Second generation effects of prenatal health shocks: disentangling social from biological pathways

Bhaskar Mazumder
Federal Reserve Bank of Chicago

André Richter
Swedish Institute for Social Research
Stockholm University

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Abstract

Animal experiments show that prenatal health shocks have repercussions even several offspring generations later. In human populations, such generational spill-over effects of prenatal health shocks represent both direct biological effects and indirect effects via the parental household environment, e.g. parental socioeconomic status. The relative importance of these two effects remain unknown. In this paper, we combine the Spanish flu as an exogenous source of variation in fetal health with an adoption design to disentangle direct biological from indirect second generation effects. We exploit the fact that adoptees do not inherit health conditions from their adoptive parents, which rules out direct effects, and are not exposed to the home environment of their biological parents, which rules out indirect post-birth effects. Our results are imprecisely estimated, but seem to suggest that direct second generation effects may be positive, whereas indirect effects are negative.

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1 Introduction

The biomedical literature has documented that prenatal health shocks to animals in one generation lead to adverse health outcomes at least one offspring generation later, suggesting that prenatal health shocks spill over to future generations. These effects are thought to occur since the primordial germ cells already develop at the prenatal stage, making them directly susceptible to prenatal health shocks. Evidence for such effects in human populations is scarce though, and while animal experiments arguably isolate biological mechanisms, estimates of such effects in human populations capture not only the biological effects of health shocks on the offspring, but also indirect effects via changes in parents' socioeconomic status. This paper combines an adoption design and the Spanish flu in Sweden as a natural experiment to shed light on the relative importance of these two mechanisms in the transmission of a prenatal health shock across generations.

The Spanish flu has previously been used by Richter & Robling (2015) to estimate second generation effects of a prenatal health shock in a human population. They find that potential prenatal flu exposure of the mother lowers her daughter's educational attainment, and potential prenatal flu exposure of the father lowers his son's educational attainment. Controlling for parental socioeconomic status indicators does not change these results overall, which leads Richter & Robling (2015) to conclude that mechanisms not accounted for by parental socioeconomic status - such as a direct biological effects - might drive the results. No conclusive tests that can distinguish between direct biological or indirect effects via the socioeconomic status of the parents are provided, though.

Adoption creates a setting suitable to disentangle direct and indirect effects of health shocks. If the placements of adoptees into adoptive households occur close to birth, and if the placements are as good as random with respect to household characteristics, then a comparison of the effects on the adoptees based on which type of parent was exposed can help us understand the underlying mechanisms. This is because exposure of the adoptive parents can have an effect only via indirect mechanisms since adoptees do not inherit biological health conditions of their adoptive parents. Similarly, exposure of the biological parents can have two effects: a biological effect, and an indirect effect through the prenatal environment of the adoptees.[1] The comparison of these effects is thus informative for the relative contribution of pre- and postbirth channels. To be more precise, consider an individual i who was subject to a prenatal health shock. i's own biological children, say b, potentially experience the consequences of i's prenatal health shock via its effect on i's socioeconomic status, but also directly by potential implications on b's health. On the other hand, if i adopts a child, say a, then a only experiences the consequences of the health shock through the effect

[1] Note that in principle differences in outcomes need not reflect health consequences per se, but could be mediated by differences in parental investments based on the child's health endowment. See e.g. Almond & Mazumder (2013).

on i's socioeconomic status. Reversely, if i gives a child, say a', up for adoption, a' will only experience the health shock via prebirth factors. This includes the biological effect through the primordial germ cells, and the indirect effect of the health shock on i's socioeconomic status, which in turn may affect a''s prenatal environment. This suggests the following comparisons:

- compare adoptees with prenatally exposed adoptive parents to adoptees with unexposed adoptive parents. This identifies effects via the postbirth environment.
- compare adoptees with prenatally exposed biological parents to adoptees with unexposed biological parents. This identifies the effect via prebirth factors.
- compare children with prenatally exposed (biological and rearing) parents to children with unexposed parents. This identifies the joint effect of pre- and postbirth environments.

In this paper, we use a comparison of the three aforementioned effects to shed light on the relative importance of each potential channel. We find that our results seem to suggest that direct second generation effects may be positive, whereas indirect effects are negative. However, large estimation uncertainty prevents us from drawing firm conclusions, and our results need to be considered suggestive at best.

The remainder of this paper is organized as follows: in section 2 we discuss related literature, in sections 3 and and 4 we present the historical context of the Spanish flu in Sweden as well as the Swedish adoption setup, respectively. A description of the data source is provided in section 5. Section 6 discusses our empirical strategy and presents our empirical findings. Section 8 ends with a discussion of the results.

2 Related Literature

This paper relates to a variety of literatures. First, we rely on insights from the literature on the effects of prenatal health shocks on later life outcomes, and on the literature using the Spanish flu in particular. Second, this paper sheds light on multigenerational effects of prenatal health shocks and is thus related to this research area. Moreover, adoption data has been used previously to shed light on the relative contribution of pre- versus postbirth factors, though to the best of our knowledge we are the first to use an adoption design to study how exogenous health shocks are transmitted across generations. In the following, we review these literatures.

2.1 First generation effects

Lasting effects of prenatal health have been widely documented. See, for instance, Behrman & Rosenzweig (2004), Black et al. (2007), and Currie & Hyson (1999) and Oreopoulos et al. (2008) who use birth weight as a summary measure of prenatal health and document detrimental effects of being born with low birth weight on several later life outcomes. Apart from observational studies, a wide range of natural experiments thta exploit exogenous variation in prenatal health has been used in the economic literature. For instance, Chen & Zhou (2007), Meng & Qian (2009), Scholte et al. (2012), Neelsen & Stratmann (2011), Almond & Mazumder (2011), Ewijk (2011) and Almond et al. (2014) use nutritional deprivation due to famines or Ramadan exposure and find effects of these events on either early or later life outcomes.[2] Similar results are obtained when circumstantial evidence for stress exposure is used, e.g. exposure to civil conflict or war (Camacho, 2008; Lee, 2014; Valente, 2011; Mansour & Rees, 2012), death of a relative (Black et al., 2014; Persson & Rossin-Slater, 2014) and natural disasters (Simeonova (2009), Currie & Rossin-Slater (2013), among others). In utero exposure to environmental pollution also has detrimental effects, see e.g. Almond et al. (2009), Nilsson (2009), Black et al. (2013) and Currie et al. (2014).

The Spanish Flu Literature: In a series of papers, Douglas Almond and coauthors were the first to exploit the 1918 influenza pandemic in the US to test the fetal origins hypothesis. In Almond (2006), Almond & Mazumder (2005) and Mazumder et al. (2010), large reductions in educational attainment, wages, socioeconomic status indices and several health measures are found for the cohorts that were prenatally exposed to the Spanish flu. Brown & Thomas (2011) show however that these results are potentially confounded by a change in parental quality due to conscription procedures for World War I. Evidence from a number of non-participating countries such as Brazil (Nelson, 2010), Taiwan (Lin & Liu, 2014) and Switzerland (Neelsen & Stratmann, 2012) has

[2] The impact of nutritional deprivation at later developmental stages has also been studied. See, for instance, Kaati et al. (2007) and Berg et al. (2012).

confirmed Almond's earlier results, though. Richter & Robling (2015) furthermore document adverse effects of potential prenatal flu exposure on educational attainment and marriage market outcomes. Bengtsson & Helgertz (2015) extend the analysis of Richter & Robling (2015) to health outcomes and find that the Spanish flu in Sweden had adverse health consequences for prenatally exposed individuals.

Other noteworthy studies are Kelly (2011), Parman (2012) and Karlsson et al. (2014). Kelly uses cross-sectional variation in the Asian flu of 1957 in the UK and finds that prenatal exposure to the flu has negative effects on cognitive test score measures. Parman uses the US influenza pandemic in 1918 to identify how a health shock to a child affects the outcomes for its siblings via parental investments. Karlsson et al. (2014) uses Spanish flu mortality in Sweden as a labor supply shock to test empirical predictions of macroeconomic growth models.

2.2 Second generation effects

Evidence for multigenerational effects of prenatal health remains scarce. An intergenerational transmission of birth weight has been documented in observational studies (e.g. Currie & Moretti (2007) and Royer (2009), among others), but twin studies suggest that the genetic component here is likely to be strong (cf. Royer & Witman, 2014). Quasi-experimental evidence linking parental prenatal health to children's outcomes is limited to only a few studies. Almond et al. (2010) use the Chinese famine from 1959 to 1961 as a natural experiment and compare mothers who were in utero during the time of the famine to mothers of adjacent birth cohorts. They find that children were more likely to be girls and to have low birth weight if their mother was prenatally exposed to the famine. Kim et al. (2014) furthermore shows that junior secondary school attendance of individuals born to mothers prenatally exposed to the Chinese famine was reduced by 5-7 percentage points. Almond & Chay (2006) use the civil rights era as a natural experiment and exploit that black women born in the late 1960s experienced better prenatal and infant health than black women born in the early 1960s. They show that children of mothers who were themselves born in the late 1960s had better birth outcomes than children of mothers born in the early 1960s.[3] Richter & Robling (2015) use the Spanish flu in Sweden to estimate an intent to treat effect on educational attainment of the second generation and find that potential prenatal flu exposure lowers the offspring's education.

Evidence for multigenerational responses of health shocks is more abundant in the biomedical and epidemiological literature. See Drake & Walker (2004) and Drake & Liu (2010) for comprehensive reviews. In particular, animal experiments have produced a substantial body of evidence for multigenerational

[3] Nilsson (2009) uses prenatal alcohol exposure induced by a policy experiment in Sweden and finds no effects on birth outcomes of the children of prenatally exposed parents.

responses in health outcomes. In a recent systematic review of this literature, Aiken & Ozanne (2014) finds that out of 48 published animal experiments looking at the second generation, 44 found effects while only 4 failed to do so. In these experiments, pregnant animals are exposed to some form of stress (e.g. under- or malnutrition, or excessive exercise) and multiple generations of offspring are observed, who are then compared to a corresponding control group. Early examples are Stewart et al. (1975) and Stewart et al. (1980), who follow rats over up to 12 generations and document that adverse health effects of in utero malnourishment perpetuate over three subsequent generations even after the reintroduction of a normal diet. Similarly, Pinto & Shetty (1995) expose pregnant rats to exercise stress and show that not only their offspring but also the second generation offspring are growth-retarded despite sedentary conditions during their gestation. In human populations, multigenerational effects of the Dutch Hunger Winter of 1944-1945 (see Roseboom et al. (2011) for a review of related studies) have been documented by Painter et al. (2008), who show that children of mothers that were in utero during the Dutch Hunger Winter were more likely to suffer from atypical conditions. Epigenetic changes have furthermore been identified (Heijmans et al., 2008; Tobi et al., 2009).

A set of related studies look at food supply during the slow growth period, another critical period in human development around the age of 8-12. These studies suggest that health outcomes of individuals might be influenced by the food supply during their parents' and grandparents' slow growth period. See Kaati et al. (2002), Bygren et al. (2001), Kaati et al. (2007) and Pembrey et al. (2006), as well as Pembrey (2002) and Pembrey (2010) for overviews. While these studies can be criticized on statistical grounds (e.g. Senn (2002)), Berg & Pinger (2016) found mental health effects of the food supply during the ancestors' slow growth period in Germany, using exposure to the German famine for identification.

Compositional effects

Prenatal health shocks may have compositional effects on the second generation through fertility responses of the parents or the prenatally exposed children, but the existing evidence for such effects is mixed. Black et al. (2013) investigate the effect of prenatal exposure to radiation on the probability of having younger siblings, and find no such effect. Nilsson (2009) does not find evidence for fertility responses due to prenatal alcohol exposure either. On the other hand, Neelsen & Stratmann (2012) finds that individuals prenatally exposed to the Spanish flu were less likely to be married, a finding similar to Almond et al. (2010) for the Chinese famine.

2.3 Adoption studies

Adoption as a natural experiment has been used to study the relative importance of prebirth and postbirth factors for the intergenerational transmission of economic outcomes in a variety of settings. Studies in economics include Sacerdote

(2002), Sacerdote (2004), Plug & Vijverberg (2003), Plug (2004), Plug & Vijverberg (2005), Björklund et al. (2006), Björklund et al. (2007), and Hjalmarsson & Lindquist (2013) and Lindahl et al. (2016). The underlying idea is that while parent-child associations based on representative data capture both pre- and postbirth environments, associations based on adoptee data only capture the importance of the postbirth environment. A comparison of both associations is thus informative for the relative importance of these two factors.

Few of these studies have looked at the transmission of health outcomes. Sacerdote (2004) uses data on Korean adoptees to American families and finds that adoptive parents transmit health habits such as smoking and drinking equally to adoptees and to own biological children, whereas height and obesity are exclusively transmitted to the latter. Lindahl et al. (2016) use Swedish adoption data containing information on both the biological and adoptive parents to study the formation of adult health and mortality, as measured by parental longevity and the children's mortality risk. They find that health outcomes are transmitted from biological parents to children, but not so from adoptive parents to adoptees.

The use of adoption as a natural experiment relies on a variety of assumptions, including random assignment of adoptees to adoptive families, early timing of adoption and generalizability of adoptees to the general population. Using data containing information on both adoptive and biological parents of the adoptees, Björklund et al. (2004) evaluate the validity of these assumptions for Sweden and argue that potential biases due to violations of these assumptions are likely to be small. Björklund et al. (2006) furthermore show that the placement of adoptees appears non-random along dimensions such as income and education, but that this affects estimated intergenerational transmission parameters only marginally. Note that the Swedish setting has also been used by Hjalmarsson & Lindquist (2013) for crime outcomes, Lindquist et al. (2015) for entrepreneurship, and Lindahl et al. (2016) for health outcomes.

All of the above mentioned studies look at associations between parental characteristics and offspring outcomes. To the best of our knowledge, we are the first study in a human population that uses an adoption design combined with a natural experiment to look at how an exogenous (health) shock is transmitted across generations.

3 Historical context

3.1 The Spanish flu as a natural experiment

We follow the setup in Richter & Robling (2015) and use the Spanish flu as an exogenous source of variation in the parental generation's prenatal health. Four characteristics of the Spanish flu facilitate its use for this purpose: First,

it was a severe health shock. Over the course of the pandemic, at least 10% of the Swedish population had been infected, and some figures indicate that about 25% of all women of child-bearing age between 20 and 30 were affected.[4]

Second, the Spanish flu happened unexpectedly and its timing as well as its intensity was unforeseen by authorities and medical professionals of that time, see e.g. Barry (2005). In Sweden, the medical community started to raise concerns as late as August 1918, but these were largely ignored by the authorities who did not believe the Spanish flu to be a substantial threat (Åhman, 1990). See also Karlsson et al. (2014) and references therein for an excellent overview regarding the Swedish case.

Third, the pandemic ended after just a couple of months, which lends credibility to a birth cohort design. This can be seen in figure 1, where we plot the aggregate influenza morbidity per month. As in other countries, we see a distinct and relatively sudden spike in influenza morbidity in the last quarter of 1918, which exhibits an equally sudden drop to almost normal levels in the beginning of 1919. We see two smaller waves of influenza infections, though. One shortly after the peak in 1918, and one in the beginning of 1920.

Fourth, it is widely believed that the Spanish flu was a socially neutral disease and infected people essentially at random. This is important insofar as a social gradient would imply that resulting estimates are confounded by the social origin of those who got infected. In this respect, note that while regular influenza strains mainly affect vulnerable populations (e.g. the very young, the elderly, and immunocompromised individuals), the Spanish flu affected primarily healthy adults. For this reason, contemporary scientists even argued whether the Spanish flu was a flu at all (Barry, 2005).

3.2 World War I and parental quality

The Spanish flu episode is an attractive historical event for the study of prenatal insults, but its validity relies on the assumption that the timing of the flu does not coincide with any other historical event which could potentially confound the effects. Since the flu episode occurred during the end of 1918 and lasted until the beginning of 1919, it is quite natural to ask if the effects of the Spanish flu might be confounded by the end of World War I in November 1918. Even though Sweden remained neutral during the war, the repercussions of the hostilities in Europe certainly affected the country. Most importantly, maritime warfare and trade blockades interfered with imports to Sweden, which led to a general scarcity of certain goods, and in combination with poor harvests in 1917 also to a food shortage in that year (Montgomery, 1955). Moreover, Sweden and

[4] While the official records by Medicinalstyrelsen (*National Board of Health*) indicate that roughly 10% of the Swedish population had been infected, it also reports that this is likely a lower bound (Medicinalstyrelsen, 1920).

Norway as non-participating countries were surrounded by opposing war participants, and it seems likely that concerns about Sweden's safety emerged in the population. Contemporary political events support this conjecture. For instance, in February 1914, when political tensions between the participating countries grew and war was widely anticipated, plans of the Swedish government to reduce the defense budget were an important factor contributing to the "Courtyard Crisis" (*Borggårdskrisen*), in which 32000 farmers gathered in Stockholm to protest against the government's plans, demanding higher defense spending instead.

It is likely that fear of war involvement and the economic hardship due to WWI has affected parents' fertility decisions. An indication for this is the evolution of the cohort size, which is depicted in figure 2. The important aspect to note is the sharp increase approximately one year after the influenza peak, whose timing coincides with the end of World War I (plus 9 months) as indicated by the shaded area.[5] Taking the evolution of the cohort size at face value, we suspect that the dramatic increase in fertility starting 9 months after the armistice reflects deferred fertility.[6] Most importantly, individuals conceived in late November onwards experienced the Spanish flu as a prenatal insult at an early stage during pregnancy, but are potentially born to parents who decided to wait because of the war.

If there was a social gradient in deferral behavior, then Brown & Thomas (2011)'s argument that the Spanish flu coincides with a change in the quality composition of parents also applies in Sweden. Unfortunately, we have no individual level data on the parents of the cohort born then, but historical population statistics contain the mother's marital status per month. The fraction of in-wedlock births increased for births conceived during the WWI period from about 83% to 88%, as shown in figure 3. Taking marital status as an indicator for parental quality, this suggests that, if anything, parental quality *increased* rather than decreased during the war. Hence, individuals conceived during the war are potentially born to parents of better quality. The drop after the end of WWI can either be due to a normalization of fertility behavior, or it could reflect family disruption due to the flu, i.e. pregnancies of initially married couples where the husband died due to the flu. Either case implies that individuals conceived after the armistice were "worse off" compared to individuals conceived before.[7]

It seems safe to say that a comparison involving war and post-war times is difficult to make, but the fact that individuals conceived in November and December 1918 might have experienced the flu as a prenatal insult requires us to find a

[5] The sharp drop in cohort size in May and June 1919 can be explained by family disruption and an increase in miscarriages due to the flu. For the former, see Åhman (1990). For the latter, note that in aggregate annual data on miscarriages, defined as the end of a pregnancy before the end of the second trimester, we find an increase for 1919. No increase in the number of stillbirths are found, though. See Richter & Robling (2015).

[6] See also Mamelund (2004) for an alternative interpretation.

[7] Note that this applies to the exposed group, but also to the comparison group.

compromise. In our analysis, we will therefore focus on the period from 1916 up to the third quarter of 1919. We thus capture everyone conceived during WWI or the Spanish flu episode while excluding people conceived both after the peak and after the armistice.

4 The Swedish Adoption System 1940-1967

In the following we will highlight key features of the Swedish adoption system which was in place during 1940 to 1967. More comprehensive reviews can be found in Björklund et al. (2006) and Hjalmarsson & Lindquist (2013) as well as Lindahl et al. (2016). This section is based on these references.

The Adoption Process: The Swedish adoption process during the relevant time period was handled by local social authorities and decided by a formal court ruling. The biological mother contacted the local authority which initiated the process, but the final decision to give up the child could only be taken after the mother recovered from delivery. Both biological parents had to consent to the adoption if they were married, and only the biological mother otherwise. After birth, the child in question was placed in a special nursery home and a health screening took place. Children with physical or mental health conditions that were thought to be hereditary were excluded from adoption and kept either in foster or institutional care instead.[8] After a positive health screening and once the biological mother (and father, in case they were married) made the decision to complete the adoption, the child was placed into an adoptive household for an initial trial period of three to six months. For the majority of cases, this initial placement occurred within one year of birth. After the trial period, the adoptive parents filed an adoption request with the local court, which then ruled on the request after consultation with the local social authority.

The adoptive parents were selected by social workers from a pool of parents who registered with the local social authority as seeking to adopt. Guidelines for the selection of adoptive households stated that adopting parents should be married, at least 25 years old, but young enough to be the biological parent. They were required to have adequate housing and the adoptive father should have a steady income. It was furthermore required that they should be childless and not expect to have a child in the future, though this condition was abolished in 1944. Few conditions rendered a family ineligible to adopt, among which were sexually transmitted diseases and tuberculosis.

The local authority also advised against placing adoptees into families with alcohol problems, mental illnesses or criminal records, but discretionary judgment of the corresponding social worker based on the timing and severity of these conditions was allowed. It was furthermore specified that adopting parents should

[8] According to Bohman (1970), hereditary conditions refer to whether or not the biological parents exhibited a marked social or mental handicap, including alcoholism, criminality, mental disease or low intelligence.

resemble biological parents along dimensions such as height, eye and hair color, though finding parents who were able to provide a positive environment for the child was prioritized. Up until 1959, adoptions could be canceled by mutual consent between the adoptive parents and the adoptee, in case of misbehavior of either party, or in case of major health problems and defects of the adopted child. Such cancellations were rare though, see Lindahl et al. (2016).

Individual Characteristics: Most adoptions during the relevant time period were domestic, and only few international adoptions took place. We only consider domestic adoptions in this paper. The biological mother giving up her child for adoption was typically an unmarried young women with few economic resources. The biological father was typically unknown or came from a socioeconomically disadvantaged background. The adoptive parents, on the other hand, were marginally more advantaged due to the selection criteria described above. It is worthwhile pointing out, though, that the positive selection of adoptive parents during the relevant time period is not nearly as strong as it is the case with modern adoptions.

The health status of the adoptee is a particular concern. While the biological parents' low socioeconomic status would suggest that at least the prenatal environment of the adoptees was suboptimal, the health screening that took place before the adoption was finalized implies that adoptees represent a positively selected subset of an overall negatively selected group. Importantly, the screening focused on potentially hereditary conditions such as schizophrenia or other mental illness. A large medical literature on prenatal influenza exposure has established a link between prenatal exposure and the incidence of schizophrenia and related conditions. See e.g. Brown & Susser (2008), Brown & Derkits (2010), Parboosing et al. (2013), and Canetta & Brown (2012).

We do not know how many children were filtered out in this process, but data from smaller communities may be indicative. For instance, Bohman (1970) studies a sample of children born in two consecutive years in Stockholm whose mothers applied to the adoption agency during pregnancy to give them up for adoption after birth. Out of 624 such children, 164 were eventually placed into adoptive homes by the corresponding authority in Stockholm. For 64 of the remaining children, parental consent to the adoption was also given, but they were considered unsuitable for adoption on account of the health screening.[9] This implies that for 228 cases the parents decided to give up their children for adoption via the social authority in Stockholm[10] but 28% did not pass the medical screening. We do not know if this fraction is representative for places other than Stockholm, but this rather large fraction stresses that our estimates are based on positively selected samples.[11]

[9] Medical indications were reported for 25 cases, hereditary reasons for 45 and late psychomotor development was indicated 32 times, with multiple indications per child possible.

[10] Bohman (1970) indicates that another 126 children were placed in adoptive families without the social authority.

[11] The same positive selection applies to adoptive parents, as conditions such as mental illnesses made it less likely that a family was chosen as adoptive household. We lack information

However, we reason that this selection renders our estimates conservative. This notion is corroborated by Bohman (1971), who compares schooling outcomes of a subset of the above-mentioned 624 children based on their mode of placement. He finds that a group of children that consist of about 50% of children deemed unsuitable for adoption[12] performed worse in school at age 10-11 in comparison to both their classmates but also to adopted children and to children who stayed with their biological mother despite an initial adoption request, i.e. children whose mothers changed their mind after delivery.

on the importance of the selection for adoptive households, though.

[12]The remaining 50% represent *otherwise placed* and foster children.

5 Data

Administrative register data are available in the Swedish Multigeneration Register, administered by Statistics Sweden (SCB, 2011). These data contain information on all individuals born in Sweden from 1932 onwards, as well as on their biological and, if applicable, adoptive parents. We use a 35% draw from this sample, which is based on so-called index individuals born between 1932 and 1967 and all adoptees available in this sample. We restrict the sample of adoptees to include adoptees born between 1932 and 1967 that were adopted by both parents, i.e. individuals who were adopted by only one parent are excluded. Years of education are constructed from data in the 1970 and 1990 census and the education registers from 1999 and 2003. If the educational information differs across censuses or registers, we use the highest value.

As in Richter & Robling (2015), we consider the impact of potential exposure to the Spanish flu on educational attainment. The Spanish flu occurred at the end of 1918 and the beginning of 1919, implying that individuals born in the first, second or third quarter of 1919 were likely to have been prenatally exposed to the Spanish flu in the third, second and first trimester, respectively. Individuals born in previous cohorts serve as a control group. In this respect, we restrict our attention to the period between January 1916 and September 1919.

In total, 23496 individuals born between 1932 and 1967 have both an adoptive mother and an adoptive father. Since we infer potential prenatal flu exposure by the timing of birth, we need to restrict our samples to adoptees with known parental time of birth. For 8% of the sample, the time of birth of the adoptive mother, and for 9% the time of birth of the adoptive father is missing. Similarly, for 26% the identity of the biological mother, and for 54% the identity of the biological father is missing. Information on the time of birth for the biological parents is partially missing, so that we have information on time of birth of the biological mother for 65% of the sample, and for 41% for the biological father. Table 1 shows the number of adoptees for which time of birth information is available and where the parents were born in the time period of interest.

Sample sizes are depicted in figures 4 and 5. Figure 4 displays sample sizes by year of birth of the corresponding child. Note that the majority of adoptees are born after 1940. Figure 5 presents sample sizes by parental quarter of birth, expanded until 1921 to facilitate a comparison with surrounding cohorts. For the adoptees, we present these sample sizes as a fraction of the cohort size of the non-adoptee sample to gauge potential selection effects. We find that although there is a small spike for adoptive mothers born in the third quarter of 1919, the corresponding fractions are well-aligned with surrounding cohorts.

The main aim of this paper, to distinguish between direct and indirect effects, relies on the assumption that flu exposure of biological parents is uncorrelated with flu exposure of adoptive parents. For the subset of individuals for which we have information on the time of birth of both adoptive and biological parents,

we can assess if flu exposure tends to occur simultaneously. As can be seen in table 1, only very few such cases exist. Even in the full sample of all adoptees, the biological and adoptive parents' quarter of birth is identical only for 0.7% for mother-pairs and 0.55% for father-pairs. Although we cannot rule out that flu exposure between adoptive and biological parents correlates in cases where we lack information on the biological parents, we take these figures as evidence that exposure across parental types is sufficiently uncorrelated.

Descriptive Statistics: Descriptive statistics are presented in table 2. The first three columns present sample statistics for women, and the last three columns present statistics for men. In each block, we present information on the representative (non-adoptee) sample in the first column. The second column contains information on the adoptees with known adoptive parents and the third column contains information on the adoptees with known biological parents.

Comparing adoptees and their adoptive parents to the non-adoptee sample, notable differences are that adoptive parents tend to be about 3-4 years older than the parents of the non-adoptee sample. Similarly, a comparison of adoptees and their biological parents to the non-adoptee sample reveals that adoptees' biological parents tend to be somewhat less educated. Comparing adoptive parents to adoptees' biological parents shows that adoptive parents are on average more educated and older. However, all above mentioned differences are well within one standard deviation of the corresponding variables, which reinforces the notion that differential selection in the corresponding time period is less of a concern than it is in more recent years.

Table 2 furthermore shows that adoptees for which we have information on biological parents are not noticeably different from the full set of adoptees, though they were born somewhat earlier. Moreover, a comparison of women and men reveals that there are very few and only marginal gender differences in outcomes.

Sample Restrictions: The choice of sample restrictions in our setting deserves particular attention. We need to construct a sample ensuring that the potentially exposed group is compared to a suitable control group, and as outlined in section 3, we have argued that individuals (i.e. parents) conceived after the end of World War I do not comprise a suitable control group. This would require us to restrict the time of birth of both parents simultaneously, as in Richter & Robling (2015).[13] Restricting the time of birth of both parents diminishes the sample size in our adoption sample quickly, though. For this reason, we keep the time of birth of the partner unrestricted in our preferred specification.

[13] An objection to this approach may be that prenatal exposure of one parent might affect the time of birth of the other parent via changes in marriage market outcomes. While Richter & Robling (2015) found that partner quality was affected by prenatal exposure, they did find no evidence for an effect on the likelihood that the other partner was born in the corresponding time period (not reported).

6 Empirical strategy

To establish baseline results, we estimate the following equation:

$$y_i = \alpha + \beta_1 q_{1i}^{1919} + \beta_2 q_{2i}^{1919} + \beta_3 q_{3i}^{1919} + \gamma_i + x'\eta + \epsilon_i \tag{1}$$

where y_i denotes the outcome of individual i, q_{ji}^z are indicators for individual i's parent being born in quarter j of year z. γ_i are fixed effects for the time of birth of individual i, measured in five year intervals. x indicates a vector of control variables, such as a gender dummy. Note that we define exposure by the time of birth, which means that $\beta_i, i = 1, 2, 3$ capture intent-to-treat effects.

We estimate equation 1 in three different samples: the representative non-adoptee sample; the sample of adoptees with known adoptive parents, where exposure correspondingly indicates exposure of the adoptive parent; and the sample of adoptees with known biological parents, where exposure indicates exposure of the biological parent. Our main outcome of interest y_i is educational attainment, measured by individual i's years of schooling.

Equation 1 aids presentation of differences in coefficients, but for the purpose of statistical inference, it is preferable to reformulate equation 1 as follows:

$$y_i = \alpha + \sum_{j=1}^{3} \beta_j q_{ji}^{1919} + \sum_{j=1}^{3} \delta_j q_{ji}^{1919} * adoptee_i + \gamma_i + x'\eta + \epsilon_i \tag{2}$$

where $adoptee_i$ indicates individual i's adoptee-status. We estimate equation 2 on different combinations of the three above-mentioned samples: the first combines the non-adoptee sample with the sample of adoptees with known adoptive parents, the second combines the non-adoptee sample with the sample of adoptees with known biological parents, and the third combines both adoptee samples. In the latter case, the sample of adoptees with known adoptive parents is used as reference group, and the adoptee-interaction refers to the sample of adoptees with known biological parents. In this setup, standard statistical tests for $\delta_j, j \in \{1, 2, 3\}$ can be used to determine if there are statistically significant group differences.

A concern common to adoption studies is that the different types of parents may be drawn from different parts of the parental distribution, so that differences in parameter estimates may be confounded by treatment effect heterogeneity. As we have shown in section 5, observable differences between biological and adoptive parents are small, so that the quantitative importance of this concern remains unclear. The literature typically addresses this problem by controlling for the propensity to become an adoptive parent, but the potential endogeneity of the propensity score renders this strategy problematic in the current context.

To provide more comparable estimates, we therefore complement the above analysis with within-family comparisons. We exploit the fact that some adoptive parents also have biological children, and some biological parents of adoptees have children that were not given away for adoption.[14] While these parents may be different from the full sample of parents, restricting the sample to parents that have at least one of each offspring type implicitly controls for the adoption propensity. We can therefore estimate equation 1 on this sample to produce internally consistent estimates.

Within-family comparisons furthermore facilitate a distinction between direct and indirect effects if we assume that indirect effects are similar for children growing up in the same household. For instance, consider adoptees and their non-biological siblings. If their rearing parent was prenatally exposed to a prenatal insult, then the adoptee is affected by indirect effects, whereas the biological child is affected by both indirect and direct effects. The difference thus reveals the direct effect. Similarly, adoptees with prenatally insulted biological parents experience direct effects only, whereas their biological non-adoptee siblings experience both indirect and direct effects. The difference thus reveals indirect effects.

This idea can be exploited in a difference-in-difference setting, which also accounts for natural differences between adoptees and non-adoptees. That is, we compare the difference between adoptees and non-adoptees for prenatally insulted parents to the corresponding difference in unexposed households and estimate the following equation[15]:

$$y_{ik} = adoptee_i + \sum_{j=1}^{3} \beta_j q_{ji}^{1919} + \sum_{j=1}^{3} \delta_j q_{ji}^{1919} * adoptee_i + \gamma_i + \mu_k + x'\eta + \epsilon_{ik} \quad (3)$$

where μ_k refers to a fixed effect indicating that individual i grew up in household k. $\delta_j, j \in \{1, 2, 3\}$ refer to the difference-in-difference parameters of interest. All other variables are defined as above.

7 Results

In table 3, we present results for parental exposure on offspring's educational attainment. The first column reports baseline results for a representative non-adoptee sample of Swedes whose biological parents were potentially prenatally exposed to the Spanish flu. The second and third columns report estimates for exposure of the adoptees' adoptive and biological parents, respectively. The upper panel indicates maternal exposure, and the lower panel indicates paternal exposure.

[14] See table 8 for the relevant frequency distributions.

[15] To be precise, we observe relationships via legal status, i.e. we observe that someone is registered as the adoptive or biological parent. We do not observe actual cohabitation.

In the non-adoptee sample, our baseline estimates indicate that potential parental exposure in the second trimester lowers their offspring's educational attainment. For the sample of adoptees, we find a similar effect of potential second trimester exposure of the adoptive mother, though with a larger magnitude. The magnitude for the adoptive father is more aligned with the counterpart in the non-adoptee sample, but it is imprecisely estimated and null effects can thus not be ruled out. For the effect of potential exposure of the adoptees' biological parents, we do not find any statistically significant effect due to large estimation uncertainty. However, it is noteworthy that the point estimates for second trimester exposure are large and positive, which suggests that if we take these point estimates at face value, biological effects could be positive.

To gauge statistical significance of the group differences, we present regression results according to equation 2 in table 4, where standard t-tests on the interaction terms represent formal tests for group-wise differences. We see that while the point estimates indicate meaningful differences, standard errors are large and with one exception none of the interactions are statistically significant, implying that we cannot rule out that there are no differences between groups.

In table 5, we present results from estimating equation 1 on a sample based on parents with at least one adoptee and non-adoptee. The first two columns refer to adoptive parents. The last two columns refer to the biological parents of an adoptee who have at least one biological child that is not registered as an adoptee. We first report the effects for maternal and then for paternal exposure, and for each first the effect on the biological offspring growing up in the same household and then the effect on the adoptee.

Overall, we find only one statistically significant effect, which corresponds to the effect of biological mothers' first trimester exposure on adoptees. All other estimated coefficients are statistically insignificant, so that definite statements are difficult to make. Having said that, given the results of table 3, paying some attention to the magnitudes for maternal second trimester exposure seems worthwhile. In particular, the effect for exposure of the adoptive mother on the adoptee is large, negative and similar in magnitude to the results in table 3. The corresponding effect on the biological child is, however, large and positive. If we take these estimates at face value, they seems to suggest that indirect effects are negative, while direct effects are positive. The same holds true for mothers with both types of biological offspring: the effect on the biological child that was given up for adoption as well as the effect on the biological child that stayed with the mother are positive.[16]

In table 6, we present results from difference-in-difference estimates. The first two columns indicate the interaction effects for exposure of the adoptive par-

[16] While it is surprising to see a positive effect for the latter since it was exposed to both direct and indirect effects, one explanation could be that the home environments in the exposed and unexposed groups are more similar due to negative selection of mothers that give up children for adoption. This would imply that direct effects are more pronounced.

ents, i.e. we compare the difference between adoptees and their non-biological siblings in exposed households to the difference between adoptees and their non-biological siblings in unexposed households. The last two columns indicate interaction effects for exposure of the adoptees' biological parents, i.e. we compare the difference between adoptees and their biological siblings for exposed biological parents to the difference between adoptees and their biological siblings for unexposed biological parents. None of the estimated coefficients is statistically significant, so that firm conclusions cannot be drawn from table 6. However, in line with previous results, the interaction terms for exposure of adoptive mothers are large and negative, suggesting potentially larger differences between adoptees and their non-biological siblings in these households.

Discussion:

Notwithstanding questions of statistical significance, the unexpected signs and magnitudes of our point estimates lead to the question as to whether flu exposure might have induced selection effects which could explain the patterns found above. Richter & Robling (2015) have discussed selection effects of the Spanish flu for the general population, but the adoption setting requires a separate discussion. To start with, the adoption process involved a medical screening that would divert children with hereditary conditions to foster- or institutional care instead of adoptive families. While this might be one explanation for the positive effects of exposure of the biological parents, it does not explain the large negative effect of adoptive mothers' exposure, nor the large positive effects on adoptees' non-biological siblings.

Fertility effects of prenatal flu exposure may provide an alternative explanation: Suppose severe prenatal flu exposure leads to infertility. Then, the biological parents that we observe and that give their children up for adoption would be positively selected since the most severely affected individuals would not have had any children. Along similar lines, women with impaired fertility may be more likely to seek adoption of a child to satisfy the desire to raise children, implying that the adoptive mothers are negatively selected. The subset of these women who then conceive a biological child after an adoption took place might channel parental investments into the biological child rather than the adoptee, which may explain the divergent signs. Note that this explanation hinges on the idea that adoption took place before the birth of a biological child.

To test these ideas, we estimate versions of equation 1 on the level of the parent, reported in table 7. The first two columns indicate whether or not parents were more likely to adopt a child and the third and fourth columns indicate whether parents were more likely to give their child up for adoption. The third and fourth column indicate whether adoptive parents had at least one biological child. The last two columns indicate whether the biological child was born after the adoptee. The latter is estimated for the subset of adoptive parents who have biological children.

For adopting a child, we find a significant effect of first trimester exposure of

women, but no effect in the second trimester that would correspond to our earlier results. Exposure in the first and second trimester increases the likelihood of giving a child up for adoption, although figure 5 suggests that this is due to a general increase in observing biological parents of adoptees, which is supported by the fact that these results are not robust to the inclusion of a linear trend as a control variable (not reported). Focusing on adoptive parents, we find that second trimester exposure increases the likelihood of also having a biological child. For the sample of adoptive parents with both types of children, we find no evidence that the biological child was conceived after the adoption took place, though.

While these results indicate that prenatal flu exposure may potentially have induced parents to give their children up for adoption, they do not explain the large negative effect of second trimester exposure of adoptive mothers. In particular, second trimester exposure does not seem to increase the likelihood of adopting a child and adoptive parents are even more likely to have own biological children. These children are, however, not necessarily born after the adoption took place. Overall, these results are difficult to reconcile with the notion that negative selection due to impaired fertility explains the unexpected pattern found in the previous section.

Overall, it is difficult to draw concrete conclusions from our results due to the lack of statistical power, so that the signs and magnitudes of our results can be considered suggestive at best. However, if we disregard questions of statistical significance, our results seem to suggest that direct biological second generation effects may be positive, while indirect second generation effects may be negative. Alternative explanations based on the notion of impaired fertility are not supported by our results.

8 Conclusion

The biomedical literature has documented that prenatal health shocks in one generation can spill over to future generations, but most evidence for such effects come from animal experiments. Evidence in human populations is scarce, and while animal experiments arguably isolate biological mechanisms, estimates of such effects in human populations capture not only direct biological effects of health shocks on the offspring, but also indirect effects via changes in parents' socioeconomic status.

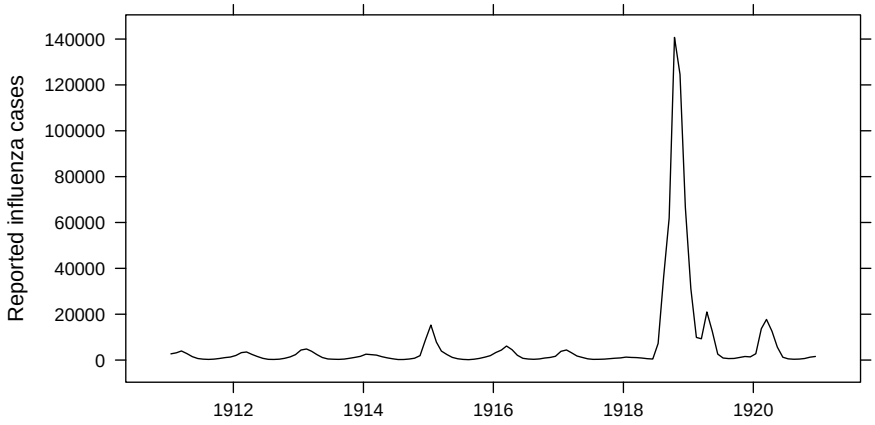
Richter & Robling (2015) have previously used the Spanish flu in Sweden to estimate second generation effects of a prenatal health shock in a human population. We extend their analysis with an adoption design to shed light on the relative importance of these two mechanisms in the transmission of a prenatal health shock across generations. Adoption creates a setting suitable to disentangle direct and indirect effects of health shocks since adoptees only experience

direct biological effects when their biological parents were prenatally exposed. Similarly, when adoptive parents were exposed to a prenatal insult, adoptees only experience indirect but no direct effects.

We find a negative effect of maternal exposure on offspring's educational attainment, and the corresponding magnitude is larger in the sample of adoptees, although the group difference is not significant due to large estimation uncertainty. In general, the combination of a natural experiment with an adoption framework is very data-demanding. Natural experiments require narrow sample restrictions to ensure that control and treatment groups are sufficiently similar, while the adoption framework is limited by the number of adoptions. For these reasons we lack statistical power to draw firm conclusions about the relative importance of direct and indirect effects and can only present results that need to be considered suggestive at best. With this caveat in mind and taking signs and magnitudes at face value, our results suggest that direct biological second generation effects may be positive, while indirect second generation effects may be negative.

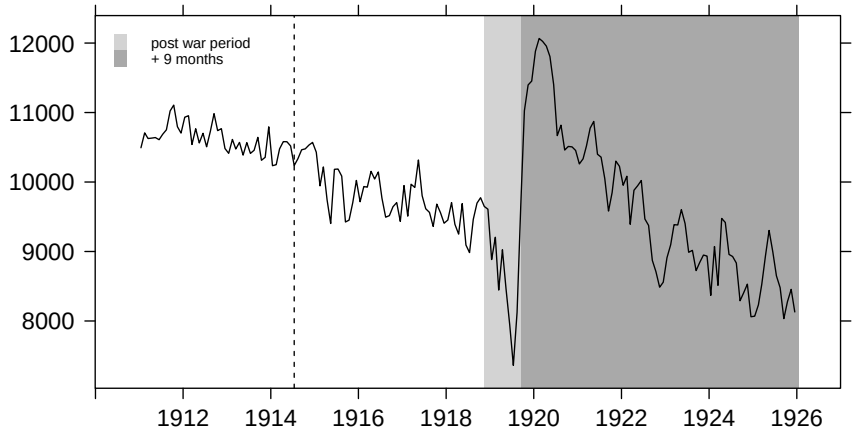
Positive biological second generation effects are in line with arguments brought forward by e.g. Pembrey et al. (2014), though more research is needed to confirm positive second generation effects. In particular, future research needs to overcome issues of statistical power due to the combination of a natural experiment and adoption setting.

Figure 1: Influenza morbidity by month in Sweden, 1911-1920



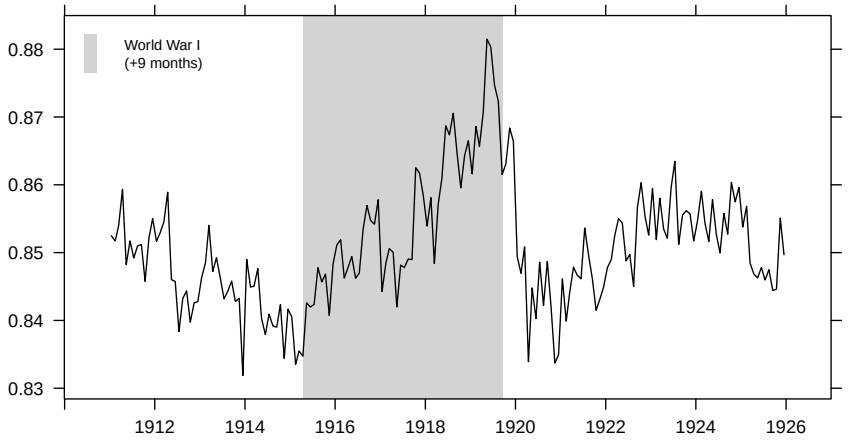
Source: Medicinalstyrelsen 1911-1920

Figure 2: Cohort size of newborns in Sweden, 1911-1925



Note: Adjusted for (pre-1918) seasonal patterns. The dashed line indicates the beginning of World War I. *Source:* Statistics Sweden

Figure 3: Births in wedlock (in %) 1911-1925



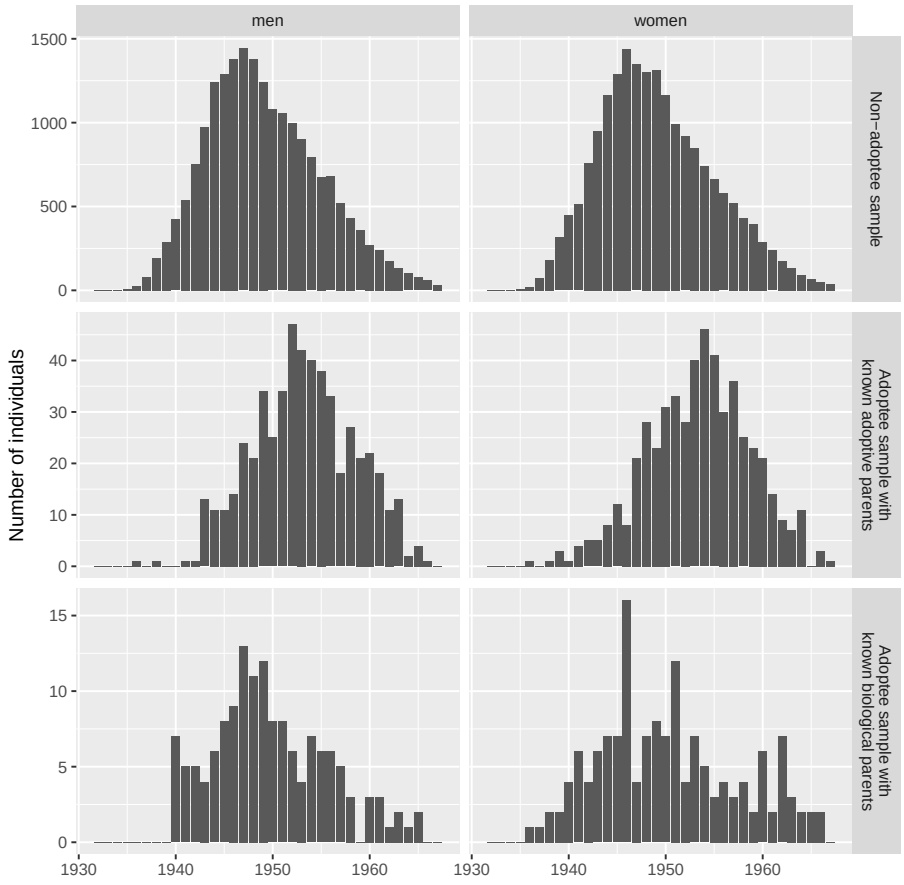
Source: Medicinalstyrelsen 1920. Adjusted for seasonal effects.

Table 1: Adoptee Sample

Number of adoptees:	23496		
	adoptive	biological	both
Mother unknown (%)	0.08	0.35	0.04
Father unknown (%)	0.09	0.59	0.07
Parents born 1916-Sep 1919:			
mother	2545	624	88
father	2732	687	86
both	753	61	2
Mother born in:			
q1 1919	175	32	0
q2 1919	153	55	1
q3 1919	190	53	2
Father born in:			
q1 1919	176	45	0
q2 1919	188	58	1
q3 1919	186	56	1

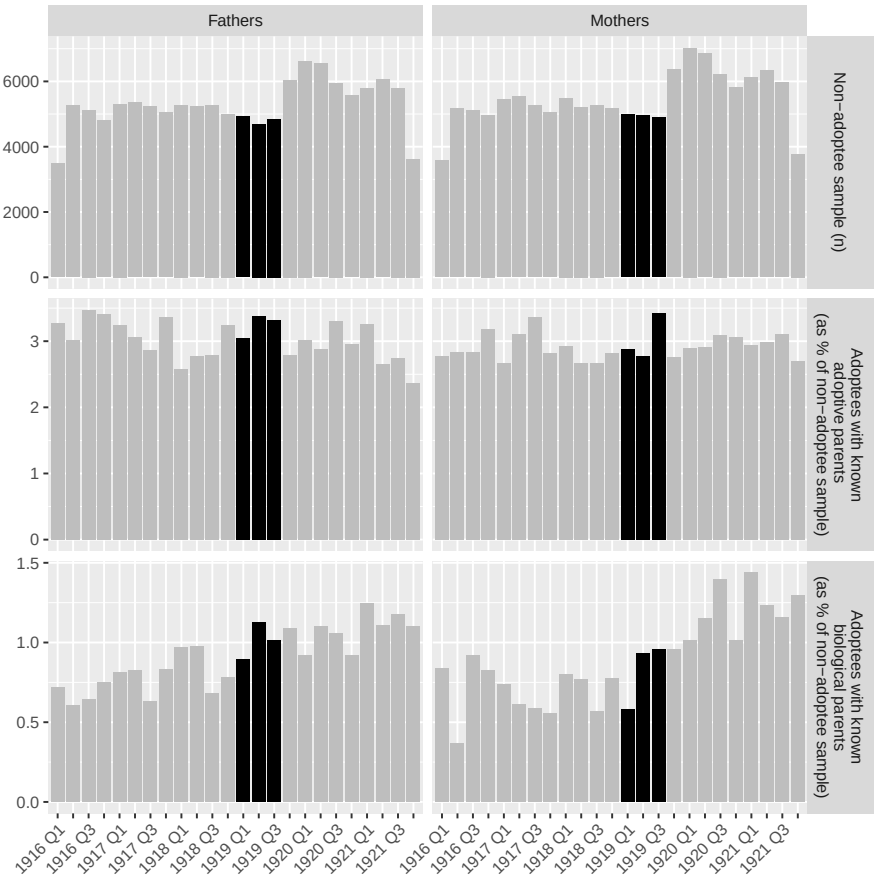
Sample size information on adoptive parents as well as biological parents of the adoptees born between 1932 and 1967. The first column refers to the adoptive parents of the adoptees, and the second column refers to the biological parents in case they are known. The third column refers to the subset of adoptees where both the adoptive and biological parents are known. Note that *unknown* above indicates that either the identity or the time of birth of the corresponding parent is missing.

Figure 4: Sample sizes by year of birth



Number of individuals with at least one parent born in the period from January 1916 to September 1919, by year of birth. The upper panel indicates the reference group. The middle and lower panel indicates adoptees with at least one adoptive or biological parent born during the relevant time period.

Figure 5: Cohort sizes by parental quarter of birth



Sample sizes by parental quarter of birth. The upper panel indicates absolute sample sizes for the non-adoptee group. The middle and lower panel indicate adoptees with known adoptive parents and adoptees with known biological parents as a percentage of the cohort size in the non-adoptee sample, respectively.

Table 2: Descriptive Sample Statistics

	Women			Men		
	Non-Adoptees	Adoptee Sample		Non-Adoptees	Adoptee Sample	
		adoptive parents	biological parents		adoptive parents	biological parents
Years of schooling	11.47	11.45	11.20	11.27	11.27	11.15
	(2.71)	(2.33)	(2.38)	(2.88)	(2.35)	(2.65)
Birthyear	1948	1952	1949	1948	1952	1949
	(6)	(6)	(7)	(6)	(6)	(6)
Years of schooling mother	8.19	8.53	7.98	8.20	8.51	7.90
	(2.08)	(2.30)	(1.84)	(2.09)	(2.29)	(1.83)
Years of schooling father	8.76	9.35	8.34	8.77	9.31	8.36
	(2.69)	(2.99)	(2.05)	(2.70)	(3.02)	(2.17)
Age at birth mother	28.75	33.50	29.27	28.85	33.27	29.21
	(5.58)	(5.40)	(6.54)	(5.57)	(5.52)	(6.21)
Age at birth father	32.70	36.47	32.76	32.80	36.26	32.83
	(6.26)	(5.82)	(7.23)	(6.25)	(5.72)	(6.98)
exposed mother	0.11	0.12	0.14	0.11	0.11	0.13
	(0.32)	(0.33)	(0.35)	(0.32)	(0.32)	(0.34)
exposed father	0.11	0.13	0.17	0.11	0.12	0.18
	(0.32)	(0.33)	(0.38)	(0.31)	(0.33)	(0.38)
n	92032	2162	621	95001	2362	629

Descriptive statistics for individuals with at least one parent born in the period from January 1916 to September 1919, by sex and adoption status.
Standard deviations are reported within parentheses.

Table 3: Effect of parental exposure on offspring's educational attainment

	Maternal exposure			Paternal exposure		
	Non-Adoptees	Adoptees		Non-Adoptees	Adoptees	
	biological	adoptive	bio non-rearing	biological	adoptive	bio non-rearing
janfebmar19	−0.39	−0.52	−2.14	−0.33	−0.47	−1.57
	(0.46)	(2.48)	(4.15)	(0.46)	(2.25)	(4.45)
aprmayjun19	−1.19**	−4.29*	2.52	−0.75	−0.54	5.16
	(0.47)	(2.58)	(3.62)	(0.46)	(2.07)	(4.79)
julaugsep19	−0.36	0.86	−4.89	0.51	−2.07	5.99
	(0.49)	(2.11)	(4.07)	(0.46)	(2.02)	(4.06)
Mean(Dep)	135	136	134	138	137	134
Num. obs.	106194	2502	617	104053	2660	676

$^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$

The effect of potential parental exposure on offspring's educational attainment, by adoption status. The dependent variable is years of schooling, scaled to indicate months. Note that each regression is based on a sample in which the corresponding parent is restricted to being born in the period from January 1916 to September 1919, but the other parent is not. Standard errors robust to clustering on the level of the exposed parent are presented within parentheses.

Table 4: Testing differences in estimates across groups

	I vs II		I vs III		II vs III	
	Mother	Father	Mother	Father	Mother	Father
janfebmar19	−0.38	−0.33	−0.38	−0.33	−0.45	−0.41
	(0.46)	(0.46)	(0.46)	(0.46)	(2.47)	(2.23)
aprmayjun19	−1.19**	−0.75	−1.19**	−0.75	−4.28*	−0.54
	(0.47)	(0.46)	(0.47)	(0.46)	(2.58)	(2.07)
julaugsep19	−0.35	0.51	−0.36	0.51	0.89	−2.02
	(0.49)	(0.46)	(0.49)	(0.46)	(2.12)	(2.02)
janfebmar19 x adoptee	−0.26	−0.34	−1.71	−1.15	−1.55	−0.69
	(2.52)	(2.27)	(4.03)	(4.38)	(4.79)	(4.91)
aprmayjun19 x adoptee	−3.52	−0.08	3.39	6.29	6.73	5.97
	(2.60)	(2.12)	(3.69)	(4.75)	(4.41)	(5.20)
julaugsep19 x adoptee	0.81	−2.73	−4.30	5.07	−5.06	7.63*
	(2.15)	(2.07)	(4.22)	(4.08)	(4.59)	(4.54)
Mean(Dep)	135	138	135	138	135	136
Num. obs.	108696	106713	106811	104729	3119	3336
of which adoptees:	2502	2660	617	676	617	676

$^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$

Group I represents the reference sample children with their parents. Group II represents adoptees with their adoptive parents, and group III represents adoptees with their biological parents. Each column represents the coefficients of interest of regression equation 1 where the quarter of birth dummies have been interacted with adoptee status, on a sample in which group A and group B have been pooled. The interaction terms indicates group differences in coefficients. In the third block, *adoptee* denotes the sample of adoptees and their biological parents, and the reference sample are adoptees with their adoptive parents. Standard errors robust to clustering on the level of the exposed parent are presented within parentheses.

Table 5: Effect of parental exposure on offspring's educational attainment: Two Margins

	Adoptive Parents				Biological Parents of Adoptees			
	Mother		Father		Mother		Father	
	non-adoptee	adoptee	non-adoptee	adoptee	non-adoptee	adoptee	non-adoptee	adoptee
janfebmar19	2.17	−8.61	−1.84	−4.06	4.12	2.79	−1.96	−2.92
	(6.97)	(5.46)	(4.91)	(5.70)	(6.57)	(5.64)	(4.33)	(5.64)
aprmayjun19	5.15	−5.46	0.20	1.03	3.99	4.59	−0.55	4.61
	(5.55)	(5.55)	(6.32)	(5.36)	(6.30)	(5.73)	(5.85)	(7.55)
julaugsep19	3.89	−4.21	−5.38	4.04	1.31	−12.59**	4.21	6.16
	(6.21)	(5.79)	(6.65)	(5.57)	(5.74)	(4.87)	(5.87)	(6.60)
Mean(Dep)	136	132	137	133	122	135	128	134
Num. obs.	408	343	433	365	446	344	552	398

$^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$

The effect of potential parental exposure on offspring's educational attainment, by adoption status. The first two columns indicate results for exposure of the rearing mother of adoptees and their non-biological siblings, and the last two columns indicate results for exposure of the adoptees' biological mothers and their biological non-adoptee siblings. The dependent variable is years of schooling, scaled to indicate months. Each regression is based on a sample in which the corresponding parent is restricted to being born in the period from January 1916 to September 1919, but the other parent is not. Standard errors robust to clustering on the level of the exposed parent are presented in parentheses.

Table 6: Difference-in-Difference Estimates

	Adoptive Parents		Biological Parents	
	Mother	Father	Mother	Father
adoptee*janfebmar19	−10.88	−2.76	2.03	−0.90
	(11.05)	(9.10)	(10.25)	(9.70)
adoptee*aprmayjun19	−6.89	1.39	0.80	8.41
	(10.72)	(10.37)	(8.85)	(10.94)
adoptee*julaugsep19	−7.92	2.76	−8.01	1.20
	(10.35)	(9.71)	(7.47)	(9.57)
Mean(Dep)	134	135	128	130
Num. obs.	751	798	790	950

$^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$

Interaction terms for difference-in-difference estimates as defined in equation 3. The sample is based on adoptive parents who have at least one biological child. Standard errors robust to clustering on the level of the exposed parent are presented in parentheses.

Table 7: Prenatal flu exposure and Selection into Adoption

	Adopting children		Giving up for adoption		Own and adopted child		Own child after adoption	
	Women	Men	Women	Men	Women	Men	Women	Men
janfebmar19	−0.0000	−0.0004	−0.0010	0.0013	−0.0078	0.0213	−0.1781	0.1504
	(0.0024)	(0.0025)	(0.0011)	(0.0013)	(0.0300)	(0.0320)	(0.1109)	(0.0978)
aprmayjun19	−0.0012	0.0026	0.0024*	0.0035**	0.0650*	0.0115	−0.0453	−0.0034
	(0.0024)	(0.0026)	(0.0014)	(0.0015)	(0.0359)	(0.0305)	(0.0977)	(0.1023)
julaugsep19	0.0051*	0.0021	0.0026*	0.0024*	0.0140	−0.0091	0.0275	−0.0686
	(0.0026)	(0.0026)	(0.0014)	(0.0014)	(0.0295)	(0.0288)	(0.1004)	(0.1074)
Mean(Dep)	0.0284	0.0303	0.0069	0.0078	0.1512	0.1535	0.5160	0.5095
Num. obs.	79959	78879	79959	78879	2269	2391	343	367

$^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$

The effect of potential prenatal flu exposure on adoption outcomes. The first two columns correspond to being registered as an adoptive parent as the dependent variable and the third and fourth column correspond to being registered as the biological parent of an adoptee. Columns 5 and 6 correspond to the likelihood of adoptive parents having biological children as well. For cases where adoptive parents have a biological child, the last two columns indicate the likelihood of the biological child being born after the adoptee. Robust standard errors in parentheses. All regressions are carried out using a linear probability model and with no additional control variables.

Table 8: Number of non-adoptees associated with different types of parents

Number non-adoptees Time of Birth	0	1	2	3	4	5
	Adoptive Mothers					
before 1919	1553	221	37	6	3	0
Q1 1919	124	17	3	0	0	0
Q2 1919	108	21	8	0	0	0
Q3 1919	141	23	4	0	0	0
	Adoptive Fathers					
before 1919	1629	242	39	10	1	0
Q1 1919	124	20	6	0	0	0
Q2 1919	133	24	1	1	0	0
Q3 1919	138	18	5	0	0	0
	Biological Mothers of Adoptee					
before 1919	180	170	55	20	6	1
Q1 1919	11	15	1	1	1	0
Q2 1919	22	17	6	1	0	0
Q3 1919	20	20	6	1	0	0
	Biological Fathers of Adoptee					
before 1919	185	163	89	28	7	1
Q1 1919	15	19	6	2	1	1
Q2 1919	30	12	8	3	0	0
Q3 1919	23	23	1	2	0	0

Frequency distribution of the number of own children for adoptive parents (i.e. own biological children) and for the biological parents of adoptees (i.e. biological siblings of adoptees that were not given away for adoption). Frequencies correspond to parents born in the period from January 1916 to September 1919.

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