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ABSTRACT

Pre- and Post-Birth Components of Intergenerational Persistence in Health and Longevity: Lessons from a Large Sample of Adoptees*

We use data on a large sample of Swedish-born adoptees and their biological and adopting parents to decompose the persistence in health inequality across generations into pre-birth and post-birth components. We use three sets of measures for health outcomes in the second generation: mortality, measures based on data on hospitalization and, finally, measures using birth outcomes for the third generation. The results show that all of the persistence in mortality is transmitted solely via pre-birth factors, while the results for the hospitalization measures suggest that at least three quarters of the intergenerational persistence in health is attributable to the biological parents.

JEL Classification: I10, I14

Keywords: health inequality, intergenerational transmission, nature and nurture

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1. Introduction

There is a long tradition of studies on intergenerational persistence in longevity and other health outcomes dating back to Beeton and Pearson (1899).[1] However, it is not clear from these papers to what extent this persistence can be attributed to genetic, factors, or to environmental ones, e.g., that healthier parents transmit behavior promoting good health to the next generation or have better economic resources to invest in their children's health development. Although there is a vast literature in epidemiology on the hereditability of a large number of health conditions, the focus in these studies is on the etiological background of diseases, rather than understanding the causes of the intergenerational persistence in overall health.

In this paper, we study the importance of pre- versus post-birth factors in the intergenerational persistence in health by using a large sample of Swedish adoptees for whom we observe health measures of both biological and adopting parents. We study how the health of the biological parents – related to genetic factors and in-utero health ("pre-birth factors")– and the health of the adopting parents – related to health formation during childhood and adolescence ("post-birth factors") – affect the child's health later in life. Our dataset is constructed by matching several different administrative registers containing information on health outcomes for biological and adopting parents and their children. We study adopted children born between 1940 and 1967 in Sweden and are able to follow the health of the adoptees up until 76 years of age. For comparison, we also present results on the same outcomes obtained using the population of 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 sets of measures of this outcome: (i) mortality and premature death; (ii) health indices based on hospitalization data; and (iii), for females in the sample, birth outcomes of their first-born child obtained from the Swedish birth register. Mortality is either measured by longevity, using Cox regression to account for censoring, or by premature death, as captured by a binary variable of dying before age 60, 65 and 70, for children and parents, respectively. The health

[1] Intergenerational longevity associations are typically estimated positive but less than 0.15 (Beeton and Pearson, 1899, Pearl, 1931, Cohen, 1964, Wyshak, 1978, Iachine *et al*., 1998, and Gavrilov and Gavrilova, 2001) whereas intergenerational correlation in earnings and educational attainments differ between countries, but are rarely below 0.25 (see, e.g., Solon, 1999, and Black and Devereaux, 2012). Studies of intergenerational associations in overall health are quite rare (e.g., see Pascual and Cantarero, 2009, and Halliday, Mazumder and Wong, 2018), although there is a larger literature that has used infant and child health outcomes as measures of the health in the child generation (see e.g., Bhalotra and Rawlings, 2011, and Currie and Moretti, 2007).

indices are based on hospitalization data from the Swedish In-patient register, one is based on hospitalization visits and the other is based on hospitalization causes, where each cause is weighted by the probability of dying of that cause. Both measures are standardized by year and gender, and transformed to percentile ranks. By residualizing out age-specific effects they can be interpreted as measures of lifetime health. The third measure is motivated by the fact that birth outcomes 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.

In our analysis of non-adoptees, we report strong evidence in favor of the intergenerational transmission of health, although the strength of the persistence is weaker than the intergenerational transmission of education or income (see Solon, 1999, and Black and Devereux, 2011). Our mortality estimates are in line with findings in the literature on parent-child associations in longevity, including those in the epidemiological literature, which are often based on findings from samples of twins in the child generation. For our two health indices we estimate rank correlations of about 0.13-0.15. We also find evidence of positive health associations across three generations, where the health status of the grandparents is positively associated with the birth outcomes of their grandchildren.

Our decomposition results show that the intergenerational association in mortality and premature death 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, nor in the intergenerational association of death by age 60, 65 and 70, respectively. In addition, we show that these results survive several sensitivity tests on sample selection and selective placement. Hence, we are able to confirm results on general mortality from papers by epidemiologists using Danish data on adoptees (Sørensen *et al.*, 1988; Petersen *et al.*, 2005). The decomposition results for the intergenerational association in the health indices based on hospitalization attribute some of the health status to post-birth factors in the decomposition. However, a much larger share (75-85%) is still attributed to pre-birth factors captured by the health measure of the biological parents.

Our strategy of separating pre- and post-birth effects for intergenerational associations follows previous research that has used the regression-based approach using Swedish data for

adopted children and their biological and adoptive parents. This approach has been applied to a number of outcomes such as education and income (Björklund, Lindahl and Plug, 2006), financial risk taking (Black *et al.*, 2017), wealth, savings and consumption (Black *et al.*, 2019), crime (Hjalmarsson and Lindquist, 2013); entrepreneurship (Lindquist, Sol and van Praag, 2015) and voting (Cesarini, Johannesson and Oskarsson, 2014). Most recently, Black *et al.* (2019) presents a coherent analysis for nine different outcomes including wealth, risky investments, and years of schooling. Post-birth factors are much more important than pre-birth factors for outcomes such as wealth and savings rate, and somewhat more important for outcomes such as income, risky market participation and consumption. The sole exception is years of schooling, where pre-birth factors are slightly more important. The findings in our paper constitute an important complement to their results, as we find pre-birth factors to be much more important than post-birth factors for premature death and health as measured by the hospitalization indices. Because of our results, we now have additional estimates that contribute to our understanding of the degree of genetic versus environmental factors in explaining the intergenerational transmission in well-being that can be compared to results on other important economic and social outcomes from earlier papers using the same adoption design and study population.

There are additional economic motivations for the research question of this study. First, the recent interest in health inequality (see e.g. Chetty *et al.*, 2016) relates closely to the question on the importance of pre- versus post-birth factors in the child-parent association in health, since this intergenerational persistence is an element of the formation of health inequality. Second, our research question is closely related to the intergenerational persistence in human capital outcomes, income and wealth. Previous research has shown that health is very important for the formation of human capital, strongly associated with earnings ability in the labor market and indeed an important determinant of individual well-being (see e.g. Deaton, 2003). A strong element for pre-birth factors in intergenerational health persistence would limit the possibilities to affect intergenerational mobility in economic outcomes and well-being through policy measures that affects post-birth environmental transmission channels.[2] Finally, the research question relates to the literature on the effect of

[2] It is important to emphasize that a dominant role for pre-birth factors do not eliminate the role for policy, although it makes it more important to design policies so that it limits the role for genetic and prenatal environmental factors in transmitting health inequality across generations. A famous example is given in Goldberger (1979), criticizing heritability studies estimating the share of variance in an outcome that are due to nature or nurture, where he makes the point that variation in eye-sight that are due to genetic differences can be remedied by supplying eye glasses. Hence, finding nurture to be dominating as explaining variation in an

various health- and family-related interventions on later outcomes (see e.g. Almond and Currie, 2011; Campbell *et al*., 2014), as to whether or not they are implemented early in life or during the prenatal period.

We are aware of only two previous studies, from the same research group (Sørensen *et al*., 1988; Petersen *et al*., 2005), that analyze the intergenerational transmission of premature death using data on adopted children and their adopting and biological parents. The authors use Danish data on 960 and 2,365 adoptees, respectively, and find a significant association between the likelihood that the biological parents are still alive at age 50 or at age 70 and the child being alive at age 58 (Sørensen *et al*., 1988) or at age 70 (Petersen *et al*., 2005). For the adopting parents, no such associations were found.[3] In addition to the studies on mortality there is an extensive epidemiology literature on the heritability of specific diseases and psychological conditions using (also Swedish) data on adoptees.[4] The studies on cancer and circulatory diseases show that adoptees with at least one biological parent suffering from the disease under study have a significantly elevated risk of getting the disease. No such associations were found for the adopting parents. The study on suicides gives similar results. Research on drug abuse and alcohol usage, however, shows significant associations for both the biological and the adopting parents.

There are only a few studies on the intergenerational transmission of health that use data on adoptees in economics. Sacerdote (2007) uses data on 1,650 Korean-American adoptees placed by Holt International Children's Services during 1964-1985. He finds physical outcomes (height, overweight) to not be transmitted at all from the adopting parents, whereas health-related behaviors (drinking alcohol and smoking) are transmitted from the adopting parents. 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. He finds a significant association in the prevalence of medical diagnoses between adopting parents and their adoptive children. Classen and Thompson (2016) use the

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outcome does not mean that policies necessarily are ineffective. A central issue in this discussion is the importance of nature-nurture interactions, something we test for later in the paper.

[3] In addition, there is a small literature on mortality using data on adoptees and their biological siblings (such as Petersen *et al.*, 2008) that essentially confirms the findings from the intergenerational adoption studies. A separate but 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).

[4] Zöller *et al*. (2014) studies prostate, breast and colorectal cancer; Sundquist *et al.* (2011) coronary heart disease; Zöller *et al.* (2015) chronic obstructive pulmonary disease; von Borczyskowski *et al*. (2011) suicides; Kendler *et al.* (2012) drug abuse; and Kendler *et al.* (2015) alcohol use disorders.

same data set as in Thompson (2014) and perform a similar analysis on BMI and obesity measures. For these outcomes, they find (similar to Sacerdote, 2007) no association between adoptees and their adoptive parents.

Our study is able to reconcile the findings from the previous literature. We replicate the findings from the literature on premature death that shows environmental factors not to be important for intergenerational transmission. At the same time, there are studies in the epidemiological and economic literature that find that, although genetic factors are the explanation for many health measures, there is also a role for the environment especially regarding some health-related behaviors (such as smoking, drug and alcohol abuse). To reconcile these findings requires a longer follow-up compared to the study of hereditary diseases, which has been the focus in the epidemiological literature, and a richer set of health outcomes measured throughout the lives of the adopted children. By using hospitalization-based health measures capturing health status through decades of health-care utilization, we are able to estimate the importance of genetic and environmental factors for overall health. Although genetic factors account for a larger share in the intergenerational transmission of health, we do find some evidence that environmental factors are also important. Another notable difference with the previous epidemiological literature is that we compare our estimates for adoptees to those obtained for the population of children raised by their biological parents.

Our study differs from Sacerdote (2007), Thompson (2014) and Classen and Thompson (2016) along several dimensions. The most important difference is that our data include information on the biological parents of the adoptees, which enables us to decompose the pre- and post-birth parental influences on child health.[5] Because we have a much longer follow-up period, we are able to study long-run health outcomes rather than self-reported health outcomes and health-related behavior measured at younger or middle ages.[6] Finally, our sample size is much larger than those used in these past studies, potentially allowing us to identify smaller effects due to improved statistical power.

The rest of the paper is organized as follows. Section 2 presents our econometric models. Section 3 presents the data and descriptive statistics. The main results, as well as the

[5] Sacerdote (2007) has information on approximately 100 biological parents. This information is not used in the main analysis of his study.
[6] In Thompson (2014), the outcomes are measured for children, on average, at age 10 and in Sacerdote (2007), when those in the child generation are, on average, age 28.

sensitivity analyses, are laid out in Section 4. Section 5 concludes the paper. Finally, the paper contains two Appendices. Appendix A provides a brief historical background and a description of institutions related to the adoption process in Sweden. Appendix B presents the results of various sensitivity analyses.

2. Empirical Specifications

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

$$H_j^{bc} = \beta_0 + \beta_1 H_j^{bp} + \upsilon_j^{bc}, \qquad (1)$$

where H_j^{bc} represents adult health status for the biological child and H_j^{bp} the biological parents' health. 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 H_j^{bp}. The coefficient β_1 measures the strength of the association between the adult health of the child and the health 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:[7]

$$H_i^{ac} = \alpha_0 + \alpha_1 H_j^{bp} + \alpha_2 H_i^{ap} + \upsilon_i^{ac}, \qquad (2)$$

where H once more measures health that is transmitted from the biological parent *bp*, or the adoptive parent *ap*, to the adopted child *ac* born in family j and adopted and reared in family i; υ_j^{ac} is a child-specific error term uncorrelated with H_j^{bp} and H_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;

[7] Our strategy of separating pre- and post-birth effects follows Björklund *et al*. (2006), who estimated their relative importance for the intergenerational transmission of education and income. The same approach has been applied to other outcomes such as financial risk taking (Black *et al*., 2015a), wealth, consumption and savings rate (Black *et al.,* 2019), voting (Cesarini *et al.,* 2014), crime (Hjalmarsson and Lindquist, 2013); entrepreneurship (Lindquist *et al*., 2015) and political candidacy (Oskarsson *et al.,* 2018).

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 home) 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 provide internally valid estimates of the share of the intergenerational association in health status that is due to pre- and post-birth factors by estimating equation (2) by OLS using data on adopted children and their biological and adoptive parents. Since α_2 captures not only the importance of adoptive parental health, H_i^{ap}, but also everything else in the adoptive family that is correlated with H_i^{ap}, we do not interpret an estimate of α_2 as a causal effect, but instead as a measure of the importance of transmission channels stemming from the post-birth influences (a similar interpretation can be made for α_1).

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 the discussion in Section 4.4.2). As we will see in section 3.4, we find evidence of less selective placement for our longevity and health measures than what has been found for most other outcomes analyzed in previous adoption studies (such as education and income). Nevertheless, we perform two 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.

Second, we restrict the sample to include only adoptees who moved away from their municipality of birth. We cannot directly observe whether relatives or friends of the biological parents adopted some of the 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 or other reasons that might make the biological parents unable to accommodate a large family, 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 into established families.

Note also that equation (2) can easily be extended to account for "nature-nurture-interactions" by adding the product of H_j^{bp} and H_i^{ap} to this specification (see Björklund *et al.*, 2006).[8] We investigate the importance of such interactions in Section 4.4.3.

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 degree of generalizability of the 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 was instead reared by the biological mother (see section 4.4.1).

3. Data and Descriptive Statistics

3.1 Sample Definition

We use data from different national registers in Sweden and include all males and females born in Sweden between 1940 and 1967.[9] We use the Multigenerational Register (see Statistics Sweden, 2012) to identify whether a person was adopted as a child. This register also contains a personal identifier of the biological mother and father (if known to the authorities) as well as of 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

[8] There can be various reasons for nature-nurture interactions to be present. One of these is epigenetic mechanisms: environmental factors can affect gene expression in that genes are present, but either "switched on" or "switched off" depending on environmental factors.

[9] 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.

process. In total, there are 64,889 adoptees who we can identify in our data. Approximately 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 all samples used in this study. For the main analysis, we restrict the sample for whom we have information on both the biological parents. Since the In-patient register starts in 1987, we require that members of the family have not died before then.[10]

Table 1. Number of observations remaining after different sample restrictions

Born in Sweden 1940-1967	Non-adoptees	Adoptees
Not adopted	3,061,504	
Adopted		64,889
Adopted by two parents		33,312
Biological mother identified	3,016,646	24,274
Date of death is not missing*	2,923,652	22,424
Not adopted by own parents	2,923,652	22,385
Adopting parents' age restriction**	2,923,652	21,010
Not dead first year	2,912,701	21,001
Biological father is identified	2,832,475	10,728
Hospitalization records could be observed (alive in 1987)	1,937,645	6,117

*Dropping observations for which we cannot observe date of death because they have migrated or is missing in the cause of death register. We define them as missing in the cause of death register if we do not observe date of death and they are born before 1913, or they are not observed in any Censuses in 1960, 1970, 1990 or 2004.
** Adopting mother's age 25-47, and adopting father's 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 include, 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 because 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.[11] 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.

10 In Appendix B, Table B1, we display the sample restrictions for the mortality analysis sample.
11 Figure A1 in the Appendix A shows the ratio of adopted children in birth cohorts 1940-1967, which documents the same trends.

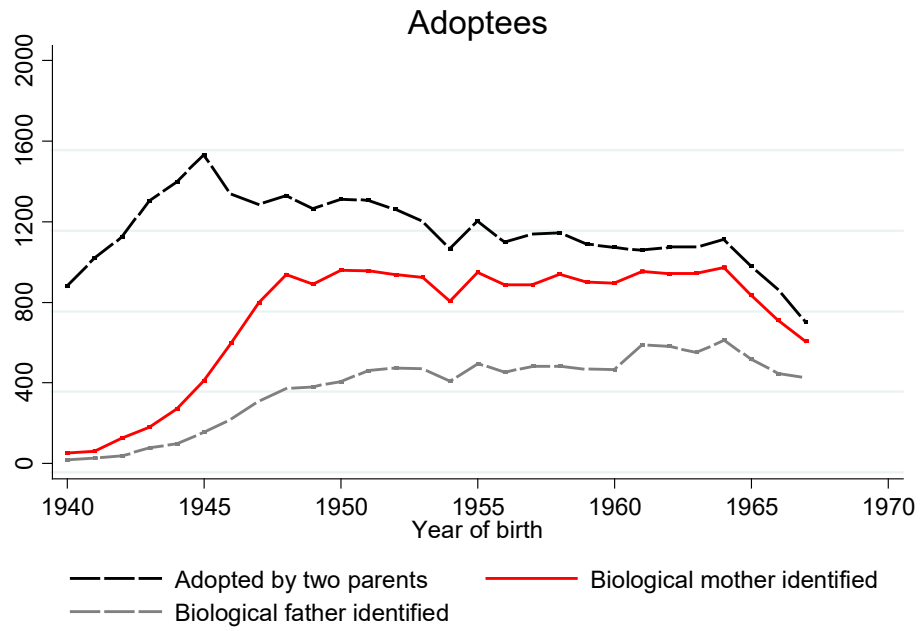


Figure 1. Swedish domestic adoptions by year of birth of the adoptees.

3.2 Measures of Health

3.2.1 Mortality

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 of death and International Classification of Diseases codes for the underlying cause of death from 1947 and with full coverage for all deaths in Sweden from 1961 onwards. Our observation period stops on August, 2016. This implies that for the child generation, we can observe the oldest person in our sample until age 76 and the youngest until age 49.

We use two different measures of mortality for both parents and children. First, longevity is constructed using dates of birth and death. Second, we construct indicator variables for the incidence of death before age 60, 65 and 70, respectively. Figure A2 in Appendix A, shows the share of individuals who died before the end of the observation window by year of birth.

3.2.2 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 offers national coverage starting in 1987, and we have access to data for the entire period until 2012.[12] Because the first birth cohort included in our data was born in 1940, we observe hospital stays for children from age 47 and until age 72. For parents, we observe hospitalizations at older ages. The In-patient Register includes ICD codes for a maximum of eight different medical causes of each hospital stay.

We construct two measures of health utilizing the hospitalization data. The first, labeled "Hospitalization", is simply the residuals from a linear probability model regression of an indicator variable for whether the individual has been in hospital care for each year separately during the observation window on calendar 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.[13] 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.[14] In a second step, we use these coefficients to 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, whereas individuals not making any hospital visits and still alive are assigned the value of 0. Then, we average over all years for each person. Based on this index, we obtain a percentile rank for each individual within each birth cohort and gender separately. The difference of this measure compared to the other hospitalization measure is that it weights the various diagnoses by severity based on how likely the person is to die within five years.

[12] This implies that only individuals that have survived until 1987 have a health measure based on the hospitalization data.
[13] The first two follow Cesarini *et al.* (2016).
[14] We use the first two digits in the ICD10 diagnosis codes (one letter and one number), which constitute approximately 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 an indicator of more than a week in hospital care. We control for gender and stratify on birth cohort.

Both "Hospitalization" and the "Health index" are ranked so as higher percentiles means better health. As the hospitalization and health index measures are adjusted for age effects and ranked by gender and cohort, we effectively compare lifetime health for individuals born in the same year. Note that in our main analysis we use the average of the health measures for mothers and fathers. In a sensitivity analysis we also show results for mothers and fathers separately.

3.2.3 Measures Based on Birth Outcomes for the Third Generation

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 females included in our sample as a health measure. Further, weight at birth, and in particular low birth weight (below 2,500 grams), 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.

Using the Multigenerational Register, we are able to link births to all children (adopted and biological) included in our sample. Our data source for studying health at birth is the National Swedish Birth Register (see Socialstyrelsen, 2009b). This birth register contains a large amount of information on all births in Sweden from 1973 and onwards.[15] We use three different birth outcome measures: (1) Birth weight measured in grams (scaled in percentile ranks); (2) An indicator for low birth weight, i.e., birth weight below 2,500 grams; and (3) an indicator of an APGAR score below 9 at five minutes after the birth.[16]

3.3 Descriptive Statistics

Table 2 contains sample means and standard deviations (within parentheses) for the main outcome and control variables in the sample of non-adoptees and adoptees, respectively. The first panel shows information on the children in the two samples. On average, the adopted children have worse health compared to the non-adopted. The same pattern can be seen for the children of the mothers in the child generation, with lower APGAR scores and birth weights for the children of the adopted mothers in the child generation, compared to the same

[15] This means that we are not able to include individuals born before 1973 in the third generation in the analysis.
[16] 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.

outcomes for the children of the population of non-adopted mothers. However, the mean differences are quite small. The third panel shows descriptive statistics for the biological parents. On average, the biological parents of adopted children have much worse health compared to the parents of non-adopted children. The fourth panel shows descriptive statistics of the adopting parents. The adopting parents have similar health as compared to the parents of non-adopted children.[17]

[17] 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.

Table 2. Summary statistics of main outcome and control variables

	Non-adoptees	Adoptees
Children		
Female	0.49	0.48
	(0.50)	(0.50)
Hospitalization (rank)	50.72	45.76
	(27.65)	(28.76)
Health index (rank)	50.78	45.87
	(27.43)	(28.36)
Year of birth	1955.98	1958.67
	(7.60)	(5.78)
Grandchildren		
Birth weight (rank)	50.28	48.91
	(28.80)	(29.66)
Birth weight<2,500g	0.05	0.07
	(0.22)	(0.25)
APGAR score 5 min<9	0.06	0.06
	(0.24)	(0.24)
Age at birth, mother	26.25	25.62
	(4.98)	(5.12)
Female	0.49	0.49
	(0.50)	(0.50)
Year of birth	1985.36	1985.62
	(7.56)	(7.40)
Biological parents		
Hospitalization (rank), Bio parents	49.63	44.96
	(21.02)	(21.79)
Health index (rank), Bio parents	50.07	41.12
	(21.11)	(20.99)
Year of birth, Bio mother	1928.95	1935.34
	(9.55)	(7.93)
Year of birth, Bio father	1925.81	1932.04
	(9.85)	(8.67)
Adopting parents		
Hospitalization (rank), Ad parents		49.87
		(20.92)
Health index (rank), Ad parents		51.41
		(21.18)
Year of birth, Ad mother		1925.25
		(7.56)
Year of birth, Ad father		1922.77
		(7.71)
Observations	1,937,645	6,117

Note: Standard deviations in parentheses. Hospitalization, health index and birth weight are within gender and birth cohort percentile rank. Higher values of Hospitalization and health index represents better health. For parents, health measures are mean of parents' health ranks.

3.4 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. There are at least two reasons why we would observe a positive correlation for characteristics of biological and adoptive parents. First, this correlation could be due to some children being adopted by relatives of one of the biological parents. Second,

there could be matching on characteristics known to the adoption agency, either because of the demands of parents or because of the view that an adopted child would be better off in an adoptive family with similar characteristics as the biological parents. One way to check the likely severity of this issue with regard to our main results – made possible by the fact that we can observe health for both adoptive and biological parents of the adoptees – is to simply correlate the health measures for these two parental types. Table 3 shows the correlations of mortality and health measures based on hospitalization data between adopting and biological parents of adoptees.

Table 3. Correlations between biological and adoptive parents' mortality and health measures

Hospitalization	Heath index	Age at death	Age at death (children born < 1953)	Dead < age 60	Dead < age 65	Dead < age70
-0.0049	0.0021	0.1272*	0.0938*	0.0482*	0.0152	0.0415

* *p*-values for significance are below 1 percent.

We obtain very small and statistically insignificant correlations for the hospitalization based measures. This differs compared to those reported for most other outcomes in adoption studies using Swedish data.[18] This finding is very important for the purpose of this study because it suggests that selective placement is unlikely to generate biased estimates of intergenerational health correlations using adoption data. That said, because selective placement is still possible on unobservable characteristics we discuss this issue and also perform some sensitivity analyses of the likely impact of selective placement on our main estimates in Section 4.4.2. The correlation of our mortality measures varies more, but are still very low for our indicators for premature death.

[18] For instance, Björklund *et al*. (2006) find a correlation of 0.14 for the mother's and father's years of schooling for children born 1962-1966.

4. Results

4.1 Mortality

We start the Results section by studying the intergenerational persistence in mortality. Sørensen *et al.* (1988) and Petersen *et al.* (2005) focus exclusively on mortality outcomes and we therefore compare our results to these previous findings as a point of departure before showing results for the other health outcomes under study. In the mortality analysis, we extend the work of the two papers mentioned above primarily by having a longer follow up period (the oldest child cohort is 76 when we stop observing them) and also by comparing our results to those obtained on the population of children raised by their biological parents born in the same birth cohorts.

Table 4 shows the results from the Cox proportional hazard model for the persistence in longevity across generations. The dependent variable in these models is age of death (measured in months) of the individual in the child generation and the independent variable the age of death of the biological and the adopting parents, respectively. The Cox model relies on the proportional hazard specification, but not on any particular functional form for the baseline hazard. The results are presented as hazard ratios and should be interpreted as the relative difference in the hazard resulting from a one-unit (one year) change in the independent variable.

Censoring, on both the dependent and the independent variables, is a main concern for our choice of econometric model as well as principles for sample selection. We use a hazard model to deal with the high proportion of right censoring on the dependent variable. However, since we, in the full sample, do not observe date of death for 36 percent of the biological mothers, 21 percent of biological fathers, 26 percent of the adopting mothers, and 15 percent of adopting fathers, we also have a problem of censoring on the independent variables. To deal with this, we have restricted the sample to those where we could observe the date of death of all parents, i.e., we impose a selection on Complete Cases (CC).

Rigobon and Stoker (2007) show, in the framework of a linear regression model, that a sufficient condition for consistency of the Complete Case regression estimates is that the selection, conditional on observables, is exogenous, i.e., that an indicator variable for sample inclusion would be conditionally independent of the error term in the linear regression. Although there are no obvious reasons to why this assumption would not apply in our

application and to a non-linear proportional hazard model, we provide a sensitivity analysis of our results. In the first column of Table 4 we present the Complete Case results from when we use the entire sample born between 1940 and 1967. In the second column, we show the results from when we restrict the sample to those born in the first half of the sampling window defined by year of birth, i.e., those born before 1953. In this sub-sample, we observe date of death for a much larger share of the parents (87 percent of children have parents that are all dead), which makes the potential inconsistency from censoring on the independent variable much smaller.

Comparing the estimates in Columns 1 and 2 of Table 4 it is apparent that the results are almost identical. This result suggests that we can maintain the hypothesis of exogenous selection conditional on the independent variables. The results furthermore suggest that there is a strong intergenerational persistence in longevity in the population of those raised by their biological parents. The hazard ratio estimate in Column 1 suggests that an additional year in average length of life of the parents corresponds to an about 1.8 percent reduction in mortality of the child.

Turning to the estimates for adoptees, Column 3 shows the results for the entire sample and Column 4 for those born before 1953. The estimates are very similar and they unambiguously suggest that the entire persistency in mortality can be attributed to pre-birth differences. The hazard ratio-estimates for the biological parents are similar to those obtained in the sample of children raised by their biological parents and the estimates for the adopting parents are all insignificantly different from the no-effect hazard ratio of 1. Since we require all four parental types to be deceased before the end of our sample period, the sample is limited to about two-thirds of all parents to adoptees born before 1953. Finally, Column 5 shows the result when we use the *Complete Cases* sample for the adopting parents only in the born-before-1953 sample. Since the adopting parents are in general older than the biological ones, we only need to exclude 4 percent. Reassuringly, the estimates from this model are very similar to the estimates for adoptive parents shown in Columns 3 and 4.

Table 4. Cox proportional hazard model estimates of the associations between child mortality and parental age at death

	(1)	(2)	(3)	(4)	(5)
	Non-adoptees		Adoptees		
Age at death, Bio parents	0.9818***	0.9813***	0.9763***	0.9705***	
	(0.0003)	(0.0003)	(0.0055)	(0.0071)	
Age at death, Ad parents			0.9960	0.9967	0.9947
			(0.0062)	(0.0082)	(0.0068)
Share of dead children	0.1216	0.1493	0.1301	0.1654	0.1577
Sample	All CC	CC born <1953	All CC	CC born <1953	CC born <1953
Observations	1,674,637	1,126,649	4,069	2,194	2,949

Note: Results from Cox proportional hazard models. Robust 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 both children and parents. Columns (1) and (2) are based on a sample of non-adopted children, columns (3)- (5) on adoptees. Column (1) and (3) is based on a sample of children with parents that are all dead (CC) in the end of the observation period. In column (2) and (4) an additional restriction is imposed that children are born before 1953. In column (5) the sample is restricted to adoptees born before 1953 with dead adopting parents.

Table 5. Linear probability model estimates of intergenerational association of dying before age 60, 65 and 70, respectively.

	(1)	(2)	(3)	(4)	(5)	(6)
	Dead < age 60		Dead < age 65		Dead < age 70	
	Non-adoptees	Adoptees	Non-adoptees	Adoptees	Non-adoptees	Adoptees
Bio parents	0.0109***	0.0505***	0.0170***	0.0755***	0.0258***	0.0870**
	(0.0006)	(0.0132)	(0.0007)	(0.0176)	(0.0009)	(0.0369)
Ad parents		0.0062		0.0021		0.0292
		(0.0135)		(0.0177)		(0.0382)
Mean dep var	0.0524	0.0661	0.0679	0.0783	0.0824	0.0861
Observations	1,638,054	4,770	1,147,746	2,470	623,366	582

Note: Results from a linear probability model. Robust 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 both children and parents. In columns (1) and (2), the sample is restricted so that all children are born before August 1956; in columns (3) and (4) before August 1951; and in columns (5) and (6) before August 1946. Columns (1), (3) and (5) are based on a sample of non-adopted children and columns (2), (4) and (6) on adoptees. The share of biological parents with one parent that has deceased before a given age threshold is 0.128 in column (1), 0.144 in column (2), 0.213 in column (3), 0.232 in column (4), 0.340 in column (5), and 0.326 in column (6). The share of adopting parents with one parent that has deceased is 0.109 in column (2), 0.185 in column (4), and 0.309 in column (6).

As an additional sensitivity analysis, Table 5 presents Linear Probability Model estimates for intergenerational persistence in deaths before ages 60, 65 and 70, respectively.[19] The advantage of these models *vis-à-vis* the hazard models presented in Table 4 is that they can be estimated without any censoring on either the dependent or the independent variables. We restrict the samples to the cohorts that allow us to follow the included individuals to each of

[19] Probit estimates for intergenerational persistence in early deaths show very similar results.

the ages. This means that for the model for intergenerational association in mortality before age 60, we restrict the sample in the child generation to those born before August 1956; for mortality before age 65 to those born before August 1951 and for mortality before age 70 to those born before August 1946.

The results for non-adoptees - shown in Columns (1), (3) and (5) - reveal that the intergenerational association in premature death becomes stronger as the age limit increases from age 60 to age 70. The results for adoptees - shown in Columns (2), (4) and (6) – suggest that the association can be fully attributed to the biological parents, which confirms our previous results as well as those obtained by Sørensen *et al.* (1988) and Petersen *et al.* (2005).

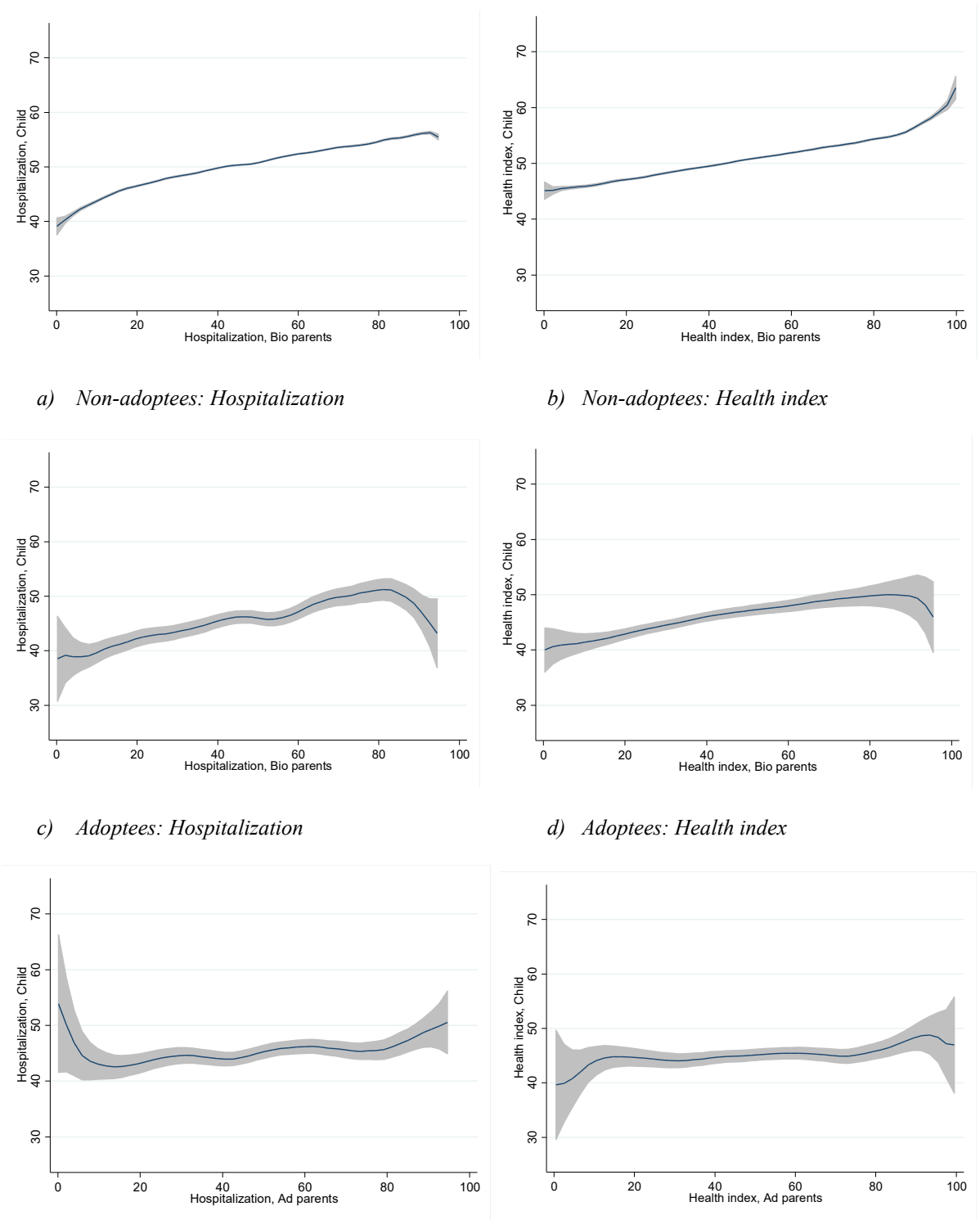
Appendix B shows the results from a number of alternative specifications and sample restrictions. Table B2 shows the estimates with mothers and fathers separately and we also include those with unknown biological father in the sample of adoptees. The results show that there is a marginally stronger association between mothers and their children's longevity than between fathers and their children's longevity (conditional on the other parent's longevity). To investigate how the 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 for the baseline hazard rather than the Cox model. The hazard ratio estimates from this model turned out to be very similar to those of the Cox model presented in Table 4, see Appendix Table B3. Using these estimates for adoptees, we find that the prediction of one additional year of longevity for the biological parents extends the child's median life expectancy by 0.25 additional years.[20] In Table B4 we show results that are obtained on the entire original sample and instead of excluding individuals with parents still alive when we stop observing them in August 2016, we include dummy variables for them being alive at that time. All results shown in these tables support our conclusion that the intergenerational persistence in mortality can be attributed to the biological parents.

4.2 Health Measures Based on Hospitalization Data

Figure 2 shows the relation between percentiles of the parental and child hospitalization and health index. We use a local linear kernel regression, instead of scatter plots, given that the adoption sample is relatively small. The graphs for non-adoptees - shown in the upper panel,

[20] For non-adoptees the corresponding figure is 0.24.

- reveal a strong intergenerational persistence in health, which is well approximated by a linear relationship (except at the very top of the distribution). The middle panel shows the graphs for the relation between child health and the health of the biological parents in the adoptee sample. The relation is almost equally strong as the one shown for the children raised by their biological parents. Finally, the figures in the bottom panel show the relation between the health status of the adopting parents and their children. The relation is slightly positive but clearly weaker than for the biological parents.



a) Non-adoptees: Hospitalization

b) Non-adoptees: Health index

c) Adoptees: Hospitalization

d) Adoptees: Health index

e) Adoptees: Hospitalization

f) Adoptees: Health index

Note: The figures show results from bivariate local linear kernel regressions using an Epanechnikov kernel and rule-of-thumb bandwidths. The shaded area represents the 95% confidence interval.

Figure 2. Relationship between percentile rank of child and parental hospitalization and health index for non-adoptees and adoptees

Table 6 reports OLS regression results from models using Hospitalization and Health index as health measures for the child and parental generations. Columns 1 and 3 report the results for non-adoptees. As both measures are scaled in percentile ranks we are estimating rank correlations. The magnitudes of the estimates are somewhat stronger for the hospitalization measure compared with the health index, suggesting that a one-percentage-point increase in the parents' relative health is associated with a 0.12-0.14 percentile increase in the child's health. Hence, confirming findings from previous research, we find that the intergenerational transmission of health in the population is positive but smaller than what is typically found for outcomes such as education and income (see Black and Devereux, 2011; Black *et al.*, 2019).[21]

The results for adoptees are reported in Columns 2 and 4. As opposed to the estimates for mortality, the coefficient estimate for the Hospitalization measure of the adopting parents is statistically significantly different from zero at the 1 percent level. These results allow us to decompose the intergenerational association in health into pre- and post-birth influences. For the Hospitalization measure, such decomposition attributes about ¾ of the association to pre- and ¼ to post-birth influences. However, for the Health index, the estimate for the adopting parents is smaller and again insignificantly different from zero. The latter result is line with our findings for mortality above, which is not surprising given that the health index partly is based on cause-of-hospitalization specific mortality probabilities.

In Appendix B, Table B5, we show results for mothers and fathers separately. We also present results from an extended sample where we include adoptees with an unknown biological father. The results show a slightly stronger association between biological mothers' health and their children's health, than between biological father's health and their children's health, both for adoptees and non-adoptees. This is similar to our results for mortality. When increasing the sample of adoptees to include adoptees with an unknown biological father the sample size more than doubles, which improves precision of our estimates. This result in the Health index measures of the adopting parents becoming

[21] The relatively smaller intergenerational health associations, compared to intergenerational schooling and income associations, found here are in line with the results in Halliday and Mazumder (2017) and Mazumder (2011) that finds smaller sibling correlations for health status than for education and family income. In Halliday, Mazumder and Wong (2018) the authors use PSID and estimate intergenerational rank correlations in health outcomes for US, using self-reported health averaged over the lifetime. The find rank correlations that are almost twice as large (0.26) compared to our estimates for Sweden. Interestingly, this finding is in line with differences of income persistence estimates for US and Sweden, which can differ up to as much as with a factor of 2.

statistically significant (*p*-value: 0.0116). In Table B6 we present separate results for males and females, respectively. The results reveal that there is a significant association between adopting parents' Health index and the health of male, but not the female, adoptees.

Table 6. OLS estimates of associations between percentile rank of parental and child lifetime health measured by indices based on hospitalization data

	(1)	(2)	(3)	(4)
	Non-adoptees	Adoptees	Non-adoptees	Adoptees
	Hospitalization		Health index	
Bio parents	0.1406***	0.1444***	0.1221***	0.1277***
	(0.0009)	(0.0169)	(0.0009)	(0.0172)
Ad parents		0.0477***		0.0248
		(0.0176)		(0.0171)
Observations	1,937,645	6,117	1,937,645	6,117

Note: Results from OLS regressions. Robust 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 both children and parents. Columns (1) and (3) are based on a sample of non-adopted children, columns (2) and (4) on adoptees. The dependent variable in columns (1) and (2) is a measure of hospitalizations, and the dependent variable in columns (3) and (4) is a health index. Both measures are ranked within-cohorts separately by mother, fathers, daughters, and sons.

4.3 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 the birth weights and APGAR scores of children as a proxy for the health among women. The second reason is that birth weight is known to correlate strongly with later-life health. It can thus serve as an additional measure of the intergenerational transmission of health going into the third generation.[22]

Table 7 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 grams), as well as an indicator for an APGAR

[22] Selection into giving birth is likely driven by maternal health status, so that healthier women are more likely to conceive and deliver live children. It is, however, not obvious that this form of selection would bias our results, or, if this is the case, in what direction if we make inference to the population of all women. We therefore confine ourselves with making inference to the population of women that give birth in the given time window.

score below 9 at five minutes.[23] Because we have to restrict the sample to females, and additionally to those who give birth, the sample sizes for these regressions are approximately halved compared to those shown in the previous sections.

We find highly statistically significant positive correlations of the hospitalization measure and health index of the biological parents on all birth outcomes of their grandchildren in the sample of non-adoptees. In Appendix Table B7 we show that these associations remain very similar if we control or the health status of the child. Hence, there is only a very weak mediating role of child's health in explaining the associations between grandchild's birth outcomes and parent's health. Since the previous literature (see e.g. Almond and Currie, 2011, or Barker, 1990 and 1995) has shown that there is a strong association between birth outcomes, in particular birth weight and adult health, these results contribute with further support that there is a multigenerational association in health outcomes[24].

Turning to the samples of adoptees, the results in Column 1, 2, and 4 show a significant association between the health of the adopting grandparents and birth weight. The estimates for low APGAR scores in the adoptee sample are in general too imprecise to give significant estimates. Only the health measure of the biological grandparents turned out significantly different from zero at the 5 percent level for this outcome measure. For all sets of results shown in Table 7, the precision of the estimates is not sufficient for a meaningful decomposition of the pre-and post-birth influences on health formation.

[23] 6% of children have an APGAR score at 5 minutes that is below 9. We choose APGAR below 9 instead of below 10 to follow the praxis from medical research of looking at the lower part of the APGAR distribution and because these estimates are more precise. Estimates are qualitatively similar for APGAR below 10.
[24] This confirms previous findings on longevity (Piraino *et al.*, 2014; Maystadt and Migali, 2017) and mental health (Johnston, Schurer and Shields, 2013).

Table 7. Associations between percentile rank of parental health index and firstborn grandchild's health at birth

	(1) Birth weight	(2) Low Birth weight	(3) APGAR<9	(4) Birth weight	(5) Low Birth weight	(6) APGAR<9
	Hospitalization			Health index		
Non-adoptees						
Bio parents	0.0091***	-0.0001***	-0.0000***	0.0255***	-0.0002***	-0.0001***
	(0.0018)	(0.0000)	(0.0000)	(0.0018)	(0.0000)	(0.0000)
Observations	623795	623795	570657	623795	623795	570657
Adoptees						
Bio parents	-0.0024	-0.0001	-0.0005**	0.0242	-0.0004	-0.0002
	(0.0307)	(0.0003)	(0.0003)	(0.0314)	(0.0003)	(0.0003)
Ad parents	0.0710**	-0.0006**	-0.0002	0.0701**	-0.0003	-0.0002
	(0.0316)	(0.0002)	(0.0003)	(0.0305)	(0.0002)	(0.0003)
Observations	2,152	2,152	1,964	2,152	2,152	1,964

Note: Results from OLS regressions. Robust standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression, and all regressions include indicators for mother's age at birth, child gender, year of birth, and grandparents' birth cohort. The dependent variable in columns (1) and (4) is birth weight measured in grams and scaled into percentile ranks, in columns (2) and (5) it is a binary variable capturing if birth weight <2,500 grams, and in columns (3) and (6) the dependent variables is a binary variable capturing if APGAR score at 5 min is below 9.

4.4. Sensitivity Analyses

4.4.1 External validity

As we discuss in Section 2, a way of assessing the similarity between the adoptees and the rest of the population is to compare the sum of the estimates for biological and adoptive parents with those obtained for non-adoptees for the biological parents.[25] The results in Tables 4-6 reveal that the sums of the estimates of adoptees are always larger than the population estimates. This is in particular true for the estimates using premature death as outcome variable.[26]

We do two different checks of the similarity between the adopted and non-adopted children. First, we compare the results for the decomposition of pre- versus post-birth factors for adoptees, with the intergenerational association for the non-adopted children of the mothers who gave up their first-born child for adoption, in the subsample of adoptees with at least one biological sibling reared by the biological mother. Second, we compare the causes

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

[26] We note that Sørensen *et al.* (1988) and Petersen *et al.* (2005) present results for premature death using a sample of (Danish) adoptees, but that they did not perform population-based estimations. Hence, we don't know the degree of external validity of their adoption estimates.

of death for adoptees with those of non-adoptees and do the pre- and post-birth decomposition in the framework of a competing risk analysis.

The results shown in Table 8 from the first exercise for the two health indices reveal two interesting results. First, the results for the importance of pre- versus post-birth factors are qualitatively very similar to the main ones in Table 6. Second, we now find that the sum of the estimates in the second column is very similar to the magnitude of the population-based estimate in the first column, for both health indices. Hence, for our main health outcomes, previous conclusions are unchanged.

Table 8. Comparison of the intergenerational association in health for the non-adopted and adopted children with the same biological mother.

	(1) Non-adoptees	(2) Adoptees	(3) Non-adoptees	(4) Adoptees
	Hospitalization		Health index	
Bio parents	0.1593***	0.1280***	0.1528***	0.1286***
	(0.0094)	(0.0197)	(0.0095)	(0.0202)
Ad parents		0.0468**		0.0219
		(0.0203)		(0.0197)
Observations	19,997	4,582	19,997	4,582

Note: Results from OLS regressions. Robust 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 both children and parents. The samples are overall restricted to children with a biological mother that has given at least one child up for adoption and raised at least one child of her own. Columns (1) and (3) are based on a sample of non-adopted children, columns (2) and (4) on adoptees. The dependent variable in columns (1) and (2) is a measure of hospitalizations, and the dependent variable in columns (3) and (4) is a health index. Both measures are ranked within cohort separately by mother, fathers, daughters, and sons.

In Appendix Tables B8 and B9 we show mortality results for this sample of adoptees and non-adoptees with the same biological mothers. Our result that intergenerational association in mortality can be attributed to pre-birth factors is maintained in these samples. However, the sum of the estimates for the adoption sample is still much larger than the population-based estimates.

Appendix Tables B11 and B12 show the results for the Competing risk analysis for different causes of death.[27] The results show evidence of some important differences between adoptees and non-adoptees. For instance, the positive intergenerational association for the mortality measures between the adoptees and their biological parents is to a high degree due to death from External causes, Circulatory diseases and Treatable conditions, whereas for non-adoptees, the positive intergenerational association is mostly due to associations in

[27] Table B10 shows the ICD codes for the disease categories used in the competing risk analysis.

Cancer and Circulatory diseases. Hence, we posit that for mortality outcomes, at least based on premature death, external validity is limited, possibly because of differences in the causes of death between adopted and non-adopted children

4.4.2 Parameter Robustness with respect to selective placement

In section 3.4, we mentioned two reasons for selective placement of adoptees. First, some adoptions could be made by relatives of one of the biological parents. Second, there could be matching on characteristics known to the adoption agency but unknown to us as researchers. As discussed in Appendix A Section A.4, the empirical importance of the first reason – adoptions by relatives – is likely to be very limited because of the rule prohibiting people with their own biological children from adopting. This rule, to a large extent, precluded parents and siblings of the biological parents from adopting.[28]

The second reason, matching, is possibly a more important mechanism. However, as reported in Table 3, health status (measured either as hospitalization or the health index) is not correlated for the adopting and biological parents, supporting the absence of selective placement on observable health characteristics. Note also that for mortality, where the results reported in Table 3 suggest a marginally significant positive selection implying a positive bias for the estimates for the adopting parents, we do not observe any significant effects for adopting parents in the results. We will, nevertheless, test for parameter robustness with respect to matching based on broad set of characteristics observable in the data.

A simple, and informal, way of empirically the assumption of independence between the biological and adopting parents is to include and exclude the observable parental characteristics to check the stability of the coefficient estimates of main interest (see Björklund, Lindahl and Plug, 2006). Table 9 reports results from such a robustness check for the two key results obtained in Section 4.2 for our health measures based on hospitalization data. Column 1 shows the results for hospitalization for the biological parents when we include no other parental controls except indicators for the birth cohort of the biological parents, and Column 2 reports the results when we successively add variables for the observable characteristics of the adopting parents: hospitalization, years of education, cohort

[28] As further discussed in Appendix A, Section A.4, Nordlöf (2001) estimated these adoptions to be around 1 percent of the total number of adoptions in the Stockholm area. Brandén, Lindahl and Öckert (2018) confirm this conclusion, although their estimate of the share of adoptions by close relatives is slightly higher at 5.4 percent, applicable to the whole country. They are also able to eliminate those adopted by close relatives from their sample, and they find that the correlation in years of schooling between (unrelated) adoptive and biological parents of adoptees remains virtually unchanged.

indicators and regional indicators of both adopting parents. The estimates for the biological parents barely change with added controls. Column 3 shows the results for the adopting parents when we only include indicators for year of birth of the adopting parents in the model. Column 4 shows the results when we add variables measuring the characteristics of the biological parents. Columns (5)-(8) report the corresponding results for the health index. The estimates for the adoptive parents remain unchanged with these added controls. Hence, we conclude that there is no evidence that selective placement on observables affects our results.

Table 9. Sensitivity analyses among adoptees

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Hospitalization				Health index			
Bio parents	0.1454***	0.1393***		0.1421***	0.1315***	0.1242***		0.1285***
	(0.0169)	(0.0171)		(0.0170)	(0.0172)	(0.0175)		(0.0175)
Ad parents		0.0410**	0.0469***	0.0448**		0.0179	0.0260	0.0196
		(0.0177)	(0.0177)	(0.0176)		(0.0173)	(0.0172)	(0.0172)
Cohorts, Bio mother	Yes	Yes	No	Yes	Yes	Yes	No	Yes
Cohorts, Bio father	Yes	Yes	No	Yes	Yes	Yes	No	Yes
Cohorts, Ad mother	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Cohort, Ad father	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education, Bio mother	No	No	No	Yes	No	No	No	Yes
Education, Bio father	No	No	No	Yes	No	No	No	Yes
Education, Ad mother	No	Yes	No	No	No	Yes	No	No
Education, Ad father	No	Yes	No	No	No	Yes	No	No
Region, Bio parents	No	No	No	Yes	No	No	No	Yes
Region, Ad parents	No	Yes	No	No	No	Yes	No	No
R^2	0.0222	0.0304	0.0080	0.0263	0.0175	0.0250	0.0078	0.0213
Observations	6,097	6,097	6,097	6,097	6,097	6,097	6,097	6,097

Note: 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.

Another 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., adoptive parents with better health are somehow able to "pick out" healthier children. While we cannot directly test for this because we lack data on health at birth for the index cohorts, 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), using a sample of adoptees mostly born in the 1970s, show that there is no significant correlation between adoptive parents' education, and 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.

4.4.3 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 the health measures of the adoptive parents and the health measures of the biological parents.

The results are reported in Appendix Table B13. For the Hospitalization index, the interaction between biological and adopting parents' hospitalization is significantly negative, but the magnitude of the coefficient estimate is not very large. Since the interaction effect is negative it means that adoptive parents' health becomes relatively more important, with lower health of the biological parents. For an adoptive child with biological parents of mean health, the adoptive parents would have to be in the 98th percentile of health in order for pre- and post-birth factors to be equally important for intergenerational health transmission, suing the hospitalization measure.[29] For the Health index, the estimate is insignificantly different from zero. Taken together, the results suggest that the additive model provides a good approximation of the relation between child health and the health of the biological and adopting parents.

4.4.4 First-born Adoptees and Adoptees who Move from their Municipality of Birth

A concern discussed in Section 2 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 away from their municipality of birth after the adoption. The results shown in Appendix Table B14 are almost identical to the one obtained for the entire sample shown in Table 6.

[29] This can be seen by equalizing two first derivatives of an extended version of equation (2) where an interaction term is added, $H_i^{ac} = \alpha_0 + \alpha_1 H_j^{bp} + \alpha_2 H_i^{ap} + \alpha_3 H_j^{bp} \cdot H_i^{ap} + \upsilon_i^{ac}$. First, take the derivative with respect to H_j^{bp}. Second, take the derivative with respect to H_i^{ap}. Third, equalize the two derivatives and set H_j^{bp} equal to the mean in the adoption sample (44.96 according to Table 2). Fourth, solve for H_i^{ap}, which gives 98.5.

In the final sensitivity analysis, we restrict the sample to include first-born adoptees only. As discussed in Section 2, 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. Again, Table B14 shows very similar results to the original ones shown in Table 6.

5. Conclusions

This study uses data on adoptees and decomposes the intergenerational persistence in health outcomes into pre-birth factors – reflected by the health outcome of the biological parents – and post-birth factors – reflected in the health outcomes of the adopting parents. Our results for mortality confirm previous findings – primarily obtained in epidemiology studies - that intergenerational persistence in longevity can be fully attributed to pre-birth factors. Our main contribution is to decompose the association in intergenerational health status. The results for the hospitalization measure suggest a significant effect of post-birth influences. However, a decomposition of the overall health persistence still attributes a much larger share (75-85%) to pre-birth factors captured by the health measure of the biological parents.

Our data do not allow us to distinguish between the possibility that if we would have observed mortality for the entire life cycle, we would have been able to estimate a significant effect of post birth-factors as well and the competing possibility that the health measure using hospitalization data captures a broader aspect of health than mortality. Although this is a limitation, our results are still able to reconcile the previous evidence on and results obtained in the economics literature that health related behavior are affected by the adopting parents (see Sacerdote, 2007, for drinking and smoking behavior and Thompson, 2014, on health problems related to environmental exposure) as well as the epidemiology literature on health related behavior (see e.g. Kendler *et al.*, 2012; and Kendler *et al.*, 2015).

Similarly to other empirical studies, the results could to some extent depend on the social and physical environment of the country where the data were obtained. In particular, Sweden's universal and practically free-of-charge health care system, low poverty rate and, compared to most industrialized countries, small income differences, can be important in this context. One could argue that the genetic influences may be more important in such environment. For example, Turkheimer *et al*. (2003) finds that genetic differences are more

important in IQ determination for high SES children than for low SES ones, since high SES children are more equal on other IQ determinants. In the same spirit, one could argue that genetic differences is “all that is left”, or at least given a more prominent role, in equal societies such as Sweden and Denmark. Following this line of argument, our results should be interpreted as upper bounds for share of pre-birth influences on the intergenerational persistence in health outcomes.

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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, they were 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. (2017), 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.[30]

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

[30] 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.

Bohman (1970) and Nordlöf (2001) give a fairly consistent description of the mothers who gave up their children for adoption: 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.

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 married or being in a steady relationship, the increased availability and usage of contraceptives, and the liberalization of the legislation for abortion.[31]

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, Dawes and Lindgren (2018) 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.[32] 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,

[31] 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.

[32] In Section 4.4.5 we compare the health status in our sample of adoptees to non-adoptees in the same age group.

which are circulatory diseases, cancer and all other causes of death.[33] The left-hand panel corresponds to the sample of adoptees and the right-hand panel to the comparison group of non-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.

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 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.[34]

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 disadvantage 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

[33] 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).

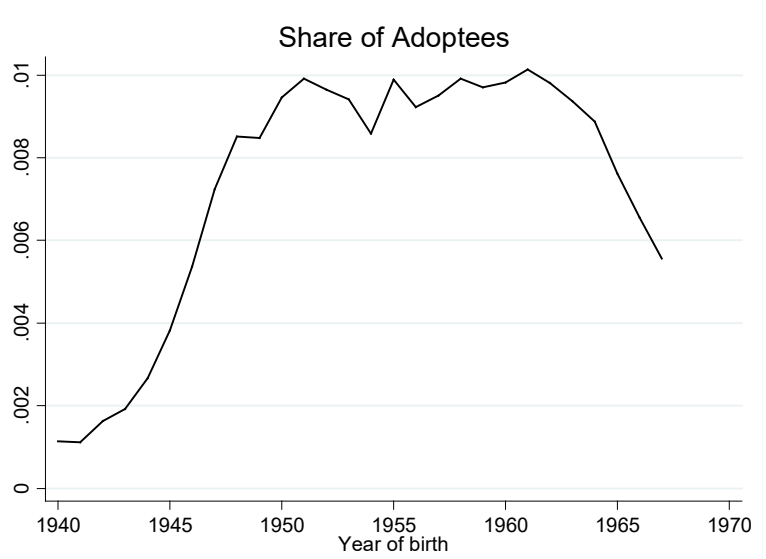
[34] The results in Table 2 are not adjusted for educational attainment or other measure of SES, which could explain the differences. We also know from previous research that adoptees have worse mental health than non-adoptees (see e.g. Miller et al., 2000). Included in digestive causes are K70 (ICD 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.

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."[35]

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.

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



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

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

Figure A2. Share of individuals in the child-generation sample who died before August, 2016.

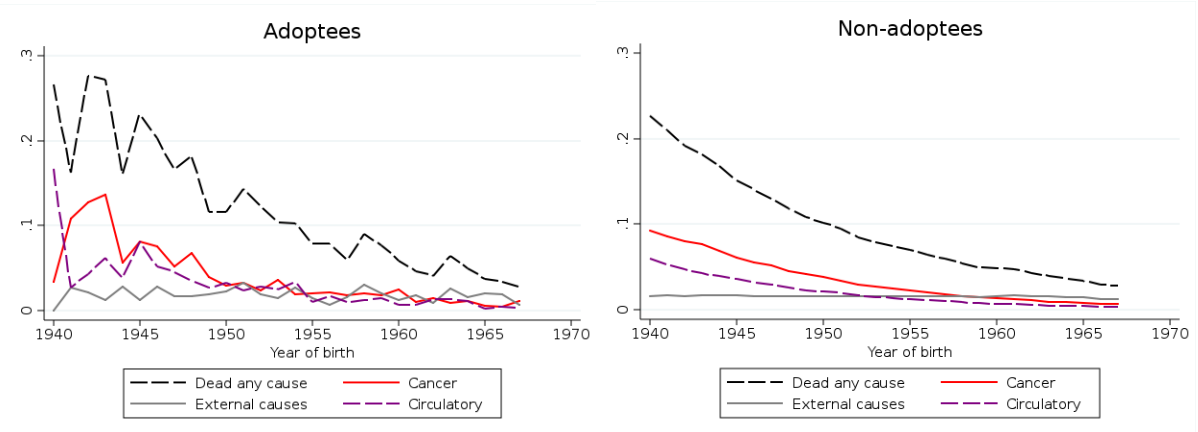


Table A1. Share of deaths by cause

	Adoptees	Non-adoptees (weighted)	*p*-values mean diff
Causes of death			
Cancer	0.278	0.329	0.0000
External causes	0.206	0.222	0.6612
Circulatory	0.215	0.180	0.1155
Digestive	0.070	0.042	0.0012
Mental	0.037	0.024	0.0169
Respiratory	0.036	0.034	0.6933
Other	0.159	0.168	0.5284
Share of deaths	0.088	0.069	0.0001
Tot # of deaths	965	258,860	

Note: 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

Table B1. Sample restrictions, mortality sample

Born in Sweden 1940-1967	Non-adoptees	Adoptees
Not adopted	3,058,697	
Adopted		65,034
Adopted by two parents		33,276
Biological mother identified	3,016,784	24,248
Date of death is not missing*	2,926,756	22,807
Not adopted by own parents	2,926,756	22,766
Adopting parents' age restriction**	2,926,756	21,356
Not dead first year	2,915,162	21,343
Biological father is identified	2,833,838	10,913
Parents are dead by August 2016	1,674,637	4,069

*Dropping observations for which we cannot observe date of death because they have migrated or is missing in the cause of death register. We define them as missing in the cause of death register if we do not observe date of death and they are born before 1913, or they are not observed in any Censuses in 1960, 1970, 1990 or 2004.
** Adopting mother's age 25-47, and adopting father's age 25-66, at birth of adopted child.

Table B2. Cox proportional hazard model estimates of the associations between child mortality and parental age at death for mothers and fathers separately and the sample of adoptees with unknown biological father

	(1)	(2)	(3)	(4)	(5)	(6)
	Non-adoptees		Adoptees			
Age at death, Bio Mother	0.9903***	0.9900***	0.9859***	0.9814***	0.9872***	
	(0.0002)	(0.0002)	(0.0038)	(0.0052)	(0.0032)	
Age at death, Bio Father	0.9914***	0.9912***	0.9904**	0.9886**		
	(0.0002)	(0.0002)	(0.0040)	(0.0050)		
Age at death, Ad Mother			0.9962	0.9962	0.9950	0.9969
			(0.0042)	(0.0054)	(0.0032)	(0.0046)
Age at death, Ad Father			0.9995	0.9998	1.0040	0.9977
			(0.0043)	(0.0054)	(0.0032)	(0.0046)
Sample	All CC	CC born <1953	All CC	CC born <1953	Children with unknown bio fathers included	CC born <1953
P-value joint significance						
Biological parents	0.0000	0.0000	0.0001	0.0001	0.0001	
Adoptive parents			0.6662	0.7874	0.1273	0.7322
Observations	1,674,637	1,126,649	4,069	2,194	5,694	2,949

Note: Results from Cox proportional hazard models. Robust 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 both children and parents. Columns (1) and (2) are based on a sample of non-adopted children, columns (3)- (5) on adoptees. Column (1) and (3) is based on a sample of children with parents that are all dead (CC) in the end of the observation period. In column (2) and (4) an additional restriction is imposed that children are born before 1953. In column (5) the sample is expanded to include adopted children with unknown biological fathers. In column (6) the sample is restricted to adoptees born before 1953 with dead adopting parents.

Table B3. Proportional hazard model (Gompertz distribution) estimates of the associations between child mortality and parental age at death

	(1)	(2)	(3)	(4)	(5)
	Non-adoptees		Adoptees		
Age at death, Bio parents	0.9818***	0.9813***	0.9761***	0.9702***	
	(0.0003)	(0.0003)	(0.0055)	(0.0072)	
Age at death, Ad parents			0.9960	0.9969	0.9947
			(0.0063)	(0.0083)	(0.0069)
Share of dead children	0.1216	0.1493	0.1301	0.1654	0.1577
Sample	All CC	CC born <1953	All CC	CC born <1953	CC born <1953
Observations	1,674,637	1,126,649	4,069	2,194	2,949

Note: Results from a proportional hazard model based on the Gompertz distribution. Robust 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 both children and parents. Columns (1) and (2) are based on a sample of non-adopted children, columns (3)- (5) on adoptees. Column (1) and (3) is based on a sample of children with parents that are all dead (CC) in the end of the observation period. In column (2) and (4) an additional restriction is imposed that children are born before 1953. In column (5) the sample is restricted to adoptees born before 1953 with dead adopting parents.

Table B4. Cox proportional hazard model estimates of the associations between child mortality and parental age at death

	(1)	(2)
	Non-adoptees	Adoptees
Age at death, Bio mother	0.8824***	0.8560***
	(0.0019)	(0.0311)
Age at death, Bio father	0.8971***	0.8815***
	(0.0019)	(0.0320)
Alive 2013, Bio Mother	0.8191***	0.9916
	(0.0050)	(0.0779)
Alive 2013, Bio Father	0.8382***	0.8405*
	(0.0067)	(0.0782)
Age at death, Ad mother		0.9811
		(0.0389)
Age at death, Ad father		0.9885
		(0.0373)
Alive 2013, Ad Mother		0.9272
		(0.0934)
Alive 2013, Ad Father		1.0083
		(0.1300)
P-value joint significance		
Biological parents	0.0000	0.0000
Adoptive parents		0.9175
Observations	2,833,838	1,0913

Note: 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 (August 2016).

Table B5. OLS estimates of associations between percentile rank of parental and child lifetime health measured by indices based on hospitalization data for mothers and fathers separately and the sample of adoptees with an unknown biological father

	(1) Non-adoptees	(2) Adoptees	(3) Adoptees – Bio father unknown	(4) Non-adoptees	(5) Adoptees	(6) Adoptees – Bio father unknown
	Hospitalization			Health index		
Bio Mother	0.0807***	0.0752***	0.0795***	0.0682***	0.0769***	0.0734***
	(0.0007)	(0.0123)	(0.0083)	(0.0007)	(0.0124)	(0.0084)
Bio Father	0.0600***	0.0691***		0.0540***	0.0500***	
	(0.0007)	(0.0122)		(0.0007)	(0.0126)	
Ad Father		0.0331**	0.0258***		0.0155	0.0167*
		(0.0131)	(0.0090)		(0.0128)	(0.0087)
Ad Mother		0.0150	0.0195**		0.0097	0.0183**
		(0.0128)	(0.0087)		(0.0127)	(0.0086)
Biological parents	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
Adoptive parents		0.0161	0.0008		0.3261	0.0116
Observations	1,937,645	6,117	13,095	1,937,645	6,117	13,095

Note: Results from OLS regressions. Robust 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 both children and parents. Columns (1) and (4) are based on a sample of non-adopted children, columns (2) and (5) on adoptees with all parents that are all known, in column (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. Both measures are ranked within cohort separately by mother, fathers, daughters, and sons.

Table B6. OLS estimates of associations between percentile rank of parental and child lifetime health measured by indices based on hospitalization data by gender

	(1) Non-adoptees	(2) Adoptees	(3) Non-adoptees	(4) Adoptees
	Women		Men	
A. Health index				
Bio parents	0.1197***	0.1160***	0.1245***	0.1361***
	(0.0013)	(0.0260)	(0.0013)	(0.0232)
Ad parents		-0.0078		0.0520**
		(0.0254)		(0.0236)
B. Hospitalization				
Bio parents	0.1456***	0.1271***	0.1359***	0.1626***
	(0.0014)	(0.0251)	(0.0013)	(0.0231)
Ad parents		0.0576**		0.0414*
		(0.0263)		(0.0239)
Observations	946,017	2,933	991,628	3,184

Note: Results from OLS regressions. Robust 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 both children and parents. Column (1) an (2) shows results for women and column (3) and (4) for men. Columns (1) and (3) are based on a sample of non-adopted children, columns (2) and (4) on adoptees. In panel A, the results for hospitalization is shown, and in panel B, the results or health index is shown. Both measures are ranked within cohort separately by mother, fathers, daughters, and sons.

Table B7. Associations between percentile rank of parental health index and firstborn grandchild's health at birth, controlling for percentile rank of child's health

	(1) Birth weight	(2) Low Birth weight	(3) APGAR<9	(4) Birth weight	(5) Low Birth weight	(6) APGAR<9
	Hospitalization			Health index		
A.Non-adoptees						
Child	0.0105***	-0.0003***	-0.0003***	0.0146***	-0.0003***	-0.0003***
	(0.0014)	(0.0000)	(0.0000)	(0.0014)	(0.0000)	(0.0000)
Bio parents	0.0077***	-0.0001***	-0.0002***	0.0240***	-0.0001***	-0.0002***
	(0.0018)	(0.0000)	(0.0000)	(0.0018)	(0.0000)	(0.0000)
Observations	623,153	623,153	570,148	623,153	623,153	570,148
B.Adoptees						
Child	0.0309	-0.0005**	-0.0000	0.0500**	-0.0005**	-0.0001
	(0.0229)	(0.0002)	(0.0003)	(0.0234)	(0.0002)	(0.0003)
Bio parents	-0.0056	-0.0000	-0.0006	0.0195	-0.0004	-0.0006
	(0.0308)	(0.0003)	(0.0005)	(0.0315)	(0.0003)	(0.0005)
Ad parents	0.0692**	-0.0006**	-0.0002	0.0711**	-0.0004	-0.0002
	(0.0316)	(0.0002)	(0.0005)	(0.0305)	(0.0002)	(0.0004)
Observations	2,152	2,152	1,964	2,152	2,152	1,964

Note: Results from OLS regressions. Robust standard errors in parentheses; *** significant at 1%, ** at 5%, * at 10%. Each column represents a separate regression, and all regressions include indicators for mother's age at birth, child gender, year of birth, and grandparents' birth cohort. Panel A is based ona sample of non-adoptees, and panel B on a sample of adoptees. The dependent variable in columns (1) and (4) is birth weight measured in grams and scaled into percentile ranks, in columns (2) and (5) it is a binary variable capturing if birth weight <2,500 grams, and in columns (3) and (6) the dependent variables is a binary variable capturing if APGAR score at 5 min is below 9.

Table B8. Cox proportional hazard model estimates of the intergenerational association in mortality for the non-adopted and adopted children with the same biological mother

	(1)	(2)	(3)	(4)	(5)
	Non-adoptees		Adoptees		
Age at death, Bio parents	0.9871***	0.9822***	0.9764***	0.9693***	
	(0.0026)	(0.0035)	(0.0061)	(0.0075)	
Age at death, Ad parents			0.9935	0.9933	0.9915
			(0.0067)	(0.0087)	(0.0073)
Sample	All CC	CC born <1953	All CC	CC born <1953	CC born <1953
Observations	15,439	6,039	3,411	1,832	2,500

Note: Results from Cox proportional hazard models. Robust 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 both children and parents. The samples are overall restricted to children with a biological mother that has given at least one child up for adoption and raised at least one child of her own. Columns (1) and (2) are based on a sample of non-adopted children, columns (3)-(5) on adoptees. Column (1) and (3) is based on a sample of children with parents that are all dead (CC) in the end of the observation period. In column (2) and (4) an additional restriction is imposed that children are born before 1953. In column (5) the sample is restricted to adoptees born before 1953 with dead adopting parents.

Table B9. Linear probability model estimates of intergenerational association of dying before age 60, 65 and 70, respectively, for the non-adopted and adopted children with the same biological mother

	(1)	(2)	(3)	(4)	(5)	(6)
	Dead < age 60		Dead < age 65		Dead < age 70	
	Non-adoptees	Adoptees	Non-adoptees	Adoptees	Non-adoptees	Adoptees
Bio parents	0.0103	0.0520***	0.0272***	0.0734***	0.0304*	0.0580
	(0.0066)	(0.0145)	(0.0097)	(0.0186)	(0.0175)	(0.0400)
Ad parents		0.0017		0.0026		0.0311
		(0.0139)		(0.0185)		(0.0423)
Mean dep var	0.0759	0.0678	0.0897	0.0806	0.0974	0.0892
Observations	12,532	4,069	6,106	2,096	2,117	470

Note: Results from a linear probability model. Robust 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 both children and parents. The samples are overall restricted to children with a biological mother that has given at least one child up for adoption and raised at least one child of her own. In columns (1) and (2), the sample is restricted so that all children are born before August 1956; in columns (3) and (4) before August 1951; and in columns (5) and (6) before August 1946. Columns (1), (3) and (5) are based on a sample of non-adopted children and columns (2), (4) and (6) on adoptees.

Table B10. Diagnoses codes for different diagnose categories

Diagnoses	ICD10 codes
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 B11. Linear probability model estimates of intergenerational association of dying before age 65, by child cause of death.

	(1) Cancer	(2) External causes	(3) Circulatory	(4) Digestive	(5) Mental	(6) Respiratory	(7) Other	(8) Preventable	(9) Treatable
Panel A. Non-adoptees									
Dead < age 65,	0.0040***	-0.0003	0.0089***	0.0013***	0.0004***	0.0009***	0.0019***	0.0021***	0.0026***
Bio parents	(0.0004)	(0.0003)	(0.0004)	(0.0002)	(0.0001)	(0.0001)	(0.0002)	(0.0002)	(0.0002)
Observations	1,147,746	1,147,746	1,147,746	1,147,746	1,147,746	1,147,7,46	1,147,746	1,147,746	1,147,746
Panel B. Adoptees									
Dead < age 65,	0.0149	0.0202**	0.0338***	0.0040	-0.0020	0.0078*	-0.0032	-0.0027	0.0110*
Bio parents	(0.0104)	(0.0083)	(0.0102)	(0.0051)	(0.0022)	(0.0043)	(0.0057)	(0.0044)	(0.0061)
Dead < age 65,	-0.0006	0.0012	-0.0079	-0.0002	0.0007	-0.0001	0.0090	-0.0010	-0.0027
Ad parents	(0.0108)	(0.0077)	(0.0082)	(0.0048)	(0.0033)	(0.0034)	(0.0077)	(0.0051)	(0.0050)
Observations	2,470	2,470	2,470	2,470	2,470	2,470	2,470	2,470	2,470

Note: Results from a linear probability model. Robust 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 both children and parents. The sample is restricted so that all children are born before August 1951. In Panel A results for non-adoptees are shown, and in Panel B results for non-adoptees are shown. The grouping of the different diagnoses is displayed in Table B10.

Table B12. Competing risk Cox proportional hazard model estimates of the associations between percentile rank of parental longevity and child mortality by child cause of death.

	(1) Cancer	(2) External causes	(3) Circulatory	(4) Digestive	(5) Mental	(6) Respiratory	(7) Other	(8) Preventable	(9) Treatable
Panel A: Non-adoptees									
Age at death,	0.9885***	0.9671***	0.9951***	0.9785***	0.9759***	0.9812***	0.9707***	0.9753***	0.9692***
Bio parents	(0.0004)	(0.0005)	(0.0008)	(0.0012)	(0.0016)	(0.0006)	(0.0012)	(0.0010)	(0.0008)
Observations	1,674,637	1,674,637	1,674,637	1,674,637	1,674,637	1,674,637	1,674,637	1,674,637	1,674,637
Panel B: Adoptees									
Age at death,	0.9822*	0.9693***	0.9678**	0.9878	0.9636	0.9938	0.9191***	1.0040	0.9552**
Bio parents	(0.0106)	(0.0101)	(0.0159)	(0.0210)	(0.0374)	(0.0122)	(0.0239)	(0.0254)	(0.0185)
Age at death,	1.0003	1.0094	0.9987	0.9717*	1.0278	0.9817	0.9577	1.0209	1.0084
Ad parents	(0.0112)	(0.0144)	(0.0183)	(0.0165)	(0.0378)	(0.0137)	(0.0283)	(0.0229)	(0.0223)
Observations	4,069	4,069	4,069	4,069	4,069	4,069	4,069	4,069	4,069

Note: Results from a Cox proportional hazard models. Robust 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 both children and parents. In Panel A results for non-adoptees are shown, and in Panel B results for non-adoptees are shown. The grouping of the different diagnoses is displayed in Table B9. The sample consists of children with parents that have all died (CC) in the end of the observational period (August 2016).

Table B13. Interaction effects between health of biological and adopting parents

	(1) Hospitalization	(2) Health index
Bio parents*Ad parents	-0.0020**	-0.0010
	(0.0008)	(0.0008)
Bio parents	0.2462***	0.1795***
	(0.0438)	(0.0445)
Ad parents	0.1391***	0.0663*
	(0.0405)	(0.0370)
Observations	6,117	6,117

Note: Results from OLS regressions. Robust 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 both children and parents. In column (1) results for hospitalization is shown, and column (2) the results for health index is shown. Both measures are ranked within cohort separately by mother, fathers, daughters, and sons.

Table B14. Sample restricted to first-born adoptees and adoptees who moved out from the municipality of birth

	(1) Hospitalization	(2) Health index	(3) Hospitalization	(4) Health index
	Different municipalities		First-borns	
Bio parents	0.1402***	0.1233***	0.1998***	0.1292***
	(0.0182)	(0.0187)	(0.0247)	(0.0245)
Ad parents	0.0446**	0.0273	0.0490*	0.0315
	(0.0190)	(0.0184)	(0.0257)	(0.0253)
Observations	5,240	5,240	2,908	2,908

Note: Results from OLS regressions. Robust 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 both children and parents. In column (1) and (2) the sample consists of adoptees with biological mothers living in a different municipality than their adopting mothers in the 1960. In column (3) and (4) the sample consists of first-born children that has been given up for adoption. Column (1) and (3) shows results for hospitalization and (2) and (4) for health index.