Nature versus Nurture in the Intergenerational Transmission of Health: Lessons from a Large Sample of Adoptees[1]

by

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This draft: March 30th, 2015

Very preliminary. Please do not cite

Abstract:

We study the importance of pre- and post-birth factors in the formation of adult health and life expectancy using a data on about 22,000 adoptees born between 1940 and 1967 including information on both biological and adopting parents. We find that the health status of the biological mother has significant impact on the probability of premature mortality of her children. We also find that the educational attainment of the adopting mother is very important for the adoptee's long-term health. We can thus reject the hypothesis that parental resources and the environment during upbringing has no effect on adult health.

[1] We are grateful for comments from Orazio Attanasio, Richard Blundell and Gabriella Conti as well as for those from participants on seminars at the University College London and Uppsala University. Mikael Lindahl is a Royal Swedish Academy of Sciences Research Fellow supported by a grant from the Torsten and Ragnar Söderberg Foundation, the Scientific Council of Sweden and the European Research Council [ERC starting grant 241161]. Mårten Palme gratefully acknowledges financial support from the Swedish Council of Social Research. Emilia Simeonova gratefully acknowledges support from the Swedish Research Council and the National Science Foundation.

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1. Introduction

Health is a crucial determinant of education and labor market earnings (Grossman, 2000; Case, Fertig and Paxson, 2005; Currie and Madrian, 1999). Inequalities in health status and their immediate and long-term consequences, how health gets transmitted across generations and its relation to educational attainments have been heavily debated in both scientific and policy circles. The extent to which health status is inherited from previous generations through immutable genetic factors as opposed to the formative influence of the pre-natal and childhood circumstance has obvious implications for the potential of policies designed to address health inequalities.

A large literature has established that experiences in the pre-natal period have long-lasting effects on infant health, children's health and socio-economic wellbeing later in life (Almond 2006, Almond, Edlund and Palme, 2007; Nilsson, 2014). Thus, at least part of the characteristics that appear innate at birth may be determined by suboptimal physical or socio-economic environment while in utero. A separate strand of studies have shown that health endowments at birth are strongly predictive of youth and adult success (Black et al., 2007, Figlio et al., 2014), suggesting that at least part of the strong inter-generational correlation of low socio-economic circumstance is due to the inter-generational transmission of bad health. However, relatively little is known about the importance of changes in the childhood environment that happen after birth for long-term health. In particular, the extent to which positive changes in family background post-birth can counter-balance or reinforce the effects of endowments at birth is not well-understood.

The aim of this paper is to separate out the importance of pre-birth factors from post-birth factors in the formation of adult health. We use a large sample of Swedish adoptees. We study how the health status and educational attainments of the biological parents – related to genetic factors and in utero health investments– and the corresponding characteristics of the adopting parents – related to health formation during childhood and adolescence – affect the child's health later in life.

The benefit of the adoption design is that, given assumptions of conditional random assignment of children to adoptive families and no interaction between the adoptive and biological families, it can be used to estimate the effect of the post adoption family environment on long-term outcomes for the adopted child. We control of a number of health

inputs pertinent to the pre-natal period, making it possible to attribute adult health outcomes to random variation in the (adoptive) family environment during childhood and adolescence.

Although the adoption design should not be used to infer causal effects from exogenously changing parental education on childhood outcomes, our study is of course related to studies of the effect of extra education in childhood health. There is a growing literature on the effects of maternal education on infant health that has delivered different conclusions across types of extra education attained and country of origin (Currie and Moretti, 2007; McCrary and Royer, 2012; Lindeboom et al, 2009). These studies make use of exogenous changes in the policy or educational environment that induce future mothers to obtain extra education. Such experiments do not, however, exogenously affect the education of women with children, presenting a serious challenge to the identification of post-birth changes in maternal knowledge and family social standing on long-term children's health. Conditional on the appropriate caveats, the random placement of adoptees post-birth could provide plausible upper bound estimates of these effects (see Holmlund et al, 2011 for a discussion of the adoption strategy in relation to estimates from IV and Twin fixed effect strategies).

This study utilizes a unique dataset, constructed by matching several different administrative registers, and containing information on health and educational attainments for biological and adopting parents for about 22,000 individuals born between 1940 and 1967. The main outcome of interest is the health status of the adopted children as adults, as measured by adult mortality and hospitalization.

We first show estimates of the association between non-adopted parental characteristics and adult health. We find that adult mortality and hospitalization is strongly associated with parental health status and with parental education. When we focus on adoptees we find that length of the biological mothers' lifespan is strongly predictive of children's premature mortality. We also find that the adopting mothers' educational attainments are significantly associated with the adult mortality of their adoptive children. We find no significant association between the biological mothers' educational attainment and adopted children's adult health. We can thus reject the hypothesis that parental resources and environments post-birth have no effect on adult health status.

The rest of the paper is organized as follows. The next section describes the conceptual framework for identifying pre- and post-birth factors in the association between parental

education and child adult health. Section 3 presents the econometric model. Section 4 shows descriptive statistics and Section 5 the results. Finally, Section 6 concludes.

2. Background and Related Literature

A robust familial influence on premature mortality has been well established in the epidemiology literature (see review by Christensen and Vaupel, 1996). There are two competing channels that may drive this correlation – shared genetic and physical environment both in utero and after birth; or similar socio-economic environment and parental investments in children's health and wellbeing after birth. Twin studies have attempted to separate the effects of genetic as opposed to post-birth factors using differences between monozygotic and dizygotic twin pairs. A crucial assumption in these studies is that the post-birth environment does not differ across types of twin pairs. A series of studies using the Danish Adoption Register attempt to disentangle genetic from environmental factors affecting premature mortality by studying the competing effects of biological and adoptive parents' mortality patterns and health characteristics. The main takeaway is that genetic influences, as measured by biological parents' early mortality, have a strong influence on adoptees' premature deaths but the same variables constructed for adoptive parents' have much weaker effects or no influence at all (Sorensen et al, 1988; Petersen et al. 2005). The same set of authors report elevated probability of premature mortality among biological siblings of a prematurely deceased adopted child and no correlation with premature mortality of the adoptive siblings of the same individual (Petersen et al., 2008).

These studies provide solid evidence in favor of genetic and other pre-birth influences on human longevity that correlated across generations of biological parents and siblings. Research in economics has found that socio-economic circumstance at different stages of life – in utero, in infancy and childhood , and even in adult life – also have substantial effects on long-term health. As the socio-economic background of biological parents likely affects their own long-term health, it is hard to disentangle the relative contributions of these two channels. Further, to our knowledge there are no studies that examine the relative contribution of pre- vs post-birth exposure to socio-economic circumstance. There are several related strands of the economics literature.

2.1 Studies on the Relation between Parental Resources and Child Health

Case, Lubotsky and Paxson (2003) study the relation between parental resources and the prevalence of chronic conditions among their children as well as the health effect of these conditions. The study finds that the prevalence of chronic conditions is inversely related to parental income. However, more importantly, more than 60 percent of the protective health effect from income is attributed a less severe health effect among high-income children conditional on having a chronic condition. The study makes no attempts to separate out a causal effect of parental income from the possibly confounding effects of unobservables correlated with parental income.

Palme and Sandgren (2008) study the relation between parental income and child adult mortality. They find that there is an independent effect of parental income even after conditioning on own lifetime income. This result supports the "life course" or "critical period" hypothesis over the "pathway" hypothesis (see e.g. Marmot et al., 2001). The pathway hypothesis attributes adverse health outcomes for low SES children as adults to own inferior educational attainments due to intergenerational correlations in education.

An example of yet another hypothesis on the relation between parental resources and health is Case, Fertig and Paxson (2005) which shows how poor health during childhood and adolescence, associated with inferior parental resources, may transform into inferior school performance and human capital accumulation. In turn, suboptimal human capital potentially reinforces into an inferior health status in adult life.

Our main analysis relates to all these strands of the previous literature as it studies the adopting parents' resources to child adult health. We do not, however, attempt to separate out the association between parental resources and the prevalence of health conditions from the protective effect of these resources on the health effect of these resources. Nor do we attempt to distinguish between the direct effect of parental education on child adult health from the indirect one through the children's own educational attainments. Since the estimated effect of the adopting parents' resources in our study is independent of the potential association between parental educational attainments and genetic factors as well in utero health, our estimates are only partially comparable to those from the previous literature summarized above.

2.2 Studies on Pre-Natal and In Utero Health on Adult Health

Our study relates naturally to the very large epidemiology literature on heritability of diseases. We will, however, make no attempt to summarize this literature in this context. It also relates to a growing literature in economics on the relation between in utero health and later outcomes. This literature can, in turn, be divided into two main strands. First, there is a substantial body of research on the effects of health shocks or release of toxic substances injurious to the child's physical environment very early in life or in utero (see Almond and Currie, 2010, for an overview). Second, there is a separate literature on the effects of health measured at birth - such as birth weight, gestation lengths or APGAR scores - on later educational attainments and health.

An example of the first strand of the literature mentioned above is Almond (2004), which relates outcomes on educational attainments and labor earnings to exposure in utero to the Spanish influenza epidemic in the US in 1991. Almond et al. (2009) show that school performance of children exposed in utero to radioactive downfall from the Chernobyl nuclear power plant accident in Ukraine 1986 were significantly inferior compared to those not exposed. Finally, Nilsson (2008) shows that an experiment with more accessible alcohol strong beer in the Swedish city of Gothenburg resulted in increased alcohol consumption of pregnant mothers and worse labor market outcomes for the cohort in utero at the time of the experiment.

Adverse health status at birth, related to parental SES, is predictive for future health. Black et al. (2007) report that the twin with the lowest birth weight has worse outcomes on height, BMI, IQ, educational attainments and labor earnings. Oreopoulos et al. (2008) look at within sibling and twin pair differences in several birth outcomes and show that they are strongly predictive of several long- and medium term outcomes including high school completion and welfare take-up. Figlio et al. (2014) show that within sibling advantage in birth weight through various stages in the school system and at the labor market. The differences seem accentuated among children with highly educated parents suggesting complementarity between child and parental resources.

Currie (2011) summarizes previous research and discusses several sources for health inequality at birth. Less educated mothers are exposed to more environmental risks and have fewer resources to compensate for adverse health at birth. Our study relates to this strand of the literature as we are able to separate out the effects of pre-birth differences and investigate to what extent parental resources alone have an effect on long term health outcomes.

2.3 Studies on the Causal Effect of Parental Education on Child Health

Currie and Moretti (2003) use college openings as exogenous variation in educational attainments and find significant effect of educational attainments on child health measured by birth weight and gestational age. They also examine potential channels for this impact, including prenatal care, smoking, marriage, and fertility. McCrary and Royer (2011) uses age-at-school-entry policies to identify the effect of female education on fertility and infant health. They find heterogeneous effects on both outcomes. Finally, Lundborg et al. (2013) uses the staggered implementation of the Swedish comprehensive school reform as exogenous variation in maternal schooling. They find mixed results on health outcomes obtained at the enlistment for military service.

Although our study obviously has much in common with this literature it differs in one important aspect. We do not aim at estimating the causal effect of parental education on child health, since we not are able to identify the effect of education separate from unobservable parental characteristics potentially correlated with parental education. Our aim is more modest: to estimate the combined effect of parental education and all parental characteristics correlated with parental education on child long-term health separate from influences from pre-birth factors.

3. Conceptual Framework

Most of the literature on the relationship between parental socioeconomic status and health has focused on child health, where its determinants can be captured by a production function such as

$$H_1^c = f\left(CI_1^p, G_0; \varepsilon_1\right) \qquad (1)$$

where H_1^c is childhood (1) health for an individual in the child (c) generation, which is determined by inputs stemming from human and health capital investments of the parents (CI_1^p), genetic and prenatal environmental factors (G_0) and exogenous shocks during childhood (ε_1).

Among the challenges that are present in inferring the effect of CI_1^p on childhood health is how to control for genetic endowments, how to accurately measure parental inputs and to more generally understand the mechanisms of the "Parental SES gradient". Currie and

Goodman (2010), drawing on Grossman (2000), distinguishes three channels: budget constraint limiting investments in inputs, preferences/beliefs determining what parents choose to do with these inputs and the health status at birth. They argue that parent's inputs utilization is mostly associated with parent's education and that health status at birth is determined by genetic endowment and prenatal environment.

We can (given some important assumptions discussed below) use the adoption design to purge any association between child health and parental SES from genetic endowment and prenatal environmental factors. Given that we have enough proxies for the latent variable CI_1^p and that these proxies are predetermined (so that we avoid reverse causality bias), estimating the effect of CI_1^p on H_1^c, within the sample of adoptees, is then fairly straightforward.

Ultimately, however, we are interested in the "Parental SES gradient" for long-term health status of the individuals in the child generation. Let us then modify equation (1) to what can represent an adult health production function:

$$H_2^c = f\left(E_2, CI_1^p, G_0; \varepsilon_1\right), \qquad (2)$$

where $E_2 = g\left(AI_2^c, AI_2^p, CI_1^p, G_0; \varepsilon_2\right)$captures factors during adulthood that are relevant in determining H_2^c, for the individuals in the child generation: AI_2^c is the inputs stemming from the individual's own health protective investments during adulthood, and AI_2^p is the inputs stemming from the parental characteristics when the child has become an adult.

In what way do the modified production function in (2) complicates what we can estimate using the adoption design? Since adoptions place babies into different family environments, we cannot use the adoption design to estimate effects of AI_2^c or AI_2^p on the adult health status of the child (conditional on childhood environment and G_0). Hence, we need to focus on estimating the impact of CI_1^p on the adult health status of the child. An additional complication, related to data constraints, is that we can only observe health indicators of the parents when the child is an adult. Using such proxies for CI_1^p means that we need to assume that adult investments do not reinforce or compensates for any effects for CI_1^p: the investments during adulthood are determined entirely by CI_1^p , G_0 and some random shocks.

We will focus on a model for lifetime health

$$LH^c = f(CI_1^p, G_0; \varepsilon_1) \qquad (3)$$

where the aim is to estimating the importance of the latent variable CI_1^p, mainly consisting of human and health capital investments made by parents during childhood and adolescence, holding G, a latent variable consisting on those genetic and pre-natal environmental background factors that are relevant for LH^c, constant.

We start by running simple regressions for the population of families, and then restrict attention to those families where the child is adopted. We argue that the latter will estimate the reduced form impact of CI_1^p on LH^c conditional on genetic endowment and prenatal environment. Assuming that the adoptees are a random draw of the population of individuals (we get back to this issue below) we can infer how much of the association between LH^c and CI_1^p that is due to genetic endowment and prenatal environment and how much that is due to the importance of health and human capital inputs that accrue post-adoption(working either through more inputs or through the parental use of these inputs).

We will use two main indicators of CI_1^p: the health status/life expectance (also for different conditions and causes of death) of the adoptive parents and years (and type of) education of the adoptive parents. The importance of both these indicators is supported by the Grossman model. Such comprehensive information on the biological and adoptive parent also makes it possible to distinguish channels (also because schooling has been shown empirically to be the most important SES variable for own health).

4. Empirical Specifications

4.1 Intergenerational Transmission Models

Having settled for a modeling framework, where we aim at estimating the degree of transmission from parental health and human capital inputs of the parents for lifetime health of the next generation, we now discuss how we use data to estimate this relationship.

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

$$H_j^{bc} = \beta_0 + \beta_1 Y_j^{bp} + \upsilon_j^{bc} \qquad (4)$$

where H_j^{bc} denotes adult health status for the biological child, Y_j^{bp} denotes the biological parents' health status and/or educational attainment. Subscript *j* indexes the family in which the child is born and raised, superscripts *bc* and *bp* denote the biological child and parent,

respectively, and v_j^{bc} is the child-specific error term assumed to be uncorrelated with Y_j^{bp}. An estimate of β_1 measures the strength of the association between adult health of the child and health and 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

$$H_i^{ac} = \alpha_0 + \alpha_1 Y_j^{bp} + \alpha_2 Y_i^{ap} + v_i^{ac} \qquad (5)$$

where health and human capital inputs are indirectly transmitted from the biological parent *bp* or directly transmitted from the adoptive parent *ap* to the adopted 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 (see ..): 1) Adoptees are conditionally randomly assigned to adoptive families, 2) The adoption should have taken place close to birth to be able to separate pre and post birth effects accurately, 3) Post natal pre-adoption environment is uncorrelated with the genetic background and post adoption environment or the adoptive family background has no influence on the health of for the adopted child, and 4) the biological parents have no contact with the adopted child post adoption.

Under these four assumptions, we are able to separately estimate the association between adult health status and the observable pre- and post-birth characteristics *Y* by estimating equation (5) using data on adopted children and their biological and adoptive parents. Assuming that adoptees and non-adoptees are drawn from the same distribution we are also able to decompose an estimate of β_1 to separate entities of pre- and post-birth factors, captured by estimates of α_1 and α_2 interpretable for the population of children.

When can an estimate of α_1 be given a causal interpretation? Unless we can observe and control for all relevant indicators of the childhood adoption family environment, we cannot infer causal effects of these indicators: an estimate of α_2 does not only capture the importance of the parental characteristic Y_i^{ap} but also everything else in the adoption family

that is correlated with Y_i^{ap}. However, if we can observe enough relevant indicators of Y_i^{ap} so as to accurately capture the variation in post adoption family environment, we can infer the causal effect of broad family environment on adult health status (see Sacerdote, 2011, for a general discussion of this interpretation). As now we interpret the estimates as indicating the importance of transmission channels stemming either from pre- or post-birth environment.[3]

4.2 Siblings Models

An alternative approach to decompose pre- and post-birth factors using adoptees is to estimate sibling correlations. This is an approach rarely used in the economics literature.

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

$$H_j^{bc_1} = \beta_0 + \beta_1 H_j^{bc_2} + u_j^{bc_1} \quad (6)$$

where $H_j^{bc_1}$ denotes adult health status for one biological child and where $H_j^{bc_2}$ denotes adult health status for another biological child from the same family *j* where the children are born and raised, and $u_j^{bc_1}$ is the child-specific error term which, per definition, is assumed to be uncorrelated with $H_j^{bc_2}$. An estimate of β_1 measures the strength of the association between adult health of the two biological children and is therefore due to the combined effect of many different factors such as genetics, prenatal environment and environment during childhood and adolescence, that the siblings share. Under weak assumptions β_1 can be interpreted as the fraction of the overall variance in adult health status that is due to factors that siblings share (genes, prenatal environment, family environment etc).[4]

As we have data on the characteristics of both adopted and biological children of their biological and adoptive parents we can decompose the sibling correlation into the part which is due to pre-birth factors (nature and pre-natal environment) and post-birth factors (nurture) by estimating the following two models.

First we estimate a model using data on separated biological children

$$H_{ji}^{ac_1} = \alpha_0 + \alpha_1 H_{j/jk}^{bc_2} + u_{ji}^{ac_1} \quad (7)$$

[3] One could possibly use factor analysis methods to infer the impact of the latent variable "childhood environment" on adult health status.
[4] See Solon, 1999, for a discussion of siblings correlations and regressions.

where ac_1 denotes an adopted child born in family *j* and adopted and reared in family *i* and bc_2 denotes a biological sibling that is born and reared in family *j* (i.e., stayed with the biological mother) or born in family *j* and adopted and reared in family *k.* Hence these siblings are always born in the same family (i.e., they have the same biological mother) but grow up in different families. The parameter α_1 captures the importance of shared genes and pre-natal environment.

Second, we estimate a model using data on siblings that share the same rearing family but that are born in different families

$$H_{ji}^{ac_1} = \alpha_0 + \alpha_2 H_{i/ki}^{c_2} + u_i^{ac_1} \quad (8)$$

where ac_1 denotes an adopted child born in family *j* and adopted and reared in family *i* and c_2 denotes a sibling that is born and reared in family *i* (i.e., stayed with the biological mother) or born in family k and adopted and reared in family j. Hence these siblings are always reared in a family with at least two children and where at least one is adopted.

The key assumptions are similar as when we estimate intergenerational transmission models: 1) Adoptees are conditionally randomly assigned to adoptive families, 2) The adoption should have taken place close to birth to be able to separate pre and post birth effects accurately, 3) Post natal pre-adoption environment is uncorrelated with the genetic background and post adoption environment or the adoptive family background has no influence on the health of for the adopted child, and 4) the biological parents and have no contact with the adopted child post adoption, and *the biological siblings have no contact post adoption*.

Under these assumptions, we are able to separately estimate the association between adult health status among different sibling types and infer the importance of family background (β_1), the importance of genes and pre-natal environment (α_1) and the importance of post-birth factors (α_2). Assuming that adoptees and non-adoptees are drawn from the same distribution we are also able to decompose an estimate of β_1 to separate entities of pre- and post-birth factors, captured by estimates of α_1 and α_2 interpretable for the population of siblings.

5. Data

We use data from different national registers in Sweden. The sample is drawn from the Multigeneration Register (see Statistics Sweden 2009) and includes all males and females born in Sweden between 1940 and 1967. The Multigeneration Register includes information on whether or not a person was adopted as a child. It also contains information on the identity 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 samples used in this study - the adoptees and, as a comparison, the non-adoptees – at different stages of the sample selection process. There are in total 64,825 adoptees in our data. Of these, about 30,000 were adopted by only one parent, in most cases the husband of the child's biological mother. We have excluded these children from the analysis. For 22,589 of the remaining children we have information on both the identity and educational attainments of the biological mother as well as information on the educational attainments of both the adopting parents. This sample is used for the main empirical analysis in this paper.

For about half of this sample, we have, in addition, information on the identity of the biological father, his educational attainments, whether or not he is still alive and, if he has passed away, date of death. The resulting, substantially small sample is used for sensitivity analysis.

Table 1. Sample restrictions and summary statistics

Sample restrictions:

Born in Sweden 1940-67	Non-adoptees	Adoptees
Non-adopted	3,059,184	
Adopted by at least one parent		64,825
Adopted by both parents		33,286
Not adopted by own parents		33,240
Not died or emigrated first month	3,051,341	33,238
Biological mother is identified	3,006,629	24,202
Parents' education is known	2,911,220	22,589
Biological father is identified	2,829,632	11,616

Summary Statistics:

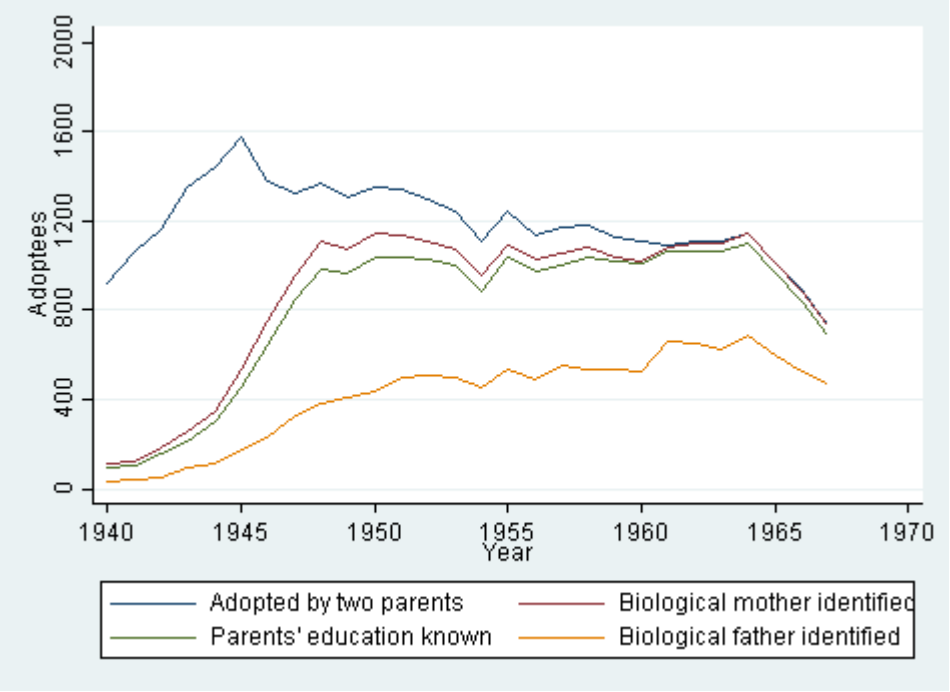
	Non-adoptees		Non-adoptees (weighted by cohort)		Adoptees	
			Children			
Female	0.488	(0.500)	0.488	(0.500)	0.476	(0.499)

Dead by April 2013	0.072	(0.258)	0.059	(0.235)	0.078	(0.268)
			Biological parents			
Dead by April 2013, mother	0.568	(0.495)	0.433	(0.496)	0.519	(0.500)
Age at death, mother	77.538	(12.191)	74.203	(12.245)	71.013	(13.421)
Years of schooling, mother	8.235	(2.140)	8.439	(2.272)	8.113	(1.804)
Dead by April 2013, father	0.741	(0.438)	0.642	(0.480)	0.683	(0.465)
Age at death, father	74.508	(11.822)	72.479	(12.061)	70.456	(11.591)
Years of schooling, father	8.781	(2.774)	9.078	(2.865)	8.374	(2.161)
			Adoptive parents			
Dead by April 2013, mother					0.671	(0.470)
Age at death, mother					79.351	(11.362)
Years of schooling, mother					8.113	(1.804)
Dead by April 2013, father					0.801	(0.399)
Age at death, father					76.907	(10.741)
Years of schooling, father					9.261	(3.094)
Observations	2,829,632		2,829,632		22,589	

Note: standard deviations in parentheses

Figure 1 shows the number of adoptees in the sample by cohort of birth. The figure contains four graphs. The upper graph shows the number of children adopted by two parents. The increase between 1940 and 1945 reflects the increase in the overall fertility rate in Sweden. The most likely reason behind the decline between 1945 and 1967 is increased contraceptive use and more liberal rules for abortions. The increase in the number of adopted children for whom we can identify the biological mother increases during the 1940's.

Figure 1. Swedish domestic adoptions by birth year



Data on the outcome variables - date and ICD codes for cause of death and dates and ICD codes for causes of hospital stays – are obtained from the national Cause of Death Register (see Statistics Sweden, 2009) and the national In-patient Register (see Statistics Sweden, 2009). The Cause of Death Register records on all deaths in Sweden from 1952 and onwards the underlying cause ICD codes for the underlying cause of death. The national In-patient register includes dates for all hospital stays on Swedish hospitals. This register has a national coverage starting from 1987. Since the first birth cohort included in our data were born in 1940, we observe all their hospital stays since age 47. The In-patient Register includes ICD codes for the maximum of eight different medical causes of each hospital stay.

The bottom part of Table 1 contains sample means and standard deviations (within parentheses) for the sample of adoptees and non-adoptees. The first panel shows information on the children of the two samples. The second panel shows descriptive statistics for the biological parents. It can be seen that the biological parents of the adopted children have on average slightly less education and shorter life expectancy compared to the non-adopted. The third panel shows descriptives of the adopting parents. It can be seen that the adopting fathers

have on average almost a year longer education than the biological fathers of the adopted children.

6. Results

6.1. The Association between Parental Life Expectancy and Child Health

In the first two columns of table 2 we present basic correlations between parental and child mortality in the Swedish population of children born between 1940 and 1967 who resided with at least one biological parent after birth. We use two sets of controls for parental health – the actual age at death for the parent and a dummy indicating whether the parent was still alive in 2013 (parents still alive in 2013 are coded as having died at the age they were in 2013); and the predicted age at death. The predicted age at death is gender-specific and imputed based on the number of overnight visits to the hospitals we observe in the inpatient register, the type of diagnoses that triggered these hospitalizations, and the birth cohort of the individual. More details on the imputation are available in the Appendix.

The results indicate that an extra year of life for the biological parent increases the probability of survival for the biological child by about 1 percentage point or a 14 percent reduction. The longevity of the adoptive parents does not seem to have significant effects on the life expectancy of adoptive children. This suggests that the physical fitness of the biological parents affects genetic and early-life (in utero) conditions that in turn influence the long-term health of children even if these children grow up in a different family. All results shown in Table 2 are obtained from Cox proportional hazard models where we also have included birth cohort fixed effects as well as an indicator for gender.

Table 2. The association between biological as well as adopting parents' mortality and child mortality. Samples of non-adopted children, adopted children with both parents known and adopted parents including children with unknown biological fathers. Cox Proportional Hazard Models.

	Non-adoptees		*Adoptees- Bio father known*		*Adoptees – Full sample*	
VARIABLES	(1) Actual	(2) Predicted	(3) Actual	(4) Predicted	(5) Actual	(6) Predicted
Age at death, Bio Mother	0.9916***	0.9887***	0.9832***	0.9869***	0.9870***	0.9880***
	(0.0002)	(0.0002)	(0.0038)	(0.0029)	(0.0027)	(0.0020)
Age at death, Bio Father	0.9929***	0.9912***	0.9877***	0.9854***		
	(0.0002)	(0.0002)	(0.0037)	(0.0030)		
Age at death, Ad Mother			1.0002	0.9986	0.9990	0.9985
			(0.0041)	(0.0035)	(0.0026)	(0.0023)
Age at death, Ad Father			0.9991	0.9998	1.0011	0.9998
			(0.0037)	(0.0035)	(0.0025)	(0.0023)
Alive 2013, Bio Mother	0.7751***		0.9341		0.8757**	
	(0.0050)		(0.0772)		(0.0481)	
Alive 2013, Bio Father	0.7984***		0.7615***			
	(0.0065)		(0.0764)			
Alive 2013, Ad Mother			0.8922		0.9674	
			(0.0927)		(0.0695)	
Alive 2013, Ad Father			1.0095		0.8727	
			(0.1308)		(0.0800)	
P-value joint significance						
Adoptive parents			0.8636	0.9219	0.5739	0.7928
Biological parents	0.0000	0.0000	0.0000	0.0000	0.0000	
Observations	2,829,632	2,829,632	11,616	11,616	22,589	22,589

All models include cohort of birth dummies for parents and children and gender dummies.
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

In Table 3 we consider a different health outcome – the probability of an overnight inpatient hospital stay between ages 43 and 47. The age restriction is driven by the fact that we observe hospitalizations for the oldest cohort (1940) first at the age of 47 and we observe hospitalizations for the youngest cohort (1967) last at the age of 47. To save space, we only report results from specifications using the predicted age at death for the parents. In column (1) we consider all children living with their biological parents and find that the probability of having been hospitalized decreases by about 0.1 percent for each extra year of life for the biological mother and after. For adoptees the same correlation persists across biological mothers and children; however we also find a significant negative effect of the longevity of the adoptive father and a marginally significant effect of the longevity of the adoptive mother.

Table 3. The association between biological and adopting parents' mortality and child hospitalization age 43-47. Samples of non-adopted children, adopted children with both parents known and adopted parents including children with unknown biological fathers. OLS regressions.

VARIABLES	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Full sample
Age at death, Bio Mother	-0.0011***	-0.0016***	-0.0014***
	(0.0000)	(0.0004)	(0.0003)
Age at death, Ad Mother		0.0000	-0.0008*
		(0.0005)	(0.0005)
Age at death, Ad Father		-0.0009**	-0.0009**
		(0.0004)	(0.0004)
Age at death, Bio Father	-0.0009***	-0.0004	
	(0.0000)	(0.0004)	
P-value joint significance			
Adoptive parents		0.0727	0.0315
Biological parents	0.0000	0.0007	
Observations	2,803,091	11,577	22,444
R-squared	0.0027	0.0085	0.0042

All models include cohort of birth dummies for parents and children and gender dummies.
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

The mother's health is reflected in the birth weights of her children (see e.g. Currie, 2011). In Table 4 we show results where we use the probability of low birth weight (<2500gr) and birth weight for the first born child as outcome variables and health indicators of the mothers. We find small but highly significant positive effects of the longevity of the biological grandmother even for the children of individuals who have been given up for adoption. The magnitude of the effect on the probability of low birth weight is about 0.75- 1.5 percent reduction given the mean low birth weight of 4%. There are no significant effects of the longevity of the adoptive mother or adoptive father, which indicates that any habits and life choices that might affect adoptive parents' longevity do not carry over to influence the health status at birth of their grand-children.

Table 4. The association between biological as well as adopting parents' mortality and prevalence of low birth weight (<2,500 gram) and birth weight, respectively among firstborn grandchildren. Samples of non-adopted children, adopted children with both parents known and adopted parents including children with unknown biological fathers. OLS regressions.

	Low birth weight (<2,500)			*Birth weight*		
VARIABLES	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Full sample	(4) Non-adoptees	(5) Adoptees - Bio father known	(6) Adoptees- Full sample
Age at death, Bio Mother	-0.0002***	-0.0006*	-0.0003	0.6303***	1.6464**	1.2576**
	(0.0000)	(0.0003)	(0.0003)	(0.0618)	(0.7730)	(0.5931)
Age at death, Bio Father	-0.0003***	-0.0004		1.2032***	0.1897	
	(0.0000)	(0.0004)		(0.0574)	(0.8548)	
Age at death, Ad Mother		-0.0000	-0.0000		1.0415	1.1496
		(0.0004)	(0.0003)		(0.9541)	(0.7038)
Age at death, Ad Father		0.0006	0.0004		-1.1845	-0.5247
		(0.0004)	(0.0003)		(0.9405)	(0.6914)
P-value joint significance						
Adoptive parents		0.3023	0.3016		0.2395	0.1996
Biological parents	0.0000	0.1325		0.0000	0.0965	
Observations	789,924	3,666	6,742	789,924	3,666	6,742
R-squared	0.0005	0.0106	0.0107	0.0015	0.0158	0.0102

All models include cohort of birth dummies for parents and children and gender dummies.
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

6.2. The Association between Parental Education and Child Health

Children's health is strongly related to family socio-economic status. There are at least two channels that determine this link – family SES and in particular maternal education positively affects maternal investments in-utero; family SES affects children's health after birth by providing a safer nurturing environment and better investments in the children's own human capital formation. The question we investigate in the next set of tables relates to the relative effects of these two channels in determining the child's long-term health outcomes.

In the first column of Table 5 we report the correlation between parental years of education and the longevity of biological children. An additional year of maternal or paternal education increases the survival rate for own children by 2.5% - almost a 33 percent reduction in mortality estimated at the mean mortality rate in this sample. Comparing these effects to

those reported in Table 2 above, we note that an extra year of education has more than twice the effect of an extra year of life for the biological parents. When we consider the set of adoptees in columns (2) and (3) a different picture emerges. For these children the education of the biological parents is less significant, while the education of the adoptive mother in particular plays a much more important role. We see that an extra year of education for the biological mother decreases the mortality probability by at least as much as an extra year of education for the biological mother in the full sample of biological children.

Table 5. The association between biological as well as adopting parents' years of schooling and child mortality. Samples of non-adopted children, adopted children with both parents known and adopted parents including children with unknown biological fathers. Cox Proportional Hazard Models.

VARIABLES	(1) Non-adoptees	(2) Adoptees - Bio Father Known	(3) Adoptees - Full sample
Years of education, Bio Mother	0.9768***	1.0407*	0.9886
	(0.0015)	(0.0236)	(0.0145)
Years of education, Bio Father	0.9749***	0.9794	
	(0.0010)	(0.0173)	
Years of education, Ad Mother		0.9612*	0.9694**
		(0.0202)	(0.0134)
Years of education, Ad Father		0.9861	0.9993
		(0.0153)	(0.0099)
P-values joint significance			
Adoptive parents		0.0196	0.0378
Biological parents	0.0000	0.1488	
Observations	2,829,632	11,616	22,589

All models include cohort of birth dummies for parents and children and gender dummies.
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table A5 in the Appendix presents the corresponding results when we consider the probability of hospitalization between ages 43 and 47. The findings are qualitatively similar even though the statistical significance of the effects, particularly those associated with the adoptive parents, decreases.

The educational attainment of the biological grandparents has an economically small but significant effect on the health endowment at birth of grandchildren as measured by the probability of low birth weight and the actual birth weight of those children. Table 6 reports the results from the subset of female biological children and their own first children. As we saw in Table 5, an extra year of education has a larger effect of an extra year of longevity for

biological grandparents, reducing the probability of a low-birth-weight biological grandchild by 1.75 percent and increasing actual birth weight by 5 grams (3 grams for the biological grandfather).

The corresponding coefficient for the biological mother in the sample of adoptees is not statistically significant and has the wrong sign. The effect of an extra year of education for the adoptive mother is negative and of the same magnitude as the coefficient on the biological mother's years of education in column (1), however it is imprecisely estimated. We get similar results for the years of education of the adoptive father and a positive and significant 6 gram increase in birth weight associated with an extra year of education for the adoptive father in the full sample of adoptees in column (6). The p-values of the joint test of significance for the coefficients on the adoptive parents' years of educational attainment is 0.136 for the probability of low birth weight and 0.106 for the actual birth weight as an outcome.

Taken together these results suggest that the education of the adoptive parents positively affects the health endowment at birth of the children of their adoptive children, while the education of the biological parents of adoptive children has very little effect on the health of these children's children.

Table 6. The association between biological as well as adopting parents' years of schooling and prevalence of low birth weight (<2,500 gram) and actual birth weight, respectively among firstborn grandchildren. Samples of non-adopted children, adopted children with both parents known and adopted parents including children with unknown biological fathers. OLS regressions.

	Low birth weight (<2,500)			*Birth weight*		
VARIABLES	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Full sample	(4) Non-adoptees	(5) Adoptees - Bio father known	(6) Adoptees- Full sample
Years of education, Bio Mother	-0.0007***	0.0019	0.0016	4.0776***	6.5841	5.8110
	(0.0001)	(0.0024)	(0.0017)	(0.3351)	(5.9684)	(4.0687)
Years of education, Bio Father	-0.0008***	-0.0008		2.8049***	-4.3860	
	(0.0001)	(0.0021)		(0.2658)	(4.9499)	
Years of education, Ad Mother		-0.0009	-0.0006		1.8771	-3.3490
		(0.0020)	(0.0014)		(4.8358)	(3.4136)
Years of education, Ad Father		-0.0013	-0.0016		5.0646	5.7606**
		(0.0016)	(0.0011)		(3.7599)	(2.7234)
P-value joint significance						
Adoptive parents		0.4093	0.1361		0.1894	0.1061
Biological parents	0.000	0.6949		0.0000	0.4152	
Observations	789,924	3,666	6,742	789,924	3,666	6,742
R-squared	0.0004	0.0094	0.0107	0.0015	0.0153	0.0102

All models include cohort of birth dummies for parents and children and gender dummies.
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

6.3. Conditional Associations

There are several possible mechanisms behind the associations between the health of the biological parents and child mortality as well as the relation between adopting parents' educational attainment and child mortality. From previous research we know that there is a strong association between education and health and also between parental and child education. This means that the result on adopting parents' education on child health can come through the parental parent's health status, or, indirectly, through children's educational attainments (the so called pathway hypothesis), rather than directly through influences during childhood and adolescence.

One way to study these possibilities is to include independent variable for parental and child education along with the ones measuring parental health status. An obvious problem with this approach is that it introduces obvious bias or inconsistencies by including endogenous regressors (mediating factors) into the model. Being aware of this, such model may nevertheless give us important information on the possible mechanisms behind the results obtained in the previous two sub-sections.

The first two columns in Table 7 show the conditional associations for the sample of non-adoptees. The first column shows that the inverse effect of parental education is robust for the inclusion of controls for parental health. An interpretation of this result is that there is an independent effect of parental education on child health. However, when we also include an independent variable for the child's educational attainment, we see that see that the effect of parental education is wiped out and even change sign. This result supports the pathway hypothesis that there is no direct effect of parental SES on child adult health, but only through the children's educational attainment.

Columns 3 through 6 show the corresponding results for adoptees. The first two of these columns the restricted sample where we have information on both biological parents and the latter two for the extended sample where we also include observations where we have information on the biological mother only. The results in columns 5 and 6 differ somewhat for those obtained from the sample of non-adoptees. Although, the effect of adopted parents education still remains significant when we add controls for adopting mother's health status, the effect is robust also for adding the variable child's education, i.e., the results suggest a direct effect from adopting parent's education.

Table 7. The association between child mortality and biological as well as adopting parents' mortality and years of schooling. Samples of non-adopted children, adopted children with both parents known and adopted parents including children with unknown biological fathers. Cox Proportional Hazard Models.

VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
	Non-adoptees		Adoptees - Bio father known		Adoptees - Full sample	
Years of education, Bio Mother	0.9809***	1.0009	1.0542**	1.0740***	0.9958	1.0146
	(0.0015)	(0.0019)	(0.0239)	(0.0287)	(0.0147)	(0.0178)
Years of education, Bio Father	0.9797***	1.0056***	0.9909	1.0207		
	(0.0010)	(0.0014)	(0.0175)	(0.0205)		
Years of education, Ad Mother			0.9646*	0.9623	0.9717**	0.9623**
			(0.0203)	(0.0240)	(0.0135)	(0.0166)
Years of education, Ad Father			0.9904	1.0278	1.0014	1.0337***
			(0.0154)	(0.0185)	(0.0100)	(0.0126)
Age at death, Bio Mother	0.9892***	0.9882***	0.9866***	0.9885***	0.9883***	0.9887***
	(0.0002)	(0.0002)	(0.0029)	(0.0033)	(0.0020)	(0.0024)
Age at death, Bio Father	0.9917***	0.9886***	0.9857***	0.9875***		
	(0.0002)	(0.0002)	(0.0030)	(0.0035)		
Age at death, Ad Mother			0.9990	0.9981	0.9988	0.9990
			(0.0035)	(0.0041)	(0.0023)	(0.0027)
Age at death, Ad Father			1.0002	0.9996	0.9999	0.9988
			(0.0035)	(0.0041)	(0.0023)	(0.0027)
Years of education, Child		0.9145***		0.8725***		0.8898***
		(0.0010)		(0.0171)		(0.0113)
P-value joint significance						
Adoptive parents			0.2126	0.4898	0.2338	0.0649
Biological parents	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
Observations	2,829,632	2,689,178	11,616	11,116	22,589	21,533

All models include cohort of birth dummies for parents and children and gender dummies.
Robust se form in parentheses
*** p<0.01, ** p<0.05, * p<0.1

In the Appendix we show results corresponding to those shown in Table 7 for male and female adoptees separately. As expected, the precision in these estimates are somewhat inferior compared to the pooled sample. The result on the mortality of the biological mother remains significant for both males and females sub-samples, while the precision is only sufficient in the female sample to make the result on the adopting mothers' educational attainments significant.

The Appendix also includes tables (Table A6 through A8) where we instead of using child mortality as a dependent variable use hospitalization, birth weight of grandchildren and incidence of low birth weight among grandchildren.

6.4. The Association in Health between siblings

In tables X.a-e we estimate regression models using data on siblings ("sibling correlations"). We start with models using the population of siblings, where the outcome of the first child is regressed on the outcome of the second child. We show regression estimates for the following outcomes: hospitalization at ages 43-47, dead or not by April 2013 and (for purpose of comparison) years of schooling.

Table 8a. Non-adoptees, first and second child.

VARIABLES	(1) Hospitalization age 43-47 - first	(2) Dead - first	(3) Years of schooling - first
Hospitalization age 43-47 – second	0.0239***		
	(0.00162)		
Dead – second		0.0400***	
		(0.00161)	
Years of schooling - second			0.440***
			(0.00115)
Constant	0.112***	0.146***	5.803***
	(0.0191)	(0.0145)	(0.116)
Observations	858,586	858,586	793,590
R-squared	0.002	0.031	0.194

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

The results indicate small but statistically significant associations between siblings for the health measures whereas the association in years of schooling is much higher. This suggests either that adult health/life expectancy is to a very high degree determined by factors that siblings do not share or that there is a lot of noise in these measures. If a sibling has died, the probability of dying increases by 4 percentage points.

Next we estimate model (7) using data on separated biological children. Table 8b use data on siblings where the first child that has been given up for adoption and the first child that stayed with biological mother, and Table 8c use data on siblings where both have been given up for adoption but to different biological mothers. The purpose of these estimations is to find out how much of the sibling correlations in Table X.a that is due to "nature" and prenatal environment.

Table 8b. Separated biological children - first child that has been given up for adoption and first child that stayed with biological mother.

VARIABLES	(1) Hospitalization age 43-47	(2) Dead	(3) Years of schooling – adopted
Hospitalization age 43-47 – adopted	0.0132*		
	(0.00788)		
Dead – adopted		0.0179*	
		(0.0100)	
Years of schooling – adopted			0.133***
			(0.00871)
Constant	0.335**	0.218***	8.388***
	(0.141)	(0.0532)	(0.379)
Observations	14,495	14,495	13,229
R-squared	0.005	0.038	0.071

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 8c. Separated biological children - both have been given up for adoption but to different biological mothers. Cox proprtional hazard models

VARIABLES	(1) Hospitalization age 43-47 - first	(2) Dead - first	(3) Years of schooling - first
Hospitalization age 43-47 – second	0.0176		
	(0.0225)		
Dead – second		0.0293	
		(0.0312)	
Years of schooling - second			0.105***
			(0.0306)
Constant	0.662***	0	9.303***
	(0.00613)	(6.46e-07)	(0.318)
Observations	1,612	1,612	1,501
R-squared	0.026	0.076	0.053

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

In table 8c we see that the health estimate are always statistically significant and of about half the size as the estimates for the population of siblings (in Table X.a). Hence, although these biological siblings have been separated from their biological parenst at a very early age and

(most likely) have never met each other, they are still more likely to end up having similar health. The estimates in Table 8c, although statistically insignificant, confirm this picture.

Next we estimate model (8) using data on biologically unrelated children that have experienced the same rearing environment. Table 8d use data on rearing siblings where one is adopted and the other is the biological child of the adoptive mother. Table 8e instead use rearing siblings with the same upbringing mother but different biological mothers. The purpose of these estimations is to find out how much of the sibling correlations in Table 8a that is due to "nurture".

Table 8d. Siblings sharing same upbringing environment, one is adopted and one is the biological child of an adoptive mother.

VARIABLES	(1) Hospitalization age 43-47	(2) Dead	(3) Years of schooling
Hospitalization age 43-47 - adopted	0.00126		
	(0.00803)		
Dead - adopted		-0.0187	
		(0.0157)	
Years of schooling - adopted			0.254***
			(0.0222)
Constant	0.111	0.108	8.379***
	(0.0766)	(0.103)	(1.040)
Observations	3,570	3,570	3,291
R-squared	0.024	0.050	0.092

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 8e. Siblings with the same upbringing mother but different biological mothers.

VARIABLES	(1) Hospitalization age 43-47 - first	(2) Dead - first	(3) Years of schooling - first
Hospitalization age 43-47 – second	-0.00154		
	(0.0109)		
Dead - second		-0.00765	
		(0.0125)	
Years of schooling - second			0.185***
			(0.0144)
Constant	0.285*	0.119**	8.576***
	(0.172)	(0.0480)	(0.529)
Observations	5,659	5,659	5,199
R-squared	0.012	0.036	0.049

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

The results indicate small and always statistically insignificant associations between siblings for the health measures. Hence, although these rearing siblings have been experienced much of the same family environment, they are not more likely to have similar health.

Note that for years of schooling, the sibling estimates are always statistically significant and large. The pattern is different compared to health through, as "nurture" seems to be more important than "nature".

We conclude that the importance of family background for inequality in health is mainly due to nature (or prenatal environment). Hence, the exercise using siblings confirm the conclusions for health and mortality using parents and children.

6.4. Sensitivity analyses

A potential threat to our identification strategy may be that the adoptee might still maintain significant contact with the biological parents (mother) even after adoption and thus the characteristics of the biological parents would have effects above and beyond the in-utero period. A related concern is that the biological parents may have pre-adoption contact with

the adopting parents and thereby are able to intervene in the adoption process. As a sensitivity analysis we have restricted the sample to only include the adopted children who move from their municipality of birth when they were adopted. The results from this exercise are presented in Table 9. As is apparent from these results, this additional sample restriction has a very small effect on the results.

Table 9. Mortality among adoptees - Cox Proportional Hazard Models; Biological and adoptive mothers living in different municipalities in 1960

	(1)	(2)	(3)	(4)
Age at death, Bio Mother	0.9878***		0.9879***	0.9880***
	(0.9821 - 0.9935)		(0.9822 - 0.9936)	(0.9823 - 0.9937)
Age at death, Ad Mother	0.9967			0.9969
	(0.9914 - 1.0021)			(0.9916 - 1.0023)
Age at death, Ad Father	1.0009			1.0008
	(0.9957 - 1.0062)			(0.9956 - 1.0061)
Alive 2013, Bio Mother	0.8618**		0.8720**	0.8717**
	(0.7673 - 0.9680)		(0.7757 - 0.9803)	(0.7754 - 0.9799)
Alive 2013, Ad Mother	1.0393			1.0465
	(0.8937 - 1.2087)			(0.8998 - 1.2171)
Alive 2013, Ad Father	0.8994			0.9068
	(0.7433 - 1.0882)			(0.7494 - 1.0971)
Years of education, Ad Father		1.0036	1.0052	1.0051
		(0.9826 - 1.0250)	(0.9842 - 1.0267)	(0.9840 - 1.0267)
Years of education, Bio Mother		0.9825	0.9892	0.9893
		(0.9523 - 1.0136)	(0.9588 - 1.0206)	(0.9589 - 1.0207)
Years of education, Ad Mother		0.9690**	0.9701**	0.9710**
		(0.9410 - 0.9977)	(0.9422 - 0.9989)	(0.9430 - 0.9998)
Observations	19,389	19,389	19,389	19,389

All models include cohort of birth dummies for parents and children and gender dummies. Robust CI in parentheses; *** p<0.01, ** p<0.05, * p<0.1

In a second sensitivity analysis we restrict the sample of adoptees to first-born children only. The motivation for this analysis is to exclude adoptions where the child was given up because of poverty, inability of accommodate a large family, or parental illness. Table 10 presents the estimates. Again, these additional restrictions have very small effects on the results. In fact, the main results came out even stronger in this group of adoptees.

Table 10: Mortality among adoptees. Cox proportional hazard models; restricting the sample to first born children given up for adoption.

	(1)	(2)	(3)	(4)
Age at death, Bio Mother	0.9859***		0.9858***	0.9861***
	(0.9779 - 0.9939)		(0.9778 - 0.9939)	(0.9781 - 0.9942)
Age at death, Ad Mother	0.9968			0.9972
	(0.9897 - 1.0039)			(0.9901 - 1.0044)
Age at death, Ad Father	0.9981			0.9980
	(0.9914 - 1.0049)			(0.9912 - 1.0048)
Alive 2013, Bio Mother	0.8523**		0.8650**	0.8658**
	(0.7399 - 0.9818)		(0.7505 - 0.9969)	(0.7511 - 0.9981)
Alive 2013, Ad Mother	1.0468			1.0647
	(0.8554 - 1.2810)			(0.8701 - 1.3028)
Alive 2013, Ad Father	0.7454**			0.7535**
	(0.5661 - 0.9815)			(0.5721 - 0.9924)
Years of education, Ad Father		1.0075	1.0091	1.0108
		(0.9801 - 1.0357)	(0.9816 - 1.0373)	(0.9833 - 1.0390)
Years of education, Bio Mother		0.9857	0.9909	0.9914
		(0.9495 - 1.0232)	(0.9546 - 1.0286)	(0.9551 - 1.0291)
Years of education, Ad Mother		0.9416***	0.9435***	0.9436***
		(0.9063 - 0.9781)	(0.9084 - 0.9799)	(0.9086 - 0.9800)
Observations	11,097	11,097	11,097	11,097

All models include cohort of birth dummies for parents and children and gender dummies.
Robust confidence intervals in parentheses
*** p<0.01, ** p<0.05, * p<0.1

7. Conclusions

To understand how health is related to parental resources has obvious policy implications and is important for understanding how health inequalities are formed and passed on between generations. In this paper use a large sample of adoptees containing detailed information on educational attainments and health status of both biological and adopting parents to separate the effects of pre- and post-birth parental characteristics.

We find that health status of the biological mother is important for health status of the adopted children. We also find that the educational attainment of the adopting mother has a significant effect on child adult mortality. This result is robust in a smaller sample where we also have information on characteristics of the biological fathers and where we restrict the sample to those who were adopted to live in another municipality than they were born in.

The result on the adopting mothers' educational attainments does not necessarily tell us that there is a causal effect of parental education on child health, since parental education

is not in any sense exogenous and the result may originate from a “third factor” correlated with the educational attainments of the adopting mother. However, given the assumption that the genetic set up of the biological parents are adopting ones are independent, it does tell us that we can reject the hypothesis that environments during upbringing and parental resources do not affect mortality in adult life.

References

Almond, Douglas (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, Douglas, Lena Edlund, and Mårten Palme (2009) "Chernobyl's subclinical legacy: prenatal exposure to radioactive fallout and school outcomes in Sweden" *Quarterly Journal of Economics* 124(4), 1729-1772.

Björklund, Anders, Markus Jäntti, and Gary Solon, "Influences of Nature and Nurture on Earnings Variation: A Report on a Study of Sibling Types in Sweden," in Samuel Bowles, Herbert Gintis, and Melissa Osborne, eds., *Unequal Chances: Family Background and Economic Success* (New York: Russell Sage Foundation, 2005).

Björklund, Anders, Mikael Lindahl, and Erik Plug (2006) "The origins of intergenerational associations: Lessons from Swedish adoption data" *The Quarterly Journal of Economics*, 999-1028.

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

Black, S. E. and P. Devereux (2011) "Recent Developments in the Study of Intergenerational Mobility"

Case, A., A. Fertig and C. Paxson (2003) "From Cradle to Grave? The Lasting Impact of Childhood Health and Circumstance" NBER Working Paper 9788.

Case, A., D. Lubotsky and C. Paxson (2002) "Economic Status and Health in Childhood: The Origins of the Gradient" *The American Economic Review* 92 (5), 1308-1334.

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

Christensen, K. and J.W. Vaupel (1996) "Determinants of Longevity: Genetic, Environmental and Medical Factors" Journal of Internal Medicine, Vol. 240 Issue 6, pp. 333-341

Currie, J. (2000) "Child health in developed countries", in: A.J. Culyer and J.P. Newhouse, eds., Handbook of Health Economics (Elsevier, Amsterdam) Chapter 19.

Currie, Janet. *Inequality at birth: Some causes and consequences*. No. w16798. National Bureau of Economic Research, 2011.

Currie, Janet, and Goodman, Joshua. 2010. Parental Socioeconomic Status, Child Health, and Human Capital, in International Encyclopedia of Education, 2:253-259.

Currie, Janet, and Enrico Moretti. *Mother's education and the intergenerational transmission of human capital: evidence from college openings and longitudinal data*. No. w9360. National Bureau of Economic Research, 2002.

Deaton, A. (2003) "Health, Inequality and Economic Development" *Journal of Economic Literature* 41, 113-158.

Figlio, David, *Jonathan Guryan, Jeffrey Roth* (2014) "The Effects of Poor Neonatal Health on Children's Cognitive Development" *The American Economic Review* 104(12), 3921-55.

Grossman, M. (2000), "The Human Capital Model", in: A.J. Culyer and J.P. Newhouse, eds., Handbook of Health Economics (Elsevier, Amsterdam) Chapter 7.

Holmlund, Helena, Mikael Lindahl, and Erik Plug. "The causal effect of parents' schooling on children's schooling: A comparison of estimation methods." *Journal of Economic Literature* 49.3 (2011): 615-651.

Lindeboom, Maarten, Ana Llena-Nozal, and Bas van Der Klaauw. "Parental education and child health: Evidence from a schooling reform." *Journal of Health Economics* 28.1 (2009): 109-131.

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

Lindquist, M., and R. Hjalrmarsson, "The Origins of Intergenerational Associations in Crime: Lessons from Swedish Adoption Data", *Labour Economics* 20, January 2013, 68-81.

McCrary, Justin, and Heather Royer. "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 (2011): 158-195.

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

Palme, Mårten, and Sofia Sandgren. "Parental income, lifetime income, and mortality." *Journal of the European Economic Association* 6.4 (2008): 890-911.

Petersen, Liselotte, Per Kragh Andersen and Thorkild Sorensen (2005) "Premature Death of Adult Adoptees: Analyses of a Case-Cohort Sample" Genetic Epidemiology 28; 376-382

Petersen, Liselotte, Per Kragh Andersen and Thorkild Sorensen (2008) "Genetic and Environmental Effects on Mortality before Age 70 Years" Epidemiology, Vol. 19, No 3, 472-476.

Plug, Erik, and Wim Vijverberg, "Schooling, Family Background, and Adoption: Is It Nature or Is It Nurture?" *Journal of Political Economy,* CXI (2003), 611–641.

Rietveld CA, S Medland, J Derringer, Authors from the Social Science Genetic Association Consortium, PM Visscher, DJ Benjamin, D Cesarini & PD Koellinger (2013). "GWAS of 126,559 Individuals Identifies Genetic Variants Associated with Educational Attainment." *Science,* 340: 1467–1471.

Sacerdote, B. (2011), " Nature and Nurture Effects On Children's Outcomes : What Have We Learned From Studies of Twins And Adoptees?", in: Jess Benhabib, Alberto Bisin and Matthew O. Jackson, eds., Handbook of Social Economics (Elsevier, Amsterdam) Chapter 1.

Smith, J. (1999) "Healthy Bodies and Thick Wallets: The Dual Relationship between Health and Economic Status" *Journal of Economic Perspectives* 13, 145-166.

Solon, Gary, "Intergenerational Mobility in the Labor Market," in Orley Ashenfelter and David Card, eds., *Handbook of Labor Economics,* Volume III (Amsterdam: Elsevier, 1999).

Sorensen, Thorkild, Gert Nielsen, Per Kragh Andersen and Thomas Teasdale (1988) "Genetic and Environmental Influences on Premature Death in Adult Adoptees" NEJM, Vol. 318 No. 12

Appendix

Table A1: Mortality Among Female Non-Adoptees - Cox Proportional Hazard Models

VARIABLES	(1) Women	(2) Women	(3) Women
Age at death, Bio Mother	0.9907***		0.9909***
	(0.9901 - 0.9914)		(0.9902 - 0.9916)
Age at death, Bio Father	0.9939***		0.9941***
	(0.9933 - 0.9946)		(0.9935 - 0.9948)
Alive 2013, Bio Mother	0.7724***		0.7778***
	(0.7567 - 0.7884)		(0.7620 - 0.7939)
Alive 2013, Bio Father	0.8049***		0.8126***
	(0.7840 - 0.8263)		(0.7915 - 0.8343)
Years of education, Bio Father		0.9844***	0.9893***
		(0.9811 - 0.9877)	(0.9859 - 0.9926)
Years of education, Bio Mother		0.9815***	0.9868***
		(0.9769 - 0.9861)	(0.9823 - 0.9915)
Observations	1,381,500	1,381,500	1,381,500

Robust cieform in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table A2: Mortality Among Male Non-Adoptees - Cox Proportional Hazard Models

VARIABLES	(1) Men	(2) Men	(3) Men
Age at death, Bio Mother	0.9921***		0.9925***
	(0.9916 - 0.9927)		(0.9919 - 0.9930)
Age at death, Bio Father	0.9922***		0.9927***
	(0.9917 - 0.9927)		(0.9921 - 0.9932)
Alive 2013, Bio Mother	0.7765***		0.7873***
	(0.7642 - 0.7890)		(0.7748 - 0.8000)
Alive 2013, Bio Father	0.7949***		0.8103***
	(0.7792 - 0.8110)		(0.7942 - 0.8268)
Years of education, Bio Father		0.9690***	0.9745***
		(0.9664 - 0.9716)	(0.9719 - 0.9772)
Years of education, Bio Mother		0.9739***	0.9783***
		(0.9702 - 0.9775)	(0.9747 - 0.9820)
Observations	1,448,132	1,448,132	1,448,132

Robust cieform in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table A3: Mortality Among Female Adoptees - Cox Proportional Hazard Models

VARIABLES	(1) Women	(2) Women	(3) Women	(4) Women
Age at death, Bio Mother	0.9850***		0.9849***	0.9846***
	(0.9766 - 0.9934)		(0.9766 - 0.9933)	(0.9763 - 0.9931)
Age at death, Ad Mother	1.0004			1.0007
	(0.9918 - 1.0092)			(0.9921 - 1.0095)
Age at death, Ad Father	1.0065			1.0066
	(0.9980 - 1.0151)			(0.9981 - 1.0152)
Alive 2013, Bio Mother	0.8837		0.8882	0.8874
	(0.7342 - 1.0637)		(0.7373 - 1.0699)	(0.7369 - 1.0686)
Alive 2013, Ad Mother	1.0043			1.0148
	(0.7927 - 1.2724)			(0.8006 - 1.2862)
Alive 2013, Ad Father	0.8125			0.8223
	(0.6052 - 1.0908)			(0.6127 - 1.1037)
Years of education, Ad Father		0.9998	1.0015	1.0007
		(0.9693 - 1.0312)	(0.9708 - 1.0331)	(0.9700 - 1.0323)
Years of education, Bio Mother		1.0200	1.0270	1.0275
		(0.9763 - 1.0657)	(0.9832 - 1.0728)	(0.9837 - 1.0733)
Years of education, Ad Mother		0.9544**	0.9541**	0.9552**
		(0.9142 - 0.9964)	(0.9137 - 0.9963)	(0.9148 - 0.9973)
Observations	10,743	10,743	10,743	10,743

Robust cieform in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table A4: Mortality Among Male Adoptees - Cox Proportional Hazard Models

VARIABLES	(1) Men	(2) Men	(3) Men	(4) Men
Age at death, Bio Mother	0.9887***		0.9889***	0.9891***
	(0.9821 - 0.9953)		(0.9823 - 0.9956)	(0.9825 - 0.9957)
Age at death, Ad Mother	0.9984			0.9986
	(0.9922 - 1.0046)			(0.9924 - 1.0048)
Age at death, Ad Father	0.9983			0.9982
	(0.9925 - 1.0041)			(0.9924 - 1.0041)
Alive 2013, Bio Mother	0.8676**		0.8771*	0.8783*
	(0.7597 - 0.9909)		(0.7671 - 1.0028)	(0.7681 - 1.0042)
Alive 2013, Ad Mother	0.9596			0.9672
	(0.8054 - 1.1434)			(0.8113 - 1.1529)
Alive 2013, Ad Father	0.9035			0.9079
	(0.7199 - 1.1339)			(0.7235 - 1.1392)
Years of education, Ad Father		0.9993	1.0009	1.0019
		(0.9746 - 1.0247)	(0.9761 - 1.0263)	(0.9770 - 1.0275)
Years of education, Bio Mother		0.9696	0.9755	0.9755
		(0.9335 - 1.0071)	(0.9391 - 1.0133)	(0.9391 - 1.0133)
Years of education, Ad Mother		0.9782	0.9801	0.9805
		(0.9446 - 1.0130)	(0.9465 - 1.0149)	(0.9468 - 1.0153)
Observations	11,846	11,846	11,846	11,846

Robust cieform in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table A5. The association between biological as well as adopting parents' years of schooling and child hospitalization age 43-47. Samples of non-adopted children, adopted children with both parents known and adopted parents including children with unknown biological fathers. OLS regressions.

VARIABLES	(1) Non-adoptees	(2) Adoptees - Bio father known	(3) Adoptees - Full sample
Years of education, Bio Mother	-0.0020***	-0.0018	-0.0033*
	(0.0001)	(0.0028)	(0.0019)
Years of education, Bio Father	-0.0022***	-0.0065***	
	(0.0001)	(0.0020)	
Years of education, Ad Mother		-0.0001	0.0003
		(0.0024)	(0.0019)
Years of education, Ad Father		-0.0009	-0.0022*
		(0.0018)	(0.0012)
P-value joint significance			
Adoptive parents		0.8128	0.1406
Biological parents	0.000	0.0015	
Observations	2,803,091	11,577	22,444
R-squared	0.0015	0.0074	0.0028

All models include cohort of birth dummies for parents and children and gender dummies.
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table A6. The association between child hospitalization age 43-47 and biological as well as adopting parents' mortality and years of schooling. Samples of non-adopted children, adopted children with both parents known and adopted parents including children with unknown biological fathers. OLS regressions.

	(1)	(2)	(3)	(4)	(5)	(6)
VARIABLES	Non-adoptees		Adoptees - Bio father known		Adoptees - Full sample	
Years of education, Bio Mother	-0.0017***	-0.0000	-0.0007	0.0002	-0.0025	-0.0010
	(0.0001)	(0.0001)	(0.0029)	(0.0029)	(0.0019)	(0.0019)
Years of education, Bio Father	-0.0017***	0.0004***	-0.0062***	-0.0051**		
	(0.0001)	(0.0001)	(0.0020)	(0.0020)		
Years of education, Ad Mother			0.0000	0.0008	0.0006	0.0019
			(0.0024)	(0.0025)	(0.0019)	(0.0020)
Years of education, Ad Father			-0.0003	0.0012	-0.0016	0.0000
			(0.0019)	(0.0019)	(0.0013)	(0.0013)
Age at death, Bio Mother	-0.0011***	-0.0009***	-0.0015***	-0.0014***	-0.0013***	-0.0012***
	(0.0000)	(0.0000)	(0.0004)	(0.0005)	(0.0003)	(0.0003)
Age at death, Bio Father	-0.0008***	-0.0007***	-0.0003	-0.0001		
	(0.0000)	(0.0000)	(0.0004)	(0.0005)		
Age at death, Ad Mother			0.0000	0.0002	-0.0008*	-0.0006
			(0.0005)	(0.0005)	(0.0004)	(0.0005)
Age at death, Ad Father			-0.0009**	-0.0007*	-0.0009**	-0.0007*
			(0.0004)	(0.0004)	(0.0004)	(0.0004)
Years of education, Child		-0.0072***		-0.0112***		-0.0123***
		(0.0001)		(0.0019)		(0.0015)
P-value joint significance						
Adoptive parents			0.2570	0.4120	0.0468	0.1865
Biological parents	0.0000	0.0000	0.0002	0.0041	0.0000	0.0002
Observations	2,803,091	2,689,161	11,577	11,116	22,444	21,533
R-squared	0.0030	0.0049	0.0093	0.0116	0.0043	0.0066

All models include cohort of birth dummies for parents and children and gender dummies.
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table A7. The association between birth weight among first born grandchildren and biological as well as adopting parents' mortality and years of schooling. Samples of non-adopted children, adopted children with both parents known andadopted parents including children with unknown biological fathers. OLS regressions.

VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
	Non-adoptees		Adoptees - Bio father known		Adoptees - Large sample	
Age at death, Bio Mother	0.5369***	0.3753***	1.3385*	1.5003*	1.1058*	1.1340*
	(0.0619)	(0.0623)	(0.7911)	(0.7975)	(0.5947)	(0.5997)
Age at death, Bio Father	1.1041***	0.9940***	-0.0068	0.1033		
	(0.0576)	(0.0580)	(0.8731)	(0.8819)		
Age at death, Ad Mother			1.0717	1.0874	1.1148	1.0056
			(0.9574)	(0.9688)	(0.7058)	(0.7124)
Age at death, Ad Father			-1.2958	-1.2739	-0.6717	-0.7328
			(0.9543)	(0.9651)	(0.6954)	(0.6987)
Years of education, Bio Mother	3.8819***	1.6460***	4.7961	4.6076	5.0388	3.3000
	(0.3355)	(0.3428)	(6.0201)	(6.1593)	(4.0778)	(4.1589)
Years of education, Bio Father	2.3217***	0.3078	-4.0685	-4.6754		
	(0.2667)	(0.2742)	(5.0500)	(5.1032)		
Years of education, Ad Mother			1.6997	1.8594	-3.6974	-4.1057
			(4.8678)	(4.9463)	(3.4202)	(3.4709)
Years of education, Ad Father			4.5343	4.9442	5.7047**	4.8765*
			(3.8481)	(3.8915)	(2.7368)	(2.7608)
Years of education, Child		10.3604***		-0.7543		11.0889***
		(0.2832)		(4.8413)		(3.3244)
P-value joint significance						
Adoptive parents			0.2482	0.2244	0.1043	0.1851
Biological parents	0.0000	0.0000	0.3669	0.2755	0.0689	0.1094
Observations	789,924	778,997	3,666	3,615	6,742	6,638
R-squared	0.0016	0.0033	0.0229	0.0234	0.0112	0.0132

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 9. The association between prevalence of low birth weight (<2,500 gram) among first born grandchildren and biological as well as adopting parents' mortality and years of schooling. Samples of non-adopted children, adopted children with both parents known and adopted parents including children with unknown biological fathers. OLS regressions.

	(1)	(2)	(3)	(4)	(5)	(6)
VARIABLES	Non-adoptees		Adoptees - Bio father known		Adoptees – Full sample	
Age at death, Bio Mother	-0.0002***	-0.0001***	-0.0005	-0.0005	-0.0003	-0.0003
	(0.0000)	(0.0000)	(0.0003)	(0.0004)	(0.0003)	(0.0003)
Age at death, Bio Father	-0.0002***	-0.0002***	-0.0004	-0.0004		
	(0.0000)	(0.0000)	(0.0004)	(0.0004)		
Age at death, Ad Mother			-0.0001	-0.0001	-0.0000	0.0000
			(0.0004)	(0.0004)	(0.0003)	(0.0003)
Age at death, Ad Father			0.0007*	0.0007*	0.0005*	0.0005*
			(0.0004)	(0.0004)	(0.0003)	(0.0003)
Years of education, Bio Mother	-0.0006***	-0.0002	0.0029	0.0023	0.0018	0.0023
	(0.0001)	(0.0001)	(0.0024)	(0.0025)	(0.0017)	(0.0017)
Years of education, Bio Father	-0.0007***	-0.0003***	-0.0011	-0.0011		
	(0.0001)	(0.0001)	(0.0021)	(0.0021)		
Years of education, Ad Mother			-0.0007	-0.0009	-0.0005	-0.0003
			(0.0020)	(0.0020)	(0.0014)	(0.0014)
Years of education, Ad Father			-0.0013	-0.0014	-0.0017	-0.0015
			(0.0016)	(0.0016)	(0.0011)	(0.0011)
Years of education, Child		-0.0020***		0.0017		-0.0029**
		(0.0001)		(0.0021)		(0.0014)
P-value joint significance						
Adoptive parents			0.4003	0.3204	0.1677	0.2463
Biological parents	0.0000	0.0000	0.3028	0.3558	0.2823	02234
Observations	789,924	778,997	3,666	3,615	6,742	6,638
R-squared	0.0005	0.0009	0.0186	0.0190	0.0113	0.0120

All models include cohort of birth dummies for parents and children and gender dummies.
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1