Transgenerational Effects of Childhood Conditions on Third Generation Health and Education Outcomes

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Abstract

This paper examines the extent to which pre-puberty nutritional conditions in one generation affect productivity-related outcomes in later generations. Recent findings from the biological literature suggest that the so-called slow growth period around age 9 is a sensitive period for male germ cell development. We build on this evidence and investigate whether undernutrition at those ages transmits to children and grandchildren. Our findings indicate that third generation males (females) tend to have higher mental health scores if their paternal grandfather (maternal grandmother) was exposed to a famine during the slow growth period. These effects appear to reflect biological responses to adaptive expectations about scarcity in the environment, and as such they can be seen as an economic correctional mechanism in evolution, with marked socio-economic implications for the offspring.

Key words: Nutrition, epigenetics, mental health, height, education.
JEL Codes: I12, J11.

1. Introduction

Inequalities in health and social status tend to persist across generations, but the extent to which this is a causal process is still an open question. Do strong intergenerational correlations of health and social status variables imply that human capital shocks transmit from parents to children and grandchildren? To find an answer to this question, several recent studies have examined the intergenerational effects of policy changes (see e.g. Oreopoulos et al., 2006, Dahl and Lochner, 2012, Black and Devereux, 2011), while others have focused the effects of health-related shocks on child outcomes (Almond et al., 2012, Andreella et al., 2014). Yet, multigenerational studies spanning more than two generations are rare in social sciences and the existing studies mostly investigate the persistence of socio-economic variables (Lindahl et al., 2014, Sacerdote, 2005, Behrman and Taubman, 1985, Clark, 2014). However, as shown in this paper, studies with more than two generations can be useful to separate the effects of biological and social processes. From a policy perspective it is essential to understand whether human capital shocks and invest-

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ments causally affect later generations. Knowing that there are intergenerational returns would imply that the costs and benefits of any policy measure had to be reevaluated to take their long-term effects into account.[1] In this paper, we take a step into this direction by providing evidence of environmentally induced transgenerational biological effects of adolescent undernutrition on children and grandchildren.

While transgenerational effects of nutritional deprivation during adolescence may seem far-fetched from the point of view of a social scientist, there exists ample evidence from mice models (and some on humans) showing that the effects of nutritional shocks can indeed persist for several generations. For example Zamenhof et al. (1971) and Cowley and Griesel (1966) have shown that if rats are malnourished before or during gestation, brain sizes, maturation and cognitive performance of two subsequent generations are reduced even if all descendants are fed a normal diet. More recent evidence shows that the offspring of male mice, who consumed a low protein diet during late childhood (from weaning until sexual maturity), have elevated expression of many genes in liver tissue and exhibit changes in cytosine methylation (Carone et al., 2010). Similarly, it has been shown that the offspring of Holocaust survivors had altered gene expression levels related to cortisol (Yehuda, 2015). Many studies argue that the most likely mechanism behind such phenomena are environmentally induced but heritable changes in the epigenome (Morgan et al., 1999, Rakyan et al., 2003, Ng et al., 2010). In Section II. of the paper we discuss this mechanism in detail.[2] Heritable epigenetic modifications may depend on the sex of the parent who transmits it and can lead to transgenerational non-genetic inheritance of lifetime experiences (Hochberg et al., 2011).

Evidence on whether the above findings on mice translate to human probands is rare. Studies focusing on later-life health and cognitive outcomes among offspring have to rely on non-experimental data, which leads to identification problems if parental conditions are endogenously related to unobserved characteristics that also influence outcomes in another way. To identify causal biological effects of food deprivation on subsequent generations, one needs to observe an exogenous shock in nutrition in the first generation as well as the relevant outcomes in subsequent generations. By now, a consensus has emerged that the study of transgenerational epigenetic inheritance that is induced by environmental shocks early in life requires the observation of at least three generations (see Grossniklaus et al., 2013). After all, effects of parental exposure to environmental shocks on their children's health may not only result from inheritance but can have many other biological and behavioral explanations. Moreover, environmental shocks during pregnancy affect the mother, the fetus, *and* the fetus's primordial germ cells that will produce the grandchildren of the mother.[3] Along these lines, it is particularly interesting to consider environmental shocks that may occur before reproductive ages, and to simultaneously analyze any effects of conditions faced by each grandparent.

The transmission of a nutritional shock over three generations of humans has only been studied in a single line of research papers using historical harvest data and church registers from the remote Överkalix region and connected regions in Northern Sweden (Bygren et al., 2001, Kaati, Bygren

[1] See also Mare (2011) for a discussion on the importance of multigenerational studies.

[2] Epigenetic inheritance is the most plausible explanation for why nutritional shocks may persist across generations and one which has become a focal point in biological and epidemiological research on the long run effects of nutrition, stress and other early life circumstances (see Gräff and Mansuy, 2008, Masterpasqua, 2009, Lundborg and Stenberg, 2010, Hochberg et al., 2011, Kuzawa and Thayer, 2011, and Low et al., 2012, for overview articles).

[3] For a study on the intergenerational effects of in-utero exposure to undernutrition see Lumey (1992).

and Edvinsson, 2002, Pembrey, 2002, Pembrey et al., 2006, Kaati et al., 2007, Pembrey, 2010, Kaati, 2010). Due to its pathbreaking nature, the sequence of studies based on the Överkalix data has evoked great interest in the biological literature (see e.g. Zeisel, 2007, Gräff and Mansuy, 2008, Masterpasqua, 2009, Francis, 2011, Low et al., 2012, and Grossniklaus et al., 2013). The authors find that low paternal grandfather's food supply in the years just before adolescence is associated with a lower mortality risk of grandsons, while low paternal grandmother's food supply is linked to a lower mortality risk of their granddaughters. Low food supply during the paternal grandfather's pre-puberty phase is also associated with lower third generation mortality from cardiovascular diseases, and higher diabetes mortality with a surfeit of food. The authors postulate that these effects are triggered by methylation of epigenetic marks during the ancestor's slow growth period (SGP) which takes place at ages 8-10 for girls and at ages 9-12 for boys. The SGP is a sensitive period for the methylation of male sperm, and the authors hypothesize that the resulting methyl tags are transmitted to subsequent generations via epigenetic imprinting (Pembrey et al., 2006). This mechanism could be an evolved transgenerational response to developmental conditions. Adverse grandparental SGP conditions may then cause an improvement in the offspring's capability to face certain living conditions. Note that the sign of the effect within the first generation members' lives (as typically found in single-generation studies of long-run effects of early-life conditions) is then opposite to the sign of effects on certain later generations.

In this paper we build on the above finding that the slow growth period might be a critical period for sperm development implying that shocks during that age period can have a biological effect on the descendants. Our goal is hence to investigate whether a nutritional shock at that age causally affects health, schooling and mental health outcomes of later generations. Specifically, we examine adult outcomes of subsequent generations following first generation exposure to the severe German famine of 1916-1918 during the slow growth period. Clearly, such a study requires unusual data, containing the date of birth of the first generation as well as relevant outcome measures for the third generation. Currently available administrative register data do not date back sufficiently far to meet these requirements. The same applies to longitudinal panel survey data. However, surveys may gather retrospective information obtained from second or third generation individuals about the relevant variables from the first generation. This is the approach we follow in this paper.

We use data from the German Socioeconomic Panel (SOEP), a large longitudinal household panel since 1986 that is representative for the German population. The data allow us to identify whether a first generation of individuals (usually the parents of SOEP respondents) was exposed to the famine during the SGP. Furthermore, they contain information on a wide range of health information, longevity, education and economic outcomes for the second and third generation. We expect a first-generation individual's famine exposure during the SGP to be positively associated with favorable second and, in particular, favorable third generation outcomes. Furthermore, if famine exposure during SGP affects methylation of the male gametes but not the female ones, we expect only male SGP famine exposure to affect offspring results.[4] Moreover, any causal impact of the famine, biological or not, should be stronger for individuals who suffered from the famine for a longer period of time during their SGP.

[4]The female oocytes fully develop during fetal development.

As an exogenous shifter of nutritional conditions in the SGP, the German famine has several advantages. Specifically, it is well documented, it was severe, and it is sharply delineated in time. Yet, its usage also involves two limitations. First, following the famine, Germany was hit by the Spanish influenza, so that famine and influenza effects are hard to disentangle. Secondly, the famine was a single event, so that exposure during the SGP is equivalent to birth around 1907. Moreover, historical events, including World War II, may have affected the SGP famine cohort differently than adjacent cohorts. This should however not be confused with cohort effects in the third generation, because of random variation in age at childbirth among the first and second generation.

A number of additional empirical issues are common across studies of transgenerational effects. Notably, the study of three consecutive generations of a family requires that family members of the first two generations survive until reproductive ages and that they actually get children. This condition may be selective in that it relates to evolutionary fitness. We deal with many of these issues by carefully selecting the multigenerational sample and by estimating models that include a large number of indicators of secular changes in society and background controls.

As third-generation outcomes we examine adult height, adult mental health outcomes, as well as the highest attained level of education. Adult height is a universally accepted summary measure of pre-adult conditions that influence late-life health outcomes, notably longevity (Waaler, 1984, Steckel, 2008). It has been shown to be sensitive to nutritional deficits in the SGP (see van den Berg et al., 2014). Mental health has been shown to be particularly sensitive to biological shocks and it has been argued that it is sensitive to epigenetic imprinting (see the above overview articles as well as McGowan et al., 2008, and Radtke et al., 2011). Moreover, just like mortality and cardiovascular disease, the mental health outcome has been shown to be sensitive to early-life conditions (see Lumey et al., 2011, for a systematic overview of the evidence on the effects of in utero exposure to famines on schizophrenia and other mental disorders). Notice that health outcomes such as dementia, mortality or cardiovascular diseases are virtually absent in the third generation up to the end of the observation window, so that we cannot analyze them. Education is closely linked to cognitive and noncognitive skills and to social class. It is not clear why this outcome should reflect transgenerational biological shocks, but as a determinant of economic well-being later in life it is interesting to consider it nevertheless. Moreover, cognitive skills at high ages have been shown to depend on early-life conditions, and the underlying mechanism may be related to the dependence of cardiovascular diseases on early-life conditions (Doblhammer et al., 2013).[5] Concerning the second generation, we also investigate late-life mortality.

Our data lack information on biological measures such as methylation patterns (which prove epigenetic modifications). This means that even if the transgenerational associations that we focus on all have the expected sign and significance, it cannot be completely ruled out that other transgenerational mechanisms are at play. In particular, exposure to a famine at pre-pubertal age may lead to more mature behavior. Elder (1999), investigating the impact of the Great Depression on children born in 1920-1921, finds that economic hardship around the age of 10 leads to more

[5]To avoid confusion, notice that it is not an aim of our paper to study long-run effects of *in-utero* exposure to adverse conditions on the health of the individual him- or herself. As mentioned above, such conditions are known to lead to epigenetic changes (see e.g. Heijmans et al., 2008, and Tobi et al., 2009, for evidence on individuals exposed to the Dutch hunger winter around birth). It is not inconceivable that these are transgenerational. Heijmans et al. (2008) find different methylation levels of several genes almost 60 years after exposure, compared to unexposed siblings. Potentially, this could be inherited by subsequent generations.

resilience and psychological strength. This may translate into a different style of upbringing of children. Moreover, some research suggests that economic preferences are transmitted more strongly through gender specific lines (Altonji and Dunn, 2000), while other research finds no gender differences (Dohmen et al., 2012). However, if such social or cultural mechanisms were important in our setting then effects on the second generation should be at least as large as on the third. Apart from that, if such effects resulted from differences in upbringing, we should expect that any third generation results are driven by those individuals who were close to their grandparents during childhood. We can show that this is not the case.

The paper proceeds as follows. Section 2 discusses recent developments in relevant branches of literature. Section 3 describes the 1916-1918 famine and summarizes the existing empirical evidence on its impact. Section 4 describes the empirical approach. We discuss a range of selection issues that may affect the results and that may have plagued the existing evidence. In Section 5 we describe our data. Section 6 presents our main results and section 7 provides a critical discussion of our findings. We investigate how results change if we extend the basic set of covariates with additional background variables that potentially account for non-biological pathways. We also present robustness checks in which the famine intensity is taken into account and its presumed time interval is adjusted. Section 8 concludes.

2. Epigenetic Imprinting

Possible epigenetic mechanisms motivate our study, which is why we discuss them in some detail in this section. Epigenetics is defined as the process by which patterns of gene expression are modified through methylation of the chromatin. Methylation involves the addition of a methyl group to the DNA base, which can turn down a gene's activity or switch it off entirely. This may be driven by environmental shocks such as exposure to malnutrition or stress. In this context, the possibility of transgenerational transmission is particularly interesting (see e.g. Harper, 2005, Gräff and Mansuy, 2008, Masterpasqua, 2009, Grossniklaus et al., 2013, Bohacek and Mansuy, 2015). *Epigenetic imprinting* is the phenomenon that shortly after conception, when stem cells are formed, some of the methyl tags from previous generations remain, causing heritable changes in gene functioning that are not driven by changes in the DNA sequence. Methyl markers are passed on through the germ line, with potentially different expressions of the maternal and paternal alleles in the offspring. Epigenetic modifications may depend on the sex of the parent who transmits it and can lead to transgenerational non-genetic inheritance of lifetime experiences across generations (Hochberg et al., 2011).

The transgenerational stability of epigenetically induced phenotypic patterns is diverse and seemingly depends on the species, exposure and affected tissue. On the one hand, there exist a large number of papers which find that environmental shocks lead to epigenetically induced phenotypic changes that become less pronounced from one generation to the next (Geoghegan, 2014, Schmitz et al., 2011, Remy, 2010). On the other hand, there exist several studies which suggest that epigenetic changes may skip the child generation or may predominantly manifest

among the grandchildren of the affected individuals (Padmanabhan, 2013, Pembrey et al., 2006, Zeybel, 2012).[6]

Therefore, epigenetics potentially has important implications for the effects of health interventions and the economic modeling of human capital formation. Epigenetic marks play an important role in normal development because methylation patterns determine how stem cells develop into certain types of tissue which then maintain cell identity over the lifetime of an individual. In addition, epigenetic alterations cause gene regulation and changing phenotypes, and thus help cells to adopt to different purposes or environments in an evolutionary manner. Such epigenetic patterns are formed over the entire life-course (Fraga et al., 2005) and are strongly influenced by nutritional shocks (Heijmans et al., 2008, Tobi et al., 2009). Empirical evidence suggests that several health dimensions are related to epigenetic modifications. Epigenetics play an central role for the development of cancer cells and cardiovascular diseases and thus determine old-age mortality in humans (Ordovás and Smith, 2010, Jones and Baylin, 2002). Moreover, epigenetic changes influence mental health and cognition with behavioral implications (Bale, 2014, Gräff and Mansuy, 2008, McGowan et al., 2008, Radtke et al., 2011, Bohacek and Mansuy, 2015).

Epigenetic imprinting and epigenetic inheritance imply that adaptive methylation patterns in one generation influence gene expression in the next. How such epigenetic transmissions or inheritance in humans works biologically is not fully resolved (Harper, 2005). Shortly after conception, when the first cell divisions are taking place, the stem cells are generally cleared of all methylation (Farooq, 2010, Mayer et al., 2000). However, recent evidence suggests that some loci associated with metabolic and neurological disorders are also resistant to DNA demethylation (Tang et al., 2015). If epigenetic modifications take place on the part of the genome that is genetically imprinted, this could explain sex-specific epigenetic inheritance. "Imprinted genes" keep their methyl tags (about 1% of genes), which function as a biological marker to flag up their maternal or paternal origin (Masterpasqua, 2009). Epigenetic inheritance may thus be a biological means for humans to adapt to changing environments and to transmit environmental information to the next generation. This is effectively an adaptive expectations response to the degree of scarcity in the environment. As such, this comes as close as it gets to an economic mechanism in evolution, with possible beneficial economic implications for the offspring.

The authors of the Överkalix studies argue that the slow growth period of a child may be a sensitive period for epigenetic modifications on the male gametes and thus for epigenetic imprinting.[7] In this period, the first sperm cells mature, which may make it an important period for the reprogramming of methylation imprints (Pembrey, 2002). The latter part of this period of childhood is also known as the 'fat spurt': growth is low and the body is accumulating reserves for in anticipation of the puberty-related development spurt (Marshall and Tanner, 1968, Gasser et al.,

[6] If phenotypic variations do not manifest among the second generation, this may be the result of two countervailing effects, namely the negative (direct) effect of an adverse shock on the gametes, and the positive effect of the adaptive response.

[7] Indeed, this growth period has previously been found to be a sensitive period for development. Sparén et al. (2004) find that a famine at this age increases cardiovascular problems later in life and Lindeboom et al. (2010) and van den Berg et al. (2014) find this age period to be a sensitive period for life expectancy and adult height, respectively.

1994, Gasser, 1996). Therefore is plausible that limited food availability during pre-adolescence leads to worse pubertal development and to epigenetic modifications on the sperm or egg.[8]

While the Överkalix studies have evoked great interest, it is fair to state that from a statistical point of view the analysis of the data has some limitations. The studies consider up to three different degrees of food availability, among six ancestors (4 grandparents and 2 parents), during several parental and grandparental pre-adult age periods, and they examine their associations with several outcomes among grandchildren distinguished by sex. In the absence of strong theoretical priors, this amounts to the detection of a large number of associations. Even if no transgenerational transmission exists, a statistical analysis would typically result in a few numbers of false positives. Simply put, under the null hypothesis of no effects, and with a 5% test size, one finds an effect in 5% of the cases. Furthermore, samples of third generation members are rather small, ranging from ca. 100 to 300 individuals. For such a large number of parameters and given the small sample size, it is possible that the authors found effects that prove unimportant in other samples. Hence, in a sense this paper also aims to assess the external validity of these findings.

Economists and social scientists have since long been interested in intergenerational correlations of outcomes and in the intergenerational transmission of adverse experiences by way of behavior, upbringing and learning. Similarly, there has been a wide interest in genetic determinants of intergenerational associations. Epigenetic transmission across generations is biological but not genetic, and it may be triggered by economic shocks.[9] As such, it constitutes a potentially valuable framework to understand intergenerational patterns, and it may have distinct implications for the costs and benefits of policies with effects on subsequent generations. At the same time, it should of course borne in mind that observed intergenerational associations in general can potentially be explained in many different ways, including non-biological and other biological explanations.

3. The Famine

The World War I famine which we use to study the effects of an exogenous variation in nutrition among the first generation was sharply delineated in time and extremely severe. In fact, it is said to be the severest famine experienced in Europe outside of Russia since Ireland's travail in the 1840s (Raico, 1989). At the end of the war, the German 'Reichsgesundheitsamt' (Health Office) calculated that 763,000 German civilians died from starvation.[10] The period of food scarcity started in June 1915 when bread began to be rationed. In early 1916, food rationing became severe. From 1916 to mid-1919, the German population on average had to live on less than 1500 calories per day (Starling, 1919). Because the portion of bran in the bread was very large, the caloric value was further reduced by about 15 to 20 percent.[11] Most Germans had to live on a meagre diet of dark bread, slices of sausage without fat, three points of potatoes per week and turnips (Vincent,

[8] So, effects along the female line could also be driven by maternal nutrition during SGP. Indeed, mice models indicate that epigenetic imprinting may take place along the female germline (Cooney, 2006).

[9] For an overview of molecular genetics and economics see Lundborg and Stenberg (2009). For the role of epigenetics in psychology see Harper (2005).

[10] The overall population of the German empire at that time was about 65 million. In addition there were about 2 million military deaths, who in a conventional ground-based war like World War I, were almost exclusively men of age 17-60.

[11] Typically, a man needs about 2500 calories a day and a women about 2000.

1985). Table A.1 in the Online Appendix displays an overview over the amount of food consumed during the famine as compared to prewar times.[12] While these amounts are well below subsistence to begin with, the situation was aggravated by the mere length of the famine which started in 1916 and extended into 1919. At the height of the famine, purchasing foodstuffs on the black market was the only way to prevent starvation. Black market prices in cities skyrocketed (see Table A.3 in the Online Appendix) and many families had to rely on excursions to the countryside to feed their children.

Four factors led to the extreme shortage of food. First, by mid-1916 the Allied Powers had successfully enacted a complete naval blockade of Germany restricting the maritime supply of raw materials and foodstuffs. Before the war Germany had imported one third of its food, but after the blockade Germany was cut from foodstuff imports of all sorts: fodder for livestock, grain and potatoes. The blockade continued even after the Armistice and until June 1919 to force Germany to sign the Treaty of Versailles. In fact, throughout 1919 rationing was maintained in many parts of the country at a rate of 1000-1300 calories per day (Vincent, 1985).[13]

Second, due to the general war mobilization, around 40% of the male agricultural labor force was absent, with a similar fraction of horses and cattle. This reduction in the male work force was not adequately compensated by employment of prisoners of war, women, adolescents and children (Huber and Fogel, 1920). As a consequence, between 1913 and 1919, annual production of crops, potatoes and milk decreased to about 50% when compared to pre-war levels (Blum, 2011).

Third, in the summer of 1916, the root crop and grain harvest were particularly bad and the potato crop failed almost completely. The latter was particularly detrimental, because much of the German food supply was based on potatoes and during the war more agricultural crop land had been shifted away from turnip cultivation and towards potatoes (Klein, 1968). The Winter of 1916-1917 thus marked the peak of the famine and is today remembered as the 'turnip winter' (Steckrübenwinter), because the only food in sufficient supply during that winter were turnips.

Last, food storage was a concern. Before the war most of the potato crop was stored in the countryside and only supplied to the cities on demand. After the start of the war, transportation and dislocation became more difficult, and potatoes had to be stored in larger quantities by individuals unschooled in the proper techniques of storage, which led to spoilage and waste (Vincent, 1985).

The famine had a large impact on the German population and in particular on young adolescents. On average, individuals lost 15-25 percent of their weight between 1916 and 1919 and height in the male population born in the period between 1914 and 1917 is around 1.5cm less than for adjacent cohorts (Blum, 2011).[14] Children were less likely to die than adults, but often suffered from hunger-diseases such as edema, tuberculosis, rickets, influenza, scurvy, and keratomalacia (Roesle, 1928).[15] Under-consumption of food during that time was particularly dramatic among male adolescents. As a consequence, late-life mortality is higher for male (but not female) individuals who were around age 15 at the end of the famine (Horiuchi, 1983).

[12] For further evidence see also Table A.2 in the Online Appendix.

[13] The reason for continued food rationing was that even after the end of the blockade in June 1919 Germany could not import freely, since all funds had to be saved for war reparations.

[14] Individuals who had lost 30 percent or more mostly died.

[15] The number of occurrences of epidemic diseases such as typhoid, rabies, trichiniasis and dysentery stayed roughly constant in the population.

The first wave of the Spanish Influenza pandemic hit Germany in June 1918, the second one in the Fall and the third one in January 1919 (Witte, 2008). In Germany, about 150,000 individuals died in this period as a result of the disease (Vincent, 1985). This number is low when compared to the overall number of deaths that resulted from starvation, but it is still considerable. Because identification in our study relies on comparing outcomes of the descendants of adjacent first-generation cohorts, it is difficult to separate the effects of the famine from the effects of the influenza pandemic. However, evidence from Norway suggests that, for the most part, the Spanish Influenza was lethal only for individuals of ages 20-40, such that selective survival is not an issue for our cohorts of interest (Mamelund, 2003). Nevertheless, since the continuation of the blockade and the third wave of the Spanish influenza extended well into 1919, we conduct sensitivity checks that include the year 1919 in our famine period.

4. Identification and Outcome Models

We use common coefficient models and matching to identify the effect of SGP famine exposure of first generation (G1) ancestors on second (G2) and third generation (G3) individuals. We thus calculate a famine effect among G2 and G3 individuals with the same background and birth year, who differ with respect to exogenous first generation famine-SGP exposure. Because, first generation famine-SGP exposure is a historical incident that is exogenous at the individual level, our approach allows us to identify the impact of having ancestors of a certain age during the famine on the second and third generation.[16]

4.1. Famine Exposure

To investigate systematically how adult outcomes of G2 and G3 vary with first generation SGP exposure to the famine, we focus on individuals who have at least one ancestor born during the years 1902-1913. Note that male individuals in this cohort were too young to have been drafted and females were too young to have conceived a child during Word War I. Also, none were born during the war.

Table 1 displays the number of years the famine affected different birth cohorts of first generation individuals during their SGP, defining 1916–1918 as the famine period. Note that all first-generation individuals have been affected by the famine, but that exposure occurred at different ages. We thus identify the effect on the third generation of having a first generation ancestor who was exposed during SGP as compared to having a first generation ancestor affected by the famine at a different point in time.

While using cross-sectional variation in adverse famine exposure in one-generation studies may lead to the confounding of causal and cohort effects, this should be less of a problem in multigeneration studies. If adverse early-life circumstances universally affect some cohorts but not others, any observed difference in first-generation outcomes may potentially result from cohort effects that orig-

[16]Note that our measure of famine exposure is whether someone had reached a certain age by the time of the famine. This measure thus reflects the intention to treat (ITT) and not the actual treatment effect (TE). Our data do not contain information on whether a first generation individual actually went hungry during the famine (for a discussion of ITT and TE effects in famine studies see van den Berg et al., 2016). Recall however that the famine was widespread and more severe than other famines in Europe in the past 150 years.

inate from a different source than the cause of adversity. With three generations, random variation in the timing of fertility allows us to identify outcome effects within second and third generation birth cohorts. In addition, as a robustness check, we use geographic variation to see whether there is heterogeneity in effects across the degree of urbanization, and we exclude individuals who were famine-affected in utero or during early childhood.

Nevertheless, our analysis relies on the assumption that there are no systematic differences in famine survival between individuals affected by the famine during the SGP and the control groups. Hence, we assume that children in their slow growth period were about equally likely to die from the famine than children that were slightly younger or older at the time. Historical sources seem to back this claim. Vincent (1985) reports that death rates of children between the ages of one and five had risen by fifty percent during the famine, while for children from five to fifteen they were only slightly higher (55 %). Historical data displayed in Figure A.1 even show no dramatic increases in child mortality at all. Nor do they indicate an impact on the life expectancy at birth of individuals born around 1907 (see Figure A.2).[17] In addition, selection into fertility would be a problem if parents from different social classes had been more or less likely to conceive children in the periods 1902-1903 (1902-1904) or 1910-1913 (1911-1913) than during the years 1904-1909 (1906-1910). Figure A.3 however shows that for the time period of births we are analyzing (1901-1914), overall birth rates do not show any systematic pattern. Of course, it was impossible to anticipate the famine a decade earlier.

[Table 1 about here.]

In G1, $Z \in \{0, 1\}$ is defined as an indicator of SGP exposure to the famine, at ages 8-10 (females) and at ages 9-12 (males) in 1916-1918. For G2, Z is a 2x1 vector with the first entry indicating whether the mother was affected by the famine during her SGP and the second indicating whether the father was affected during that same period. Following the same logic, for G3, Z has four entries: whether paternal grandfather (PGF) was SGP famine affected, whether paternal grandmother (PGM) was SGP famine affected, whether maternal grandfather (MGF) was SGP famine affected and whether maternal grandmother (MGM) was SGP famine affected.

4.2. Outcome Models

We estimate three different types of outcome models to account for the different distributional properties of the respective outcome variables: a duration model for individual mortality, a discrete choice probit model for the decision to obtain a higher secondary school degree and a linear regression model for the continuous outcomes height and mental health. In all models, x denotes an individual-specific vector of observable characteristics, which always comprises some basic control variables and in some specifications an additional set of background controls. f_y is a vector of birth year dummies for the respective generation that captures any variation that may be cohort or birth year specific.

[17] Figure A.2 does show a decrease in life expectancy among individuals born during the famine. We address this issue in the empirical part of this paper.

4.2.1. Duration Model

To estimate the impact of the famine on G2 longevity, we model the hazard of G2 mortality at any given point in time as being composed of a baseline hazard and a systematic part. Following the standard biological literature on modeling mortality, the baseline hazard has a Gompertz[18] functional form with ancillary parameter γ, so that

$$h(t) = \exp(\gamma t)\exp(Z_i'\delta + x_i'\beta + f_y'\eta). \tag{1}$$

Being a sample member requires participation in at least one interview. By necessity, then, longevity exceeds the age at the first interview. We therefore correct the likelihood function for left truncation of longevity at the age at the first interview.[19] In addition, longevity spells are right-censored at the age at the last interview in which the individual participates, unless we observe that this interview is succeeded by mortality before the assigned date of the subsequent interview, in which case we observe the realized longevity.

4.2.2. Probit Model

We model binary outcomes such as upper secondary schooling as a binary outcome latent index model with $Y_{it} = 1_{[Y_{it}^*>0]}$, where Y_{it}^* denotes the latent continuous variable. The latent variable in turn is determined by famine exposure, birth year fixed effects and observable control variables. We assume a linear structure and additive separability in the error term:

$$Y_i^* \quad = \quad Z_i'\delta + x_i'\beta + f_y'\eta + \epsilon_{it}.$$

The observed binary variable Y_i is an indicator variable that is assumed to equal one if the latent variable crosses zero as a threshold $Y_i^* > 0$. We estimate a probit model, assuming that $P(Y_{it} = 1|x_i, f_y, Z_i) = \Phi(Z_i'\delta + x_i'\beta + f_y'\eta)$ where Φ denotes the normal cdf.

4.2.3. Linear Regression Model

For continuous outcomes, we estimate the following linear model between outcomes Y_{it}, famine effects and covariates for adult i born in year t:

$$Y_i = Z_i'\delta + x_i'\beta + f_y'\eta + \epsilon_{it}.$$

again x_i denotes a vector of control variables and the equation comprises a vector of own birth-year fixed effects (f_y) to capture any variation that may be cohort or birth year specific.

[18] As shown in Table D.2 of the Appendix, our results are robust when relaxing the Gompertz assumption in a Cox Proportional Hazard setting.

[19] We also conduct robustness checks where we introduce an individual-specific frailty term (α) that enters the hazard function multiplicatively:

$$h(t) = \alpha\mu_0. \tag{2}$$

We find that the introduction of unobserved heterogeneity hardly affects the results.

5. Data

The German Socioeconomic Panel is a representative longitudinal survey data set. Since 1984, around 12,000 households, comprising more than 20,000 individuals, are followed over time. The sampling unit is the household, and all household members aged 17 or above participate in the survey. Individuals who leave the household to form a new household are kept in the sample, as are their new household members. At the age of 17, children of participating households become full-blown survey participants. Attrition from the panel is compensated by refreshment samples aimed at maintaining representativeness for the full population.

The survey participants are interviewed once a year. An average interview takes about 1.5 hours. The survey is a broad multi-purpose survey. It aims to obtain extensive information on socioeconomic outcomes, demographic conditions, living conditions, opinions, behavior, consumption, etc. of the respondents and their household. In certain waves of the survey, individuals are exposed to modules covering special topics such as health status, cognitive abilities, or family trees. In their respective first SOEP interviews, the adult respondents were asked about the birth years of their parents if the latter were not SOEP respondents themselves. In case a parent had died, the death year was also asked for.

The data design leads to a natural choice of what constitute the three generations in our analysis. The second generation (G2) are adult SOEP respondents. The first generation (G1) are their parents, while G3 are their children. This is the only choice that leads to a substantial number of G1 individuals who experience the 1916-1918 famine in their SGP. By relating their birth year to the famine period we infer whether they were exposed to the famine during their SGP or indeed during any other age interval.

Our G3-sample (G2-sample) contains 2670 (6548) G1 individuals who were born in 1902-1913.[20] In addition, 580 (855) and 1914 (873) G1 individuals are born before 1902 and after 1913, respectively. The data contain 4138 G2 individuals and 1291 G3 individuals.[21] See Table 2 for more details.[22]

[Table 2 about here.]

5.1. Outcome Variables

Our outcome measures (Y_{it}) are height, longevity, mental health and whether an individual has obtained an upper secondary school degree. In the SOEP, age at death can be obtained for individuals who have participated in the survey at least once and who dropped out of the survey because they died. The death year is provided by the SOEP in the so-called person-based metafile. Mental health and height measures are obtained using the most recent information from the biannual SOEP health module. Height is self-reported and mental health is measured by the Mental Component Summary Scale (MCS), one of the two sub-dimensions of the SF-12 questionnaire.

[20] See Section Appendix B of the Online Appendix for a detailed description of the actual selection of the G1, G2 and G3 samples.

[21] In addition we conduct robustness checks where we restrict attention to only those G1 individuals who were born in 1902-1913. That is, we then focus *only* on G2 and G3 individuals if *all* their G1 ancestors were born in that period. This leaves us with much smaller sample sizes of 1232 G2-males and 1261 G2-females and of 178 G3-males and 137 G3-females, respectively.

[22] In the G3 generation sample we count 659 siblings who mostly have the same ancestors.

The MCS is measured on a scale that ranges from 0 to 100 with mean 50 and standard deviation 10. It results from a factor analysis comprising the dimensions 'general mental health', 'emotional functioning', 'social functioning' and 'vitality' each measured on separate scales (for details see Andersen et al., 2007). We define whether an individual has obtained the German university or technical college entrance diploma (German "Abitur" or "Fachhochschulreife") using the international Comparative Analysis of Social Mobility in Industrial Nations (CASMIN) classification. Mental health and schooling measures are exactly the same for generations two and three, which makes our results directly comparable across generations. Table 3 comprises summary statistics of all outcome variables.[23]

[Table 3 about here.]

5.2. Control Variables

We define two sets of control variables: basic control variables and background control variables. Basic control variables comprise all variables that account for any bias that may arise because famine-affected G1 individuals (and their offspring) are (may be) born in different years than the corresponding control group members. Most importantly, in accordance with research showing that wars and famines can have lasting scars on those individuals who were born at that time (see e.g. Lumey et al., 2011), we are including indicator variables for whether G1 individuals were born during World War I and for whether G2 individuals were born during World War II. Note however that our sample comprises rather few of those individuals (68 G1 and 140 G2). Second, we are controlling for individual birth year fixed effects to capture cohort or birth year specific variation in outcomes. Last, we include detrended GDP per capita during the year of birth of G1 (see Figure A.4) in line with a strand of literature which demonstrates that economic and health conditions at birth and during infancy can have long-run mortality and height effects. (For the effect of business cycle variation on outcomes see e.g. van den Berg et al., 2011, Woitek, 2003 and Sunder and Woitek, 2005.)

We include background variables as additional covariates in some specifications to detect behavioral, non-biological pathways. One of the potentially most important pathways goes through parental education, reflecting parental cognitive ability, parenting skill, social class and family earnings potential all of which are essential for health and schooling outcomes. We define parental education dummies for different educational degrees in Germany. In addition, we include the number of siblings, as a proxy for parental resources. Descriptive statistics of all included background variables can be found in Table C.1.

6. Empirical Results

The results are discussed in two stages. First, in Subsection 6.1 we discuss how grandparental SGP famine exposure affects outcomes in the third generation. These are the findings that most reliably allow us to draw conclusions about epigenetic pathways and that can be connected directly to the main findings in the Överkalix studies. We investigate how the results change if we move

[23] We do not consider wages, because our data are a cross section of individuals who are sampled at different points in their lives, which makes the computation and comparison of (permanent) income problematic.

from controlling only for a basic set of variables to controlling for additional background variables, which potentially account for non-biological channels. We also conduct robustness checks where we vary famine intensity and period, e.g. by excluding individuals who experienced the famine for only one year and by extending the famine period to 1919. If famine exposure has a causal effect on outcomes, we expect this effect to become larger and more significant if individuals with weak famine exposure are excluded from the analysis. Last, we restrict the sample to individuals for whom all G1-ancestors were born during the period 1902-1913.

Second, in Subsection 6.2 we discuss the results of parental SGP famine exposure on the second generation, keeping in mind that these findings may be driven by a range of mechanisms including epigenetic mechanisms but also direct behavioral or environmental processes. Nevertheless, we expect these results to be informative about how any potential effect transmits from the first to the third generation.

6.1. Third Generation

The coefficients in Table 4 display the effect sizes of grandparental SGP-famine exposure on height, mental health and schooling, conditional on the basic set of control variables and on the combined set of basic and background controls. Since epigenetic inheritance may be sex specific, we perform all analyzes separately for males (left panel) and females (right panel) (Pembrey et al., 2006). We report p-values that correspond to robust standard errors clustered at the original household level, because individuals with the same ancestors share genes, environment, and history of ancestral famine exposure.[24] Note that there are four ancestors who have potentially been affected by the famine during their SGP: the paternal grandfather, the paternal grandmother, the maternal grandfather and the maternal grandmother. In line with our initial hypothesis that individuals should be affected along the male line, we highlight the line with coefficients on paternal grandfather exposure. If our results are driven by transgenerational epigenetic mechanisms, we should expect positive and significant effects on G3 height, schooling and mental health.[25]

The coefficients displayed in row 1 of Table 4 do indeed indicate positive effects on height along the male line, but the effect sizes are small and insignificant. Similarly, the effects on schooling are close to zero. Hence, for these two outcomes we do not find evidence in support of epigenetic transmission mechanisms. For mental health, the case is different: in line with the findings by Kaati et al. (2007) our results displayed in Columns 3 of Table 4 indicate that paternal grandfather SGP exposure does indeed have a positive effect on mental health of grandsons. Moreover, interestingly, row 4 of column 9 reveals that maternal grandmother SGP exposure positively affects granddaughters' mental well-being. Thus, as predicted by the theory, mental health is positively affected by a nutritional shock to the first generation, and the effect transmits along the male and female line, respectively. The right columns in Table 4 display coefficients for models with additional controls for parental education and sibship size. If our findings were driven by changing opportunities, fertility or first generation cognitive ability, we would expect changes to our coefficients' size and significance after the introduction of these additional background variables. We conduct a like-

[24] The standard error are clustered on original household numbers. That is, any G3-individuals with the same G1 and G2 ancestors (siblings and cousins) are part of the same cluster.

[25] Our data do not allow us to look at sibling fixed effects, because the number of siblings who do not share the exact same ancestral line is very low.

lihood ratio test for the improvement in model fit due to the additional variables and find that controlling for parental background improves the fit to the data, but does not reduce the size and significance of mental health coefficients. The results indicate that having a paternal grandfather or a maternal grandmother who has been affected by a famine during SGP improves mental health by about 1.6 points, or 16% of a standard deviation, for males and by about 2.2 points, or 22% of a standard deviation, for females.[26]

[Table 4 about here.]

6.2. Second Generation

The analysis so far has focused on the third generation, where any findings most reliably point towards transgenerational epigenetic effects. For two reasons, we also present results for the second generation. First of all, the fact that about 10% of the G2 individuals have died enables us to examine mortality effects, albeit to a limited extent. Second, for the interpretation of the G3 results it is interesting to examine whether the effects on mental health are also present in the second generation.

Table 5 displays the main results for G2 and Table D.1 contains the same robustness checks as for G3 discussed above. Overall, we do not find robust effects on G2 mortality or height if a parent has been exposed during the famine during SGP. If at all, males tend to be slightly shorter if the mother, and slightly taller if the father has been affected by the famine. Furthermore, the coefficient on parental SGP famine exposure on schooling is negative and significant in some specifications for males. We suspect that the negative schooling effects of paternal famine exposure on male individuals are nonbiological that come about because fathers who experienced the famine during adolescence received less rigorous educational training. This presumption is supported by the fact that the effect is insignificant in Column 7 of Table D.1 where the less severely affected fathers are excluded from the analysis. Turning to second generation mental health, we neither find supportive evidence that paternal SGP famine exposure raises the son's mental health, nor do we find that maternal SGP famine exposure has positive mental health effects for their daughters.[27]

[Table 5 about here.]

6.3. Robustness Analysis

To investigate the robustness of our results we conduct a large number of additional analysis: We vary the degree of exposure to the famine, use different model specifications and varying definitions of control and treatment groups. Last, we increase our sample by allowing for up to one missing grandparent.

[26] It might seem surprising to find significant results for mental health but no significant effects of schooling. However, after conditioning on basic control and background variables, the correlation between mental health and schooling turn out to be small and insignificantly different from zero ($\rho = 0.023$ for males and $\rho = -0.008$ for females).

[27] Similarly, if we restrict the sample to only those G2 individuals for whom both parents were born during 1902-1913, we do not find any significant mental health effects.

6.3.1. Varying degrees of exposure

To test the robustness of our findings, we begin by investigating if coefficients increase when only the more highly affected individuals are included in the analysis. Table 6 shows the famine effect of at least two years of famine exposure, and the right column shows the SGP famine effect with the famine period extended to 1919. We find that point estimates for mental health mostly increase if we only include the most highly affected individuals. If the paternal grandfather is exposed to the famine during his SGP, grandsons' mental health now tends to be 21% of a standard deviation higher. The increase in mental health for granddaughters is only slightly smaller than in the previous specification, but now turns insignificant. Effects remain large and significant when the famine period is extended to the year 1919. We interpret these findings as supportive for the evidence that the pathway starts with a nutritional shortage in the SGP.

[Table 6 about here.]

To further vary the degree of exposure, we investigate whether there are heterogenous effects across degrees of urbanization. The rationale for doing so is historical evidence, which suggests that individuals living in cities were more heavily affected by the famine than individuals living in the countryside. Unfortunately, our data do not contain information on the place of G1 childhood residence. Instead, we can only use urbanization of the childhood place of residence of the second generation. Given the long time lag and many migration movements related to the history of Germany, urbanization of the G2 childhood place of residence is arguably a rough proxy for whether the first generation lived in a city while young. Therefore, we conduct an additional robustness check, which investigates how our results change if we restrict the sample to G1 individuals who have completed an upper secondary school degree. This yields a sample of individuals from urban areas, because in former times, schools which offered an upper secondary school track were usually located in cities. Again this sample restriction is imperfect, because as shown in the following subsection (Subsection 6.4), G1 males affected by the famine during SGP were less likely to obtain a secondary schooling certificate in the first place. The results of these two robustness checks are presented in Table 7, where in columns 1-2 of the table we restrict the sample to individuals with paternal grandfathers who are likely to have lived in cities, and in columns 4-5 of the table we do the same with a restriction on maternal grandmothers. We find that significance is reduced due to the much smaller sample size, but that our point estimates are unaffected. For males, the effect of paternal grandfather SGP famine affectedness remains strong and increases considerably for G1 paternal grandfathers with upper secondary schooling. For females the case is less clear, and the effect vanishes when restricting the sample to city dwellers.

[Table 7 about here.]

Next, we investigate wether our effects are driven by a control group of first generation individuals which was (at least in part) affected by the famine at very young ages. By now, a large and convincing literature exists, which shows that adverse in utero and early childhood conditions can be detrimental to health outcomes (see Almond and Currie, 2011, van den Berg et al., 2016, among others). Since our control group partly consists of children affected by the famine at young ages, adverse outcomes for the descendants of this first generation of infants, who are part of the control group, might drive the effect. The third and sixth column of Table 7 therefore show the results for

a sample of individuals which excludes any paternal grandfathers (maternal grandmothers) who were born or in infancy during the famine (corresponding to birth years 1913-1919). Our results remain robust to excluding these individuals from the sample.

6.3.2. Model specification

Our results are robust to varying sets of background control variables. One concern might be, for example, that the indicator for G2-individuals being born during World War II might be endogenous, since it is a function of age-at-childbirth of G1, which in turn may be affected by the socioeconomic status of the lineage.[28] In columns 1, 2, 7 and 8 of Table 8, we therefore report the results of a restricted model where all G2 war-birth controls are excluded. Comparing the coefficients for paternal grandfather exposure (maternal grandmother exposure) on third generation mental health to those reported in our main specification (Table 4) we find that our point estimates are hardly affected by this change in specification. The same holds true if we exclude other background control variables, such as detrended GDP per capita at rst generation birth or parental age at birth.[29]

6.3.3. Definition of control and treatment groups

All results reported so far rely on the identifying assumption that our treatment group (the descendants of the cohort of individuals born in or around 1907) is not systematically "better" in terms of unobservable characteristics related to mental health than the control group. Therefore, we show in Table C.2 that our treated first generation individuals are very similar in terms of observable characteristics related to their age at death and sex-specific fertility. If at all, male G1 individuals that are part of the treatment group seem to die at slightly younger ages than those that are part of the control group. Nevertheless, in the above analyses, the control group is comprised of individuals who are G3 descendants of G1 individuals born both before and after the cohort of treated G1 individuals. The famine, however, may have had adverse effects outside of the SGP. We therefore run robustness checks using a control group of G1 paternal grandfathers (maternal grandmothers) born entirely after the famine.[30] The results of this exercise are displayed in columns 3, 4, 9 and 10 of Table 8. Although this reduces our sample size by around one third, we find that our results are largely robust to this change in the control group. While the precision of the estimated effects is reduced for males, the point estimates remain very similar for both males and females.

As an additional sensitivity test, we use G1-individuals born after 1919 as a placebo treatment group. That is, we use the same sample of individuals as in our main specification, but we assign

[28] We are grateful to one of the anonymous referees for pointing this out to us.

[29] We also tested the robustness of our results with respect to the addition of a large number of different control variables (results not displayed). The set of variables comprised the age at the time of measurement, population growth, indicator variables for whether G1 individuals were born before 1902 or after 1913 as well as parental (G1 and G2) age at the birth of the children. For G3, the results also robust with respect to the inclusion of parental (G2) birth year fixed effects that capture business cycle fluctuations and variation in the probability for an individual to be part of the sample.

[30] See, e. g. Klemp and Weisdorf (2012) for a similar approach to construct control groups. Also note that our previous analysis of restricting the G1 sample to individuals with upper secondary education is similar to their approach, which is to subdivide the sample by paternal occupation.

all descendants of G1 individuals born after the famine to the treatment group and all other individuals to the control group. Note that individuals born after 1919 should not directly have been affected by the Spanish Influenza. As shown in columns 5, 6, 11 and 12 of Table 8, we find that for males the point estimate switches signs. Moreover, all mental health effects turn insignificant.

[Table 8 about here.]

6.3.4. Missing observations

The above analysis is restrictive in a sense that the sample is composed exclusively of G3 individuals with non-missing information on all four G1 ancestors. In Table 9 we therefore investigate the robustness of our results with respect to inclusion of observations with one missing grandparent (in each respective column it is the one for which no coefficient is displayed). This almost doubles our sample size and leads to a considerable increase in the precision of our estimates. While the point estimates remain largely unchanged, we now obtain coefficients that are significant at the five and one percent significance level, respectively.[31]

[Table 9 about here.]

6.3.5. Multiple hypothesis testing

The possibility of incorrect statistical inference due to ignoring the multiple-hypothesis testing nature of the empirical analysis is a concern in any study where the exposure to an adverse shock may potentially affect a wide range of observed outcomes. In our setting, the hypotheses follow from theoretical priors according to which the first generation exposure to a famine leads to positive outcomes among the third generation along sex-specific lines. These specific hypotheses are also targeted by the studies using the Överkalix data. Moreover, the set of outcomes we consider is rather small and is motivated by the existing literature described in earlier sections. All this leads us to conclude that our approach is not highly sensitive to the risk that we ex post overstate the significance of estimated coefficients.

To proceed, we apply the ultra-conservative Bonferroni adjustment according to which critical p-values are corrected for the number of tests performed, to our results. The small set of null hypotheses concerning paternal grandfather SGP exposure (maternal grandmother SGP exposure) on third generation males (females) is not jointly rejected if we perform this adjustment where the number of tests performed is three (corresponding to the number of outcomes) in our case (Abdi, 2007). In this sense, it is important that our point estimates stand up to all kinds of robustness analyses. We have seen that, fortunately, the point estimates remain largely unchanged if we use different exposure variables, sample definitions, various kinds of control groups, and model

[31] If we restrict the sample to only those individuals with all G1-ancestors born during 1902-1913 (results not displayed), we find positive height effects if the paternal grandfather was exposed for both males and females. Moreover, we find highly significant positive mental health effects for females whose maternal grandmothers were exposed but no significant effects for males whose paternal grandfathers were exposed. A problem with this sample is, however, that it is rather small and that almost all individuals in it have at least one first generation ancestor who was exposed to the famine during the SGP. Consequently, the control group of completely unexposed individuals becomes tiny (2 males and 6 females) and the result may be affected by that.

specifications. Essentially these robustness checks mean that a large number of different (albeit correlated) tests reveal the same underlying effects in the data. The validity of our results in the presence of such a considerable number of robustness checks leads us to believe that the lack of statistical significance in our main specification is mainly a problem of power due to the limited sample size. If we focus on those models that are estimated on samples with up to one missing ancestor (see Table 9) our results fully survive in the presence of a Bonferroni-type adjustment of significance levels.

6.4. Potential Channels

The above findings hint towards a transgenerational biological transmission of effects triggered by a reduction in food access during the ancestors' slow growth period, similar to those in the above-described series of papers on the Överkalix data. However, concerning the channels through which the transgenerational effect operates, we cannot be certain that our estimated effects are of biological (let alone epigenetic) origin. In this subsection we thus perform some additional analyses to shed light on the plausibility of the presence of a biological pathway.

Experiencing a famine during SGP might have affected education levels or social competencies of G1. The results on the G3 educational outcome may be affected by causal effects of G1's level of education on G3's level of education. Evidence of positive associations between G3 and G1 levels of education, controlling for G2 levels of education, is provided by Lindahl et al. (2013), using multigenerational data from Sweden. In our setting, it is conceivable that SGP famine exposure has a negative effect on the highest attained level of education of G1. This by itself could then generate a negative effect of G1's SGP famine exposure on G3's level of education. Such a pathway runs opposite to the epigenetic pathway that we focus on. This may explain our empirical finding that the net over-all effect is not significantly different from zero. We thus start out by examining whether G1 SGP famine exposure has a negative impact on G1 education. Table 10 presents average marginal effects from a probit analysis. It shows that the effects are negative for males but not for females. The average effects on male upper secondary and secondary education amount each to a two percentage point reduction. In relative terms, this is quite considerable (around 20 percent), since the share of individuals with upper secondary and secondary education for these cohorts is around 10% each. We proceed by investigating whether adding G3's realized education levels to our preferred specification impacts the mental health results displayed in Table 4. Table 11 displays the mental health results for G3 if we do not only account for parental (G2), but also for grandparental (G1) education. The results indicate that the SGP famine effect for males becomes stronger and more significant, while for females it becomes less significant. That we do not find a major change in the results for females is not very surprising given that the realized level of education of G1 females was not affected by the famine in the first place. Conversely, controlling for G1's level of education reinforces the mental health effect for males.[32] Another striking result is that paternal grandfather upper secondary education seems to affect male mental health while maternal grandmother secondary education seems to affect female mental health.

[32]The estimated G1 SGP famine effects on G3 height and education hardly change if G1 education variables are included in the main specification.

This result suggests that both investments and shocks on the first generation affect mental health outcomes of the third generation and that the effect operates along sex-specific lines.

[Table 10 about here.]

[Table 11 about here.]

Next, we assess whether our results reflect better grandparental caregiving. After all, it might be the case that first-generation individuals who experienced the famine during adolescence show more resilience and psychological strength and are therefore better caregivers to their grandchildren. To see whether this is indeed the case we re-estimate our main model, but exclude all third generation individuals who (at age 18) reported a "close" or "very close" relationship with their grandparents.[33] The mental health results of this robustness check are displayed in Table 12. We find that our results are completely invariant to this change in sample composition.

[Table 12 about here.]

7. Discussion

Using data on three generations and an exogenous shock to the nutritional environment of G1, this paper provides evidence in support of the hypothesis that there exists a transgenerational effect of undernutrition during the slow growth period on third generation offspring mental health, but not on third generation body height or schooling. The positive effect of G1 undernutrition during the slow growth period on G3 mental health is robust to using different sets of control and exposure variables, and to different definitions of treatment and control groups. Therefore, although our G3 findings should be considered alongside the parallel tests of three different outcome variables, it seems unlikely that they are driven purely by chance.

Nevertheless, it should be borne in mind that our data are non-experimental and it is difficult to rule out that our results are affected by that. A potential limitation is that the 1916-1918 famine was a single adverse event, so that exposure during the SGP is equivalent to birth in or around 1907. Most importantly, randomness in the assignment to treatment and control groups arises only due to randomness in the spacing of births. Random spacing then ensures that parental grandfathers and maternal grandmothers of very similar G3 individuals were of different ages when affected by the famine. Arguably, our design would be flawed if 1907 and the years around it were special in the sense that the male (female) descendants of male (female) individuals born at that time were systematically different.[34] While this in itself is unlikely, we address this concern by carefully designing the sample by choosing a control group that is heterogenous in a sense that about half of it is composed of older G1 cohorts and half of it is composed of younger G1 cohorts.

[33] The reply to the question on closeness to the grandparents is conditional on having living grandparents at age 18.

[34] However, macro-economic statistics of the years 1900-1910 do not suggest that the time around 1907 was special. For example, the German economy grew at a rather steady rate until World War I. In addition, we point out that if the cohorts born in and around 1907 are selective in a way that is unrelated to the effects we are interested in then we would expect this to affect the results for each of the four grandparents. It is unlikely that the presence of an effect of the parental grandfather's SGP exposure is caused by a mechanism that is unrelated to the effects we are after if we do not find an effect of the maternal grandfather's SGP exposure.

If cohort effects were important, the model would thus need to yield negative effects at very young and very old ages. A similar potential problem could arise if the descendants of the 1907 cohort were systematically exposed to different cohort-specific shocks later in life than the descendants of adjacent G1 cohorts. This would be a problem for example if World War II affected the well-being of the SGP famine cohort or their descendants differently than the one of adjacent cohorts. For example, it is likely that many G1 individuals, although mostly older than 30 years of age during World War II, were drafted in that war. However, this holds true for both the treatment and the control group, such that there should be no systematic differences in battlefield exposure. Also, World War II occurred after most G1 individuals (whether in the SGP famine cohort or not) had obtained their first children and before the G2 individuals reached ages at which they were drafted. Nevertheless, we deal with potential differences in exposure to historical events such as World War II by including indicators for G2 birth years and a large number of indicators of secular changes in society and background controls in the models we estimate.

An additional issue originates from the combination of the single adverse famine shock and the fact that the sampling starts with potential G2 members. The latter come from the cross section of individuals alive at some point during the SOEP observation window. Because of the age composition of the population, this means that they were born on average in the late 1930s. Any G1 parent of such an individual who was exposed to the famine in his/her SGP can not have been a young father in the late 1930s (on average they were around 30 years old). This could affect the G2 upbringing. Moreover, epidemiological studies have shown a positive association between higher paternal age at birth and leukocyte telomere length in adulthood (see e.g. Kimura et al., 2008). The latter is known to be associated with beneficial health outcomes later in life such as a lower mortality rate. We address again by conditioning on indicators of secular trends and background control variables. Moreover, among the G1 members who were not famine-exposed in their SGP we also observe many who received offspring in their thirties or after that.

Given the recurrence of the sex-specific mental health effects in a large number of robustness checks, it seems unlikely that our results were obtained by chance. In Section 6.3 we report evidence of a stronger sex-specific famine effect for descendants of G1 individuals who were more strongly affected by the famine. Moreover, we find that the effect persist for varying sets of background controls and if we change the composition of the control group to earlier or later born G1 individuals, respectively.

Similar to most datasets on several generations of humans our data do not allow us do distinguish a potential epigenetic pathway from other mechanisms. Instead, behavioral mechanisms are another possible explanation for our findings. The experience of economic hardship around the age of 10 has been found to increase resilience and psychological strength (Elder, 1999) and may therefore have positive mental health effects. However, it is not evident why behavioral mechanisms should work exclusively along sex-specific lines. Moreover, if third generation mental health was elevated due to an increase in first generation resilience and psychological strength, we would expect the effect to transmit to the third generation via elevated mental health levels among the second generation. Instead all mental health coefficients are insignificant for the second generation. From this we conclude that the mental health effects we find for the third generation are more likely biological in origin and plausibly the result of epigenetic transmission. Moreover, the analysis in subsection 6.4 hints towards a biological explanation behind our findings. Most importantly, we

find that our results persist after conditioning on G1 education and if we exclude those individuals who report a close or very close relationship with their grandparents.

8. Conclusion

This paper investigates whether undernutrition during adolescence causally affects the descendants of the second and third generation. We build on a recent line of literature in biology which finds that low food availability during the slow growth period of male individuals positively affects health outcomes of subsequent generations. The studies involved argue that such effects are potentially triggered by methylation of epigenetic marks in the sperm, with methyl tags being transmitted to subsequent generations via epigenetic imprinting.

We find that paternal grandfather SGP-famine exposure is associated with higher mental health of third generation sons, while maternal grandmother SGP-famine exposure has a positive effect on her granddaughters' mental health. We conclude that these third-generation mental health effects are causally related to a nutritional shortage during the SGP. The estimated effects are larger if famine exposure is redefined such that only the most severely affected individuals are included. Furthermore, the analyses indicate that it is implausible that the results are driven by social mechanisms. For example, mental health effects are largely absent among the second generation and do not seem to result from grandparent-grandchild interactions. They take place along sex-specific lines and are stronger and more robust for males than for females.

Interestingly, mental health is among the most responsive adult health outcomes as a function of nutritional conditions in utero, according to the literature on famine exposure (see the survey in Lumey, Stein and Susser, 2011). This effect should not be confounded with the effect detected in our study. The latter is driven by nutritional conditions in the slow growth period, and it works on subsequent generations. Nevertheless, the results provide a further confirmation that adult mental health is affected by past nutritional shocks within the family.

Concerning the literature on the intergenerational persistence of socio-economic status and health, our results indicate that the transmission dynamics may be more complex than what is often assumed. Causal effects that "jump" one generation can not be described by autoregressive models of order one (Solon, 2015). Such dynamic patterns may explain some of the high degree of persistence in outcome variables and direct grandparental effects found in recent studies (Clark and Cummins, 2014, Lindahl et al., 2013, Lindahl et al., 2014).

Research on transgenerational biological effects is only starting. Further studies are needed that provide evidence on how nutritional shocks transmit across generations, preferably including evidence on biological changes in the germ cells of first generation individuals. The present study may therefore motivate the construction or usage of data sets that contain elaborate retrospective information on nutritional shocks during childhood and adolescence, as well as biological measures of gene expressions and health outcomes for several generations.

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Table 1: Number of years of famine exposure during the slow growth period, first generation males and females

Birth year	1902	1903	1904	1905	1906	1907	1908	1909	1910	1911	1912	1913
Males	0	0	1	2	3	3	2	1	0	0	0	0
Females	0	0	0	0	1	2	3	2	1	0	0	0
Age during famine	14–16	13–15	12–14	11–13	10–12	9–11	8–10	7–9	6–8	5–7	4–6	3–5

Note: Famine years: 1916-1918. The slow growth period ranges from ages 8-10 and 9-12 for females and males, respectively.

Table 2: Sample size and ancestor famine affectedness

Variables	Second generation				Third generation			
	Males		**Females**		**Males**		**Females**	
Father famine in SGP	0.40	(0.49)	0.42	(0.49)	.	(.)	.	(.)
Mother famine in SGP	0.35	(0.48)	0.33	(0.47)	.	(.)	.	(.)
PGF famine in SGP	.	(.)	.	(.)	0.29	(0.45)	0.35	(0.48)
PGM famine in SGP	.	(.)	.	(.)	0.23	(0.42)	0.24	(0.43)
MGF famine in SGP	.	(.)	.	(.)	0.26	(0.44)	0.22	(0.42)
MGM famine in SGP	.	(.)	.	(.)	0.19	(0.39)	0.16	(0.37)
Birth year	1938.86	(6.36)	1938.76	(6.63)	1973.20	(7.85)	1974.41	(7.15)
N	2061		2077		715		576	

Source: SOEP.

Table 3: Descriptive statistics: Age at death, height, mental health and schooling outcomes by sex and generation

Variables	Second generation				Third generation			
	Males		**Females**		**Males**		**Females**	
Age at death	71.91	(6.35)	72.90	(6.24)				
Height	175.52	(6.63)	163.72	(6.03)	180.51	(7.05)	167.76	(6.06)
Mental health	52.50	(9.77)	50.45	(10.69)	49.44	(9.18)	47.57	(9.98)
Upper secondary school degree	0.36	(0.48)	0.19	(0.39)	0.49	(0.50)	0.52	(0.50)
N	2061		2077		715		576	

Source: SOEP.

Table 4: Main results: Third generation, parental SGP famine effects on height, mental health and schooling

Variables	**Males**						**Females**					
	Height		**Mental health**		**Schooling**		**Height**		**Mental health**		**Schooling**	
Paternal grandfather famine in SGP	0.191	0.358	1.577*	1.749**	-0.0187	0.0506	0.555	0.608	-0.0246	0.0482	-0.00395	0.0527
	(0.78)	(0.61)	(0.06)	(0.04)	(0.70)	(0.24)	(0.41)	(0.36)	(0.98)	(0.96)	(0.93)	(0.22)
Paternal grandmother famine in SGP	0.550	0.330	-0.133	-0.0825	0.0513	0.00737	-0.559	-0.645	0.425	0.580	0.0555	0.0423
	(0.45)	(0.65)	(0.88)	(0.92)	(0.30)	(0.87)	(0.43)	(0.37)	(0.68)	(0.59)	(0.30)	(0.37)
Maternal grandfather famine in SGP	-1.101	-1.097	0.459	0.385	-0.0415	-0.00850	0.805	0.743	0.297	0.397	0.0305	-0.00706
	(0.20)	(0.20)	(0.62)	(0.68)	(0.44)	(0.86)	(0.26)	(0.30)	(0.80)	(0.74)	(0.57)	(0.88)
Maternal grandmother famine in SGP	0.154	0.257	-0.535	-0.476	0.0434	0.0561	0.0685	0.101	2.195*	2.135*	0.00609	0.0499
	(0.87)	(0.77)	(0.62)	(0.65)	(0.45)	(0.26)	(0.93)	(0.89)	(0.08)	(0.10)	(0.92)	(0.34)
Observations	715	715	715	715	715	715	576	576	576	576	576	576
pval LLR-test		0		.03		0		.13		.13		0
Basic control variables	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
Background variables	NO	YES	NO	YES	NO	YES	NO	YES	NO	YES	NO	YES
Log lik.	-2387.6	-2380.8	-2577.3	-2573.6	-478.4	-410.0	-1836.1	-1834.0	-2121.7	-2119.6	-359.4	-303.3
R-squared	0.0630	0.0808	0.0588	0.0685			0.0611	0.0678	0.0671	0.0737		

Source: SOEP. **Note**: *p*-values in parentheses. Standard errors are robust and clustered on the household level. For schooling models, coefficients reported are average marginal effects. *pval LLR-test* is the p-value of a likelihood ratio test for the improvement in model fit due to the background variables. **Basic control variables**: detrended GDP per capita at first generation birth, parental age at birth, individual (G3) birth year fixed effects, indicator variables for whether the mother/father was born during World War II (until currency reform). **Background variables**: Parental education, number of siblings. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 5: Main results: Second generation, parental SGP famine effects on longevity, height, mental health and schooling

Variables	**Males**								**Females**							
	Mortality		**Height**		**Mental health**		**Schooling**		**Mortality**		**Height**		**Mental health**		**Schooling**	
Father famine in SGP	1.098	1.073	0.290	0.519*	-0.346	-0.198	-0.0838***	-0.0534***	1.304	1.315	-0.368	-0.278	0.726	0.780	0.00736	0.0271
	(0.50)	(0.62)	(0.36)	(0.10)	(0.47)	(0.68)	(0.00)	(0.01)	(0.13)	(0.12)	(0.20)	(0.33)	(0.16)	(0.13)	(0.70)	(0.14)
Mother famine in SGP	0.809	0.813	-0.390	-0.496	0.283	0.226	0.0372	0.0225	1.188	1.198	-0.243	-0.356	0.422	0.388	0.0277	0.0127
	(0.14)	(0.15)	(0.25)	(0.14)	(0.59)	(0.66)	(0.14)	(0.33)	(0.34)	(0.31)	(0.44)	(0.26)	(0.46)	(0.50)	(0.21)	(0.52)
Observations	2061	2061	2061	2061	2061	2061	2061	2061	2077	2077	2077	2077	2077	2077	2077	2077
pval LLR-test		0		0		0		0		.05		0		0		0
γ	0.169	0.170							0.190	0.191						
Basic control variables	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
Background variables	NO	YES	NO	YES	NO	YES	NO	YES	NO	YES	NO	YES	NO	YES	NO	YES
Log lik.	-188.228	-181.673	-6.8e+03	-6.7e+03	-7.6e+03	-7.6e+03	-1.3e+03	-1.2e+03	-178.558	-175.655	-6.6e+03	-6.6e+03	-7.8e+03	-7.8e+03	-965.675	-842.614
R-squared			0.068	0.108	0.035	0.046					0.036	0.059	0.038	0.045		

Source: SOEP. **Note**: *p*-values in parentheses. For the mortality models, coefficients are hazard rate coefficients. For schooling models, coefficients reported are average marginal effects. *pval LLR-test* is the p-value of a likelihood ratio test for the improvement in model fit due to the background variables. γ is an ancillary parameter that parametrizes the Gompertz baseline hazard function. Standard errors are robust and clustered at the household level. **Basic control variables**: detrended GDP per capita at first generation birth, individual (G2) birth year fixed effects, indicators for whether G1 born during World War II (until currency reform). **Background variables**: Parental education, number of siblings. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 6: Robustness checks: different definitions of the famine exposure variable

Variables	Males						Females					
	Height		**Mental health**		**Schooling**		**Height**		**Mental health**		**Schooling**	
Paternal grandfather famine in SGP ($\geq$ 2 yrs)	-0.0745		2.109**		0.00945		-0.0939		-0.617		0.0852	
	(0.93)		(0.05)		(0.86)		(0.91)		(0.61)		(0.10)	
Paternal grandmother famine in SGP ($\geq$ 2 yrs)	1.057		-0.819		-0.00392		0.0577		0.907		0.0513	
	(0.22)		(0.49)		(0.95)		(0.95)		(0.51)		(0.42)	
Maternal grandfather famine in SGP ($\geq$ 2 yrs)	-2.864***		0.506		0.0163		0.152		-0.0959		0.0223	
	(0.00)		(0.70)		(0.78)		(0.88)		(0.96)		(0.72)	
Maternal grandmother famine in SGP ($\geq$ 2 yrs)	0.994		-0.0326		0.0143		-0.318		1.649		-0.0800	
	(0.35)		(0.98)		(0.85)		(0.78)		(0.41)		(0.28)	
Paternal grandfather famine in SGP (incl. 1919)		0.264		1.585**		0.0408		0.582		-0.0762		0.0511
		(0.69)		(0.05)		(0.33)		(0.35)		(0.94)		(0.21)
Paternal grandmother famine in SGP (incl. 1919)		0.399		0.208		0.0393		-0.481		1.479		-0.00481
		(0.56)		(0.80)		(0.35)		(0.47)		(0.14)		(0.91)
Maternal grandfather famine in SGP (incl. 1919)		-1.264		0.458		0.0143		0.673		1.031		0.00746
		(0.12)		(0.61)		(0.76)		(0.35)		(0.37)		(0.86)
Maternal grandmother famine in SGP (incl. 1919)		0.730		-0.832		-0.00463		0.549		2.181*		0.0265
		(0.39)		(0.43)		(0.92)		(0.50)		(0.09)		(0.61)
Observations	516	715	516	715	516	715	416	576	416	576	416	576
Hunger period	$\geq$ 2 yrs	incl 1919	$\geq$ 2 yrs	incl 1919	$\geq$ 2 yrs	incl 1919	$\geq$ 2 yrs	incl 1919	$\geq$ 2 yrs	incl 1919	$\geq$ 2 yrs	incl 1919
Basic control variables	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
Background variables	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
Log lik.	-1680.4	-2378.5	-1839.8	-2573.0	-284.8	-409.4	-1303.8	-1832.3	-1512.7	-2117.2	-204.9	-299.0
R-squared	0.126	0.0867	0.0981	0.0700			0.102	0.0734	0.105	0.0814		

Source: SOEP. **Note**: *p*-values in parentheses. Standard errors are robust and clustered on the household level. For schooling models, coefficients reported are average marginal effects. *pval LLR-test* is the p-value of a likelihood ratio test for the improvement in model fit due to the background variables. **Basic control variables**: detrended GDP per capita at first generation birth, parental age at birth, individual (G3) birth year fixed effects, indicator variables for whether the mother/father was born during World War II (until currency reform). **Background variables**: Parental education, number of siblings. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 7: Third generation, robustness checks for different subsamples

Variables	**Males**			**Females**		
	City	**Upper sec**	**excl. inf.**	**City**	**Upper sec**	**excl. inf.**
Paternal grandfather famine in SGP	1.636	3.411	1.846**	0.204	-3.155	-0.508
	(0.14)	(0.16)	(0.04)	(0.89)	(0.69)	(0.65)
Paternal grandmother famine in SGP	0.879	-2.393	0.136	1.236	-2.657	0.758
	(0.43)	(0.23)	(0.88)	(0.41)	(0.59)	(0.54)
Maternal grandfather famine in SGP	1.055	-3.941	-0.0528	1.659	9.717*	0.0342
	(0.41)	(0.22)	(0.96)	(0.31)	(0.08)	(0.98)
Maternal grandmother famine in SGP	1.247	4.758	-0.0273	-0.278	4.668	2.100
	(0.37)	(0.12)	(0.98)	(0.89)	(0.57)	(0.13)
Observations	390	68	608	313	49	450
Basic control variables	YES	YES	YES	YES	YES	YES
Background variables	YES	YES	YES	YES	YES	YES
Log lik.	-1384.5	-196.3	-2191.3	-1139.2	-124.7	-1649.8
R-squared	0.123	0.637	0.0640	0.149	0.879	0.105

Source: SOEP Data. **Notes**: Standard errors are clustered at the household level. City denotes whether the second generation was raised in the city to proxy place of living of the first generation. Upper sec denotes G1 *upper secondary school education* (mostly provided in cities). excl. inf. denotes that the sample excludes G1 individuals who were born or in infancy during the famine (birth at or after 1920). **Basic control variables**: detrended GDP per capita at first generation birth, parental age at birth, individual (G3) birth year fixed effects, indicator variables for whether the mother/father was born during World War II (until currency reform). **Background variables**: Parental education, number of siblings. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 8: Robustness checks: excluding basic control variables, later cohorts, and placebo test

Mental health	Males						Females					
	Excl. WW dummy		**Control post 1918**		**Placebo**		**Excl. WW dummy**		**Control post 1918**		**Placebo**	
Paternal grandfather treated	1.537*	1.700**	1.270	1.577*	-1.556	-1.599	-0.0378	0.0387	0.00596	-0.0246	0.0563	0.213
	(0.06)	(0.04)	(0.18)	(0.05)	(0.30)	(0.29)	(0.97)	(0.97)	(1.00)	(0.98)	(0.98)	(0.92)
Paternal grandmother treated	-0.0774	-0.0364	-0.256	-0.133	1.303	1.466	0.532	0.702	0.698	0.425	-2.090	-2.207
	(0.93)	(0.97)	(0.78)	(0.87)	(0.28)	(0.22)	(0.61)	(0.51)	(0.53)	(0.68)	(0.17)	(0.14)
Maternal grandfather treated	0.487	0.394	-0.0972	0.459	0.913	1.036	0.464	0.560	0.862	0.297	-0.557	-0.779
	(0.58)	(0.65)	(0.92)	(0.60)	(0.44)	(0.38)	(0.69)	(0.63)	(0.51)	(0.80)	(0.67)	(0.55)
Maternal grandmother treated	-0.570	-0.507	-0.677	-0.535	-0.952	-0.959	2.146*	2.109*	2.344*	2.195*	0.447	0.636
	(0.56)	(0.60)	(0.52)	(0.58)	(0.36)	(0.35)	(0.09)	(0.09)	(0.08)	(0.08)	(0.70)	(0.59)
Observations	715	715	459	459	715	715	576	576	389	389	576	576
pval LLR-test		.03		1		.03		.13		1		.12
Basic control variables	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
Background variables	NO	YES	NO	YES	NO	YES	NO	YES	NO	YES	NO	YES
Log lik.	-2578.6	-2574.9	-1642.3	-2577.3	-2578.2	-2574.8	-2124.2	-2122.2	-1430.0	-2121.7	-2122.2	-2120.1
R-squared	0.0554	0.0650	0.0811	0.0588	0.0563	0.0654	0.0589	0.0655	0.109	0.0671	0.0655	0.0723

Source: SOEP. **Note**: *p*-values in parentheses. Standard errors are robust. In columns 1, 2, 7 and 8 G2 war-birth controls are excluded. In columns 3, 4, 9 and 10 the sample is selected, such that all control group paternal grandfathers for the male G1 sample (maternal grandmothers for the female G1 sample) are born after the famine. In columns 5, 6, 11 and 12 descendants of G1 individuals born after the famine are assigned to be the treatment group as a placebo test. For schooling models, coefficients reported are average marginal effects. *pval LLR-test* is the p-value of a likelihood ratio test for the improvement in model fit due to the background variables. **Basic control variables**: detrended GDP per capita at first generation birth, parental age at birth, individual (G3) birth year fixed effects. **Background variables**: Parental education, number of siblings. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 9: Robustness checks for G3: allowing for one missing ancestor per model

Variables	**Males**				**Females**			
Paternal grandfather treated	1.378**	1.293**	1.510***		-0.198	0.329	0.584	
	(0.02)	(0.03)	(0.01)		(0.76)	(0.64)	(0.38)	
Paternal grandmother treated	-0.214	-0.103		0.337	0.578	0.633		0.642
	(0.72)	(0.87)		(0.57)	(0.43)	(0.40)		(0.39)
Maternal grandfather treated	-0.109		0.258	-0.0253	0.794		0.695	0.453
	(0.85)		(0.67)	(0.97)	(0.30)		(0.39)	(0.57)
Maternal grandmother treated		-0.389	-0.548	-0.571		2.379***	2.305***	2.250**
		(0.54)	(0.41)	(0.39)		(0.00)	(0.01)	(0.01)
Observations	1371	1270	1360	1221	1127	1039	1090	957
Basic control variables	YES	YES	YES	YES	YES	YES	YES	YES
Background variables	YES	YES	YES	YES	YES	YES	YES	YES
Log lik.	-4917.5	-4569.6	-4902.6	-4376.6	-4143.8	-3822.5	-4011.6	-3525.5
R-squared	0.0697	0.0608	0.0684	0.0615	0.0697	0.0667	0.0707	0.0757

Source: SOEP. **Note**: *p*-values in parentheses. We allow for missing information for the ancestor on for whom we do not estimate the effect of being famine exposed during SGP. Standard errors are robust. In columns, 5, 6, 11 and 12 individuals born after the famine are assigned to be the treatment group. For schooling models, coefficients reported are average marginal effects. *pval LLR-test* is the p-value of a likelihood ratio test for the improvement in model fit due to the background variables. **Basic control variables**: detrended GDP per capita at first generation birth, parental age at birth, individual (G3) birth year fixed effects. **Background variables**: Parental education, number of siblings. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 10: Famine effect on first-generation education outcomes

Variables	**Father**		**Mother**	
	Upper sec	**Secondary**	**Upper sec**	**Secondary**
Male individual treated in SGP by hunger	-0.0240***	-0.0176**		
	(0.01)	(0.05)		
Female individual treated in SGP by hunger			0.00567	0.00942
			(0.36)	(0.37)
Observations	4138	4138	4138	4138
Birth year trend	YES	YES	YES	YES
Log lik.	-1334.9	-1213.9	-572.9	-1399.3
Pseudo R-squared	0.00473	0.00162	0.00838	0.00335

Source: SOEP Data. **Notes**: *p*-values in parentheses. Table displays average marginal effects. Standard errors are robust and clustered on the household level. Upper sec and Secondary refer to *upper secondary school education* and *secondary school education* respectively (the base category being lower secondary school education). * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 11: Effects of first-generation SGP famine and first-generation education on third-generation mental health outcomes

Variables	**Males**		**Females**	
	Mental health			
Paternal grandfather famine in SGP	1.647**	1.801**	0.0876	0.0996
	(0.04)	(0.03)	(0.93)	(0.92)
Paternal grandmother famine in SGP	-0.197	-0.148	0.430	0.611
	(0.82)	(0.86)	(0.68)	(0.56)
Maternal grandfather famine in SGP	0.449	0.377	0.525	0.690
	(0.63)	(0.69)	(0.65)	(0.56)
Maternal grandmother famine in SGP	-0.703	-0.636	1.807	1.693
	(0.52)	(0.55)	(0.15)	(0.18)
Paternal grandfather upper secondary school	3.631**	3.526**	-0.195	-0.258
	(0.01)	(0.01)	(0.93)	(0.91)
Maternal grandfather upper secondary school	-1.797	-1.733	-1.421	-1.287
	(0.27)	(0.28)	(0.38)	(0.44)
Paternal grandmother upper secondary school	-1.804	-1.837	3.178	3.101
	(0.35)	(0.33)	(0.27)	(0.30)
Maternal grandmother upper secondary school	1.442	2.022	-5.272*	-5.095
	(0.55)	(0.41)	(0.10)	(0.11)
Paternal grandfather secondary school	-0.0867	0.00125	1.908	2.125
	(0.95)	(1.00)	(0.23)	(0.18)
Paternal grandmother secondary school	-1.147	-1.463	-0.817	-0.564
	(0.44)	(0.33)	(0.63)	(0.74)
Maternal grandfather secondary school	-0.140	0.0142	0.582	0.843
	(0.91)	(0.99)	(0.72)	(0.62)
Maternal grandmother secondary school	-0.0805	0.581	4.303***	4.291***
	(0.95)	(0.66)	(0.00)	(0.00)
Observations	715	715	576	576
Basic control variables	YES	YES	YES	YES
Background variables	NO	YES	NO	YES
Log lik.	-2573.5	-2569.9	-2114.4	-2112.6
R-squared	0.0687	0.0781	0.0904	0.0960

Source: SOEP Data. **Notes**: Standard errors are robust and clustered at the household level. **Basic control variables**: detrended GDP p.c. at first generation birth, parental age at birth, individual (G3) birth year fixed effects, indicators for mother/father born during World War II (until currency reform). **Background variables**: Parental education, number of siblings. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 12: Third generation, parental SGP famine effects on third-generation mental health outcomes (excluding individuals who report a close relationship to their grandparents)

Variables	**Males**		**Females**	
	Mental health			
Paternal grandfather famine in SGP	1.911**	1.935**	-0.137	-0.115
	(0.03)	(0.03)	(0.90)	(0.92)
Paternal grandmother famine in SGP	0.0317	-0.0102	0.688	1.001
	(0.97)	(0.99)	(0.57)	(0.41)
Maternal grandfather famine in SGP	0.360	0.242	0.560	0.831
	(0.72)	(0.81)	(0.64)	(0.50)
Maternal grandmother famine in SGP	-0.452	-0.370	2.274*	2.206
	(0.69)	(0.73)	(0.09)	(0.10)
Observations	592	592	462	462
pval LLR-test		.25		.02
Basic control variables	YES	YES	YES	YES
Background variables	NO	YES	NO	YES
Log lik.	-2129.9	-2128.5	-1698.4	-1694.2
R-squared	0.0806	0.0850	0.0736	0.0900

Source: SOEP Data. **Notes**: Standard errors are clustered at the household level. For schooling models, coefficients reported are average marginal effects. *pval LLR-test* is the p-value of a likelihood ratio test for the improvement in model fit due to the background variables. **Basic control variables**: detrended GDP p.c. at first generation birth, parental age at birth, individual (G3) birth year fixed effects, indicators for mother/father born during World War II (until currency reform). **Background variables**: Parental education, number of siblings. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.