or linear relationship between our health measures at birth and long-term outcomes which justifies our parameterization.

[102] We standardize the measures within the sample since we have the full population of children born these cohorts and hence a representative sample.

vantage of this procedure is that the contribution of each measure does not depend on the order in which they are included in the model. This is a very useful property in our context, since the three alternative measure of health at birth are highly correlated.

Suppose we study the effect of birth weight on later outcomes and want to measure to what extent length at birth and head circumference affect the estimates. Gelbach (2016) shows that equation (2) can be extended to

$$\beta_{bw}^{uc} - \beta_{bw}^{c} = \Gamma_{bw}^{bl}\beta_{bw}^{bl} + \Gamma_{bw}^{hc}\beta_{bw}^{hc} = \delta_{bw}^{bl} + \delta_{bw}^{hc} = \delta_{bw}, \tag{3}$$

where Γ_{bw}^{bl} is the projection of the columns of X_{bw} on X_{bl}, i.e., $(X_{bw}'X_{bw})^{-1}X_{bw}'X_{bl}$ and β_{bw}^{bl} is the regression coefficient of length at birth in the conditional (full) regression of birth weight on the outcome under study. Γ_{bw}^{hc} and β_{bw}^{hc} are the corresponding projection matrix and parameter for head circumference, respectively (see Gelbach, 2016, for a more detailed description of the decomposition).

Ordinary least square estimation of (1) will give biased estimates if there are unobserved factors (for example, prenatal care and genetic factors) that that impacts both the child's health at birth and individual outcomes. To address this concern, we estimate a twin fixed effects model:

$$Outcome_{ij} = \alpha + \beta \ln\left(Health\ at\ Birth\ _{ij}\right) + \mu_j + \gamma' \boldsymbol{X}_{ij} + \varepsilon_{ij}, \tag{4}$$

where *j* is an index for the twin-pair and μ is the twin-pair fixed effect. In the twin fixed effects model, $\boldsymbol{X}$ only controls for gender and within twin pair birth order, since twin-pairs share all mother- and birth specific characteristics.

In the twin fixed effects model β is identified from the within twin pair variation in health at birth. Generally, children might be born small because of preterm delivery or because of slow fetal growth. Since twins have the same gestational length the within differences at birth arises because of differences in fetal growth, which is often related to nutritional uptake. If there are two placentas, differences in nutritional uptake might depend on differences in position in the womb, while if sharing placenta, twin might differ because of the position of the cord to the placenta (Black et al., 2007).

In common with all previous studies using administrative data, our sample includes both monozygotic and dizygotic twins, meaning that we cannot fully control for shared genetic endowments across twins. This concern is nonetheless attenuated when we examine outcomes for which the sample is restricted to male twin pairs. We also study same-sex twins for our other outcomes in Section 5.6 to assess to what extent this might affect our estimates.

Note that while the within-twin variation may credibly identify causal effects from differences in utero, the results do not necessarily generalize to

the population in general. There are substantial differences in the different measures of health at birth between singletons and twins, with twins coming to a larger extent from the bottom of the distribution. The difference partly stems from differences in gestational length with 2.5 weeks longer gestation length for singletons. Other birth outcomes also differ, for instance lower APGAR score, higher child mortality rate etc. However, as noted in Section 5.5, comparing singleton and twin outcomes, the outcomes are very similar. Still we should be should be cautious in generalizing from within twin pair results to the general population.

4. Data

We use data on all individuals born in Sweden between the years 1973-1979. The main data source is the Medical Birth Register which contains a personal identifier for the child, the mother and the father, as well as information on the time of birth, and health measures such as birth weight, birth length, head circumference and gestational length.[103,104] The personal identifiers for both children and parents allow us to match the Medical Birth Register with other administrative registers. In particular, it allows us to link birth outcomes with children's educational achievements, data from the Military Enlistment Register, as well as the socioeconomic status of the parents.

Armed with these rich linked data, we study a set of outcomes chosen to be representative of the main outcomes studied in the literature to date – so to ease comparison, especially given it is the first time this analysis is carried out on Swedish data. The first outcome that we study is infant death, which is defined as death before the child turns one year old. Date of death is recoded in the Cause of Death Register, but death occurring very close to birth is also recorded in the Medical Birth Register. Secondly, we also study grade point average (GPA) in the end of compulsory school, which is measured

[103] Birth length and head circumference are rounded to the nearest centimeter while birth weight are measured in grams. Measuring birth length and head circumference at birth is also more difficult than measuring birth weight. Varying muscle tonus and how much the child is stretched out during measuring will affect the recorded birth length. The size of the head might be affected by the way the head is placed during birth and late pregnancy, as well as the duration of birth (Lunde et al., 2007). However, we have no reason to believe that the quality of measures would vary systematically. Hence, potential measurement error should be classical and in that case cause attenuation bias.

[104] In Sweden, ultrasounds were introduced in the 1970's but were not routinely used until the 1980's. Hence, the measure of gestational age is based on last menstrual period. This usually leads to an underestimation of age at completed gestation. This error is not a cause of concern as it should affect our three measures in the same way. However, in order to handle implausible combinations we exclude children with birth weight more (or less) than four standard deviations above (or below) the average birth weight by gestational week. This restriction excludes 2,026 observations (0.3%). In doing so, we use Swedish reference curves for estimated intrauterine growth (Marsál et al., 1996).

around age 16, and standardized by birth cohort; and educational attainment, which is measured as the highest observed attainment at age 35. Lastly, matching on data from the Military Enlistment Register, we study men's height and cognitive skills at age 18. Military enlistment was mandatory for all men in Sweden born in the 1970's. The only exception was men who were institutionalized, prisoners, living abroad, and men with severe medical conditions or disability.[105] The cognitive test score that we use is the results from a non-standard IQ test that consists of four parts testing logical, spatial, verbal and technical comprehension respectively. The results from these tests were summarized into one single measure and standardized to give a score between 1 and 9, a so-called stanine scale (Öhman, 2015).

We now turn to documenting some basic patterns we observe in the data. Table 1 contains the summary statistics for the health at birth measures, various outcomes, and maternal characteristics used in our analysis. As expected, it shows that twins are on average lighter, shorter and have smaller head circumference. Twins also have shorter gestational age, higher risk of dying in infancy, and are more often born with an intrauterine growth restriction and have older mothers.

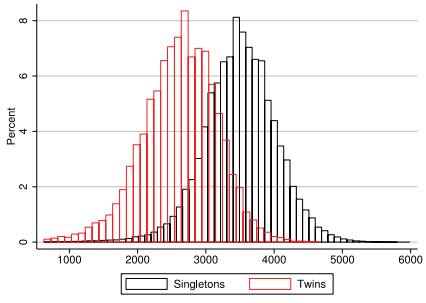
Figure 1 illustrates that twins have different distributions of birth weight, birth length and head circumference than singletons. The mean twin birth weight is 2,620 g and thereby 25 percent smaller than the mean singleton birth weight of 3,500 g. The mean twin birth length and mean twin head circumference are 7 and 5 percent smaller than the mean singleton birth length and head circumference, respectively. The fact that the difference between twins and singletons, in percentage terms, is bigger for birth weight than for other measures is partly due to the shorter gestational age at which the twins are born (given that fetuses gain most of the weight at the end of gestation).

As mentioned in Section 3 above, we identify the effect of health at birth on various outcomes by using variation in birth weights, birth length and head circumference within twin-pairs. All three measures of neonatal health vary within most twin pairs, and sometimes the difference is substantial. The average difference in twins' birth weight is 323 grams, the average difference in twins' birth length is 1.56 centimeters and the average difference in twins' head circumference is 1.06 centimeter. Figure 2 shows the distribution of difference in birth weight, birth length and head circumference for all twins. Among twin pairs, 57 percent have birth weight difference over 200 g, 22 percent have birth weight difference over 500 gr, 9 percent have a birth length difference above 3 cm and 8 percent have a head circumference differences above 2 cm.

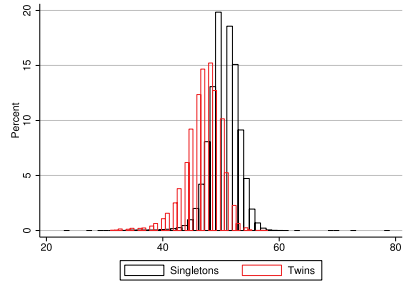
[105] Of all men in our final sample, 80 percent have enlisted. 84 percent enlisted at age 18, 15 percent at age 19, and 1 percent at age 20.

Table 1. Summary statistics

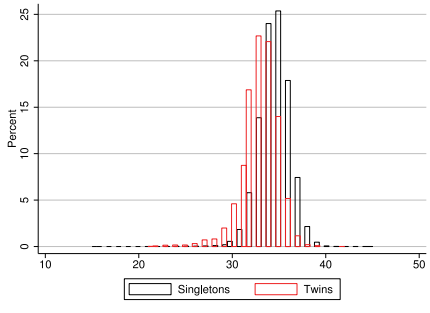
	Singletons			Twins		
	Mean	SD	N	Mean	SD	N
Female	0.486	0.50	678,848	0.500	0.50	10,400
Birth weight, grams	3498.479	527.80	678,848	2619.755	544.18	10,400
Head circ, cm	34.585	1.64	678,848	33.000	1.97	10,400
Birth length, cm	50.450	2.35	678,848	46.958	2.99	10,400
Gestational age, weeks	39.769	1.77	676,067	37.232	2.63	10,357
Infant death	0.005	0.07	678,848	0.023	0.15	10,400
Height, cm	179.550	6.56	276,572	178.994	6.64	4,022
GPA (Standardized)	0.024	0.99	641,436	0.085	0.97	9,653
Cog. Skills (Stanine)	5.083	1.91	277,266	4.938	1.90	4,029
Years of schooling, child	13.109	2.20	633,706	13.189	2.22	9,564
Intrauterine growth restriction	0.096	0.29	678,848	0.371	0.48	10,400
Asymmetric IUGR	0.064	0.24	678,848	0.256	0.44	10,400
Symmetric IUGR	0.032	0.18	678,848	0.115	0.32	10,400
Mother's age at birth	26.959	4.89	678,848	27.873	4.85	10,400
Years of schooling, mother	11.010	2.72	665,344	11.005	2.84	10,192



(a) Distributions of Birth Weight



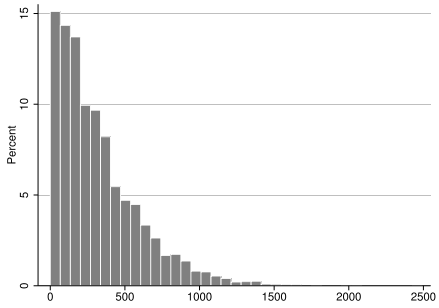
(b) Distributions of Birth Length



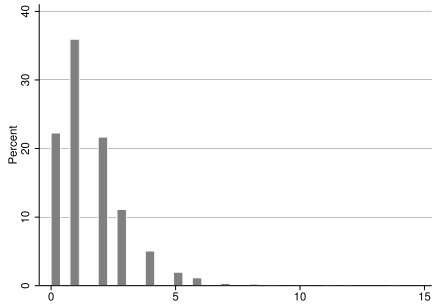
(c) Distributions of Head Circumference

Figure 1. Difference in birth weight, birth length and head circumference distributions

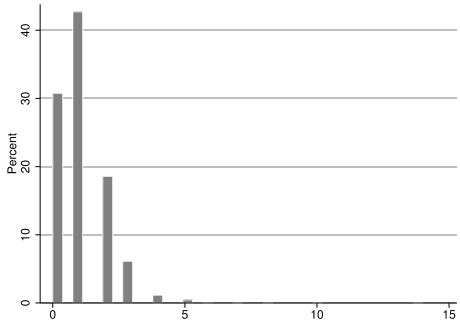
Notes: Figure 1 plots histograms of birth weight, birth length and head circumference for all twins (dashed line) and singletons (solid line).



(a) Differences in Birth Weight



(b) Differences in Birth Length



(c) Differences in Head Circumference

Figure 2. Difference in birth weight, birth length and head circumference within twin pairs

Notes: Figure 2 plots histograms of within-twin-pair difference in birth weight, birth length and head circumference for all twin births. Differences in birth weight are measured in grams, differences in birth length and head circumference in centimeter.

5. Results

In this section, we start with presenting the relationship between health at birth and our five outcomes for singletons. Secondly, we estimate the effects in a twin fixed effects framework, and then we discuss heterogeneous effects and external validity. Lastly, we study the importance of body proportionality.

5.1 Singletons

First we show the relationship between health at birth and later outcomes for all singletons births non-parametrically. Figure 3 displays the relationship between our three measures of health at birth (birth weight, length, and head circumference) and all five outcomes (infant death, height, GPA, cognitive skills, and years of schooling). The dummy variables correspond to 100 g wide bins for birth weight, and for birth length and head circumference each dummy variable corresponds to one centimeter. The first panel presents the relationship between birth weight and all outcomes, with the reference category being all children born below 1,000 g. For infant death, the risk reduces sharply up until 2,500 g, and then flattens out. For height age 18 the relationship is close to linear. However, for our human capital measures, GPA, cognitive skills (IQ) and years of schooling, the relationship seems to be positive until 4,000 g and negative thereafter.

The second panel shows the relationship between birth length and all five outcomes, using as reference category all children born with length <38 cm. It displays a pattern very similar to the one observed for the birth weight measure. Lastly, the third panel shows the relationship between head circumference and the five outcomes we study, using as reference category children born with head smaller than 26 cm. It shows less evidence of a decreasing effect at the top of the distribution for the human capital measures.

Given that boys are born heavier than girls on average, and given that girls on average have better educational outcomes than boys, we might think that the negative effect at the top of the distributions for GPA and years of schooling come from gender differences. Therefore, we split the sample by gender. Figure A1 and A2 in Appendix show that the pattern for females and males are very similar. Furthermore, in Section 5.4 we show that there is no evidence of any gender differences in our twin fixed effects specification.

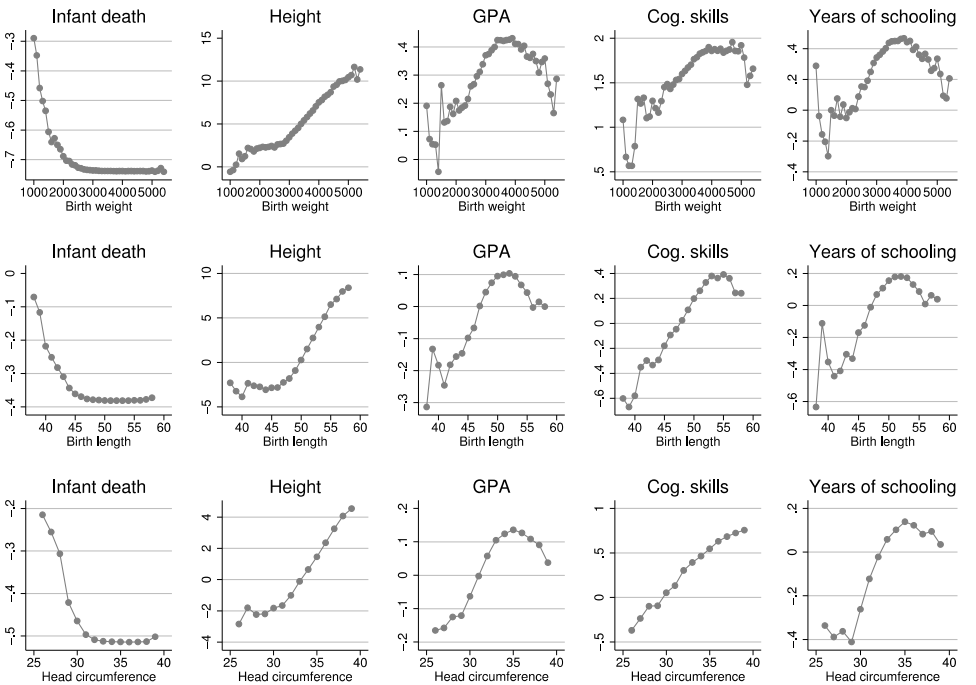


Figure 3. Non-parametric estimates for the association between different measures of health at birth and child outcomes (singletons). For birth weight the reference category is <1,000 g, for birth length it is <38 cm, and for head circumference it is <26 cm. To deal with extreme values we winsorize the data at 5,400 g for birth weight, (0.01% of the sample), 50 cm for birth length (0.02% of the sample), and 39 cm for head circumference (0.1% of the sample). GPA is standardized by cohort (mean 0, standard deviation 1). Height (cm) and cognitive skills (stanine scale) are only available for men age 18. Years of schooling is measured as highest observed educational attainment by age 35.

5.2 Decomposition

We next turn to the regression results for singletons, which are presented in Table 2. First, we present the results for OLS regressions of the log of the three health measure at birth on our five outcomes in Column (1). Since the three health measures are correlated and we are interested in the role of each of them in turn while controlling for the two others, we follow Gelbach (2016) and decompose the respective contribution of each measure in a consistent way. In Columns (2) through (6), we present the necessary components to do this evaluation, where Column (2) displays the regression results obtained including all three health measures simultaneously, Column (3) displays the difference between the conditional and unconditional estimates, and Columns (4) through (6) present the decompositions calculated from equation (3).

Focusing first on Column (1), each cell is a separate regression showing the estimate for each measure and outcome controlling for the set of standard controls (indicators for gestational week, mother's age at childbirth, mother's education, child gender, year by month of birth, and birth order). The effect of birth weight on the different outcomes is as follows. A 10 percent increase in birth weight decreases the risk of infant mortality by 0.29 percentage points. Increasing birth weight by 10 percent is associated with an increase of 1.6 cm in height among men at age 18. The results show that an increase in birth weight by 10 percent is associated with an increase in GPA by 0.04 standard deviations, an increase in test score by 0.10 points, and an increase in years of schooling by 0.07 years.

Turning to the results for length at birth and head circumference, the estimates are on average two to three times larger than those for birth weight. However, it is precarious to draw any conclusions about the relative importance of these estimates given the different distributions of the three measures.

We now turn to Gelbach decomposition starting with how the effect of birth weight on our outcomes is affected when including the two other child health measures. Table 2 Column (2) shows the results conditional on the other two measures as well as the same set of controls as the unconditional model. The effect of birth weight is reduced when the two other measures are included but unequally across the outcomes. While the coefficient of birth weight on infant mortality is only marginally reduced when the other two measures are included, the one on height is reduced by about 80 percent. For the cognitive and educational outcomes measured at ages 15, 18 and 35, the effect of birth weight is reduced by about 50 percent. The decomposition analysis shows that length at birth is by far the most important factor in reducing the effect of birth weight, with the exception of IQ scores at age 18,

where birth length and head circumference contribute equally to the reduction.

Turning to how the effects of birth length and head circumference are affected when the other two health measures are included, the overall conclusion is that the estimate for birth weight is less reduced for child mortality than for the other two measures. For the cognitive outcomes, the reductions are of a similar magnitude as for birth weight when the other two birth measures are included. From the decomposition analysis, we conclude that birth weight is the main reason for the reduction – again, with the exception of height for which birth length has the greatest predictive power.

5.3 Twins

As discussed in Section 3, using data on singletons we cannot make any causal claims since there are many unobserved factors that might affect both health at birth and later outcomes. In the absence of any exogenous variation that affects health at birth, following an established literature we use a twin fixed effect model to control for factors which are invariant within twin pairs. In addition to twin fixed effects, we only control for gender and within twin pair birth order. The results are displayed in Table 3.

Starting with Column (1) and comparing the results to those for singletons (Table 2) reveals the same basic patterns, with the exception of the effect of birth head circumference, which is driven to insignificance both in the infant death and years of schooling specifications.

Table 2. The effect of birth weight, birth length, and head circumference on child outcomes and decomposition of the effects of different measures at birth (singletons)

	(1)	(2)	(3)	(4)	(5)	(6)
				Decomposition of change in coefficients from added controls		
	Unconditional model	Conditional model	Change (Unconditional -conditional)	ln(Birth weight)	ln(Birth length)	ln(Head circ)
Infant death						
ln(Birth weight)	-0.029***	-0.023***	-0.006***		-0.006***	-0.000
	(0.001)	(0.001)	(0.001)	-	(0.001)	(0.000)
ln(Birth length)	-0.082***	-0.026***	-0.055***	-0.054***		-0.001
	(0.002)	(0.003)	(0.002)	(0.002)	-	(0.001)
ln(Head circ)	-0.051***	-0.002	-0.049***	-0.039***	-0.011***	
	(0.002)	(0.002)	(0.001)	(0.002)	(0.001)	-
Height, cm						
ln(Birth weight)	15.510***	3.111***	12.399***		11.713***	0.686***
	(0.094)	(0.190)	(0.108)	-	(0.096)	(0.057)
ln(Birth length)	61.561***	52.466***	9.095***	7.338***		1.756***
	(0.408)	(0.672)	(0.304)	(0.326)	-	(0.146)
ln(Head circ)	29.865***	3.856***	26.009***	5.111***	20.898***	
	(0.304)	(0.339)	(0.198)	(0.227)	(0.187)	-
GPA						
ln(Birth weight)	0.424***	0.222***	0.202***		0.134***	0.0680***
	(0.009)	(0.014)	(0.010)	-	(0.009)	(0.006)
ln(Birth length)	1.307***	0.606***	0.701***	0.527***		0.175***
	(0.029)	(0.042)	(0.030)	(0.032)	-	(0.014)
ln(Head circ)	1.000***	0.385***	0.615***	0.373***	0.243***	
	(0.027)	(0.032)	(0.017)	(0.023)	(0.016)	-
Cog. Skills						
ln(Birth weight)	0.951***	0.549***	0.402***		0.189***	0.213***
	(0.026)	(0.041)	(0.031)	-	(0.028)	(0.017)
ln(Birth length)	2.687***	0.847***	1.840***	1.294***		0.546***
	(0.086)	(0.123)	(0.090)	(0.096)	-	(0.043)
ln(Head circ)	2.438***	1.197***	1.240***	0.903***	0.338***	
	(0.081)	(0.095)	(0.051)	(0.067)	(0.049)	-
Years of schooling						
ln(Birth weight)	0.668***	0.321***	0.347***		0.255***	0.092***
	(0.019)	(0.031)	(0.023)	-	(0.020)	(0.013)
ln(Birth length)	2.151***	1.152***	0.999***	0.762***		0.237***
	(0.064)	(0.093)	(0.067)	(0.072)	-	(0.032)
ln(Head circ)	1.524***	0.521***	1.002***	0.539***	0.463***	
	(0.060)	(0.072)	(0.039)	(0.051)	(0.037)	-

Notes: Each cell represents results from one regression (Columns 1 and 2). The unconditional estimates include indicators for child gender, gestational age (weeks), year by month of birth, birth order, mother's age at childbirth and mother's educational attainment (years). The conditional estimates add controls for the other two measures of health at birth. Columns 4–6 show the results of a Gelbach decomposition of the contribution of the added health at birth measures between the unconditional and the conditional specifications. GPA is standardized by cohort (mean 0, standard deviation 1). Height (cm) and cognitive skills (stanine scale) are only available for men age 18. Years of schooling is measured as highest observed educational attainment by age 35. Tests for the equality across coefficients in the conditional model (Column 2) yields *p*-values=0.000 for all outcomes. Number of observations are 665,344 (infant death) 275,438 (height) 638,428 (GPA) 276,138 (Cog skills) 630,119 (Years of schooling). Robust standard errors in parenthesis, * p<0.10, ** p<0.05, *** p<0.01.

We next turn to the decomposition results, starting with how the effect of each birth measure on our outcomes in turn is affected when including the two others. Table 3 Column (2) shows the results for each measure conditional on the other two (as well as the same set of controls as in the unconditional model). First, the effect of birth weight on infant health and height becomes insignificant when including the two other health measures, while its effect on GPA and education is unchanged; in the case of cognitive skills at 18, instead, it is imprecisely estimated.

The decomposition analysis shows that birth length is the main reason for the reduction in the estimated effects of birth weight on the health outcomes (infant death and height) in the conditional models. Indeed, length at birth has a significant impact on infant mortality and height at 18 when the two other birth measures are included, even with a stronger effect on mortality. On the other hand, the impact of birth length on the cognitive and educational outcomes observed in the unconditional models (Column 1) are driven to insignificance in the conditional models (Column 2); the decomposition analysis (Column 4) shows that birth weight explains most of the reduction in the birth length coefficients. Lastly, the significant impact of head circumference in the unconditional models (Column 1) is driven to insignificance in all the conditional models (Column 2). The decomposition results show that birth length and birth weight explain most of the reduction in the coefficients for the health (Column 5) and cognitive (Column 4) outcomes, respectively. In other words, head circumference entails no additional informational content on health at birth once accounting for the other two measures, for both health and cognitive/educational outcomes.

Lastly, in order to put these magnitudes in perspective, it is useful to compare them with the results in the existing literature, to the extent possible, given that all previous papers do not report results for birth length and head circumference. Using the same log-linear specification on administrative data for Norway, Black et al. (2007), find that for singletons the association of birth weight with height for boys at 18 years of age is about 1.1 cm and with IQ of about 0.09 points - results very similar to ours. Figlio et al. (2014) - again using the same log-linear specification as we do - find that a 10 percent increase in birth weight is associated with an increase test scores in 3-8th grade by 0.03 of a standard deviation - which is also in line with our result. Furthermore, Royer (2009) finds that a 1,000 g increase in birth weight leads to about 0.16 years in education. Turning to the twin fixed effect results, our birth weight results are also in line with those in Black et al. (2007) and Figlio et al. (2014) for the height, IQ and GPA outcomes respectively, and with the Royer (2009) results for years of education.

Table 3. The effect of birth weight, birth length, and head circumference on child outcomes and decomposition of the different measures at birth (Twin fixed effects)

	(1)	(2)	(3)	(4)	(5)	(6)
				Decomposition of change in coefficients from added controls		
	Unconditional model	Conditional model	Change (Unconditional-conditional)	ln(Birth weight)	ln(Birth length)	ln(Head circ)
Infant death						
ln(Birth weight)	-0.035**	0.016	-0.052*	-	-0.050**	-0.002
	(0.016)	(0.029)	(0.027)		(0.024)	(0.013)
ln(Birth length)	-0.199***	-0.239**	0.040	0.044	-	-0.005
	(0.072)	(0.113)	(0.071)	(0.078)		(0.037)
ln(Head circ)	-0.092	-0.009	-0.083*	0.038	-0.121**	-
	(0.062)	(0.077)	(0.049)	(0.067)	(0.058)	
Height, cm						
ln(Birth weight)	6.632***	1.800	4.832***	-	3.872***	0.960
	(0.799)	(1.491)	(1.327)		(1.083)	(0.663)
ln(Birth length)	27.001***	18.910***	8.092**	5.212	-	2.880
	(3.187)	(5.157)	(3.737)	(4.277)		(1.942)
ln(Head circ)	20.083***	6.067	14.017***	4.341	9.676***	-
	(3.261)	(4.125)	(2.608)	(3.517)	(2.715)	
GPA						
ln(Birth weight)	0.298***	0.201*	0.097	-	0.059	0.038
	(0.068)	(0.114)	(0.090)		(0.074)	(0.051)
ln(Birth length)	0.950***	0.287	0.663**	0.556*	-	0.107
	(0.242)	(0.356)	(0.280)	(0.315)		(0.144)
ln(Head circ)	0.826***	0.220	0.606***	0.466*	0.141	-
	(0.240)	(0.295)	(0.197)	(0.264)	(0.175)	
Cog. skills						
ln(Birth weight)	0.701***	0.253	0.448	-	0.318	0.130
	(0.256)	(0.464)	(0.383)		(0.326)	(0.194)
ln(Birth length)	2.693***	1.563	1.130	0.740	-	0.390
	(0.949)	(1.601)	(1.267)	(1.355)		(0.583)
ln(Head circ)	2.225**	0.816	1.409*	0.615	0.794	-
	(1.036)	(1.219)	(0.736)	(1.127)	(0.815)	
Years of schooling						
ln(Birth weight)	0.767***	0.826***	-0.059	-	0.147	-0.206
	(0.179)	(0.317)	(0.264)		(0.211)	(0.161)
ln(Birth length)	2.410**	0.709	1.701**	2.291***	-	-0.590
	(0.936)	(1.017)	(0.767)	(0.881)		(0.461)
ln(Head circ)	1.041	-1.201	2.242***	1.895**	0.347	-
	(0.701)	(0.943)	(0.561)	(0.738)	(0.498)	

Notes: Each cell represents results from one regression (Columns 1 and 2). The unconditional estimates include indicators for child gender and within twin pair birth order. The conditional estimates add controls for the other two measures of health at birth. Columns 4–6 show the results of a Gelbach decomposition of the contribution of the added health at birth measures between the unconditional and the conditional specifications. GPA is standardized by cohort (mean 0, standard deviation 1). Height (cm) and cognitive skills (stanine scale) are only available for men age 18. Years of schooling is measured as highest observed educational attainment by age 35. Testing for equality across coefficients in the conditional model (Column 2) *p*-values are 0.396 (infant death) 0.177 (height) 0.991 (GPA) 0.897 (Cog. skills) 0.439 (Years of schooling). Number of observations are 10,400 (infant death) 2,874 (height) 9,653 (GPA) 2,873 (Cog skills) 9,564 (Years of schooling). Standard errors are clustered on twin pair, * p<0.10, ** p<0.05, *** p<0.01.

The log specification puts relatively more weight on the children with low birth weight, short length and small head circumference. To gain more understanding about distributional impacts, we re-estimate all the models using standardize birth measures. The results, shown in the Appendix Tables A1-A2, confirm the basic patterns observed using the semi-log specification.

5.4 Heterogeneous Effects

Distributional effects: The non-parametric results in Figure 3 showed some evidence of non-linear effects in the singleton sample. Although the log specification allows for some non-linearity, we can make the model even more flexible by estimating the twin fixed effects with indicators for health at birth only controlling for gender and within twin-pair birth order. Figure 4 displays the results from such a specification. The dummy variables correspond to 100 g wide bins for birth weight, and for birth length and head circumference each dummy variable corresponds to one centimeter. For most outcomes, except infant death, there is a clear linear pattern although small sample sizes at the tails make the pattern less smooth. In line with the previous literature we find that the risk of infant mortality rapidly reduces as birth weight increases, stabilizing at about 2,000 g.

Gender: We next study whether the effects of health at birth vary by gender. The results are presented in Appendix, Table A3, and show no evidence of any heterogeneous effects for any of the outcomes for which we have data on both gender in the twin fixed effects models.

Mother's educational attainment: Lastly, we examine heterogeneity by maternal education, whereby we define highly educated mothers those with more than 12 years of schooling. To study if the effect differs depending on mothers' educational attainment, we interact health at birth with a dummy variable indicating if the mother has more than 12 years of schooling, or less. The results are presented in Appendix, Table A4, and show no evidence of such heterogeneity.

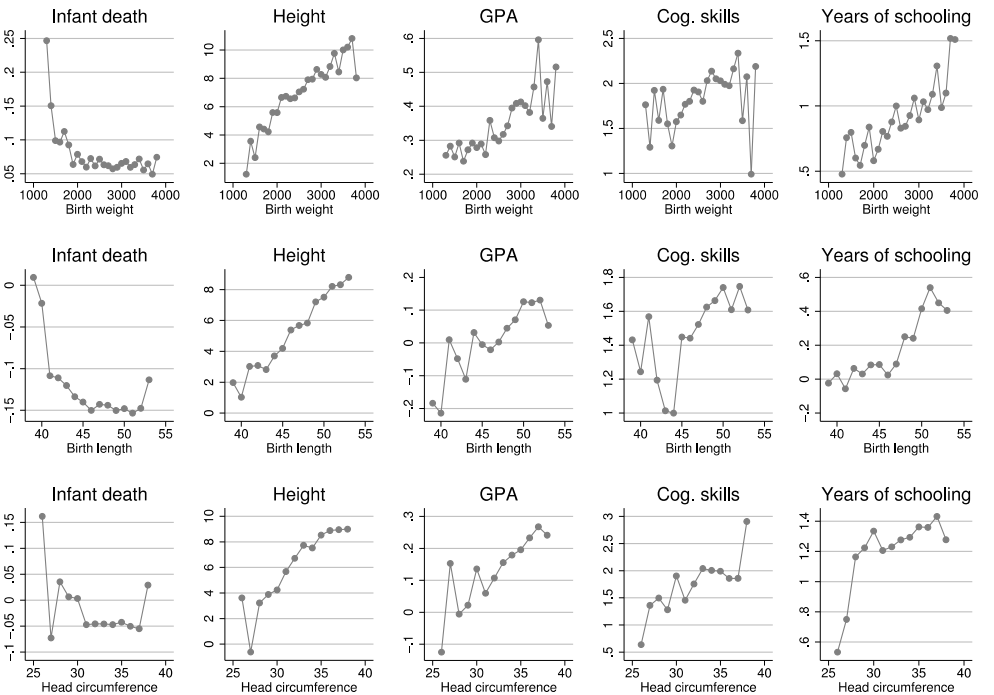


Figure 4. Twin fixed effects estimates for different measures of health at birth and child outcomes. Controlling for gender, within twin pair birth order and twin fixed effects. For birth weight the reference category is <1,000 grams, for birth length it is <38 cm, and for head circumference it is<26 cm. To deal with extreme values we winsorize the data at 3,800 g for birth weight (0.3% of the sample), at 53 cm for birth length (0.1% of the sample), and at 38 cm for head circumference (0.1% of the sample).GPA is standardized by cohort (mean 0, standard deviation 1). Height (cm) and cognitive skills (stanine scale) are only available for men age 18. Years of schooling is measured as highest observed educational attainment by age 35.

5.5 External Validity

The emphasis on twins in estimating the effects of neonatal health might come at cost of generalizability. In Section 4, we showed that twins on average have lower birth weight, shorter birth length and smaller head circumference at birth compared with singletons. The summary statistics also showed that mothers of twins on average are older than mothers of singletons. Bhalotra and Clarke (2016) show that women that give birth to twins are on average healthier, have healthier behavior, and spend more time on parenting. For the purpose of this study however, this is not a threat against our identification strategy, but the differences in distributions pose questions regarding the external validity of our results.

The analysis comparing results for singletons in Table 2 and twin fixed effects in Table 3, showed very similar estimates across birth measures and outcomes. The main differences relates to height and infant mortality. The effect of birth weight and birth length on height at age 18 is reduced by 50 percent when moving from singletons to twin fixed effects and the effect of head circumference is reduced by one third. The results for infant mortality also differs somewhat, the effect of head circumference is no longer significant, while the estimated effect of birth length is more than doubled.

The differences in height may depend on the fact that heritability of height being around 80 percent (Yang et al., 2010). The results on infant mortality on the other hand, may be related to singletons and twins being born small for different reasons; twins are generally born small because of sharing intrauterine environment, while being born small as a singleton may to a larger extent be related to the maternal health or other pregnancy related conditions. Furthermore, the differences in the estimates might arise from the differences in distributions (Figure 1). In the singleton sample, there is some evidence that being born big has negative effects on child outcomes in the singleton sample (Figure 3).[106] However, it is rare that twins are born big, which might explain why the relationship in the twin sample is linear (Figure 4).

For the sake of comparability, we limit our singleton sample to have common support with the twin sample, i.e. we restrict the sample to include children born with birth weight, birth length, and head circumference that are between the 1st and the 99th percentile of the twin sample distributions, respectively. We also estimate the singleton specification on the twin sample, i.e. not including twin fixed effects but the full set of singleton controls. The results are shown in Table 4 reveal that for our three human capital measures (Columns 4-6) the estimates for singletons and twins are very similar comparing the first and second panel. The importance of head circumference is if anything, greater in the twin sample. Adding twin fixed effects in the third

[106] This might be related to maternal health, such as diabetes.

panel slightly reduces mainly the head circumference estimates. The second column presents results for height, and shows that the effects of birth weight and birth length are very similar in the singleton and twin samples. When adding twin fixed effects, these estimates are halved.

Table 4. External validity

	(1) Infant death	(2) Height, cm	(3) GPA	(4) Cog. skills	(5) Years of schooling
Singletons (overlapping distributions)					
ln(Birth weight)	-0.042***	12.653***	0.542***	1.140***	0.841***
	(0.002)	(0.150)	(0.013)	(0.042)	(0.029)
ln(Birth length)	-0.117***	56.162***	1.601***	3.062***	2.617***
	(0.006)	(0.635)	(0.042)	(0.135)	(0.093)
ln(Head circ)	-0.068***	21.835***	1.195***	2.781***	1.821***
	(0.005)	(0.427)	(0.038)	(0.121)	(0.084)
Observations	442,059	164,392	423,194	164,894	418,036
Twins					
ln(Birth weight)	-0.090***	13.707***	0.381***	0.872***	0.906***
	(0.014)	(0.817)	(0.060)	(0.219)	(0.134)
ln(Birth length)	-0.321***	52.443***	1.374***	2.560***	3.234***
	(0.048)	(2.608)	(0.199)	(0.699)	(0.446)
ln(Head circ)	-0.246***	30.681***	1.414***	3.910***	2.084***
	(0.049)	(3.045)	(0.213)	(0.776)	(0.478)
Observations	10,192	2,853	9,597	2,852	9,496
Twin FE					
ln(Birth weight)	-0.038	6.649***	0.301***	0.711*	0.786***
	(0.024)	(1.167)	(0.098)	(0.375)	(0.260)
ln(Birth length)	-0.205**	27.340***	0.948***	2.779**	2.500***
	(0.104)	(4.647)	(0.347)	(1.387)	(0.961)
ln(Head circ)	-0.098	20.278***	0.817**	2.240	1.052
	(0.088)	(4.764)	(0.344)	(1.515)	(1.017)
Observations	10,192	2,853	9,597	2,852	9,496

Notes: Each cell represents results from one regression. The first panel (Singletons) contains a sample of singletons born with health at birth measures within the 1st-99th percentile of the twin birth distribution. The second (Twins) and third panel (Twins FE) contains a sample of twins. Number of observations for twins differs from Table 3 since in the second panel we control for mother's years of schooling which contains some missing information. To make to results comparable, we include only the non-missing observations also in the third panel. The regressions in the first and second panel include indicators for child gender, gestational age (weeks), year by month of birth, birth order, mother's age at childbirth and mother's educational attainment (years). The third panel controls for twin fixed effects, indicators of gender and within twin pair birth order. GPA is standardized by cohort (mean 0, standard deviation 1). Height (cm) and cognitive skills (stanine scale) are only available for men age 18. Years of schooling is measured as highest observed educational attainment by age 35. Robust standard errors in parenthesis, standard errors are clustered on twin pair in the third panel, * p<0.10, ** p<0.05, *** p<0.01.

Overall, the findings for singletons and twins are comparable, which suggests that our twin fixed effects estimates can be generalized to a larger population. However, this is only suggestive evidence for external validity from

the sample of twins since there may be different causal relationships for singletons and twins.

5.6 Genetic Differences

Unfortunately, our data do not contain information on whether the twin pairs are monozygotic or dizygotic. This means that the twin fixed effects results could be affected by differences in genetic traits. If genetic differences affect both health at birth and individual outcomes, and these are positively correlated, we might overestimate the effects. To assess the importance of this potential bias, we exclude different-sex twins since know that they are not monozygotic.

The results for same-sex twins in Table 5 show that the results for birth weight is very similar compared with the full sample of twins (for infant death -0.035 compared with -0.034, for GPA 0.298 compared with 0.225, and for years of schooling 0.767 compared with 0783). These findings are in line with the previous literature, Black et al. (2007) and Figlio et al. (2014) also find that the results for birth weight are stable for same-sex twins.

For birth length, the effect for infant death is reduced with one forth, and for GPA one third, while the effects on years of schooling is almost identical. Results for head circumference are also attenuated in the same-sex estimations with roughly one half for GPA. Note however that we lose precision in the same sex estimations because of the smaller sample size. The results however indicate that genetics is may be more important for birth length and head circumference than for birth weight.[107]

Table 5. The effect of birth weight, birth length, and head circumference on child outcomes among same-sex twins (Twin fixed effects)

	(1) Infant death	(2) GPA	(3) Years of schooling
ln(Birth weight)	-0.034	0.225**	0.783***
	(0.029)	(0.105)	(0.302)
ln(Birth length)	-0.157	0.674*	2.406**
	(0.124)	(0.378)	(1.117)
ln(Head circ)	-0.106	0.440	0.029
	(0.110)	(0.362)	(1.211)
Observations	7,436	6,891	6,823

Notes: Each cell represents results from one regression, controlling for indicators for child gender and within twin pair birth order. GPA is standardized by cohort (mean 0, standard deviation 1). Height (cm) and cognitive skills (stanine scale) are only available for men age 18. Years of schooling is measured as highest observed educational attainment by age 35. Standard errors are clustered on twin pair, * p<0.10, ** p<0.05, *** p<0.01.

[107] That would be in line with the findings in Lunde et al. (2007) on Norwegian register data. They find that relative to the explained variation in the different measures at birth, fetal genes were of more importance for birth length and head circumference, than for birth weight.

5.7 Body Proportionality

The importance of relative proportions of the different health at birth indicators might improve our understanding of the underlying mechanisms. In this section we focus on body proportionality and in particular on *intrauterine growth restricted* (IUGR) children, i.e. children born with birth weight <10th percentile for gestational week and gender. Comparing those who are symmetrically IUGR with the asymmetrically IUGR children can improve our understanding on the mechanisms behind the fetal origin hypothesis. A neonate is defined as symmetrically IUGR if born with birth weight <10th percentile and with head circumference <10th percentile for gestational age and gender. A neonate is instead defined as asymmetrically IUGR if born with birth weight <10th percentile, but with head circumference that is > 10th percentile for gestational week and gender.

The gross size of both symmetrically and asymmetrically growth restricted neonates is reduced. However, the fetal brain of asymmetrically growth restricted neonates gets sufficient nutrition and oxygen. This is often referred to as the brain sparing effect in the medical literature (Robinson, 2013). To test if brain sparing has any effect on child outcomes, we compare children that are born symmetrically and asymmetrically IUGR, by estimating the following model:

$$Outcome_i = \alpha + \beta_1 I_{asym} + \beta_2 I_{sym} + \gamma' \boldsymbol{X}_i + \varepsilon_i, \qquad (5)$$

where I_{asym} and I_{sym} are indicators of whether the child is asymmetrically or symmetrically growth restricted, and $\boldsymbol{X}$ includes as before an indicator for child gender, indicators for mother's age when giving birth and mother's years of schooling, indicators for birth order, and indicators for year by month of birth. The reference category is children who are not born growth restricted.

Table 6 shows results for singletons. These results suggest that it is worse to be born symmetrically than asymmetrically growth restricted. Compared with children born without growth restriction, being symmetrically growth restricted is associated with 1.6 percentage point increase in the risk of infant death. The corresponding figure for asymmetric IUGR is 0.7 percentage points. Height is reduced by 4.6 centimeters if born symmetrically growth restricted, and by 3.6 centimeters if born asymmetrically growth restricted, relative to non-IUGR. For our human capital measures, the effects are generally almost twice as large for symmetric as for asymmetric IUGR. The differences in the estimates are statistically significant.

Table 6. The effect of fetal growth restriction on child outcomes (Singletons)

	(1) Infant death	(2) Height, cm	(3) GPA	(4) Cog. skills	(5) Years of schooling
Symmetric	0.016***	-4.605***	-0.210***	-0.446***	-0.321***
	(0.001)	(0.066)	(0.007)	(0.019)	(0.014)
Asymmetric	0.007***	-3.644***	-0.121***	-0.250***	-0.190***
	(0.000)	(0.051)	(0.005)	(0.014)	(0.010)
p-value	0.000	0.000	0.000	0.000	0.000
Adj. R-Square	0.092	0.063	0.189	0.144	0.180
Observations	665,344	275,438	638,428	276,138	630,119

Notes: Each column represents results from one regression. Reference category is non-IUGR children. All specifications include indicators for child gender, gestational age (weeks), year by month of birth, birth order, mother's age at childbirth and mother's educational attainment (years). GPA is standardized by cohort (mean 0, standard deviation 1). Height (cm) and cognitive skills (stanine scale) are only available for men age 18. Years of schooling is measured as highest observed educational attainment by age 35. Robust standard errors in parenthesis, * p<0.10, ** p<0.05, *** p<0.01.

As before, we also estimate the twin fixed effects model controlling for gender and within twin-pair birth order. In our twin sample, 9.85 percent of twin pairs have variation in symmetric IUGR (one is symmetrically IUGR and the other is not IUGR) and 17.58 have variation in asymmetric IUGR (one is asymmetrically IUGR and the other is not IUGR). The results, shown in Table 7, reveal no statistically significant effects for infant death or cognitive skills. The effects on height, GPA and schooling are reduced, but still show that being symmetrically growth restricted appear to be related to worse outcomes than being asymmetrically IUGR (although the difference in the estimates is no longer statistically significant for years of schooling).

Table 7. The effect of fetal growth restriction on child outcomes (Twin fixed effects)

	(1) Infant death	(2) Height, cm	(3) GPA	(4) Cog. skills	(5) Years of schooling
Symmetric	0.004	-2.129***	-0.120***	-0.277	-0.190*
	(0.010)	(0.619)	(0.042)	(0.212)	(0.113)
Asymmetric	-0.000	-1.085***	-0.042	-0.061	-0.149*
	(0.005)	(0.396)	(0.029)	(0.142)	(0.086)
p-value	0.664	0.087	0.072	0.310	0.730
Adj. R-Square	0.425	0.788	0.739	0.695	0.599
Observations	10,400	4,022	9,653	4,029	9,564

Notes: Each column represents results from one regression. Reference category is non-IUGR children. All specifications include indicators for child gender and within twin pair birth order. GPA is standardized by cohort (mean 0, standard deviation 1). Height (cm) and cognitive skills (stanine scale) are only available for men age 18. Years of schooling is measured as highest observed educational attainment by age 35. Standard errors are clustered on twin pair, * p<0.10, ** p<0.05, *** p<0.01.

These results are in line with the brain-sparing hypothesis and show that shocks occurring early in pregnancy and leading to symmetrically growth-restricted neonates might directly impair cognitive development (see for example Weisglas-Kuperus et al., 2009).

6. Conclusions

In this paper we have examined the use of birth weight as the main measure of neonatal health, and studied the usefulness of birth length and head circumference as additional indicators. Our findings suggest that the use of these additional measures improves our understanding of the mechanisms through which early health matters. Furthermore, we find that there is a persistent effect of neonatal health on all human capital measures in adolescence and adulthood. This confirms and extends previous findings by Black et al. (2007) and Figlio et al. (2014), which were limited to birth weight.

In particular, we have used high-quality administrative data from Sweden with information on three different measures of health at birth – birth weight, length and head circumference - and studied the association of these health measures with five different outcomes later in life. Two of these outcomes – infant death and height – reflect health; and three – GPA at age 16, IQ measured at enlistment and years of schooling – measure human capital at different stages during the life course.

In cross-sectional analyses, we have found that the three measures of health at birth are strongly associated with all outcomes, also when they are simultaneously included in the estimated models. This suggests that they independently contribute to explaining variation in the life outcomes in all stages of the life course – as confirmed when using the decomposition approach proposed recently by Gelbach.

When exploiting variation within twin pairs, we have found that the length of the newborn is the main determinant of the health outcomes we study (infant death and height), while the birth weight is the main determinant of the human capital outcomes. The related decomposition results suggest that birth weight is indeed proxying for birth length when examining the health consequences of birth outcomes.

Lastly, we have shown the usefulness of measuring head circumference by categorizing growth-restricted newborns in two different types, and showing that those who are symmetrically restricted, i.e. who have not been protected by brain sparing, suffer more negative consequences, especially in terms of cognitive impairments.

In conclusion, our work shows the usefulness of collecting an extended set of birth outcomes, in particular the length and the head circumference of the newborn, as able to shed more light on the mechanisms through which birth weight matters for later health and cognitive outcomes.

References

Almond, D. (2006). Is the 1918 influenza pandemic over? Long-term effects of in utero influenza exposure in the post-1940 US population. *Journal of political Economy*, *114*(4), 672-712.

Almond, D., Chay, K. Y., & Lee, D. S. (2005). The costs of low birth weight. The Quarterly Journal of Economics, 120(3), 1031-1083.

Almond, D., & Currie, J. (2011). Killing me softly: The fetal origins hypothesis. *The Journal of Economic Perspectives*, *25*(3), 153-172.

Almond, D., Currie, J., & Duque, V. (2017). Childhood Circumstances and Adult Outcomes: Act II (No. w23017). National Bureau of Economic Research.

Almond, D., Edlund, L., & Palme, M. (2009). Chernobyl's subclinical legacy: prenatal exposure to radioactive fallout and school outcomes in Sweden. *The Quarterly journal of economics*, *124*(4), 1729-1772.

Almond, D., Edlund, L., Li, H., & Zhang, J. (2007). *Long-term effects of the 1959-1961 China famine: Mainland China and Hong Kong* (No. w13384). National Bureau of Economic Research.

Barker, D. J., Osmond, C., Winter, P. D., Margetts, B., & Simmonds, S. J. (1989). Weight in infancy and death from ischaemic heart disease. *The Lancet*, *334*(8663), 577-580.

Behrman, J. R., & Rosenzweig, M. R. (2004). Returns to birthweight. *Review of Economics and statistics*, *86*(2), 586-601.

Bhalotra, S. R., & Clarke, D. (2016). The Twin Instrument. IZA DP No. 10405

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. The Quarterly Journal of Economics, 122(1), 409-439.

Black, S. E., Devereux, P. J., & Salvanes, K. G. (2016). Does grief transfer across generations? bereavements during pregnancy and child outcomes. *American Economic Journal: Applied Economics*, *8*(1), 193-223.

Currie, J., & Hyson, R. (1999). *Is the impact of health shocks cushioned by socioeconomic status? The case of low birthweight* (No. w6999). National bureau of economic research.

Figlio, D., Guryan, J., Karbownik, K., & Roth, J. (2014). The effects of poor neonatal health on children's cognitive development. The American Economic Review, 104(12), 3921-3955.

Gelbach, J. B. (2016). When do covariates matter? And which ones, and how much?. *Journal of Labor Economics*, *34*(2), 509-543.

Lindley, A. A., Becker, S., Gray, R. H., & Herman, A. A. (2000). Effect of continuing or stopping smoking during pregnancy on infant birth weight, crown-heel length, head circumference, ponderal index, and brain: body weight ratio. American Journal of Epidemiology, 152(3), 219-225.

Lumey, L. H., & Stein, A. D. (1997). Offspring birth weights after maternal intrauterine undernutrition: a comparison within sibships. *American Journal of Epidemiology*, *146*(10), 810-819.

Lunde, A., Melve, K. K., Gjessing, H. K., Skjærven, R., & Irgens, L. M. (2007). Genetic and environmental influences on birth weight, birth length, head circumference, and gestational age by use of population-based parent-offspring data. *American journal of epidemiology*, *165*(7), 734-741.

Marsál K, Persson PH, Larsen T, Lilja H, Selbing A, Sultan B. Intrauterine growth curves based on ultrasonically estimated foetal weights. *Acta Paediatr*. 1996;85:843–848.

Martini, F. H., Timmons, M. J. & Tallitsch, R. B (2009) Human Anatomy (6th edition) Pearson Education, Inc.

Morris, S. S., Victoria, C. G., Barros, F. C., Halpern, R., Menezes, A. M., Cesar, J. A., ... & Tomasi, E. (1998). Length and ponderal index at birth: associations with mortality, hospitalizations, development and post-natal growth in Brazilian infants. *International Journal of Epidemiology*, *27*(2), 242-247.

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*(1), 88-138.

Persson, P. & Rossin-Slater, M. (Forthcoming) Family Ruptures, Stress, and the Mental Health of the Next Generation. *American Economic Review*

Petronis, A. (2010). Epigenetics as a unifying principle in the aetiology of complex traits and diseases. *Nature*, *465*(7299), 721-727.

Pietiläinen, K. H., Kaprio, J., Räsänen, M., Winter, T., Rissanen, A., & Rose, R. J. (2001). Tracking of body size from birth to late adolescence: contributions of birth length, birth weight, duration of gestation, parents' body size, and twinship. *American Journal of Epidemiology*, *154*(1), 21-29.

Ramakrishnan, U., Stein, A. D., Parra-Cabrera, S., Wang, M., Imhoff-Kunsch, B., Juárez-Márquez, S., ... & Martorell, R. (2010). Effects of docosahexaenoic acid supplementation during pregnancy on gestational age and size at birth: randomized, double-blind, placebo-controlled trial in Mexico. Food and nutrition bulletin, 31(2_suppl2), S108-S116.

Rasmussen, F., & Johansson, M. (1998). The relation of weight, length and ponderal index at birth to body mass index and overweight among 18-year-old males in Sweden. *European journal of epidemiology*, *14*(4), 373-380.

Robinson, J. J. (2013). Sound body, sound mind? Asymmetric and symmetric fetal growth restriction and human capital development. University of Alabama at Birmingham, unpublished manuscript.

Rosenzweig, M. R., & Zhang, J. (2013). Economic growth, comparative advantage, and gender differences in schooling outcomes: Evidence from the birthweight differences of Chinese twins. *Journal of Development Economics*, *104*, 245-260.

Royer, H. (2009). Separated at girth: US twin estimates of the effects of birth weight. *American Economic Journal: Applied Economics*, *1*(1), 49-85.

Shankaran, S., Das, A., Bauer, C. R., Bada, H. S., Lester, B., Wright, L. L., & Smeriglio, V. (2004). Association between patterns of maternal substance use and infant birth weight, length, and head circumference. Pediatrics, 114(2), e226-e234.

Sørensen, H. T., Sabroe, S., Rothman, K. J., Gillman, M., Steffensen, F. H., Fischer, P., & Serensen, T. I. (1999). Birth weight and length as predictors for adult height. *American Journal of Epidemiology*, *149*(8), 726-729.

Torche, F., & Conley, D. (2016). A Pound of Flesh. In The Oxford Handbook of Economics and Human Biology.

Weisglas-Kuperus, N., Hille, E. T. M., Duivenvoorden, H. J., Finken, M. J., Wit, J. M., van Buuren, S., ... & Dutch POPS-19 Collaborative Study Group. (2009). Intelligence of very preterm or very low birthweight infants in young adulthood. *Archives of Disease in Childhood-Fetal and Neonatal Edition*, *94*(3), F196-F200.

Yang, J., Benyamin, B., McEvoy, B. P., Gordon, S., Henders, A. K., Nyholt, D. R., ... & Goddard, M. E. (2010). Common SNPs explain a large proportion of the heritability for human height. *Nature genetics*, *42*(7), 565-569.

Öhman, M. (2015). *Be smart, live long: the relationship between cognitive and non-cognitive abilities and mortality* (No. 2015: 21). Working Paper, IFAU-Institute for Evaluation of Labour Market and Education Policy.

Appendix

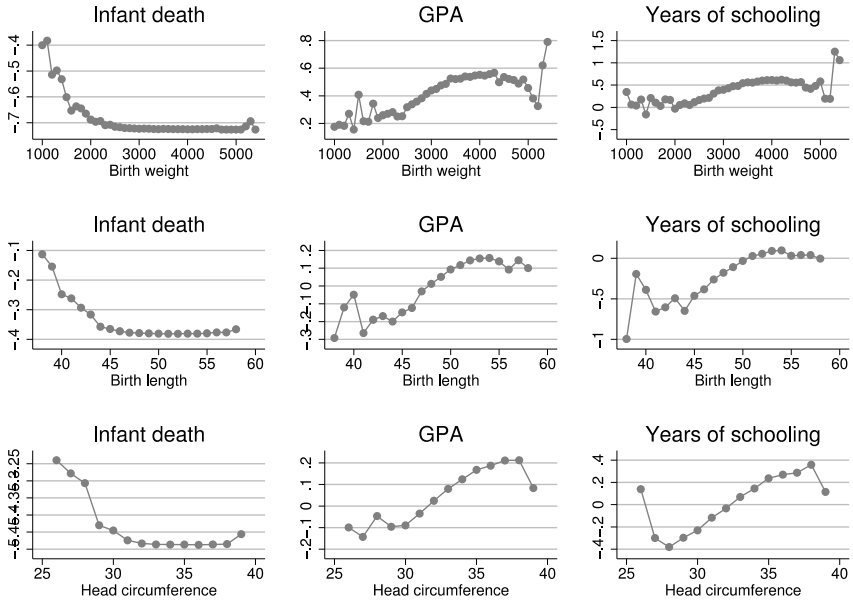


Figure A1. Singletons Females: Non-parametric estimates for the association between different measures of health at birth and child outcomes.

Notes: For birth weight the reference category is <1,000 g, for birth length it is <38 cm, and for head circumference it is <26 cm. To deal with extreme values we winsorize the data at 5,400 g for birth weight, (0.01% of the sample), 50 cm for birth length (0.02% of the sample), and 39 cm for head circumference (0.1% of the sample). GPA is standardized by cohort (mean 0, standard deviation 1). Years of schooling is measured as highest observed educational attainment by age 35.

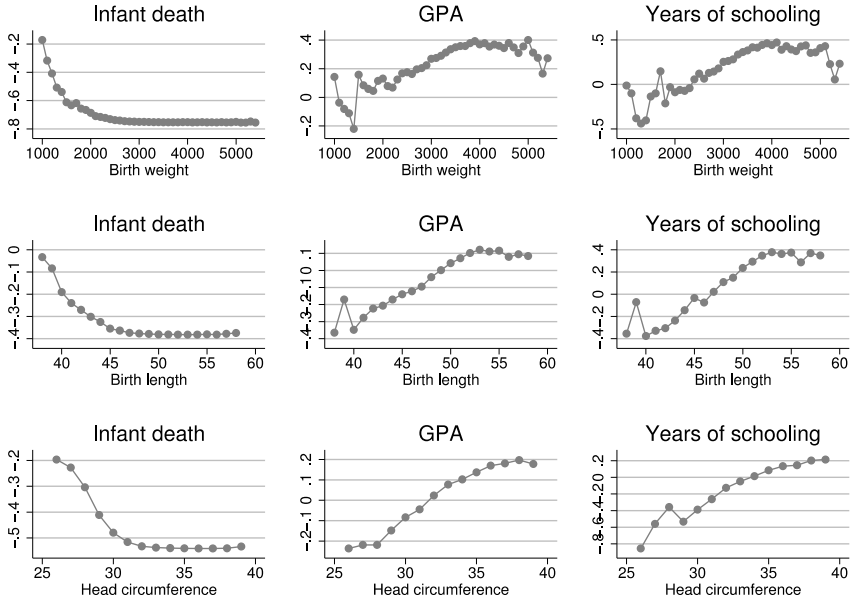


Figure A2. Singletons Males: Non-parametric estimates for the association between different measures of health at birth and child outcomes.

Notes: For birth weight the reference category is <1,000 g, for birth length it is <38 cm, and for head circumference it is <26 cm. To deal with extreme values we winsorize the data at 5,400 g for birth weight, (0.01% of the sample), 50 cm for birth length (0.02% of the sample), and 39 cm for head circumference (0.1% of the sample). GPA is standardized by cohort (mean 0, standard deviation 1). Years of schooling is measured as highest observed educational attainment by age 35.

Table A1. The effect of birth weight, birth length, and head circumference on child outcomes and decomposition of the different measures at birth, standardized measures (Singletons)

	(1)	(2)	(3)	(4)	(5)	(6)
				Decomposition of change in coefficients from added controls		
	Unconditional model	Conditional model	Change (Unconditional-conditional)	Birth weight	Birth length	Head circ
Infant death						
Birth weight	-0.003*** (0.000)	-0.002*** (0.000)	-0.001*** (0.000)	-	-0.001*** (0.000)	0.000 (0.000)
Birth length	-0.003*** (0.000)	-0.001*** (0.000)	-0.002*** (0.000)	-0.002*** (0.000)	-	0.000 (0.000)
Head circ	-0.002*** (0.000)	0.000 (0.000)	-0.002*** (0.000)	-0.001*** (0.000)	-0.000*** (0.001)	-
Height, cm						
Birth weight	2.059*** (0.012)	0.378*** (0.023)	1.681*** (0.014)	-	1.588*** (0.013)	0.092*** (0.007)
Birth length	2.517*** (0.015)	2.173*** (0.024)	0.345*** (0.012)	0.272*** (0.013)	-	0.072*** (0.006)
Head circ	1.292*** (0.013)	0.170*** (0.014)	1.122*** (0.009)	0.202*** (0.010)	0.920*** (0.008)	-
GPA						
Birth weight	0.053*** (0.001)	0.023*** (0.002)	0.030*** (0.001)	-	0.020*** (0.001)	0.010*** (0.001)
Birth length	0.052*** (0.001)	0.028*** (0.002)	0.024*** (0.001)	0.017*** (0.001)	-	0.007*** (0.001)
Head circ	0.042*** (0.001)	0.018*** (0.001)	0.024*** (0.001)	0.013*** (0.001)	0.012*** (0.001)	-
Cog. skills						
Birth weight	0.120*** (0.003)	0.060*** (0.005)	0.060*** (0.004)	-	0.030*** (0.004)	0.030*** (0.002)
Birth length	0.108*** (0.003)	0.041*** (0.005)	0.066*** (0.004)	0.043*** (0.004)	-	0.023*** (0.002)
Head circ	0.104*** (0.003)	0.054*** (0.004)	0.050*** (0.002)	0.032*** (0.003)	0.018*** (0.002)	-
Years of schooling						
Birth weight	0.084*** (0.003)	0.033*** (0.004)	0.051*** (0.003)	-	0.038*** (0.003)	0.013*** (0.002)
Birth length	0.086*** (0.003)	0.051*** (0.004)	0.034*** (0.003)	0.024*** (0.003)	-	0.010*** (0.001)
Head circ	0.064*** (0.003)	0.024*** (0.003)	0.040*** (0.002)	0.040*** (0.002)	0.022*** (0.002)	-

Notes: Each cell represents results from one regression (columns 1 and 2). The unconditional estimates include indicators for child gender, gestational age (weeks), year by month of birth, birth order, mother's age at childbirth and mother's educational attainment (years). The conditional estimates add controls for the other two measures of health at birth. Columns 4–6 show the results of a Gelbach decomposition of the contribution of the added health at birth measures between the unconditional and the conditional specifications. GPA is standardized by cohort (mean 0, standard deviation 1). Height (cm) and cognitive skills (stanine scale) are only available for men age 18. Years of schooling is measured as highest observed educational attainment by age 35. Testing for equality across coefficients in the conditional model (column 2) p-values are 0.000 for all outcomes, except cog. skills (0.082). Number of observations are 665,344 (infant death) 275,438 (height) 638,428 (GPA) 276,138 (Cog skills) 630,119 (Years of schooling). Robust standard errors in parenthesis, * $p<0.10$, ** $p<0.05$, *** $p<0.01$.

Table A2. The effect of birth weight, birth length, and head circumference on child outcomes and decomposition of the different measures at birth, standardized measures (Twin fixed effects)

	(1)	(2)	(3)	(4)	(5)	(6)
				Decomposition of change in coefficients from added controls		
	Unconditional model	Conditional model	Change (Unconditional-conditional)	Birth weight	Birth length	Head circ
Infant death						
Birth weight	-0.004	0.005	-0.009**		-0.008**	-0.000
	(0.002)	(0.004)	(0.004)	-	(0.003)	(0.002)
Birth length	-0.007***	-0.010**	0.003	0.003		-0.000
	(0.003)	(0.004)	(0.003)	(0.003)	-	(0.002)
Head circ	-0.003	-0.001	-0.002	0.003	-0.006**	
	(0.003)	(0.003)	(0.002)	(0.003)	(0.002)	-
Height, cm						
Birth weight	1.109***	0.256	0.853***		0.700***	0.153
	(0.138)	(0.253)	(0.223)	-	(0.179)	(0.117)
Birth length	1.234***	0.916***	0.318*	0.194		0.124
	(0.144)	(0.228)	(0.164)	(0.190)	-	(0.092)
Head circ	0.926***	0.264	0.661***	0.169	0.493***	
	(0.156)	(0.198)	(0.122)	(0.162)	(0.127)	-
GPA						
Birth weight	0.055***	0.042**	0.013		0.008	0.005
	(0.012)	(0.020)	(0.015)	-	(0.013)	(0.009)
Birth length	0.045***	0.010	0.035***	0.030**		0.004
	(0.011)	(0.016)	(0.013)	(0.014)	-	(0.007)
Head circ	0.040***	0.009	0.032***	0.027**	0.005	
	(0.011)	(0.014)	(0.009)	(0.013)	(0.008)	-
Cog. skills						
Birth weight	0.109**	0.029	0.080		0.061	0.019
	(0.044)	(0.080)	(0.065)	-	(0.056)	(0.033)
Birth length	0.118***	0.080	0.038	0.022		0.016
	(0.043)	(0.073)	(0.058)	(0.061)	-	(0.027)
Head circ	0.095**	0.033	0.062*	0.019	0.043	
	(0.048)	(0.057)	(0.034)	(0.053)	(0.039)	-
Years of schooling						
Birth weight	0.133***	0.145*	-0.012		0.030	-0.041
	(0.031)	(0.077)	(0.045)	-	(0.036)	(0.028)
Birth length	0.112***	0.038	0.074**	0.106***		-0.032
	(0.043)	(0.065)	(0.034)	(0.040)	-	(0.021)
Head circ	0.045	-0.066	0.111***	0.091***	0.020	
	(0.033)	(0.063)	(0.026)	(0.034)	(0.024)	-

Notes: Each cell represents results from one regression (columns 1 and 2). The unconditional estimates include indicators for child gender and within twin pair birth order. The conditional estimates add controls for the other two measures of health at birth. Columns 4–6 show the results of a Gelbach decomposition of the contribution of the added health at birth measures between the unconditional and the conditional specifications. GPA is standardized by cohort (mean 0, standard deviation 1). Height (cm) and cognitive skills (stanine scale) are only available for men age 18. Years of schooling is measured as highest observed educational attainment by age 35. Testing for equality of coefficients in the conditional model (column 2) p-values are 0.113 (infant death) 0.076 (height) 0.519 (GPA) 0.879 (Cog. skills) 0.175 (Years of schooling). Number of observations are 10,400 (infant death) 2,874 (height) 9,653 (GPA) 2,873 (Cog skills) 9,564 (Years of schooling). Standard errors are clustered on twin pair, * p<0.10, ** p<0.05, *** p<0.01.

Table A3. The effect of birth weight, birth length, and head circumference on child outcomes by gender (Twin fixed effects)

	(1) Infant death	(2) GPA	(3) Years of schooling
Birth weight			
ln(Birth weight)	-0.034	0.363***	1.031***
	(0.032)	(0.133)	(0.323)
ln(Birth weight)*Female	-0.002	-0.126	-0.515
	(0.044)	(0.160)	(0.383)
Female	0.013	1.267	4.628
	(0.351)	(1.260)	(3.017)
Adj. R-Square	0.425	0.739	0.600
Birth length			
ln(Birth length)	-0.168	1.175**	2.933**
	(0.133)	(0.462)	(1.167)
ln(Birth length)*Female	-0.061	-0.439	-1.015
	(0.158)	(0.567)	(1.359)
Female	0.228	1.969	4.488
	(0.611)	(2.187)	(5.244)
Adj. R-Square	0.427	0.739	0.599
Head circumference			
ln(Head circ)	-0.068	1.113**	2.148*
	(0.137)	(0.481)	(1.272)
ln(Head circ)*Female	-0.046	-0.525	-2.025
	(0.178)	(0.546)	(1.473)
Female	0.156	2.114	7.646
	(0.625)	(1.916)	(5.160)
Adj. R-Square	0.425	0.739	0.599
Observations	10,400	9,653	9,564

Notes: Each panel of each column represents results from one regression. Outcomes are limited to those for which we observe both females and males. GPA is standardized by cohort (mean 0, standard deviation 1). Years of schooling is measured as highest observed educational attainment by age 35**.** We control for indicators for child gender and within twin pair birth order. Standard errors are clustered on twin pair, * p<0.10, ** p<0.05, *** p<0.01.

Table A4. The effect of birth weight, birth length, and head circumference on child outcomes by mother's education (Twin fixed effects)

	(1) Infant death	(2) Height, cm	(3) GPA	(4) Cog. skills	(5) Years of schooling
Birth weight					
ln(Birth weight)	-0.036	6.855***	0.290**	0.782	0.825***
	(0.029)	(1.590)	(0.116)	(0.503)	(0.289)
ln(Birth weight)*High edu	0.001	-0.730	0.028	-0.322	-0.224
	(0.042)	(3.141)	(0.208)	(1.041)	(0.618)
Adj. R-Square	0.425	0.793	0.739	0.696	0.600
Birth length					
ln(Birth length)	-0.190	25.980***	0.940**	3.198*	2.431**
	(0.127)	(6.327)	(0.421)	(1.891)	(1.079)
ln(Birth length)*High edu	-0.034	4.831	0.037	-1.928	-0.077
	(0.189)	(12.413)	(0.711)	(3.667)	(2.246)
Adj. R-Square	0.427	0.795	0.739	0.696	0.599
Head circumference					
ln(Head circ)	-0.089	21.802***	0.868**	3.521*	1.880
	(0.109)	(6.691)	(0.416)	(2.056)	(1.154)
ln(Head circ)*High edu	-0.014	-5.963	-0.153	-4.897	-3.140
	(0.159)	(12.094)	(0.720)	(3.951)	(2.243)
Adj. R-Square	0.425	0.788	0.739	0.696	0.599
Observations	10,400	4,022	9,653	4,029	9,564

Notes: Each panel of each column represents results from one regression. High education are defined as >12 years of schooling. GPA is standardized by cohort (mean 0, standard deviation 1). Height (cm) and cognitive skills (stanine scale) are only available for men age 18. Years of schooling is measured as highest observed educational attainment by age 35. We control for indicators for child gender and within twin pair birth order. Standard errors are clustered on twin pair, * p<0.10, ** p<0.05, *** p<0.01.