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ABSTRACT

Instrumental Variable Estimation of the Causal Effect of Hunger Early in Life on Health Later in Life^{*}

Numerous studies have evaluated the effect of nutrition early in life on health much later in life by comparing individuals born during a famine to others. Nutritional intake is typically unobserved and endogenous, whereas famines arguably provide exogenous variation in the provision of nutrition. However, living through a famine early in life does not necessarily imply a lack of nutrition during that age interval, and vice versa, and in this sense the observed difference at most provides a qualitative assessment of the average causal effect of a nutritional shortage, which is the parameter of interest. In this paper we estimate this average causal effect on health outcomes later in life, by applying instrumental variable estimation, using data with self-reported periods of hunger earlier in life, with famines as instruments. The data contain samples from European countries and include birth cohorts exposed to various famines in the 20th century. We use two-sample IV estimation to deal with imperfect recollection of conditions at very early stages of life. The estimated average causal effects often exceed famine effects by a factor three.

JEL Classification: I12, J11, C21, C26

Keywords: nutrition, famine, ageing, developmental origins, height, high blood pressure, obesity, 2SLS, two-sample IV

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1 Introduction

During the past decade, the effect of early-life conditions on health outcomes late in life has become a focal point of research in economics, demography, epidemiology, biology, and related fields.¹ Empirical studies use non-experimental data of real-life individuals and typically relate indicators of early-life conditions to outcomes of the same individuals at high ages. A major concern is that observed conditions in the parents' household early in life, including conditions in utero as captured by birthweight, and outcomes later in life, are all jointly dependent on unobserved confounders. To be able to detect causal effects, one needs to observe exogenous variation in the early-life conditions, and relate this to outcomes later in life. Moreover, this variation should only affect high-age health by way of the individual early-life conditions and not through secular or cohort-specific changes in society after the early-life period of interest. As a result, candidate indicators are often not some unique characteristic of the newborn individual or his family or household, but rather a temporary state of the macro environment into which the child is born. In that case they are also called contextual variables.

In the literature, the most frequently used indicator is whether the individual has been exposed to a famine at a specific childhood age, notably in utero. Lumey, Stein and Susser (2011) provide an excellent overview. The underlying idea is that in many households birth in a famine involves nutritional shortages, in terms of quality and/or quantity of nutrition. Birth outside of a famine is expected to involve better nutritional conditions. A negative association between birth during a famine and a favorable health outcome later in life is interpreted as evidence for a causal effect of early-life conditions on that health outcome.² Studies have detected associations with adult height, schizophrenia, mortality, fertility, hospitalization, next generation birthweight and even gene expression. Interestingly, a few studies have also reported associations between exposure to a famine at the onset of puberty and late-life health outcomes. For example, Sparén et al. (2004) find that boys who had been exposed around age 9 to the Leningrad siege famine of 1941 have higher rates of cardiovascular morbidity and mortality much later in life. Oppers (1963) finds an effect of the Dutch Hunger Winter famine exposure at ages 7-14 on adult height.³

¹For overviews, see e.g. Pollitt, Rose and Kaufman (2005), Barker (2007), Lawlor (2008) and Almond and Currie (2011).

²Note that also this requires that the composition of newborns is not systematically different between famine-born and non-famine born, in terms of unobserved characteristics of the newborns.

³Many qualitative results from the famine-based studies are in agreement to those in studies using other contextual indicators of conditions around birth, such as business cycles and seasons. See e.g. Van den Berg and Lindeboom (2012) for an overview. Van den Berg et al. (2010) use immigrant siblings to study the effect of economic conditions at later childhood ages, and they find a significant effect on adult height. They also survey other studies concerning later childhood ages.

However, exposure to a famine is not equivalent to exposure to a nutritional shortage. During a famine, a fraction of all households does not face food shortages, for example because the household belongs to the ruling or wealthy class, or because it is self-sufficient in terms of food, or, in a war context, because it is allied to those responsible for the cause of the famine. Similarly, in non-famine eras, a fraction of households faces food shortages because of poverty. This means that the comparison of famine-born individuals to non-famine-born individuals does not provide a quantitative estimate of the average causal effect of nutritional shortages around birth. Most likely, the latter effect is under-estimated in absolute value by such a comparison.

To advance on this, it is necessary to observe the nutritional status in the households at the time interval in which the child is in utero or at the childhood age of interest. But this is almost impossible given that the study outcomes concern health at high ages. To observe these outcomes, the individuals in the data need to have been born a long time ago. This means that the critical period of interest necessarily occurred say before 1950. At the time, data on household conditions were not systematically collected. Moreover, for some of the sample, the critical period must have occurred during a famine. During famines, data are typically not collected, as societies are in a state of disruption.

In this paper we deal with these problems by using self-reported retrospective data on the individual occurrence of a period of severe hunger at certain childhood ages. Specifically, we relate these data to the occurrence of famines at those ages and to health outcomes of the same individuals later in life, and we use Instrumental Variable (IV) estimation techniques to estimate the average causal effects of nutritional shortages during certain childhood ages on health outcomes later in life. IV is an econometric technique to estimate a causal effect of a causal factor on an outcome, with observational data in which the cause (in our case, nutritional shortage) is not exogenous while there is a variable (famine) that affects the causal factor but that does not have an effect on the outcome except through the causal factor. In terms of the IV treatment evaluation literature, our instrumental variable is the exposure to a famine early in life, our treatment is the experience of a nutritional shortage early in life, and our outcome is health at high ages. With heterogeneous effects, the IV estimation provides so-called local average treatment effects (LATE; see Imbens and Angrist, 1994).

Our approach is novel. We are arguably the first to use the occurrence of famines to obtain an estimate of the average *causal* effect of *nutritional shortages* during specific childhood ages on health later in life. Notice that IV is generally not applicable in studies of long-run effects of early-life conditions, because of non-observability of household conditions early in life. This is not specific for famines as early-life indicators but also applies to other contextual indicators such as business cycles, seasons, weather, and infant mortality rates.

As a by-product, our study provides estimates of the strength of the association between a famine and an actual hunger episode. In terms of the IV treatment evaluation literature, we look at “compliance” to the instrument.

Our individual data are from the Survey of Health, Aging, and Retirement in Europe (SHARE), a European longitudinal survey based on a random sample of individuals aged 50+. The most recent wave asks respondents for retrospective accounts of specific aspects of their lives. The birth cohorts in the data include cohorts that were exposed to the famines in the Netherlands, Germany, or Greece, in various time intervals in the 1940s. These are the three well-studied famines in this time frame, at least in the countries participating in SHARE. Their origins are well-established in the literature, and they have been used to study long-run effects (see the literature discussion in Section 2). Notice that evidence based on multiple famines is less sensitive to culture or cohort-specific conditions.

Strictly speaking, it is the cause of the famine that is the ultimate instrumental variable, instead of the famine itself. After all, any period or era in which many people are hungry may be called a famine. What matters for the study of long run effects is that the famine is an exogenous event with no long-run impact apart from the effect running through household-specific nutritional conditions. This is more likely if the famine is due to an external intervention in society and if the famine is short and is not anticipated. The three famines in our data satisfy these requirements, as they are all due to trade blockades and destruction in the context of World War II and its aftermath.

Nutritional shortages that only took place in utero will not be reported as episodes of hunger early in life. Even with a perfect recollection of past periods of hunger (e.g. if the individual obtained this information from his or her parents), a spell in utero will only be reported if it stretches beyond the day of birth. More in general, recall of a period of severe hunger may be more difficult if this period took place around birth. Indeed, in our data, the reporting of hunger during a famine is low if the age during the famine was close to zero. We deal with this by using two-sample IV (2SIV) estimation. This method has been developed by Angrist and Krueger (1992) and Arellano and Meghir (1992); see Ridder and Moffitt (2007) for an integrated overview. Intuitively, when we consider long-run effects of nutritional shortages for newborns, we relate famines around birth to health later in life, but we may use a sample of older children to estimate the connection between famine exposure and nutritional shortage. This requires the assumption that the latter connection is the same for all children. This is not innocuous. As we shall see, there is anecdotal evidence of special food support for young children during famines that was not available for older children. In that case, our estimates provide a lower bound for the average causal effect.

The SHARE data have established a high reputation in terms of quality, and by now

many studies have been published using these data (see e.g. Börsch-Supan et al., 2008). Our particular empirical analysis faces two data design limitations. First, for our purposes, the sample is not large. The number of respondents per country is around 1500. However, to be exposed to a famine at a specific age, the respondent needs to have been a child (or in utero) in one of three countries in a birth cohort interval with a length equal to the famine, where the famine duration ranges from a few months to at most a few years. Secondly, the survey questions concerning the period of severe hunger ask for an interval in terms of full calendar years. In each analysis we therefore need to define and align three different intervals early in life: the relevant age interval in which nutritional shortage may cause long-run effects; the calendar time interval for which the individual reports severe hunger, and the calendar time interval in which the famine took place. Inevitably, we have to make several shortcuts, and it is important to address the sensitivity of the results with respect to this. In fact, given the relatively small sample size, moderate changes in the definitions of these intervals only affect the status of few respondents, and the results are often insensitive to this.

Since we aim to estimate a causal effect of nutritional shortages in general, the relevance of our findings should stretch beyond famine-stricken societies. Child hunger is not only prevalent in many parts of the developing world, but also in industrialized countries. “Feeding America” reports that even in the US 11.9 million citizens regularly suffer from hunger. From these, around a third are children under the age of 18. Undernutrition is essentially an economic problem that can be mitigated by public policy. From an economic point of view, it matters to find out at which age of the children exposure to nutritional shortages is most detrimental in the long run. This helps to address which policy measures are most efficient and cost-effective in preventing adult health problems.

The paper proceeds as follows. In Section 2 we review the explanatory frameworks to understand the long-run effects. Section 3 describes the three famines in our observation window and summarizes the evidence obtained so far for those famines. In Section 4 we describe our data. Section 5 formally presents the econometric methods. Section 6 presents our results. We do not only study effects of adverse conditions around birth but we also identify whether periods in early adolescence are critical with respect to nutritional shocks. We also consider the strength of the association between famines and actual nutritional shortages. This is of importance for the empirical literature in which the three famines have been used as indicators of early-life conditions. Section 7 concludes.

2 Explanatory frameworks for causal long-run effects of conditions early in life

2.1 Conditions around birth

Most explanations for long-run effects of nutritional conditions around birth build on Barker's fetal origins or fetal programming hypothesis (see e.g. Barker, 1994). Effects of fetal undernutrition on metabolic adaptation in utero may affect the phenotype such that the risk of cardiovascular disease later in life is increased (Hales and Barker, 1992, Bateson, 2001, Gluckman and Hanson, 2004). Underlying this model is the idea that several critical periods early in life influence the development of humans. During these periods, developing systems modify their settings in response to social and biological cues (Kuzawa and Quinn, 2009). This includes durable epigenetic changes that modify gene expressions.

Along this way, adverse conditions are known to influence inflammation, measured in terms of interleukin-6 production, in adolescence and adulthood, plausibly through changes in gene expressions (see e.g. Morozink et al., 2010). An episode of hunger early in life may thus engender a proinflammatory phenotype. Over time, this takes an allostatic toll on the body, resulting in a higher risk of chronic diseases later in life (Morozink et al., 2010, Miller and Chen, 2010, Miller et al., 2009, Zhang et al., 2006, Cole et al., 2010), notably cardiovascular diseases, diabetes and hypertension. Yet, such mechanisms should be seen as a predictive adaptive response to the future environment (Gluckman and Hanson, 2004, Cole et al., 2010). The long-run effects of reduced nutrition in utero are stronger if the affected individuals are exposed to a much more favorable environment in childhood (Schulz, 2010).

Of course, severely adverse nutritional conditions may also directly affect the build-up of organs and other body parts. This involves the postneonatal period but also puberty (see e.g. the survey by Cameron and Demerath, 2002).

The above causal pathways are all biological after the initial nutritional shortage. However, non-biological mechanisms are also possible. An episode of hunger may lead to a permanent change in the role of food in the household. After such an episode, adults may cook more greasy food or force their children to finish their plates by any means (see e.g. Hamelin, Habicht and Beaudry, 1999, for changes in household behaviors in response to food insecurity). This may cause adverse long-run health outcomes of the children. Also, an episode of hunger may induce fights for resources and thus a higher level of stress, changing within-household relationships and leading to a prolonged exposure to a higher stress level (Hadley and Patil, 2006, Whitaker, Phillips and Orzol, 2006).

2.2 Conditions in later childhood

Recently, interest has increased in long-run effects of nutritional conditions after birth. Gluckman, Hanson and Pinal (2005) and Barker (2007) give overviews of the underlying medical mechanisms. Particular attention has been given to the onset of puberty as a sensitive period. As documented and surveyed by Marshall and Tanner (1986), Gasser et al. (1994), and Zemel (2002), the earliest manifestation of puberty concerns the so-called “fat spurt” around age 9-10. In this spurt, the body collects resources in anticipation of the adolescent growth spurt. Sparén et al. (2004) argue that nutritional distortions and stress at this stage may lead to a permanent disruption of blood pressure regulation, leading to long-run cardiovascular health problems. Other studies have related the calcium intake around age 9 to adult height.

A nutritional shortage may also have an instantaneous adverse effect on schooling decisions and outcomes (see e.g. Jyoti, Frongillo and Jones, 2005) and thus affect health outcomes via realized education or adult socioeconomic status (Leigh, 1983). Lastly, adverse conditions may affect children positively by inducing more responsible behavior. For example, Elder (1999) investigates the impact of the Great Depression on children born in 1920-1921 and finds that the experience of economic hardship around the age of 10 led to more resilience and psychological strength.

3 The famines in European countries in the 1940s

3.1 The Dutch famine

The Dutch famine has been studied for decades as a cause of adverse living conditions. Therefore, the following account can be brief. The relevant literature starts with contemporaneous studies, notably Dols and Van Arcken (1946), who provide a detailed description of the occupation and the famine and who report data on rations and agricultural production, and Banning (1946), who focuses on public health issues.

Prior to World War II, food standards had been high in the Netherlands, both in terms of caloric value as well as composition of the diet. There were no notable disruptions in food availability during the first years of the occupation of the Netherlands, which started in May, 1940. In September 1944, parts of the South of the country were liberated, and the London-based Dutch Government in exile called out a railroad strike in the occupied parts of the Netherlands in order to support Operation Market Garden and in order to display its authority over the occupied nation. As a reaction, the occupying forces initiated an embargo that prohibited any food transports to the densely populated western part of the country, i.e. the provinces of North and South Holland and Utrecht. This sanction, in

combination with the early onset of the harsh winter of 1944/45, the freezing of waterways, and the generally bad state of transport infrastructure effectively closed off the western part of the country from any imports of food, fuel, medication etc. This triggered the Dutch “hungerwinter”. Individuals had to live on rations as low as 500 kcal per day. For school children, average rations amounted to 664 kcal in the first quarter of 1945. The situation lasted until the end of the occupation which coincided with the end of World War II (early May 1945). Immediately, rations rose to 2,400 kcal per day. Following most of the literature on the Dutch famine, we take November 1944 to be the onset of the famine spell. This is later than the onset of the strike in mid-September 1944.⁴

The excess death rate in the first half of 1945 over the rate in 1944 amounts to 269 percent for men and 173 percent for women (Dols and Van Arcken, 1946). Banning (1946) reports a higher incidence of tuberculosis and hunger oedema and an increased infant mortality rate. Inhabitants of large cities were struck hardest by the famine. However, Banning (1946) notes that in small towns mortality rates rose to a level almost as high as those in large cities. Special aid was targeted at starving children and young adults by the “Inter-Church Council”, an organization formed of different clerical associations (first, the focus was on children aged 5 to 16; the inclusion of children aged 3 to 5 followed later on). Help was provided in the form of additional food rations but in addition, effort was taken to send adversely affected children to districts where the food situation was somewhat better. Reports on the activities of the council yield information about the situation of the children in the famine-struck areas: Banning (1946) mentions that of the potential candidate children examined, 29% had been severely undernourished, while 31% suffered moderate undernourishment. About 27% of the children displayed a weight loss of about 10% of their weight.

Studies based on the Dutch famine indicate significant long-run effects on adult morbidity.⁵ The overview in Painter, Roseboom and Bleker (2005) lists long-run effects on the risk of cardiovascular diseases, obesity, breast cancer, cholesterol levels, diabetes, and self-perceived health. See also the survey in Lumey, Stein and Susser (2011) for effects of prenatal famine in particular. Lumey et al. (2007) find effects on anthropometric measures indicative of the reposition of fat, dermatoglyphic characteristics and a modest relationship with blood pressure. An elevated risk of schizophrenia at adult ages after prenatal exposure has been found for both genders (e.g. Susser and Lin, 1992). Susser and Stein (1994) find that adult stature is susceptible to the postnatal but not the prenatal environment.

⁴For school children, official rations dropped below 1,200 kcal in early November 1944 and to 1,000 kcal at the end of November 1944 (Dols and Van Arcken, 1946). On average, caloric consumption amounted to 1,073 kcal in the last quarter of 1944 (Dols and Van Arcken, 1946).

⁵Recall that in this section we only cite studies based on the famines we consider in our empirical analysis. Results based on other famines are discussed in the overview studies listed in Section 1.

Oppers (1963) finds a negative effect of the Dutch Hunger Winter famine exposure at ages 7-14 on adult height among men. In a landmark study, Heijmans et al. (2008) show that individuals who were exposed to the famine in the early stages of pregnancy had, 60 years later, less DNA methylation of a certain imprinted gene, compared with their unexposed same-sex siblings. The gene is the insulin-like growth factor II, which is a key factor in human growth and development. Their study provides strong empirical evidence for the epigenetic pathway discussed in Subsection 2.1 above.

3.2 The Greek famine

At the end of April 1941, Greece surrendered to Axis forces and was subsequently divided into 13 different zones occupied by Germany, Italy, and Bulgaria.⁶ These areas were isolated from each other and the transfer of goods and individuals was often close to impossible. The famine was triggered by a naval blockade of the Allies which made it impossible to supply foodstuffs to Greece. Despite being an agricultural country, Greece heavily relied on food imports (Hionidou, 2006, mentions that in 1939, over 20 percent of the wheat consumption had been imported). In addition, the blockade prohibited fishing at sea.⁷ The food situation quickly deteriorated. While the caloric value of the rations allocated by the occupiers in the Athens area had already been below any subsistence level in July 1941 (600 kcal), the rations were cut even further such that they amounted to only 320 kcal in November 1941 (Neelsen and Stratmann, 2011). The regions of Greater Athens and Piraeus were affected most severely. Mortality rates suggest that there was a general positive correlation between the famine's severity and the degree of urbanization. Furthermore, Hionidou (2006) notes that society was very unequally affected by the famine during this period; the low social classes suffered the most from deprivation, which can be attributed to the fact that soon after the rationing system had been superimposed by the occupiers, black market activity flourished with prices beyond the levels an ordinary worker could ever afford. The winter 1941/42 marked the maximum of the famine period in terms of fatalities.

The blockade was formally called off in February 1942. As of then, the Red Cross provided assistance targeted mostly at young children in Athens (Neelsen and Stratmann, 2011). At the end of March 1942, shipments by the "Joint Relief Commission" under Swedish command arrived. Subsequently, the situation in most parts of Greece improved. Hionidou (2006) reports declines in excess mortality from April 1942 onwards, even though in urban areas mortality remained high longer. We define the Greek famine to run from May

⁶This subsection relies heavily on Hionidou (2006) and Valaoras (1946).

⁷In the sense that the famine was caused by an external intervention in society, and in the sense that a large fraction of the excess mortality during the famines was due to starvation, the famines discussed in this section are "modern famines" in the terminology of O'Grada (2009).

1941 until and including June 1942. The ending date is somewhat later than in Hionidou (2006) and somewhat earlier than in Neelsen and Stratmann (2011).

Valaoras (1946) reports death rates for Athens to have been six times higher than under usual circumstances. Both Valaoras (1946) and Hionidou (2006) state that the largest fraction of the excess mortality during the famine is attributable to starvation, whereas epidemics and infectious diseases are of minor importance. Of special interest for our present study are the figures cited in Valaoras (1946) on the situation of children and adolescents. The results of surveys conducted during the famine and shortly afterwards found children aged 4 to 14 to be massively underweight, while babies and young children up to age 4 had almost normal weight. Moreover, growth retardation was found among many children 8 to 14 years old.

Neelsen and Stratmann (2011) consider long-run effects of the Greek famine on economic outcomes. They find effects of exposure during infancy on the attained level of education and on prime-aged labor market outcomes. Effects of exposure in utero are very small.

3.3 The German famine

After World War II, Germany faced various structural problems that led to the catastrophic situation we label the German Famine. Specifically, (i) many cities and much of the industry and transportation infrastructure were destroyed by Allied bombardments, (ii) millions of citizens and military had died, (iii) factories were dismantled and shipped off to the occupying powers, (iv) the agricultural land in the East which had been a major provider of food had been lost to Poland and the Soviet Union, (v) about 10 million refugees from the lost lands in the East had fled to the new German mainland, and (vi) Germany was divided into 4 occupation zones that were administered separately. Inter-zone trade was difficult because of political and bureaucratic barriers and because of a lack of transportation means. See Klatt (1950), Farquharson (1985), Trittel (1990), Häusser and Maugg (2009) and Reichardt and Zierenberg (2009) for overviews.

Before World War II, estimated daily caloric consumption amounted to about 3000 kcal, while during the war, civilians used foodstuffs of about 2500 kcal per day (Klatt, 1950). At the end of the war (European Theater) in May 1945, nutritional conditions deteriorated to levels far below those before or during the war. For May 1945, Trittel (1990) reports an average caloric ration of only 1200 kcal. After May 1945, these values decreased further. For example, in the British-administered Ruhrgebiet, only approximately 600 kcal per person was assigned in June 1945. The situation further deteriorated during the so-called “Hungerwinter” of 1946-47, which was an unusually cold winter. From mid-December 1946,

the distribution of food collapsed in many areas. Until March 1947, real average daily caloric intake was around 800 kcal per day for some cities in the Ruhrgebiet. The crop of 1947 failed to meet the demand of the starving population, so the period of hunger lasted until the spring of 1948, when a combination of foreign help and political reforms managed to improve nutritional conditions. Conditions improved spectacularly with the currency reform in June 1948. We therefore define the famine to last from June 1945 until June 1948.

As usual, the severity of the famine exhibits regional variation on top of the above-mentioned temporal variation (see Willenborg, 1979, and the references above). Notably, the different zones of occupation were affected differentially, mostly due to differences in the policies of the occupying powers. For example, the average daily food intake of a standard consumer in 1946 is estimated to equal 1610, 1430, 1535, and 1340 kcal in the US, the Soviet, the British, and the French zone, respectively, while the corresponding official food rations equalled 1330, 1083, 1050, and 900 kcal, respectively (see Cornides, 1948, and Echterkamp, 2003).

Apart from excess deaths due to starvation, sources imply that the famine also exerted additional adverse effects on the population. For instance, Klatt (1950) reports cases of retarded growth of children in the Ruhrgebiet and a weight deficit among children of about 20 percent of the normal weight. The deficit was most pronounced for children above age 10. In a study of undernutrition in the city of Wuppertal, Dean (1951) analyzes birth registers of a local hospital and reports reduced birth weights for the years of the famine with the greatest decline for the year of 1945.

Jürges (2011) analyzes the effect of birth during the German famine on educational attainment and occupation in 1970. He finds strong evidence that these outcomes are particularly adverse for those born in the winter of 1945/46. This suggests that an indirect causal pathway from early-life conditions through education and occupation to health later in life may be possible. Some epidemiological studies consider cohort-specific adult health outcomes without focusing a priori on famine cohorts. Notably, Hermanussen, Danker-Hopfe and Weber (2001) use data on male conscripts; they observe that the 1946 cohort stands out in terms of low obesity at age 19 even though the average weight is not much lower than for the surrounding cohorts. To a small extent, this also applies to the cohorts born during World War II. It is possible that this is because these cohorts faced childhood conditions that were less out of tune with conditions in utero than the cohorts later in the German famine (recall the discussion in Subsection 2.1). In this sense, individuals born in World War II may face less adverse long-run effects than those born in the 1946/47 “Hungerwinter”. Onland-Moret et al. (2005) use large samples of women from a large range of European countries. They report the average height by country and by 5-year

birth cohorts. The average height among women born in Germany in 1945-49 is much lower than that in the surrounding 5-year cohorts. For Greece and the Netherlands no such patterns are found, which is not surprising in the light of the fact that the famine spells in those countries spanned only a small part of a 5-year interval.

4 Data

For the empirical analysis we use the Survey of Health, Aging, and Retirement in Europe (SHARE), a pan-European multidisciplinary data set on individuals aged 50 and over. SHARE consists of three waves; the first two waves provide information on health, socioeconomic status, family characteristics and well-being. The third wave (called SHARELIFE), collected in 2008 and 2009, provides detailed life-cycle histories of individuals for specific thematic fields, such as employment or accommodation.⁸ The data are unique and well-suited for a causal analysis of severe undernutrition on adult health, because they do not only contain a nationally representative sample of countries that were affected by periods of famine, but also fully comparable, rich histories of retrospective information on hunger periods for each individual.

Sample construction We use 5,750 individuals of the cohorts born 1920 to 1955, who lived in Germany, the Netherlands or Greece during their youth. This excludes foreign citizens as well as individuals who, due to physical and cognitive limitations, were unable to complete the interview themselves.⁹ We also exclude individuals with missing information in the outcome variables, the hunger variable or covariates. Furthermore, we lose observations where individuals deceased ($\approx 3.5\%$) and or left the survey between waves 1 and 3 ($\approx 25\%$). Sample attrition is high in SHARE. We treat these observations as missing at random, keeping in mind that even if attrition was somehow related to health performance, this would make our estimates more conservative.

One exceptional feature of SHARE is that respondents were asked whether they had ever experienced periods of hunger in their lives and if so, in which year of their life this period began and in which year it ended. The survey question reads “Looking back at your life, was there a distinct period during which you suffered from hunger?”. Although this question alludes to an unhappy period of life, nonresponse is very small and amounts to less than 0.5%. However, the information may be prone to recall bias, since it is self-reported

⁸For general information on SHARE and SHARELIFE, see Börsch-Supan et al., 2008 and Schröder, 2011.

⁹We drop the full proxy interviews but keep assisted interview information. Moreover, we employ this criterion only for the interviews yielding the outcome measures and the undernutrition indicator.

and dates back many years.¹⁰ Moreover, recall bias is likely to be systematically higher if a period of severe hunger took place around birth. At the extreme, nutritional shortages that *only* took place in utero can not be reported as episodes of hunger.

To deal with systematic, age-related recall bias, we distinguish between two samples of different ages when we define the undernutrition indicator: an “*infant sample*” and a “*child sample*”. The infant sample is composed of individuals who potentially experienced hunger/famine in utero or at ages 0-4. They may have been too young to have formed a recollection of the hunger, so if family members or others have not informed them of any actual hunger spell in the earliest years of life, they may not recall it. The individuals in the so-called child sample potentially experienced hunger from ages 6-16. Hunger is defined as a binary indicator (or treatment variable) which equals one if a person has experienced a period of undernutrition during age interval $[age_{-1}, age_4)$ for the infant sample and during $[age_6, age_{16})$ for the child sample. A respondent is considered to have suffered undernutrition if she reported that she experienced an episode of severe hunger and if this period either started before age_{start} and ended thereafter, or if the period started within the specified interval. The intervals are defined with an eye on the small sample sizes. In sensitivity analyses we assess the robustness of the results with respect to these definitions.

Graph 1 provides evidence on the extent of recall among the infant and child samples. The graph displays the probability of reporting hunger only for those individuals who lived during a famine period. Individuals in the infant sample often do not report to have lived in a period of hunger, while individuals in the child sample report to have suffered from undernutrition at a stable rate of just under 20%. If an individual has lived during a famine, her probability to report hunger increases if the famine happened until age 6, and it remains stable for increasing ages at famine.

Famine instrument The famine periods are defined in Section 3,

1. Greece: May 1941 to end of June 1942
2. The Netherlands: November 1944 to end of April 1945, only the Western part¹¹

¹⁰Also, only one period of hunger can be reported, such that individuals are likely to choose the period where nutrition was shortest in supply.

¹¹To select the Dutch sample into those exposed to a famine or not, we use information on the accommodation a respondent lived in the year the famine started, i.e. 1944; if a respondent moved house in the very same year, we use the previous accommodation in order to rule out endogeneity due to internal migration. The SHARELIFE variable distinguishes five possible regions for the Netherlands: Noord-Nederland, Zuid-Nederland, Oost-Nederland, West-Nederland, and Midden-Nederland. The famine instrument is set to one only for West-Nederland.

3. Germany: June 1945 to end of June 1948

We construct a binary instrumental variable that takes the value one if a famine affected the individual in utero/at ages 0-4 or 6-16, respectively. This uses the information on individual's month of birth. Notice that changing the start or end months of the famine affects our results only very little, since only individuals born at the margin of a specific year and month will switch from being declared as potentially famine exposed to not being exposed and vice versa.

Figure 2 provides a graphical illustration of an example of our hunger (treatment) and instrumental variable definition for the child sample of German individuals. In this example, individual i1 did not report an episode of hunger, whereas i2 reported such a period at ages 3 to 5, that is, it has been affected by undernutrition prior to the relevant age window; the individual, however, did experience a famine in this window. For individual i3, both the treatment status and the famine instrumental variable are set to one since both the period of hunger as well as the famine occurred within the window when i3 was 6 to 16.

Figures 3 and 4 provide graphical assessments of the hunger periods we find in the data. Figure 3 shows the fraction of observations reporting to have had hunger in a given year among all individuals alive at that time. For Germany, the graph shows low propensities of hunger for the pre-war years, an increase during the war and a drastic peak toward the end of the war, which marks the beginning of the period we define as the famine; even though the fraction of those reporting hunger for this period declines somewhat after this peak, it stays high until the end of the famine. For the Netherlands, we can distinguish one single peak for the famine period while propensities for hunger amount to basically zero before and afterwards. The fraction of observations reporting hunger during the famine years is lower for the Netherlands, because only the Western part of the Netherlands was affected by the famine. For Greece we also find a spike around the famine period but the level remains relatively high for the 1920s. The impact of the famines becomes even more obvious when taking a look at the other SHARE countries: Here, the overall fraction of those reporting episodes of hunger is comparably small and increasing only somewhat during the second World War. The exclusion of Spain, which suffered from civil war in the 1930s, shows that it accounts for a relatively large fraction of those reporting hunger in the pre-war years. The graph implies that the experience of hunger in the famine countries was different from that in the other countries. In the famine countries, hunger was a severe, transitory shock, while in the other countries hunger can be ascribed to general suffering during the war.¹² This provides a rationale to exclude the other countries from the analysis.

¹²The three countries with famines were also exposed to turbulent episodes beyond the famine spells. For example, many German citizens were exposed to bombardments and stress during World War II, i.e. just before the German famine (see Akbulut-Yuksel, 2009; recall also the literature discussions in previous

Figure 4 shows the average duration of a hunger period for hunger periods starting in different years. The graph shows that hunger periods get shorter when coinciding with a famine. This supports our presumption that some individuals experience short but intense periods of severe undernutrition because of the famine. Table 1 shows that a fraction of approximately 17 percent of those having experienced a famine before age 16, report hunger for the same period. For non-famine periods, this is only 4.6 percent. Hence, famine is a powerful instrument for periods of undernutrition, although the overall propensity to report hunger remains rather low.

Outcomes We restrict our attention to those outcomes measures that are relevant in the light of the discussion in Sections 2 and 3 above and that can be reliably inferred from survey data: high blood pressure/hypertension, obesity, and adult height.

The diagnosis of high blood pressure/hypertension is a binary measure derived from self-reported information in the SHARE survey. Obesity is a binary transformation of the body-mass index (BMI) and set to one if the BMI is greater or equal to 30. Adult height is measured in centimeters and has been used in the literature as a marker of early life health, i.e. as a summary measure of the influence of the latter on health later in life. It has been seen as the best single observable indicator of an individual’s dietary history during childhood to the extent that it is of importance for health later in life (Elo and Preston, 1992). Information is taken as of the second wave of the survey.¹³ Table 2 lists the outcome variables and compares relative magnitudes between those who experienced hunger before age 16 and those who did not.

Covariates In the analysis, we control for country and gender and for a linear time trend.¹⁴ Besides, we include controls for the degree of urbanization of the place of birth or of the place where an individual lived at the time of our reference age.¹⁵ We consider this

sections), whereas Greek citizens were exposed to the Greek Civil War shortly after the Greek famine. The non-famine periods should therefore not be seen as tranquil eras of affluence.

¹³The refreshment sample entering the survey in the second wave has no information about this condition at the first wave. However, BMI and height were missing or not asked for a large group in the second survey wave. In these cases, we deviate from the initial convention and take information on the BMI as reported in the first wave. For height and BMI, this increases the sample sizes used in the analysis compared to hypertension.

¹⁴The SHARE data, especially the third wave, provides a great deal of background information. However, the information provided in the childhood module of SHARELIFE refers to a respondent’s living conditions at age ten. Since we are interested in incidents of hunger occurring before age ten, any such measure of socio-economic status may be endogenous.

¹⁵If an individual changed the accommodation in the year she turned six, information on the accommodation inhabited the year before was used in order to prevent bias from selective internal migration.

information as a proxy for socio-economic background. Table 2 provides basic summary statistics for the overall sample, for the subsample of respondents who reported an incidence of hunger sometime before age 16, and for the subgroup which never reported an incidence of hunger.

5 Empirical strategy

Our model framework is as follows,

$$Y_i = \psi(D_i, Z_i, X_i, \epsilon_i) \quad (1)$$

$$D_i = \phi(Z_i, X_i, \epsilon_i), \quad (2)$$

where $D_i \in \{0, 1\}$ denotes severe hunger during a respective childhood period of individual i . $Z_i \in \{0, 1\}$ is a binary instrumental variable, where $Z_i = 1$ if an individual has experienced a period of major exogenous food restriction in her area of residence and $Z_i = 0$, otherwise. Y is a measure of adult health. Binary outcome variables are indicator variables for chronic conditions, notably hypertension and obesity. The continuous outcome variable is adult height. The vector of covariates X comprises country and gender.

If effects of nutrition on health outcomes later in life are heterogeneous, we can identify the so-called local average treatment effect (LATE): the average effect among the "compliers", that is, those whose nutritional status is affected by the presence of a famine. The size of the complier group and the estimated effect may depend on the severity of the famine (Angrist, Imbens and Rubin, 1996). In this respect, an advantage of our approach is that the famines are regarded to be severe. Formally, the LATE is defined as:

$$\begin{aligned} LATE &= E[Y_{D=1} - Y_{D=0} | D_{Z=1} > D_{Z=0}] \\ &= \int (\psi(1, Z, X, \epsilon) - \psi(0, Z, X, \epsilon)) dF_{X,Z,\epsilon | D_{Z=1} > D_{Z=0}} \end{aligned} \quad (3)$$

Identification of the effect is based on the assumption that the famine causes are valid instruments: For example, we assume that the allied food embargo in Greece did not have a direct influence on the health status of individuals 50 years after the event, other than through the effect on individual access to nutrition. Furthermore, we assume that our sample contains a subpopulation of compliers but no defiers, and that the probability of suffering from hunger in a famine is the same for individuals who actually suffered from a famine as for those who did not. Moreover, stratifying on additional covariates requires their supports to be the same for famine and non-famine groups.

Parametric IV estimation for binary and continuous outcomes Two of our outcomes are of binary nature. We estimate nonlinear latent index bivariate probit models

to account for the nonlinearity of the conditional expectation function. Specifically, we parameterize (1) and (2) as:

$$\begin{aligned} Y^* &= \theta D + \beta' X + \epsilon_1, & Y = 1 &\Leftrightarrow \{Y^* \geq 0\} \\ D^* &= \alpha' X + \delta Z + \epsilon_2, & D = 1 &\Leftrightarrow \{D^* \geq 0\} \\ \text{with } \begin{pmatrix} \epsilon_1 \\ \epsilon_2 \end{pmatrix} &\sim \mathcal{N} \left(\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \begin{pmatrix} 1 & \rho \\ \rho & 1 \end{pmatrix} \right), \end{aligned}$$

where the error terms are distributed bivariate normal; notice that the error correlation ρ is an estimable parameter.

The LATE for the binary probit model can be computed as:

$$\begin{aligned} LATE = \\ \mathbb{E} \frac{[\Phi_b\{X'_i\beta + \theta; X'_i\alpha + \delta; \rho\} + \Phi_b\{X'_i\beta; -(X'_i\alpha + \delta); -\rho\}] - [\Phi_b\{X'_i\beta + \theta; X'_i\alpha; \rho\} + \Phi_b\{X'_i\beta; -(X'_i\alpha); -\rho\}]}{\Phi\{X'_i\alpha + \delta\} - \Phi\{X'_i\alpha\}}, \end{aligned}$$

where Φ_b denotes the bivariate normal distribution and Φ denotes the univariate normal distribution.

For adult height, our only continuous outcome variable, we use the ordinary two stage least squares (2SLS) estimator.

Nonparametric estimation of the LATE In addition, we estimate the LATE non-parametrically using a Wald estimator that allows for conditioning on covariates:¹⁶

$$LATE = \frac{\int \mathbb{E}[Y|X = x, Z = 1] - \mathbb{E}[Y|X = x, Z = 0] f(x) dx}{\int \mathbb{E}[D|X = x, Z = 1] - \mathbb{E}[D|X = x, Z = 0] f(x) dx}. \quad (4)$$

Equation (4) allows us to compute the difference in mean outcomes for the group of compliers without having to specify a functional form for the effect of hunger on health outcomes. With only discrete covariates, we compute the numerator and denominator cell-wise and then integrate over the distribution of X, where the integral is a sum over various combinations of X-values.

Moreover, we correct for recall bias. As explained above, imperfect recall in the infant sample may cause the incidence of severe hunger for that group to be misreported. We tackle this problem by replacing $\mathbb{E}[D|X = x, Z = 1] - \mathbb{E}[D|X = x, Z = 0]$ in the denominator of (4) by the estimate $[\mathbb{E}[D|X = x, Z = 1] - \mathbb{E}[D|X = x, Z = 0]]_{child}$ from the child sample but integrating over the marginal distribution of covariates in the infant sample.

¹⁶This estimator has first been proposed by Frölich (2007). He shows that equation (4) can be derived from the more intuitive (but not identified) expression $\int \frac{\mathbb{E}[Y|X=x, Z=1] - \mathbb{E}[Y|X=x, Z=0]}{\mathbb{E}[D|X=x, Z=1] - \mathbb{E}[D|X=x, Z=0]} dF_{X|D_{Z=1} > D_{Z=0}}$, using Bayes' theorem.

Note that this assumes that the true distributional effect of a famine on the probability of malnutrition is the same among infants as among older children. This assumption is debatable. On the one hand, children aged 6-16 may be better able to obtain food supplements during a famine. On the other hand, parents may direct resources towards babies, and breastfeeding may weaken the connection between famine exposure and nutritional shortage further. As discussed in Section 3, external charity aid was sometimes targeted at infants and sometimes at older children. If infants are relatively protected then the use of $[E[D|X = x, Z = 1] - E[D|X = x, Z = 0]]_{child}$ in the corrected two sample LATE estimates may yield a lower bound of the true effect.¹⁷

Linear two-sample IV As a robustness check, we estimate the effects of hunger around birth with information from two samples using *linear* models instead of the nonparametric Wald estimator. We use the two-sample two-stage least squares estimator (2S2SLS) proposed by Inoue and Solon (2010), which adjusts the original two sample IV (2SIV) estimator developed by Arellano and Meghir (1992) for use in small samples. The estimator we compute is:

$$\beta_{2S2SLS} = (\hat{D}'_{inf} \hat{D}_{inf})^{-1} \hat{D}'_{inf} Y_{inf}, \quad (5)$$

with $\hat{D}_{inf} = Z_{inf}(Z'_c Z_c)^{-1} Z'_c D_c$, where Z now includes the covariates X . The *inf* and *c* subscripts denote the infant and child samples respectively. Note that the results of this estimator are not fully equal to the LATE estimates obtained from estimating equation (4). The estimator yields a weighted LATE where the weights are given by the covariates.

Selective fertility and selective mortality Two potential sources of bias, that are typical for this literature, are selective fertility and selective mortality. First, selective fertility refers to the phenomenon of women becoming less fertile in response to the reduction in food supply. We cannot address this problem using our data, except by including region dummies, which are imperfect proxies for socioeconomic status and access to food prior to the outbreak of the famine. Selective fertility is a recurrent issue in the famine literature. The studies listed in Section 3 that examine long-run effects of the Dutch, Greek and German famines often argue that with these famines, selective fertility does not create a major source of bias. We expect any bias to make our results more conservative. The reason is that selective fertility shifts birth probabilities towards physically fitter mothers, such that during famines healthier individuals are more likely to be born. These individuals then also tend to have a lower probability of adult health problems.

¹⁷We bootstrap standard errors using 500 bootstrap iterations.

Second, we only observe individuals if they have lived until 2008, when the third wave of SHARE was administered. Hence, dynamic selection may lead to an overrepresentation of interview participants with favorable characteristics among those who experienced a famine. It is thus a second source of downward bias for our results, inducing the causal effect of undernutrition on adult outcomes to look weaker than it actually is. Hence, with respect to both selective fertility and selective mortality, our estimates can be considered conservative and represent a lower bound of the true effect.

6 Estimation results

6.1 Causal long-run effects of hunger in later childhood

In this section we estimate models in order to reveal whether hunger spells in later childhood causally affect later life health outcomes. First, we relate the outcome measures to the hunger spells as reported by the respondent. Since all outcome variables but adult height are binary, we estimate probit models in most cases. Second, we relate the outcome measures to the famine indicator while purposely dropping the actual undernutrition indicator. This corresponds to the commonly used approach of using contextual variation or, put differently, not estimating the structural effect of hunger but rather obtaining a reduced form estimate. We can thus show what the prediction would be in studies that have to remain agnostic about actual individual-specific undernutrition status, and see whether these studies yield accurate estimates of the causal effects. After this, we discuss the IV estimation results.

Table 3 shows the first set of results. The first columns show average marginal effects of hunger at age 6 to 16 on the health outcomes without controlling for possible endogeneity. The set of covariates is listed in the notes underneath the table; it is kept very limited because of the reasons detailed in the data section. For the sample merging men and women we find that hunger at these ages does not go along with worse health conditions on average. A glance at the results from a similar analysis on men and women reveals that women who report to have had hunger at this age are also 4 percent more likely to exhibit a BMI larger than or equal to 30.

The next columns concern the reduced-form estimations of outcomes as functions of famine exposure. For the general sample, we find that famine exposure at ages 6 to 16 is significantly related to a higher risk for obesity (mostly driven by women) and a higher risk of hypertension (driven by men). The effects are small in absolute value and never exceed 6 percent. The finding for obesity among women is both quantitatively and qualitatively similar to the one found when regressing these health conditions on the hunger indicator;

the effect on hypertension among men has not been observed in the previous analysis. This may already be an indication for the fact that hunger in childhood and health outcomes later are not causally related in the way simple regression results may suggest.

Next, we verify that the famine instrument is informative by presenting first-stage results. Table 4 shows the results for probit models and linear regression models where the dependent variable is the undernutrition indicator. Note that the coefficients of these models differ from those of the first stage in the nonlinear IV-models (the bivariate probit models) used in the main analysis, since in these models one typically maximizes a joint likelihood of observing the outcome at hand and the instrumental variable's realization. For the sake of clarity, we do not present estimates of all first stages. Linear probability models on the full sample of observations from all countries yield F-statistics beyond 30, i.e. values exceeding the typically recommended value of 10. We conclude that the instrumental variable is informative.

We use IV analyses to assess the causal effects of hunger at ages 6 to 16. We provide estimates of two-stage least squares estimations for all outcomes and augment this set of results by estimates of local average treatment effects and average treatment effects computed from bivariate probit models for binary health measures. The results suggest that most hunger effects are large in magnitude compared to the effects found before. The magnitude provides evidence for attenuation bias in the estimates derived from reduced-form regressions. The 2SLS estimates suggest that the risk for hypertension is raised by almost 50 percent and that obesity is 26 percent more likely to be prevalent when there has been an episode of hunger. The estimate on hypertension is not significant and much smaller in magnitude in the bivariate probit analyses. A gender-specific analysis suggests that the effect on this outcome is driven by men. Women seem to be responsible for the effects found on obesity. The risk for obesity in this subsample is raised by at least 20% when employing the conservative measure derived after bivariate probit models; this means an increase of 15 percentage points relative to the effect measured via the common approach using contextual famine exposure only. Some of the estimates are nonsensical and seem to have been affected by the various functional form assumptions that we have employed.

We test the robustness of the above results by replacing the binary famine instrument with the length of famine exposure within the defined age interval in months; we rescale this variable by dividing by twelve. We should note that we use this reformulation of the instrument to account for a further dimension of the famine exposure, namely severity. The validity of this proxy is, however, debatable. The length of exposure in our opinion is only an inferior way of including this aspect in our analysis, but it is probably the only one. Estimating the same models as before with the new instrument produces effects as depicted in Table 5. The results remain unchanged in almost every respect. We still find

a positive effect of hunger on female obesity throughout all models. In the subsample of men, hypertension appears to be significantly affected only when using linear models.

As we have documented in the descriptive statistics presented above, individuals who report experiences of hunger are on average older than those who do not. This could affect the estimated health effects. We have computed the same set of results presented above after dropping cohorts born after 1945 from the sample; doing so approximately balances the average age between both groups. On the other hand, one should keep in mind that the older cohorts remaining in the sample may to a greater extent be a dynamically selected sample. The results of this analysis suggest that the effects on hypertension among men and on obesity among women are robust and even enhanced (the LATEs and ATEs on hypertension computed after bivariate probit models are highly significant). The other IV estimates largely remain unchanged by this modification.

In a last step we use a fully nonparametric Wald estimator to assess what differences in causal effects occur when dropping any functional form or treatment effect homogeneity assumptions inherent in the models used above. We still control for country and gender. We cannot control for a linear time trend (the number of strata defined by all possible combinations of covariate values would be too large). Table 6 shows the results of this estimation approach and contrasts it with simple 2SLS estimates already presented before; the difference to the previously shown results is due to the fact that we cannot control for the urbanization of the accommodation at age 6 (some covariate cells would be empty).

New findings are an increased risk for hypertension among women. This effect is large and was not present in the 2SLS estimates. Effects on hypertension among men already found above are still present, while the effects are approximately 10 percentage points larger using the Wald estimator. Effects on female obesity remain statistically significant using the nonparametric specification.

We also notice that the Wald estimator gives large and significantly negative effects of hunger in this age interval on adult height, for both men and women. These effects are not present in the parametric results. They may be due to the parametric assumptions used in 2SLS, or it may be due to the omission of a time trend when using the Wald estimator. We may address the latter to some extent using the following ad-hoc approach. In a first step, height is regressed on a time trend. The residuals are then used as the dependent variable in regressions using the Wald estimator. As expected, doing so reduces the effects on adult height.

We have seen above that bivariate probit models produced relatively small point estimates. The Wald estimator, however, shows effects that resemble the comparably large 2SLS coefficients. This suggests that attenuation bias in reduced form coefficients of a contextual famine indicator is large; the effects of hunger on hypertension among men

(obesity among women) computed via 2SLS models reported in Table 6 are 25 (44) percentage points larger than comparable reduced form estimates from linear models.¹⁸ In relative terms, the causal effects exceed the reduced form estimates by a factor of about 3 and 8 in the case of the effects of hunger on hypertension among men and obesity among women, respectively.

In order to see what difference balancing the average age in the hunger and non-hunger groups makes, we again dropped cohorts born after 1945 and performed an analogous analysis. The effects show similar patterns as documented when using the whole sample.

6.2 Causal long-run effects of hunger around birth

The analysis so far has used retrospective information on hunger experiences in childhood. The self-reported nature of this information precludes an analogous approach when analyzing hunger effects in the very first years of life. We thus proceed as detailed above by estimating hunger propensities conditional on background factors for the first years of life by using information on the famines' impact on cohorts born earlier. We report findings for famine exposure in the first four years of life and pool this information with potential exposure in utero. Gestation is defined to begin nine months before birth. We require gestation and the famine period to overlap more than two months in order to preclude additional noise. For Dutch individuals we take the region of the accommodation at birth as a criterion for famine exposure and set it to one when this accommodation was located in the Western part of the Netherlands.

We start our discussion by presenting reduced form estimates of the effect of famines at this early age on outcomes. The results are displayed in Table 7 and show that famine exposure and accompanying potential suffering from hunger significantly raises hypertension among women by almost 8 percent. For obesity we find no effects while adult height for men is significantly reduced by almost 0.9 centimeters. If we take a crude estimate for the effect of famine exposure on the marginal propensity to report hunger to amount to 15 percent for both men and women, we expect the true underlying causal effect of hunger on female hypertension to be slightly above 50 percent (using the formula for the Wald estimator). For male adult height we find a negative effect of about 6 centimeters.

In what follows now, the reports for ages 6 to 16 are taken as the reference point. Hence, we predict hunger propensities for the early years by using conditional expectations for hunger at this later age. This analysis allows to directly compare the size of the commonly

¹⁸The reduced form estimates reported in Table 3 are not fully comparable to the effects in 2SLS models, since they are computed after probit models, i.e. under the assumption of non-linearity. The reduced form estimates implied by linear specifications are, as displayed here, slightly larger than those from non-linear models. They amount to 13 percent for hypertension among men and 6 percent for obesity among women.

computed reduced form effects we presented in Table 7 with quantitatively more reliable causal effects of actual hunger. Notice that now the famine-exposed are on average as old as the others.

Table 8 shows the results of both the corrected Wald Estimator and the 2S2SLS technique. Both approaches have limitations; as noted already, in the case of the Wald estimator we fail to control for a linear time trend since our sample is too small. For the 2S2SLS estimator we restrict both the first stage and the second stage of the outcome equation to be linear and make an implicit assumption of effect homogeneity when interpreting the effects as LATEs.

We find some evidence for a causal impact of hunger on hypertension; the effect is driven by women and amounts to approximately 50 percent which is over 40 percentage points larger than the reduced form estimate.¹⁹ Note that this result differs from the one when using mid-childhood as the age of interest. There hypertension was causally evoked by hunger among men only. For male adult height, we find that a famine-caused hunger experience early in life has a negative impact of 3 centimeters when using the Wald estimator and almost 5 centimeters when using the 2S2SLS approach; the former effect is, however, insignificant. With the latter method we can explicitly control for the secular trend in adult height such that differences in the age distribution among those with undernutrition and those without is less likely to drive the results. Hence, the reduced form estimate presented above underestimates the underlying causal effect of hunger on height in this subsample by up to 4 centimeters. For women we find again positive effects on adult height which are, however, not significant.

We analyze the relevance of a secular growth in adult height and neglecting it when using the Wald estimator further by the above introduced approach of detrending height. Using this method we still find that hunger in very early childhood significantly reduces male adult height by over 3.3 centimeters. For women we still find a positive but insignificant effect.

In addition to the sensitivity analyses discussed so far, we performed a range of estimations to assess the robustness of the results with respect to a number of assumptions and decisions concerning the operationalization of the key variables. This concerns, first of all, the precise starting and ending dates of each of the famines. For example, for Germany, one may argue that May 1945 should be included in the famine period. Secondly, we may vary the age intervals within which early-life conditions are assumed to exert their influence.

¹⁹The reduced form estimate we refer to is from a linear probability model in order to make a valid comparison to the linear 2S2SLS model. The suchlike computed reduced form estimate for the effect of famine exposure on hypertension among women is, however, approximately as large as the one implied by the non-linear models (+7.5 percent).

For example, we may exclude in utero from the age interval that merges in utero with age 0-4. or we may exclude age 4 from these intervals, or consider the age interval 7-15 instead of 6-16. Concerning the outcome measures, we may trim extreme values. It turns out that all results are insensitive with respect to such changes (results are available upon request). The “in utero” age interval for exposure (using the recording of hunger in the birth year) does not give a sufficient number of exposed individuals for a meaningful analysis. Finally, we perform separate estimations by country. It turns out that the estimates are imprecise, with large standard errors and point estimates that are insignificantly different from zero with sometimes counter-intuitive signs (results available upon request). This confirms that the current samples by country are too small for meaningful analyses. Notice also that results by country are more sensitive to effects of cohort-specific events.

7 Conclusions

This paper investigates the causal effect of undernutrition in infancy and childhood on adult health outcomes using instrumental variable estimation. We deal with the problem of selective recall and systematic underreporting of hunger periods in utero or infancy by using two-sample IV estimation. Specifically, we estimate the probability to report hunger when exposed to a famine around birth by examining the observed association between hunger and famine at teenage ages. To this end, we adjust the nonparametric Wald estimator for estimation on data from two different samples. We bootstrap the standard errors.

Our results indicate that the effects of undernutrition on adult health are heterogeneous by gender. For women aged 6-16 we find that undernutrition leads to substantially higher levels of obesity risk and blood pressure at high ages. Men also face a higher level of blood pressure at high ages. For both men and women aged 6-16, the nonparametric Wald estimator produces an average adult-height effect of undernutrition of about 6 cm. However, the estimator does not take account of the secular time trend in height, and it is likely that the estimates over-estimate the actual height reduction due to undernutrition.

If restricted in nutritional supply in the interval from in utero until age 4, women are estimated to be almost 42 percentage points more likely to suffer from hypertension in their mid-sixties. For men who suffered from low nutrition in infancy, we find a negative height effect of more than 3 cm. This effect remains after controlling for trends in height over time.

What distinguishes our results from the literature on contextual famine effects is that we are the first to use the occurrence of famines to obtain an estimate of the average *causal* effect of *nutritional shortages* during childhood on health later in life. We can thus compare the reduced-form famine effects that are usually reported in the literature with the causal

effects of undernutrition. Our estimated reduced-form famine effects are in accordance to those in the reduced-form studies of the long run effects of exposure to the famines that we consider (those studies were discussed in Section 3). However, in many cases, our estimated causal effects are at least a few times larger than our reduced-form famine effects. This emphasizes the importance of nutrition in early childhood - over and above the findings and statements in the famine literature.

The difference between contextual and causal effects is related to the rate of “compliance” to the famine. In our study, the fraction of “compliers” is about 15-18%. To the extent that reduced-form studies implicitly assume perfect compliance, this assumption seems untenable even for shocks as severe as the famines used in this study.

The analyses in this paper are restricted by some notable data limitations. First, the samples are small. Admittedly, the full SHARE data cover many European countries and include over 20,000 elderly respondents, but we only use the subsamples from the three countries with famines. Within these, the subsets of individuals who were exposed to famines during childhood are of even smaller size, because the famines had a duration of at most a few years. This implies, among other things, that we can only examine exposures within age intervals of say 4 years, since otherwise the numbers of “treated” and “controls” are insufficient for reliable inference. Another data limitation is that the individual spells of severe nutritional shortages are only measured in full calendar years. Inevitably, then, the assignment rules for the actual treatment status are open to debate. Fortunately, the results are not sensitive to moderate changes in these rules, and in general, the results are in agreement to the findings in the reduced-form literature where famine exposure is directly related to health outcomes later in life.

Nevertheless, the data limitations rule out the analysis of a number of interesting issues. This concerns, first of all, the connection between the timing of the spell of nutritional shortage and the age of the child. The famine literature distinguishes between different reduced-form effects at different stages of pregnancy, for different outcomes. More in general, the developmental origins literature finds that long-run reduced-form effects of conditions in utero and right after birth are larger than those at subsequent ages. The size of the effects may be non-monotonous during the pre-puberty and puberty ages. Our data do not enable us to go into such details. A second issue concerns the temporal and spatial variation in the strength of the famine instrument. Contextual information can be used to create an indicator of famine severity. For example, one may use official food rations per region and month. However, the meaning of a ration size depends on the country, since in some countries charities and the informal sector are more important than in others. As we have seen, the sample sizes are too small to allow for meaningful estimation by country, and on top of that, the temporal connection between a famine and the observed spell of

nutritional shortage is insufficiently tight to exploit fine temporal contextual variations. The present study may therefore motivate the construction or usage of data sets that are larger and/or more focused on specific spatial areas, where ideally, such data sets would have more elaborate retrospective information on childhood episodes of hunger.

Figure 1: Probability to Report Hunger Conditional on Famine Experience at Respective Age

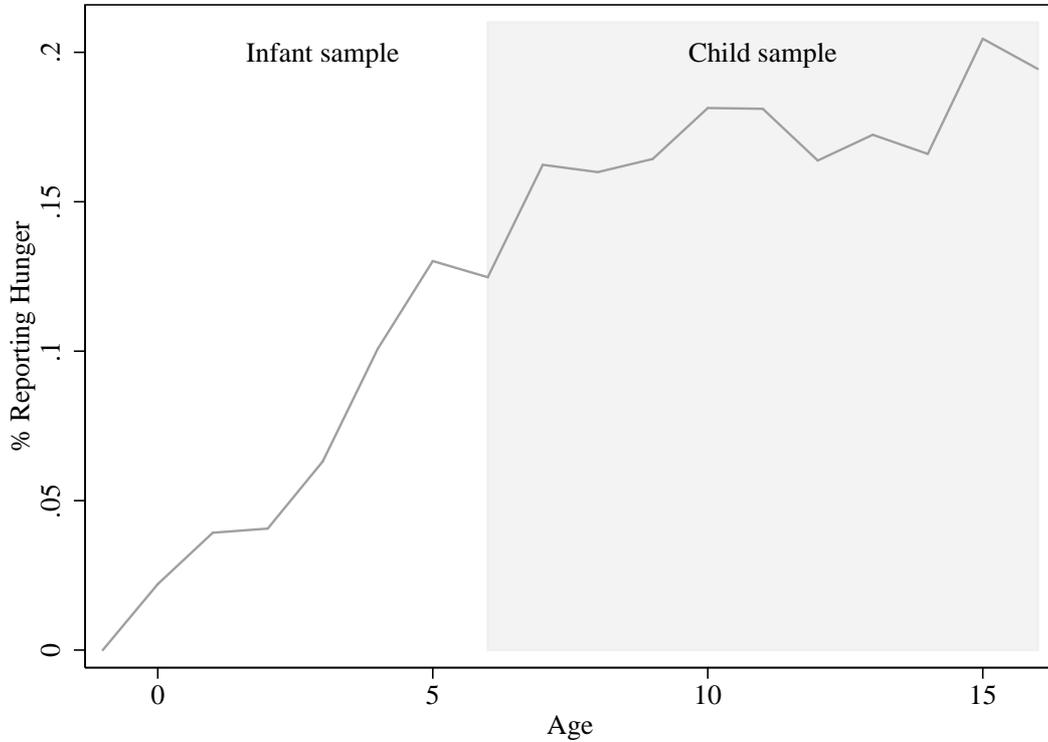


Table 1: Hunger at Age 0 – 16 During Famine Exposure (Cohorts 1920 – 1955)

	Greece		Germany		The Netherlands		Sum	
	hunger	non-hunger	hunger	non-hunger	hunger	non-hunger	hunger	non-hunger
Famine-exposed	123	881	124	664	58	180	305	1725
Not famine-exposed	46	1451	41	586	43	1553	130	3590

Table 2: Descriptive Statistics for Famine Countries (Cohorts 1920 to 1955)

Variable	All (N= 5750)		Non-hunger(N=5315)		Hunger before age 16 (N=435)		Hunger before age 4 (N=109)	
	mean	(s.d.)	mean	(s.d.)	mean	(s.d.)	mean	(s.d.)
Background information								
Year of birth	1942	(8.704)	1943	(8.700)	1936	(5.750)	1939	(6.228)
Female	0.535	(0.499)	0.538	(0.499)	0.490	(0.500)	0.505	(0.502)
Episode of hunger in utero and 0-4 (1 = yes)	0.076	(0.264)	0.000	(0.000)	1.000	(0.000)	1.000	(0.000)
Famine 0-16 (1 = yes)	0.353	(0.478)	0.325	(0.468)	0.701	(0.458)	0.670	(0.472)
Born in rural area (1 = yes)	0.414	(0.493)	0.421	(0.494)	0.332	(0.471)	0.315	(0.467)
Outcome measures								
Hypertension (yes/no)	0.335	(0.472)	0.330	(0.470)	0.396	(0.490)	0.379	(0.488)
Obesity (yes/no; 1 if BMI ≥ 30)	0.174	(0.380)	0.173	(0.378)	0.192	(0.394)	0.167	(0.374)
Male adult height in cm	175.547	(7.248)	175.669	(7.235)	174.203	(7.269)	173.426	(7.895)
Female adult height in cm	164.156	(6.229)	164.208	(6.193)	163.464	(6.674)	164.527	(6.265)

Figure 2: Example Treatment Definition

Example of critical, famine and hunger periods for 3 individuals
German Famine (Child Sample Age Period 6–16)

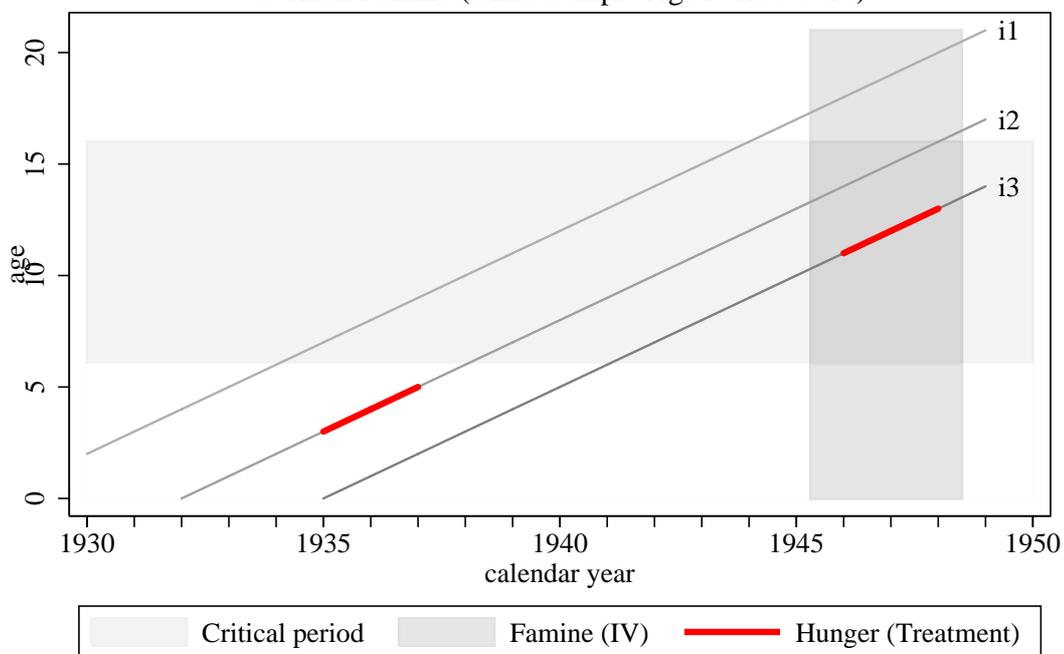


Figure 3: Probability for Episode of Hunger by Calendar Year (Given Observations of Sample Already Alive)

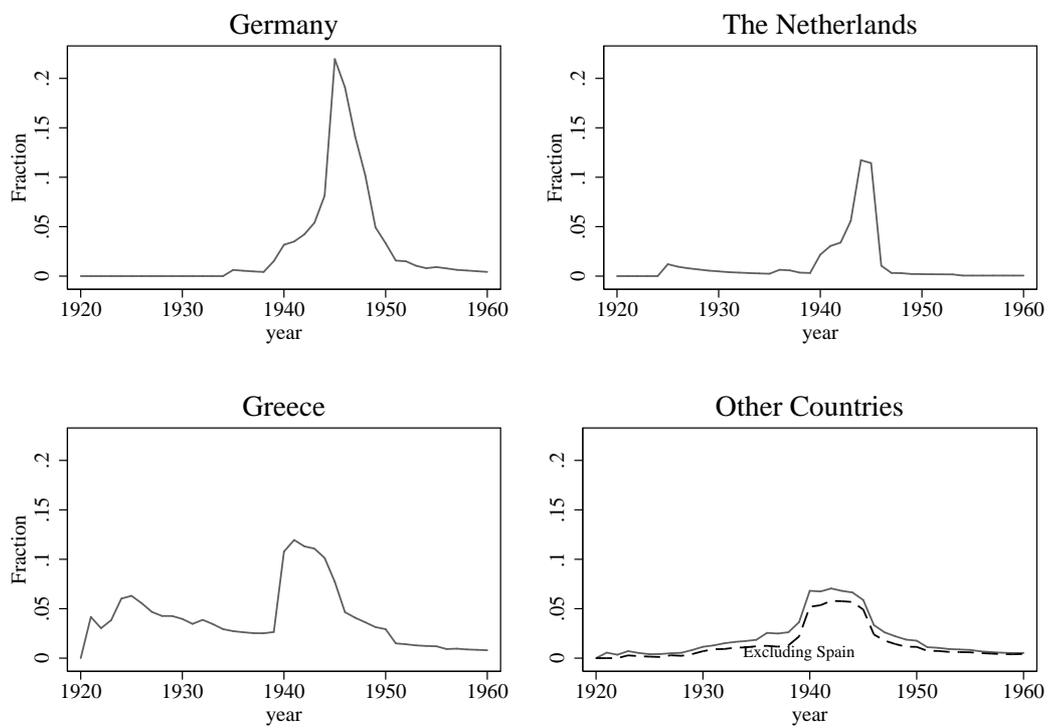


Figure 4: Average Length of Hunger Periods By Calendar Year

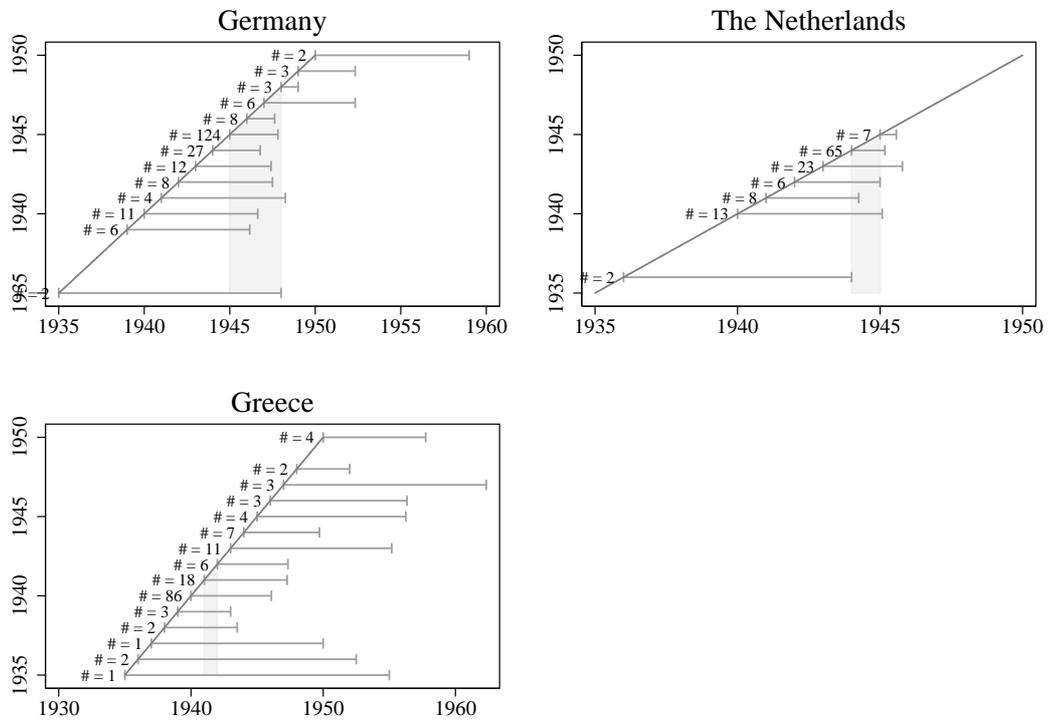


Table 3: Effects of Hunger or Famine at Ages 6 – 16 on Various Outcomes

Outcome	OLS/Probit Models			Instrumental Variables Models		
	Hunger at age 6 – 16 Marg.Eff. (z/t-stat.)	Famine at age 6 – 16 Marg.Eff. (z/t-stat.)	2SLS Marg.Eff. (z/t-stat.)	LATE from Biprobit Marg.Eff. (z/t-stat.)	ATE from Biprobit Marg.Eff. (z/t-stat.)	ATE from Biprobit Marg.Eff. (z/t-stat.)
	Whole Sample					
Hypertension (yes/no)	-0.027 (-1.136)	0.051 (2.933)	0.486 (2.718)	0.031 (0.322)	0.033 (0.356)	(0.356)
Obesity (yes/no; 1 if BMI ≥ 30)	0.024 (1.376)	0.035 (2.275)	0.264 (2.249)	0.114 (2.101)	0.149 (1.555)	(1.555)
Adult Height in cm	-0.024 (-0.064)	0.304 (1.305)	2.328 (1.345)	– (–)	– (–)	(–)
	Males Only					
Hypertension (yes/no)	-0.020 (-0.498)	0.060 (2.555)	0.409 (2.566)	0.129 (1.275)	0.152 (1.215)	(1.215)
Obesity (yes/no; 1 if BMI ≥ 30)	0.006 (0.261)	0.025 (1.205)	0.129 (1.118)	0.072 (1.094)	0.091 (0.778)	(0.778)
Adult Height in cm	-0.297 (-0.530)	-0.011 (-0.033)	-0.065 (-0.033)	– (–)	– (–)	(–)
	Females Only					
Hypertension (yes/no)	-0.033 (-0.926)	0.041 (1.632)	0.612 (1.443)	-0.127 (-0.682)	-0.114 (-0.791)	(-0.791)
Obesity (yes/no; 1 if BMI ≥ 30)	0.046 (1.860)	0.043 (2.112)	0.487 (2.241)	0.184 (2.543)	0.267 (1.734)	(1.734)
Adult Height in cm	0.326 (0.658)	0.563 (1.887)	6.145 (1.816)	– (–)	– (–)	(–)

Note: Sample sizes “hunger” ≥ 348 , non-hunger ≥ 4790 . For non-linear models, the table reports average marginal effects and ATEs and LATEs calculated by the formula as given in the text. Significance computed using standard errors clustered by country-year cells. Significance of ATEs and LATEs after Bivariate Probit has been bootstrapped (200 repetitions, using cluster sampling). Models include covariates for gender, a dummy for whether the accommodation at age 6 has been in rural area, country fixed effects, and year of birth.

Table 4: First-Stage Coefficients for Probability of Experiencing Hunger aged 6–16

Variable	Whole Sample		Men Only		Women Only	
	Probit	Linear Prob.	Probit	Linear Prob.	Probit	Linear Prob.
German	0.275*** (0.084)	0.030** (0.012)	0.196* (0.111)	0.020 (0.016)	0.321*** (0.102)	0.035** (0.014)
Dutch	-0.043 (0.117)	-0.001 (0.010)	0.018 (0.143)	0.006 (0.013)	-0.096 (0.121)	-0.007 (0.009)
Rural Accommodation Age 6	-0.202*** (0.066)	-0.025*** (0.008)	-0.176** (0.088)	-0.023** (0.011)	-0.226*** (0.085)	-0.027*** (0.009)
Famine	0.628*** (0.106)	0.120*** (0.020)	0.830*** (0.117)	0.167*** (0.024)	0.448*** (0.127)	0.079*** (0.023)
Year of Birth	-0.038*** (0.005)	-0.003*** (0.001)	-0.036*** (0.006)	-0.003*** (0.001)	-0.040*** (0.006)	-0.004*** (0.001)
Female	-0.088* (0.049)	-0.011* (0.006)				
Constant	72.978*** (9.191)	6.751*** (1.155)	68.712*** (11.936)	6.167*** (1.306)	76.514*** (10.838)	7.407*** (1.430)
R ²		0.084		0.108		0.065
F-stat.		35.42324		25.06579		32.79229
p-val.	8.90e-54	2.59e-23	4.61e-35	1.34e-16	1.12e-39	4.40e-20
N	5128	5128	2375	2375	2753	2753

Note: All estimations are conducted on minimum sample (actual samples may be larger for several outcomes due to less missing values). Standard errors clustered within country-birthyear cells in parentheses. *, **, *** indicates significance at the 10%, 5%, and 1% level, respectively.

Table 5: Effects of Hunger at age 6 – 16 on Various Outcomes, Instrument is Famine Duration in Age Interval

Outcome	OLS/Probit Model		2SLS		IV Models		ATE from Biprobit	
	Famine at age 6 – 16	Marg.Eff. (z/t-stat.)	Marg.Eff. (z/t-stat.)	Marg.Eff. (z/t-stat.)	LATE from Biprobit	Marg.Eff. (z/t-stat.)	Marg.Eff. (z/t-stat.)	Marg.Eff. (z/t-stat.)
	Whole Sample							
Hypertension (yes/no)	0.022 (2.186)	(2.186)	0.521 (2.056)	(2.056)	-0.058 (-0.428)	(-0.428)	-0.055 (-0.482)	(-0.482)
Obesity (yes/no; 1 if BMI ≥ 30)	0.010 (1.358)	(1.358)	0.178 (1.382)	(1.382)	0.048 (0.630)	(0.630)	0.052 (0.551)	(0.551)
Adult Height in cm	0.136 (0.947)	(0.947)	2.443 (1.004)	(1.004)	–	(–)	–	(–)
	Males Only							
Hypertension (yes/no)	0.035 (2.625)	(2.625)	0.619 (2.773)	(2.773)	0.129 (1.277)	(1.277)	0.152 (1.221)	(1.221)
Obesity (yes/no; 1 if BMI ≥ 30)	0.008 (0.692)	(0.692)	0.095 (0.636)	(0.636)	0.072 (1.004)	(1.004)	0.091 (0.710)	(0.710)
Adult Height in cm	-0.001 (-0.005)	(-0.005)	-0.013 (-0.005)	(-0.005)	–	(–)	–	(–)
	Females Only							
Hypertension (yes/no)	0.008 (0.533)	(0.533)	0.300 (0.579)	(0.579)	-0.127 (-0.714)	(-0.714)	-0.114 (-0.890)	(-0.890)
Obesity (yes/no; 1 if BMI ≥ 30)	0.011 (1.227)	(1.227)	0.328 (1.336)	(1.336)	0.184 (2.452)	(2.452)	0.267 (1.832)	(1.832)
Adult Height in cm	0.247 (1.771)	(1.771)	6.818 (1.638)	(1.638)	–	(–)	–	(–)

Note: Famine duration is measured in months and rescaled by dividing with the factor 12. Sample sizes “hunger” ≥ 348 ; “non-hunger” ≥ 4790 . Table reports average marginal effects and ATEs and LATEs calculated by the formula as given in the text (all for switch of famine duration from 0 to 1 year). Significance computed using standard errors clustered by country-birthyear cells. Significance of ATEs and LATEs after Bivariate Probit has been bootstrapped (200 repetitions). Models include covariates for gender, a dummy for whether the accommodation at age 6 has been in rural area, country fixed effects, and year of birth.

Table 6: Local Average Treatment Effects of Hunger at Ages 6 – 16 on Various Outcomes

Outcome	Whole Sample			
	Wald Estimator		2SLS	
	Effect	(t-stat.)	Effect	(t-stat.)
Hypertension (yes/no)	0.915	(6.246)	0.489	(2.657)
Obesity (yes/no; 1 if BMI ≥ 30)	0.140	(1.849)	0.269	(2.113)
Adult Height in cm	-6.188	(-3.523)	2.289	(1.234)
Height - Trend	1.123	(0.821)	–	(–)
Males Only				
Hypertension (yes/no)	0.572	(4.474)	0.412	(2.476)
Obesity (yes/no; 1 if BMI ≥ 30)	0.005	(0.065)	0.130	(1.109)
Adult Height in cm	-6.917	(-3.556)	-0.038	(-0.018)
Height - Trend	0.726	(0.447)	–	(–)
Females Only				
Hypertension (yes/no)	1.406	(4.048)	0.615	(0.208)
Obesity (yes/no; 1 if BMI ≥ 30)	0.319	(2.503)	0.501	(2.033)
Adult Height in cm	-5.233	(-2.237)	6.023	(1.501)
Height - Trend	3.987	(1.975)	–	(–)

Note: For sample sizes and covariates, see Table 3. T-statistics computed via bootstrap with 500 repetitions (cluster-sampling from year of birth/country clusters).

Table 7: (Average) Marginal Effects of Being Potentially Exposed to a Famine in Utero or at Ages 0 to 4

Outcome	Whole Sample		Males Only		Females Only	
	Marg.Eff.	(z/t-stat.)	Marg.Eff.	(z/t-stat.)	Marg.Eff.	(z/t-stat.)
Hypertension (yes/no)	0.046	(2.608)	0.011	(0.462)	0.078	(3.072)
Obesity (yes/no; 1 if BMI ≥ 30)	-0.001	(-0.070)	-0.006	(-0.301)	0.003	(0.214)
Adult Height in cm	-0.387	(-1.842)	-0.856	(-2.499)	-0.195	(-0.483)

Note: Sample size “hunger” ≥ 786 ; “non-hunger” ≥ 4352 . For binary outcome variables, table reports average marginal effects after probit models. Significance computed using standard errors clustered by country-year cells. Regressions include covariates for gender, country fixed effects, year of birth, and urbanization of accommodation at birth.

Table 8: Local Average Treatment Effects of Hunger at Age 0 – 4 and in Utero, on Various Outcomes

Outcome	Whole Sample			
	Wald Estimator		2S2SLS	
	Effect	(t-stat.)	Effect	(t-stat.)
Hypertension (yes/no)	0.223	(1.559)	0.281	(2.277)
Obesity (yes/no; 1 if BMI ≥ 30)	-0.036	(-0.520)	0.003	(0.036)
Adult Height in cm	-0.838	(-0.659)	-2.502	(-1.873)
Height - Trend	-0.792	(-0.865)	–	(–)
	Males Only			
Hypertension (yes/no)	0.060	(0.408)	0.034	(0.232)
Obesity (yes/no; 1 if BMI ≥ 30)	-0.006	(-0.052)	-0.023	(-0.202)
Adult Height in cm	-3.209	(-1.592)	-4.808	(-2.392)
Height - Trend	-3.354	(-1.918)	-4.808	(-2.392)
	Females Only			
Hypertension (yes/no)	0.417	(1.850)	0.569	(2.438)
Obesity (yes/no; 1 if BMI ≥ 30)	-0.068	(-0.775)	0.028	(0.237)
Adult Height in cm	1.692	(0.848)	0.084	(0.034)
Height - Trend	1.906	(1.015)	0.084	(0.034)

Note: Sample size “hunger” ≥ 5138 ; thereof famine-exposed at age 0-4 and in utero: ≥ 786 ; thereof “hunger” / famine-exposed at age 6-16 $\geq 348 / 1169$. Regressions control for gender, country fixed effects; year of birth is included for 2S2SLS. T-statistics computed using a bootstrap (500 repetitions).

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