Empirical Essays on the Long-Term Consequences of Early Infant Conditions for Health, Productivity, and Family Formation

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Eidesstattliche Erklärung

Hiermit erkläre ich, die vorliegende Dissertation selbständig angefertigt und mich keiner anderen als der in ihr angegebenen Hilfsmittel bedient zu haben. Insbesondere sind sämtliche Zitate aus anderen Quellen als solche gekennzeichnet und mit Quellenangaben versehen.

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Katharina Walliczek

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1 Introduction

In this dissertation, I analyse how experiences very early in life affect the formation of individual well-being – health, social standing, and productivity. There is plenty of evidence that living conditions during pregnancy and infancy shape long-term well-being, partly for multiple generations.
To conduct this analysis, external changes are necessary in the living conditions for children. These changes are found in a unique setting in modern history. This dissertation sheds light on a very dark chapter in history and describes how National-Socialism and the Second World War brought enormous negative consequences in the day-to-day-living for multiple generations. It details the economic and physiologic living conditions during the war and the following famine in German territory. A data set is compiled that measures these changes in early life experiences, making use of high regional and temporal variation. This data set contains economic, medical, population, health, and nutritional information at the level of German communities. To bring this data set to the individual level, it is merged with individual information from SOEP questionnaires, containing regional identifiers and reported relatively late in life. To exploit this data, various types of difference-in-differences estimations are set up and interpreted. They enable me to control for the social status of origin, and regional and temporal fixed effects, as all of these parameters affect health later in life additional to the treatment effect. With the estimations at hand I can draw conclusions on the association between early life hunger and stress and measures of well-being later in life, like general health, specific old-age diseases, satisfaction with life, and family life.
Thus, the dissertation spans several decades of German history, bringing together health and socio-economic information from very early in life with health and socio-economic conditions late in life. The findings are highly relevant for our economy, in spite of the excess in food supply and the Second World War being far away. Stress and malnutrition are omnipresent also in our modern lifestyle, which may threaten the well-being of our children and future generations and may increase inequality in their 'initial conditions'.
All these analyses are performed with the eyes of an economist, concerned with the individual's starting conditions and successes in life. Other scientific disciplines like sociology, medicine, epidemiology, history, or psychology are sometimes reflected in this dissertation to reach a better understanding of the

observed living conditions. Yet, ultimately, this dissertation is limited to the realms of economic effects of economic conditions.

In the second Chapter – a joint work with Mevlude Akbulut-Yuksel and Gerard J. van den Berg – we study long-run health effects of nutritional conditions early in life by exploiting spatial and temporal variation in those conditions. For this purpose, we consider individuals from a large set of municipalities in Germany, born in the years 1935 – 1950. Time series of local infant mortality rates are used to distinguish between affluent municipalities and municipalities with adverse early-life conditions, and these series are used to assess the local impact of the post-World War II famine in Germany. This methodology is novel and useful if the impact of a famine is strongly heterogeneous across regions and if local variation in the impact of the famine is not observed in the data. Individuals from municipalities where the famine was severe display an average realized adult height loss of almost 3 cm. This predicts adverse health outcomes late in their life.

In the third Chapter – a joint work with Gerard J. van den Berg, Anna Hammerschmid and Johannes C. Schoch – the focus lies on the underlying mechanisms, through which the early-life conditions influence well-being later in life. Long-run effects of nutritional shortages early in life are often studied using variation in contextual nutritional conditions (e.g. due to a famine). Exposure to such adverse nutritional conditions is likely to cause stress among the affected households. The combination of a lack of nutrition and an increased stress level may have different long-run effects than the occurrence of one of these factors in isolation from the other. Results in famine studies may therefore be driven by stress exposure. We advance on this by considering various types of adverse contextual conditions early in life and by exploiting the variation in temporal and regional exposure to these conditions, among birth cohorts in Germany born in 1930 – 1950. This includes exposure to bombardments on the civilian population and exposure to famine. The latter are quantified using data we collected from historical sources on daily bombardments per city and local food rations. As individual outcomes we consider measures of adult height, life satisfaction and the occurrence of high blood pressure at old ages. Moreover, we use the actual retirement pension level as an indicator of economic productivity throughout the adult working life, following the principle that it is related to the flow value of an expected present value of lifetime income. This allows us to capture long-run effects on economic outcomes in an encompassing fashion. We find that the long-run effects depend strongly on the relative importance of the different types of adversities early in life.

Finally, in the fourth Chapter, additional aspects of success in life are studied, namely social factors. Early life conditions are widely accepted as determinants of well-being and success later in life. Beside health issues and produc-

tivity, effects on family formation would be highly relevant for the society as a whole. Fertility surely is a key factor for future generations' prosperity. Additionally, the marital biography gives insights into a society's marriage market conditions and shapes health and well-being at old ages. I analyse German birth cohorts from 1930 to 1950 in their marriage behaviour and fertility. These cohorts suffered from the Second World War and the following famine during childhood. Both conditions show regional and temporal patterns which are used to identify individual exposure. I apply various measures of male and female fertility and marital biography. I only find limited effects from early life conditions on fertility. Marital biography instead seems to be substantially altered by shocks early in life. However, the direction of the estimated effects is surprising, since affected individuals seem to do particularly well.

2 Exploiting Spatial and Temporal Variation in Nutrition at Birth, Using Data on Infant Mortality and Famines: a Study of Long-run Health Effects*

2.1 Introduction

The long-term effects of health conditions during early childhood are subject of a growing literature. In accordance with Barker's fetal programming hypothesis (1990), malnutrition and poor living conditions *in utero* and during early childhood – caused by famine, disease environment, natural disasters and economic crisis – are found to have long-lasting adverse effects on individual adult health status, height, self-reported measures of mental health, schizophrenia and life expectancy. In this study, we consider the post-WWII famine as a negative shock to infants' and young children's living conditions and quantify the long-term effect of adverse health conditions in the region of birth on health much later in life. WWII was characterized by massive civilian-targeted warfare, which was followed by a severe famine in Germany. Exposure to such a severe famine during their critical period of development may have catastrophic effects especially on the well-being of children and on their long-term health and productivity. Therefore, our study focuses on infants and young children to assess the long-term health effects of famine.

The destruction of infrastructure during the war and the consequential hunger period are presumably exogenous to individual behaviour. This allows us

*This chapter is joint work with Gerard J. van den Berg and Mevlude Akbulut-Yuksel. For useful comments we thank the participants of the Junior Research Dialogue in Applied Econometrics, University of Mannheim, 2012; the CDSE seminar, University of Mannheim, 2013; the ESPE Conference, Aarhus, 2013; the ESEM Conference, Gothenburg, 2013; the EALE Conference, Turin, 2013; the Tepp Conference, Le Mans, 2013; the Masterclass by Angus Deaton, Amsterdam, 2013; and the HE Seminar, Rotterdam, 2014. The data used in this publication were made available to us by the German Socio-Economic Panel Study (SOEP) at the German Institute for Economic Research (DIW), Berlin.

to credibly isolate their effects on children's long-term health. In our paper, we apply a difference-in-differences estimation strategy by exploring the district-by-cohort variation in the exposure to the postwar famine. We conduct our analysis using a unique data set of German municipalities before and after WWII, and individual-level health variables from the Socio-Economic Panel (SOEP). To the best of our knowledge, this is the first paper that focusses on the long-term health effects of postwar famine specifically in Germany. Moreover, we exploit famine-driven regional variation in infant mortality at the very low regional level of districts, which allows us to better assess individual exposure to postwar famine.
The outcome of interest in our paper is adult height, which is widely used as an aggregate proxy for adult health and is a good predictor of adult mortality (Elo and Preston (1992)). Silventoinen (2003) documents that environmental conditions determine about 20% of adult height variation in modern Western societies. As also confirmed in Deaton (2007), the most important environmental factors are nutrition and disease load. The postwar famine significantly altered the nutrition supply and disease load in German districts, thus providing a plausibly exogenous quasi-experiment to estimate the long-term health effects of childhood environment.
Theoretically, a limited access to nutrition and health care during a critical period of development could lead to culling, immunity and scarring effects among affected children. The culling and immunity effects would suggest that under challenging conditions the observed average health quality is increased. A scarring effect would lead to a deterioration of health capital among surviving children, shifting their health endowments down in adulthood. The overall effect of childhood exposure to these adverse events on the long-term health is determined by the relative importance of the mechanisms. In our paper, we empirically test which effect dominates in the long run.

Our analyses document that exposure to famine during prenatal and early postnatal period has detrimental effects on adult health outcomes. We find that male individuals from municipalities with severe famine display an average realized adult height loss of about 2.5 cm. These results are robust to a battery of robustness checks including internal migration, the postwar reception of refugees, selective fertility and mortality, linear and quadratic time trend and a different categorization of the affected and the control cohorts. Taken together, our findings suggest that the negative scarring effect of the post-WWII famine dominates a positive effect coming from selection. Given the well-established relationship between height and socio-economic outcomes (see Case and Paxson (2008)), our findings suggest that early-life health and nutritional quality, measured by adult height, have important implications for well-being and ability in later life. The strong intergenerational correlation in height over generations also suggests that not only the directly

affected cohorts themselves suffered from long-term effects of these catastrophic events but their children are also at risk. Our results therefore provide the lower bound estimates for the harmful enduring effects of childhood exposure to hunger.

This paper makes several contributions. First, we add to the broader literature exploring the causal association between early childhood environment and health outcomes later in life.[1] Van den Berg et al. (2006) examine the effects of socio-economic conditions in early childhood, proxied by annual GDP, on individual mortality in the Netherlands. They find that being born in a recession reduces the life span by a few years. Van den Berg et al. (2011) further analyse the causal association between business cycles and health by focusing on the cardiovascular mortality later in life. They find evidence for the increased incidence of cardiovascular mortality among individuals born during a recession. The present study adds new evidence to this literature by examining the long-term health effects of famine-driven infant mortality in postwar Germany.

Our study also contributes to the studies exploring famine as health shocks to childhood environment. The 'Dutch Hunger Winter' in 1944/1945, caused by the German embargo, has been often studied as an example for adverse living conditions, see e.g. Banning (1946), Lumey et al. (2011), and Painter et al. (2005), where cardiovascular diseases, lower self-rated health, obesity and diabetes are among the consequences of malnutrition. Susser and Stein (1994) also find a decrease in adult stature among children who were exposed to the 'Dutch Hunger Winter' shortly after birth. Chen and Zhou (2007) provide similar evidence from China's Great Famine, showing that a greater exposure to famine during infancy significantly impairs height later in life.

This paper is also related to recent studies estimating the long-term micro-level effects of WWII in Europe as well as in Germany. Kesternich et al. (2014) and Van den Berg et al. (2016) examine the long-term effects of WWII on individual health and economic outcomes using data from SHARELIFE. Kesternich et al. (2014) cover 13 European countries in their analysis and find an elevated incidence of diabetes, heart diseases, depressions and worse self-reported health among individuals affected from war. Van den Berg, Pinger, and Schoch (2016) estimate the local average treatment effect of having experienced hunger during WWII on height, obesity and hypertension in Germany, Greece and the Netherlands. They find that undernourishment increased the likelihood of high blood pressure, the obesity risk and led to a shorter height in adulthood. A critical point in Kesternich et al. (2014) and Van den Berg et al. (2016) is the lack of regional variation in exposure to war or hunger within a country.

Jürges (2013) and Akbulut-Yuksel (2014) analyse the long-term effects of WWII

[1] For extensive reviews of the literature, see Lumey et al. (2011), Almond and Currie (2011b), Lawlor (2008), and Pollitt et al. (2005).

on wartime children's long-term outcomes. Jürges (2013) employs a regression discontinuity design and finds that cohorts conceived around the end of the war are more likely to only have a basic education and to hold a blue-collar occupation as adults compared to cohorts born shortly after or before due to malnutrition experienced in Germany by the end of WWII. However, to our knowledge, it is ambiguous whether nutrition shortage around the end of the war has indeed been more severe than in 1946 and 1947. Historical accounts indeed suggest that the German population was quite well supplied with food and still had some reserves until the occupational powers had reorganized the food ration system. When rations were sharply reduced in some occupation zones in 1946, however, these reserves were probably depleted. In addition, Jürges (2013) primarily exploits cohort variation in exposure to WWII; however there was a significant variation in food supply within Germany, even within districts.
Using region-by-cohort variation in rubble per capita in 1946, Akbulut-Yuksel (2014) finds that wartime children in highly destroyed regions attain fewer years of schooling, have lower future labour market earnings and are shorter as adults due to the physical destruction. The level of geographical aggregation used to assess WWII exposure is *Raumordnungsregionen* (*ROR*) in Akbulut-Yuksel (2014). Having 96 *ROR* in Germany, the study loses variation on the lower levels.
The novelty of the present study is the low-scale regional level of health conditions in infancy, which can much better capture variation in the exposure to food shortages than higher-level spatial measures such as country or regions within a country used in the previous studies. Data on infant mortality rates in the municipalities reinforce this statement. For example, in the German district of Ostholstein, the level of infant mortality in the municipality of Eutin was extremely low in 1947 with 1.9%, whereas in the municipality of Ahrensboek, it was at 6%, although both municipalities belonged to the same district. In our study, we infer the local food availability with infant mortality rates in the individual municipalities. In addition, we restrict our analysis to health outcomes later in life, which assumably are more directly affected by early life shocks and less biased through individual decisions. Finally, by using SOEP data we have access to information on the biography of the family, which allows us to account for socio-economic status before the shock appeared.

The remainder of the chapter is organized as follows: Section 2.2 provides a short overview on the children's situation during and after WWII. Section 2.3 lays out the conceptual framework. Section 2.4 describes the data used in the analysis. Section 2.5 discusses the empirical strategy. Sections 2.6 and 2.7 present the main estimation results and sensitivity analysis. Section 2.8 concludes.

2.2 Children in Post-WWII Germany

Wars and armed conflicts particularly inflict substantial direct and indirect costs to the well-being of infants and children, see Kesternich et al. (2014). The destruction of housing, production sites and infrastructure significantly impairs children's access to economic resources during the course of warfare and the postwar period. The reallocation of resources in favour of the military can further exacerbate these adverse effects on children, leading to a malnourishment of the children in the conflict regions. Moreover, warfare leaves many children without a father, which often worsens the social and economic situation of the households these children grow up in.

During WWII, the situation in German cities started to worsen with the increased Allied aerial attacks in June 1940. Millions of people lost their lives, while programs like *Kinderlandverschickung* and *Mutter-und-Kind-Verschickung* tried to protect children and their mothers by sending them to less attacked rural regions. By the end of WWII, cities, industry and the transportation system were mostly destroyed. With the loss of rural areas to Poland and the Soviets, a major part of agricultural areas was separated from Germany. At the same time the German mainland received millions of refugees from these regions. Moreover, different Allied forces separately administered the four occupational zones which rendered engaging in trade between the zones difficult.[2]

On the onset of the WWII, the government started to control the food, fuel and textile markets. Allied Forces continued this practice after the war. The (West) German households required coupons (*Lebensmittelmarken*, *Bezugsscheine*) to buy groceries until 1950. While there was a rationing in food consumption already during WWII, the food shortage acutely peaked in the postwar era. The destruction of transportation infrastructure, the loss of the agricultural land, labour shortages and lack of fertilizers in the postwar era led to a famine, which was particularly severe until 1948. However, the extent of food shortage and exposure to famine significantly varied across occupation zones depending on the policies adapted by different Allied forces and the supplies available in each Allied country after WWII. Table 2.1 illustrates the average daily calorie intake per person by occupation zone. Access to food and nutrition also varied within the zones right after the war e.g. due to destruction of roads and other means of transportation. For instance, in the British zone, the North-Rhine province received much lower food rations than the other British provinces. Even after introducing homogeneous rations within occupation zones, the administration in the 'surplus' areas re-

[2]For overview studies, see Farquharson (1985), Trittel (1990), Klatt (1950) and Reichardt and Zierenberg (2010).

fused to deliver the necessary food quantities to the areas in need and instead increased the rations for the own residents, see Schmitz (1956) p. 8.

Table 2.1: Average food rations in 1946 per zone

Occupational zone	Daily kcal
British zone	1050
US zone	1275
French zone	880
Russian zone	817 to 1167

Source: Schmitz (1956), p. 475.

Given the importance of nutrition in conception and during the early childhood years, infants and young children disproportionally bore the adverse effects of the postwar famine. School interviews in Essen 1947 say that 25% of the children got no warm lunch, 38% had no shoes, 59% had to sleep in one bed with their siblings. At a reference day, at a school of 680 pupils, 125 were missing because they had to stand in line at food stores. 106 children came to school without having eaten anything (see Schmitz (1956)). Although these historical records focus only on children of school age, similar shortages can be assumed for infants. Droese and Rominger (1950), Meier (1951) and Klatt (1950) document the impacts of these catastrophic events on children's height and weight in Schleswig-Holstein and the hard-hit Ruhr region, respectively. Moreover, infant mortality during and after WWII are presented in Beil and Wagner (1951) and Redeker (1946).

2.3 Conceptual Framework

As a shock to early childhood environment, exposure to the postwar famine provides a unique opportunity to study the impact of malnutrition *in utero* and during early childhood on long-term health outcomes. Limited access to nutrition during the critical developmental period could lead to culling (selection), immunity and scarring effects among infants and young children (see Myrskylä (2010) and Valente (2014)). On the one hand, the *culling* effect suggests that postwar malnutrition and poor living conditions combined with maternal stress deteriorate the health endowment of fetuses and infants. Only the fittest infants and children survive and recover, leading to a positive selection among surviving birth cohorts, see also Pearson (1912). In this case, the aggregate health outcomes later in life over-represent the stronger part of the birth cohort. Similarly, a negative health shock early in life can also induce immunity, increasing the individual and aggregate health quality later in life. According to this 'acquired immunity hypothesis' (Myrskylä (2010)), a negative health shock early in life leads, like culling, also to a positive effect

on health later in life.
On the other hand, a scarring effect would shift the health endowments of surviving infants and children down (see Myrskylä (2010) and Deaton (2007)). This would suggest that children suffer from the adverse effects of postwar famine for life even though they have survived and cannot fully recover. The overall effect of childhood exposure to famine is determined by the relative importance of these opposing effects. The affected cohorts experience adverse health outcomes as adults if a negative scarring effect of famine dominates the positive selection effects. Alternatively, the long-term health of these children improves if the positive culling and immunity effects overcompensate the negative effect from scarring. In practice, it is difficult to decompose the potential effects that can outweigh each other in the data. Even in cases with clearly positive estimated effects, the choice between selection and immunity inducing effect is difficult to make, because both could yield the observed positive effect. In our paper, we therefore estimate the ultimate health effects of childhood exposure to postwar famine.

2.4 Data and Descriptive Statistics

We use region-level data from West-German municipalities for the health conditions early in life and the individual-level data from the German Socio-Economic Panel (SOEP) for the health outcomes later in life. In the next subsections, we describe in detail the variables we use in our study.

2.4.1 Individual-Level Data

We utilize the German Socio-Economic Panel (SOEP) for individual and household characteristics, which contains a wide range of information on individual and household characteristics and childhood environment. Essential to our paper, we use SOEPremote, which provides confidential information on the districts (*(Land-)Kreise*) in which the respondents reside. This allows us to identify their exposure to postwar famine. Another appealing feature of SOEPremote is its compatibility with our regional historical data on infant mortality: Given that we aggregate our historical data to the district-level as well, we are able to merge our regional historical data with SOEPremote using the individuals' residential districts. Moreover, the SOEP contains detailed information on family background such as mother's and father's educational attainment. This allows us to account for pre-war socio-economic conditions in the households and to tackle the potential heterogeneity in the long-term health effects of famine.

In our study, we focus on German natives born between 1935 and 1950. Our main outcome of interest is 'adult height' measured in cm. Adult height is a widely used health indicator shown to be associated with nutrition and

infections exposed to during infancy and early childhood (see Silventoinen (2003) and Deaton (2007)). Another advantage of height as health indicator is its quantitative nature, which minimizes the subjective reporting biases to a great extent. The SOEP provides respondents' height bi-annually since 2002; therefore, we utilize the 2002, 2004, 2006, 2008, 2010 and 2012 waves of the SOEPremote for adult height.
The SOEPremote provides information on the respondents' residential districts. We estimate the long-term health effects of postwar famine at this geographical identifier. This was dictated by the fact that none of the individual-level German data sets provides information on the place of birth or childhood place of residence for cohorts of individuals we focus on in our study (see Jürges (2013) and Pischke and von Wachter (2008)).[3] Nevertheless, the SOEPremote also asks respondents whether they still or again live in the city or area where they grew up. This question helps us identify whether individuals still reside in their childhood city or area. We test the robustness of our results by restricting our sample to individuals who still or again live in their childhood city or region. In addition, it is well documented that Germany historically had low levels of geographic mobility, about two to three percent among native Germans (see Rainer and Siedler (2009), Pischke and von Wachter (2008), and Hochstadt (1999)). This low rate reflects conditions after the end of WWII: families divided by war and evacuation were attempting to reunite. With postal and telephone communication destroyed, the only way that family members could achieve reunification was by returning to their home cities (Geo Epoche Panorama (2014)). In addition, movement between occupation zones was restricted, and individuals were not allowed to travel beyond their local areas (see Allied Control Authority (1946) and Hochstadt (2011)). Taken together, historical accounts document that wartime displacement was temporary. By June 1947, the urban population had reached 80 percent of pre-war levels, then nearly 90 percent in 1948 (see Hochstadt (1999)). Therefore, internal migration should yield at most minor consequences for our estimates.

2.4.2 District-Level Data

We use the municipality-level infant mortality rate as a measure of individual famine exposure, which is plausibly exogenous to individual behaviour. The infant mortality rate is defined as the number of infant deaths below the age of one year out of 100 live births in a given year. We assume that local characteristics such as population size, degree of industrialization, rurality and the quality of health care services partly determine the local infant mortality. Moreover, more volatile factors including annual crop yield, general

[3] A notable exception is the 2012 wave of SOEP which is the first data to provide place of birth information.

food supply and average disease load also significantly affect the local infant mortality rate.[4] The volatile nature of these factors allows us to identify how shocks experienced during pregnancy and infancy impact individual long-term health. Infant mortality rate (IMR) therefore mirrors health quality and rates of illness, economic development and the social and environmental situation of the whole population, since both infants and the rest of the population are affected by the same underlying factors (see Reidpath and Allotey (2003)). Another attractive feature of IMR is that it is very well documented and thus available even in times of few resources.
The municipality-level infant mortality rate is reported in the Statistical Yearbook of German Municipalities (Zeitler, Mewes et al. (1936-1952)). The Statistical Yearbook of German Municipalities has first been published in 1890 and provides detailed information on population, public services and finances for municipalities with at least 10,000 residents in the year of observation. The municipalities recorded in the yearbook slightly change from year to year with the growth and shrinkage of cities. Due to the separation of Germany in four occupation zones in the post-WWII era and later in West and East Germany, the Statistical Yearbooks shortly after WWII only cover the Western part of Germany without Saarland. Consequently, we are forced to restrict our analysis to West German municipalities. Hence WWII interrupted the publication of the Statistical Yearbooks between 1941 and 1949, data on infant mortality is available for the years 1935, 1937-40, 1946-1948 and 1950.[5] In addition, we present the estimation results where we incorporate population size and the ratio of live births to 1,000 citizens to account for the potential differences across rural and urban areas in terms of fertility and infant mortality in the course of postwar famine.
We aggregate the contextual data to the district level by building weighted averages.[6] Now the historical data reported in the Statistical Yearbooks is merged with the SOEP data in order to obtain our final data set of the districts.[7]

2.4.3 State-Level Data

By the end of WWII, Germany received many refugees from its former Eastern territories including East Prussia, Silesia, Pomerania, and East Brandenburg. This might have affected the well-being of children for several reasons. Therefore, in addition to district level controls, we also account for the share

[4] Since penicillin was not discovered until WWII and not introduced to the German market before 1946, infectious diseases probably were much more important in the years covered here than for the timespan observed by Mackenbach et al. (1990).
[5] In 1946, infant mortality data is available only for municipalities of at least 20,000 residents.
[6] For this purpose, population data is taken from the census 1939 for the pre-war years and from the 1946 and 1949 censuses for the postwar years. For 1947, we additionally use population size calculated on the basis of 1946 census.
[7] Since the Statistical Yearbooks only provide information for municipalities with at least 10,000 inhabitants, some current-day districts are not covered or only partially covered, which yields minor consequences for our analysis.

of expellees in each federal state in our analysis. State-level data on the share of expellees is provided from Gesis Histat and was published in Besser (2007) and Besser (2008).

2.4.4 Characteristics of Data

The data on municipal infant mortality rates (IMR) covers 427 West-German municipalities and 8 years (1935, 1937-40 and 1946-1948). Table 2.2 shows that the IMR was constantly rather low in the documented municipalities. The increase in infant mortality rate is striking in the immediate postwar years, where it spiked as high as 20%, especially in big cities. For instance, in Regensburg, the infant mortality rate was 25.7% in 1946. During the same time period, on the other hand, the average IMR in England and Wales remained at 2.94%, see Pamuk (1985).

Table 2.2: IMR in 174 municipalities

Year	Mean	5% quantile	95% quantile
1935	6.990	3.788	10.294
1937	6.308	3.206	9.388
1938	5.759	2.900	9.300
1939	6.545	3.200	10.400
1940	6.866	3.400	11.500
1946	9.983	4.700	15.100
1947	8.233	3.800	12.900
1948	6.979	3.800	12.700

Source: own calculations.

Table 2.3 tabulates the infant mortality rate in 1946 by federal state. Each state had different shares of agriculture, number of refugees, degrees of wartime destruction and was located in a different occupational sector, which contributed to a significant variation in infant mortality across regions. States of Schleswig-Holstein, Lower Saxony, Hamburg and North Rhine-Westphalia formed the British zone. The US zone consisted of Bavaria, Bremen, Hesse and parts of Baden-Wuerttemberg and the French zone of Rhineland-Palatinate and the rest of Baden-Wuerttemberg.[8] Table 2.3 shows that the French zone had the highest infant mortality rate on average followed by the US and the British zones. This is in line with the nutritional levels in the occupational zones reported in Table 2.1.

Table 2.4 presents the summary statistics for individual and family background characteristics. It also shows the main individual-level and district-level control variables we use in our estimation. Our sample includes the 1935-1940 cohorts as controls and the 1946-1950 cohorts as treatment cohorts. Individuals in our sample have the average height of 176.7 centimetres as adults. 17% of their mothers and 23% fathers completed more than a basic

[8]Since the Soviet zone was located in the former East Germany, it is not included in our data.

Table 2.3: Average state-level IMR in 1946

Federal state (*Bundesland*)	Infant mortality 1946 in %
Schleswig-Holstein	11.683
Hamburg	8.100
Lower Saxony	9.791
Bremen	6.950
North Rhine-Westphalia	9.632
Hesse	8.621
Rhineland-Palatinate	11.273
Baden-Wuerttemberg	9.064
Bavaria	10.205
Berlin	11

Source: own calculations, aggregated from the municipal data.

education (*Hauptschule*). The majority of our sample resided in the British occupation zone during the postwar era. The population-weighted average infant mortality rate is around 10 %.

Table 2.4: Summary statistics at individual level

Variable	Sample size	Mean	Std. dev.	Min	Max
Year of birth	909	1942	5.41454	1935	1950
Born 1946-1950	909	.449945	.4977621	0	1
Month of birth	909	6.522552	3.53877	1	12
IMR 1946	909	10.02363	3.221309	3.4	25.7
Mean IMR 1937-1938	909	6.401665	1.845757	2.6	15.6
Height in cm	909	176.6806	6.47773	159	196
Father: more than basic education	909	.2277228	.4195936	0	1
Mother: more than basic education	909	.1727173	.3782108	0	1
Birth rate 1939	904	24.68564	9.364744	14.6	75.7
Birth rate 1946	909	14.65207	3.341705	7.3	25.4
Population in district 1939	909	521,145.3	1075167	12641	4300000
Population in district 1946	909	419,636.4	798164.1	20483	3200000
French occupation zone	909	.1353135	.3422465	0	1
American occupation zone	909	.3344334	.4720518	0	1
British occupation zone	909	.6666667	.471664	0	1

2.5 Estimation Strategy

We exploit the plausibly exogenous variation in the famine-driven infant mortality across districts and birth cohorts to identify the long-term health effects of the childhood environment. We employ a difference-in-differences estimation strategy in our empirical analysis. The 'treatment' variable is an interaction between an indicator for being born or being a young child during the postwar famine period and in a district with above-average postwar infant mortality. In particular, we estimate the following difference-in-differences equation, where δ identifies the effect of the famine-driven health conditions during conception and infancy on individual long-term health:

$$Y_{idt} = \alpha + \beta' \text{IMR46}_d + \gamma' \text{FC}_{it} + \delta(\text{FC}_{it} \times \text{IMR46}_d) + \pi' X_{idt} + \rho' Z_{dt} + \epsilon_{idt}, \tag{2.1}$$

where Y_{idt} is the adult height in centimetres for individual i in district d born in year t. IMR46_d is an indicator for being born in a district with above average infant mortality in 1946. We utilize the district-level infant mortality rates in 1946 in our analysis since the famine-driven infant mortality peaked right after WWII and gradually decreased in the later years. FC_{it} is a dummy variable indicating a famine cohort. It takes a value of 1 if individual i was exposed to famine during conception and infancy and 0 otherwise. X_{idt} controls for individual characteristics such as year and quarter of birth, parental educational attainment, and whether an individual was raised in an urban region. Z_{dt} incorporates district-level controls such as population size in 1939 and the levels of infant mortality before WWII. Population size in 1939 helps us address the level of rurality in a given district. The average IMR in 1937 and 1938 at district level is included as reference level before the war. These years were affected neither by WWII nor by the Great Depression. We further control for occupational zones to capture the occupational powers' political strategies affecting nutrition, heath quality, and services provided. Finally, we control for the birth rate defined as the number of live births per 1,000 residents in our analysis to address potential concerns of composition effects, since fertility decisions can be associated with the local living conditions.

In their seminal work, Almond and Currie (2011b) suggest that the key developmental window falls between conception and 5 years of age. Thus, individuals are more susceptible to be affected from adverse shocks during this critical period of development. Therefore, individuals born between 1946 and 1950 form the affected group in our analysis. These children either were born or were up to 5 years old during the severe postwar famine, which potentially altered the health conditions in the districts they grew up. Individuals born in 1950 represent the last affected birth cohort since the food rationing system was abolished in 1950. However, as described in the background section, the nutritional quality significantly improved starting from 1948. We therefore restrict the affected cohorts to individuals born between 1946 and 1948 to test the robustness of our results. In contrast, individuals born between 1935 and 1940 were not affected from the postwar famine in the aforementioned critical age. Allied aerial attacks on German soil were limited and mostly unsuccessful until June 1940 and the food supply became alarmingly low only in the very last period of WWII. Moreover, the food rationing was abolished in 1950 in West Germany. Therefore, cohorts born in mid-1950s were not affected by the famine. The control group therefore consists of individuals born before the war started, plus the individuals in less

affected regions (i.e. where the IMR was below average). In sensitivity analyses also individuals from after the war are added to the control group. In these observations, $FC_{it} = 0$ or $IMR46_d = 0$. Thus, δ measures the height difference between the affected and the control groups that can be ascribed to the varying exposure to the postwar famine across districts and across birth cohorts.

The validity of the difference-in-differences analysis relies on the identification assumption, which postulates that the treatment occurs randomly conditional on the control variables. More specifically, we assume that in the absence of famine the difference in health outcomes between the affected and the control cohorts would have been the same in the districts with above- and below-average postwar infant mortality rates. Thus, in order to credibly identify the long-term health effects of childhood environment, shocks experienced during early childhood should be unrelated to the unobserved individual characteristics that also directly affect long-term health outcomes (Van den Berg, Doblhammer, and Christensen (2009)). The local infant mortality has the advantage of not being directly caused by individually varying early-life health conditions. Thus, it provides a plausibly exogenous variation in the childhood environment across individuals born in different districts.

There are some potential confounding factors that can potentially bias our results. First, the long-run nature of our study presents several challenges to our estimation including selection in mortality by individual characteristics. The SOEP reports individual height in 2002 for the first time, when the birth cohorts we focus on are between 52 and 69 years old. A fraction of these cohorts thus has already died by 2002. This potential concern is partly mitigated by the birth year dummies in the difference-in-differences analysis since they control for national age-specific death rates. However, if different cohorts experience different rates of mortality in districts with high and low postwar infant mortality rates, a composition bias will occur. Moreover, if mortality rates are higher among the affected cohorts in districts with higher postwar famine, we will underestimate the true long-term health effects of childhood exposure to famine since birth cohorts with better health would survive longer. We address this potential concern in the sensitivity analysis section.

Another concern for our analysis is the positive time trend in adult height. When comparing the height of birth cohorts differing by several years, this positive time trend can bias our results in favour of later cohorts. To avoid this potential bias, we employ a difference-in-differences strategy in our analysis. Employing this approach allows us to account for the positive time trend in adult height through controlling for the improvement in adult height in the less affected districts during the same time. In addition, since all regions in Germany suffered from the famine, but to a different extent, the

time trend in adult height in the control districts is probably also lower than counter-factually without famine. Therefore, the estimated coefficients yield lower bound estimates of the true effect.
The infant mortality rates are available only for municipalities with at least 10,000 residents in 1949. Very rural areas with small population are underrepresented in our data. The exposure to famine might be different in these rural areas, as well as the capabilities to cope with nutritional shortages or destruction of health care services. For instance, food ration cards provided a lower nutritional value for residents in rural areas due to their higher capability of feeding themselves. We attempt to address this potential concern by controlling for the rurality of childhood environment in our analysis.

2.6 Estimation Results

Table 2.5 presents the difference-in-differences estimates of famine-driven postwar infant mortality on adult height. In all of our analyses, the coefficient for the interaction term ("(IMR$_{1946}$ high $\times$ (born $1946 - 1950$))") presents the estimated average treatment effect. In order to minimize the potential measurement and reporting errors in height, we utilize the individuals' height reported in several waves of our data. Thus, the outcome of interest in our analysis is the average adult height reported between 2002 and 2010. The difference-in-differences estimate in Column (1) is -2.88 cm and highly significant. This suggests that the affected cohorts in districts with high infant mortality are 2.88 cm shorter when adults compared to individuals in the control group. This is a sizeable effect since average height of West German men and women increased by almost 2.5 centimetres in the second half of the 20th century (see Heineck (2005)). Column (1) also documents that cohorts born in the postwar era are 3.19 cm taller in general than the earlier cohorts. Similarly, being born in a district with high postwar infant mortality positively contributed to adult height before the war (+1.86 cm). However, our analysis demonstrates that among the postwar affected cohorts, the cohort increase in height is almost completely eaten up when living in high-mortality-districts.
In Column (2), we include individual covariates such as quarter and year of birth, parental educational attainment and an indicator for being raised in an urban area. In addition, we add district-level controls including the average IMR of 1937 and 1938, the population size in 1939, the birthrate in 1946 and the occupation zones to which the districts belonged to in the postwar era. The difference-in-differences estimate remains statistically and quantitatively similar with these additional controls. We find that father's education significantly contributes to an individual's height later in life, while the effects of maternal education is limited. Since household income was primarily determined by father's skill level at the time, this could explain the

Table 2.5: Effect of famine exposure on adult height

Variable	(1)	(2)	(3)
Individual-level variables			
Born 1935–1940		(reference group)	
Born 1946–1950	3.192***	3.073***	
	(0.99)	(0.95)	
(IMR_{1946} high) × (born $1946-1950$)	-2.876***	-2.913***	
	(0.92)	(0.95)	
Born 1946–1948			4.295***
			(1.34)
(IMR_{1946} high) × (born $1946-1948$)			-2.624**
			(1.11)
Father: more than basic education		3.120***	2.976***
		(0.72)	(0.80)
Mother: more than basic education		-0.561	-0.895
		(0.91)	(0.96)
District-level variables			
IMR_{1946} high	1.864***	1.939***	1.964***
	(0.50)	(0.60)	(0.61)
Mean IMR 1937–1938		-0.015	-0.078
		(0.15)	(0.17)
Population 1939		0.000	0.000
		(0.00)	(0.00)
Birth rate 1946		-0.028	0.026
		(0.08)	(0.09)
American occupation zone		-0.387	-0.379
		(0.52)	(0.56)
French occupation zone		-1.143	-1.113
		(0.83)	(0.85)
State-level variables			
% Expellees in population 1946		0.032	0.022
		(0.03)	(0.04)
			(1.11)
Constant	174.655***	174.468***	174.005***
	(0.93)	(1.73)	(1.98)
Birth year FE	yes	yes	Yes
Season of birth FE	yes	yes	Yes
Adj. R-squared	0.025	0.055	0.041
Sample size	909	909	735

Note: standard errors in parentheses are clustered at district level. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

disparity in the effects of parental education on adult height. Column (2) further reveals that the occupational power in the district was also important for the future health outcomes of the affected cohorts. Individuals in the French zone attain substantially lower stature in adulthood compared to their counterparts in the British zone. These estimates confirm the historical accounts on nutritional intake. They document that nutritional quality in the French zone was considerably lower than in the later Bi-zone. We further test the validity of our results by dropping one federal state at a time (not shown in a table). Our results remain robust in this specification suggesting that they are not an artefact of postwar conditions in a particular district. As mentioned above, Column (3) shows very similar estimates for a shorter treatment window: here, only individuals who were born until 1948 were considered, since

supply of food and other goods improved in 1948 already. Our results remain robust with a different categorization of the affected cohorts.

Given that, height is a strong predictor of the general health endowment; our findings postulate that adverse local health conditions during conception and infancy significantly impair the future health outcomes of individuals. According to Hebert et al. (1993), for instance, a height loss of 2.9 cm leads to an increase in the risk of cardiovascular disease within 5 years by roughly 3% (with each additional inch, the risk is reduced by 2% to 3%). Moreover, Heineck (2005) finds an increase of 0.5% in gross monthly wages for each additional centimetre in height for men in Germany, i.e. 1.5% for the estimated height loss of 3 cm. Height is also associated with higher self-esteem (Freedman (1979)) and higher social dominance (Hensley (1993)) and is rewarded in the mating and marriage market (Belot and Fidrmuc (2010)). In addition, Akbulut-Yuksel and Kugler (2016) and Coneus and Spieß (2012) document a strong correlation in height across generations. Therefore, the detrimental and long-lasting health consequences of postwar famine clearly extend well beyond the health effects we quantify in our paper. Our results therefore provide the lower bound estimates for the harmful enduring effects of this catastrophic event.

2.7 Sensitivity Analyses

2.7.1 Famine Exposure of Control Cohorts

In this section, we discuss the potential confounding factors and test the validity of our results by formally addressing these concerns.
One of the concerns is the famine exposure among the control cohorts during their later childhood. Individuals born between 1935 and 1940 form the control group in our analysis. Cohorts born before WWII were between 6 and 18 years of age during the 1946-1950 famine. The medical literature postulates that the highest yearly height growth occurs during adolescence. Therefore, malnutrition during this period could affect the adult height of pre-war cohorts as well. Since the control cohorts in the districts with high infant mortality would be more affected than the same cohorts in the districts with low infant mortality, our results should be downward biased. Nevertheless, to circumvent this problem and test the robustness of our results to the choice of the control cohorts, we additionally include younger birth cohorts to the control group. In Column (1) of Table 2.6, we first present the estimation results including the cohorts born between 1935 and 1940 and the cohorts born between 1955 and 1960 into the control cohorts. In the second column of Table 2.6, the control cohorts only encompass individuals born after the Allied

forces repealed the food rationing practice (i.e. 1955 to 1960). In the last column of Table 2.6, we also test a control group consisting of the 1950–1955 birth cohorts. Admittedly, the sample size in this last estimation is substantially smaller than before. Possibly this explains the substantial but insignificant treatment estimate in Column (3). Taken together, analyses presented in Table 2.6 bolster our confidence that our results virtually remain quantitatively and statistically similar to a different categorization of the control group.

Table 2.6: Sensitivity analyses: inclusion of later control cohorts

Variable	(1)	(2)	(3)
Individual-level variables			
Born 1935–1940	(reference group)	–	–
Born 1946–1950	2.832***	-1.085	-1.578
	(0.97)	(1.16)	(1.60)
Born 1950-1955	–	(ref. group)	–
Born 1955-1960	2.719***	–	(ref. group)
	(0.88)		
(IMR_{1946} high) $\times$ (born $1946 - 1950$)	-2.419***	-1.935**	-2.169
	(0.85)	(0.92)	(1.68)
Father: more than basic education	2.096***	1.364**	1.601*
	(0.60)	(0.68)	(0.86)
Mother: more than basic education	-0.207	-0.093	-0.308
	(0.66)	(0.71)	(1.18)
District-level variables			
IMR_{1946} high	1.338**	0.806	1.225
	(0.55)	(0.74)	(1.56)
Mean IMR 1937–1938	0.092	0.203	0.015
	(0.13)	(0.16)	(0.21)
Population 1939	0.000	0.000	0.000
	(0.00)	(0.00)	(0.00)
Birth rate 1946	0.016	0.035	-0.015
	(0.07)	(0.10)	(0.12)
American occupation zone	-0.252	-0.400	-1.145
	(0.49)	(0.65)	(0.81)
French occupation zone	-1.527**	-2.196**	-1.574
	(0.66)	(0.96)	(1.12)
State-level variables			
% Expellees in population in 1946	0.032	0.045	0.077*
	(0.03)	(0.03)	(0.04)
Constant	173.530***	175.909***	178.013***
	(1.63)	(1.78)	(2.42)
Birth year FE	yes	yes	yes
Season of birth FE	yes	yes	yes
Adj. R-squared	0.067	0.036	0.025
Sample size	1,432	932	510

Note: standard errors in parentheses are clustered at district level. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

2.7.2 Migration

Another concern is the possibility of non-random internal migration during or after the famine across regions. In addition, Germany received refugees

and expellees from the former parts of Germany and Soviet Zone/GDR after WWII. Internal migration is unproblematic for our results as long as it is random with respect to health status. We would only overestimate the true treatment effect if healthy people move from treated to control regions, or if unhealthy people move from control to treated regions. Then, the health distribution across districts would simulate a treatment effect.
We assess the robustness of our results to internal migration in Table 2.7. First, we restrict our analysis to individuals for whom we explicitly have the regional information for childhood and adolescence. They answered positively to the question: "Do you still live in the city or area where you grew up?". If the estimated effect of famine for this restricted sample differs from the entire sample, migration will present a concern for our analysis. The first column of Table 2.7, however, shows that the treatment effect goes in the same direction for the very small sample, which lends credence to our estimation strategy. However, since this question was asked only once, in 1985, the number of respondents is low for reliable statistical inference; therefore, these results should be approached with caution. Second, following Jürges et al. (2011), we drop the city-states of Berlin, Hamburg and Bremen, where a particularly large number of individuals moved, to test robustness of our results to the potential internal migration. Similar to the previous columns, results remain statistically and quantitatively similar to the baseline specification when we exclude the city-states (Table 2.7 Column 2).
We further attempt to tackle the potential selection in migration by excluding regions to which a large number of expellees from the Eastern German regions migrated after WWII. These expellees might have a different health capital than the rest of the population due to the (partly wild) expulsion and the challenging and long migration path they were forced to take to reach West Germany. The weakest might not have undertaken or survived this journey. Our data suggests that the share of expellees in the population is highest in states of Schleswig-Holstein, Bavaria and Lower Saxony. This reinforces the historical accounts stating that the expellees stayed close to the Eastern border of West Germany and mostly settled in rural regions with hope of returning to their home soon. If the expellees drive our results, the analysis without Schleswig-Holstein, Bavaria and Lower Saxony should yield a much smaller difference-in-differences estimate. In Column (3) of Table 2.7, the difference-in-differences estimate is similar in magnitude to the baseline specification. This suggests that our results are not merely driven by the distribution of expellees.
Finally, we test the robustness of our results by excluding one district each time. We find no evidence to suggest that one particular district is responsible for our results. Taken together, analyses presented in Table 2.7 suggest that, if anything, internal migration yields minor consequences in our analyses.

Table 2.7: Sensitivity analyses: tests on selective migration

Variable	(1)	(2)	(3)
Individual-level variables			
Born 1935–1940		(reference group)	
Born 1946–1950	-1.323	2.545**	2.641**
	(3.37)	(1.11)	(1.14)
(IMR_{1946} high) × (born 1946 − 1950)	-1.772	-2.257**	-3.011***
	(2.24)	(0.99)	(1.03)
Father: more than basic education	3.185	3.751***	3.001* **
	(2.53)	(0.71)	(0.95)
Mother: more than basic education	2.778	-1.428*	-0.322
	(3.12)	(0.81)	(1.19)
District-level variables			
IMR_{1946} high	0.744	1.914***	1.836***
	(1.88)	(0.64)	(0.63)
Mean IMR 1937–1938	0.006	-0.010	-0.020
	(0.45)	(0.14)	(0.18)
Population 1939	-0.000	-0.000	0.000
	(0.00)	(0.00)	(0.00)
Birth rate 1946	-0.088	-0.081	-0.016
	(0.27)	(0.07)	(0.09)
American occupation zone	-0.630	-0.363	-0.177
	(1.40)	(0.54)	(1.00)
French occupation zone	0.354	-0.829	-1.044
	(2.03)	(0.96)	(0.86)
State-level variables			
% Expellees in population 1946	-0.128*	0.047	-0.058
	(0.07)	(0.03)	(0.15)
Constant	179.206***	175.548***	174.783***
	(5.58)	(1.74)	(2.36)
Birth year FE	yes	yes	yes
Season of birth FE	yes	yes	yes
Adj. R-squared	0.015	0.061	0.040
Sample size	114	800	679

Note: standard errors in parentheses are clustered at district level. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

2.7.3 Selective Fertility

Another concern for the interpretation of our results is the selective fertility. For instance, the prospective parents with higher socio-economic status might have deferred childbearing in the course of hardship while those with lower socio-economic status continued to conceive. This might potentially generate a selection bias and confound our results. Selective military drafting could be another source for the selective fertility. Even though there was a nationwide military draft during WWII, which even included young adults by the end of WWII, the taller men might be perceived as healthier and favoured in the military draft. At the same time, taller men are more likely to live in the districts badly affected by the war. An additional source for selective fertility might stem from the immediate postwar sexual violence towards German women by soldiers of the occupying powers. The frequency of sexual violence towards German women could vary across regions (Naimark (1995)).

Children who were conceived during sexual violence would potentially receive less parental investment during childhood.

We test the robustness of our results to the possibility of selective fertility in three different ways:
First, we run a Placebo test in Table 2.8 where the outcome of interest is paternal education. If better educated parents deferred childbearing in the course of hardship, the treatment indicator would explain the share of well-educated fathers in a district. Table 2.8 shows that a high level of infant mortality together with being born during the famine has no significant effect.
Second, we test whether father's death in combat is associated with the postwar infant mortality rates at the districts in Table 2.9. If more men – who could otherwise have had (tall) children – died in the highly affected regions than in the less affected regions, we should also observe the same pattern in father's death among the control cohorts who were born before the onset of WWII. Selective male mortality in the course of war would suggest that fathers of children in the control cohorts should have died more often during the war years, if they lived in districts with high postwar infant mortality. We find no meaningful variation in paternal death across districts; therefore it is unlikely that our results are an artefact of the potential selective fertility engendered by selective fertility and military drafting.
Third, we assess the sensitivity of our results to the immediate postwar sexual violence towards German women in Table 2.10. Historical accounts postulate that German women in all occupation zones endured sexual violence of Allied soldiers, especially during the first days and weeks of the occupation (Harrington, 2010, p. 80 and p. 84). Thus, since most of the children conceived through sexual violence were potentially born in 1946, we exclude this birth cohort from our analysis. Our difference-in-differences estimate on postwar infant mortality is nonetheless very similar to those including 1946, bolstering our confidence that these children are not driving our results.

2.7.4 Linear Treatment Definition

In Table 2.11, we replace the dummy indicating high infant mortality with the district-level IMR. This allows us to estimate the average effect of a percentage points increase in the local infant mortality on adult height. However, the validity of this specification lies in the strict assumption of linearity in the effects of infant mortality and imposes higher requirements on data quality. To the best of our knowledge, the data quality in our study is high; therefore, the latter point should not be problematic for our estimation. Table 2.11 shows a significant negative average effect of being born during the famine years in the highly affected districts. The difference-in-differences estimate suggests that a percentage point increase in district infant mortality rates leads

Table 2.8: Placebo test: famine effect on social status – paternal educational attainment (1/0)

Variable	(1)	(2)
Individual-level variables		
Born 1935–1940	(reference group)	
Born 1946–1950	0.018	0.006
	(0.04)	(0.06)
(IMR_{1946} high) × (born 1946 − 1950)	0.020	0.015
	(0.05)	(0.04)
Mother: more than basic education		0.686***
		(0.04)
District-level variables		
IMR_{1946} high	0.028	-0.008
	(0.04)	(0.03)
Mean IMR 1937–1938		0.005
		(0.01)
Population 1939		0.000
		(0.00)
French occupation zone		0.017
		(0.05)
American occupation zone		0.024
		(0.03)
Birth rate 1946		-0.005
		(0.00)
Federal state-level variables		
% Expellees in population 1946		0.002
		(0.00)
Constant	0.200***	0.030
	(0.02)	(0.09)
Birth year FE	yes	yes
Month of birth FE	yes	yes
Adj. R-squared	-0.000	0.396
Sample size	909	909

Note: standard errors in parentheses are clustered at district level. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

to a 2.96 mm shorter stature in adulthood among individuals born during the postwar famine. Hence the average postwar infant mortality is around 10 %, the difference-in-differences estimate with infant mortality measure in Table 2.11 is quantitatively and statistically similar to the main specification summarized in Table 2.5.

2.7.5 Linear and Quadratic Time Effects

Finally, in Table 2.12, we present the estimation results where we replace the birth year dummies with a linear time trend in Column (1), and with both linear and quadratic time trends in Column (2). We define the time trend as the natural logarithm of the respondent's year of birth minus 1925. Our results remain robust to the inclusion of the linear and polynomial time trends. Moreover, Column (2) shows that the effect of linear time trend in adult height is negative while the estimate for quadratic time trend is positive

Table 2.9: Placebo test: famine effect on paternal death 1939–1945

Variable	(1)	(2)
Individual-level variables		
Born 1935–1940	(reference group)	
Born 1946–1950	-0.153***	-0.189***
	(0.02)	(0.05)
(IMR_{1946} high) × (born 1946 − 1950)	-0.015	-0.013
	(0.03)	(0.03)
Father: more than basic education		0.014
		(0.03)
Mother: more than basic education		-0.042*
		(0.02)
District-level variables		
IMR_{1946} high	0.015	0.012
	(0.03)	(0.03)
Mean IMR 1937–1938		0.001
		(0.01)
Population 1939		0.000
		(0.00)
French occupation zone		-0.020
		(0.03)
American occupation zone		0.003
		(0.02)
Birth rate 1946		-0.002
		(0.00)
State-level variables		
% Expellees in population 1946		-0.001
		(0.00)
Constant	0.153***	0.241***
	(0.02)	(0.08)
Birth year FE	yes	yes
Month of birth FE	yes	yes
Adj. R-squared	0.077	0.066
Sample size	909	909

Note: standard errors in parentheses are clustered at district level. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

suggesting a U-shape relation between birth year and adult height. This U-shape pattern implies that individuals born in the medium cohorts suffered more from the famine than the surrounding cohorts, on average, and attained shorter height as adults.

2.8 Conclusion

In this study, we quantify the long-term health consequences of adverse health shocks experienced early in life. We identify these long-term health effects by exploiting a plausibly exogenous variation in the famine exposure across districts and across birth cohorts engendered by the postwar food shortage in Germany. These devastating conditions left especially infants and young

Table 2.10: Sensitivity analysis: births 1946 excluded

Variable	Height
Individual-level variables	
Born 1935–1940	(reference group)
Born 1947–1950	3.020***
	(1.02)
(IMR_{1946} high) × (born $1947 - 1950$)	-2.866***
	(0.96)
Father: more than basic education	3.092***
	(0.78)
Mother: more than basic education	-0.250
	(0.94)
District-level variables	
IMR_{1946}high	1.963***
	(0.61)
Mean IMR 1937–1938	-0.053
	(0.14)
Population 1939	0.000
	(0.00)
Birth rate 1946	-0.044
	(0.07)
American occupation zone	-0.386
	(0.52)
French occupation zone	-0.793
	(0.87)
State-level variables	
% Expellees in population 1946	0.043
	(0.03)
Constant	174.872***
	(1.74)
Birth year FE	yes
Season of birth	yes
Adj. R-squared	0.057
Sample size	845

Note: standard errors in parentheses are clustered at district level. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

children vulnerable. They suffered from severe malnutrition, stress and generally adverse living conditions during their key developmental window. Therefore, we utilize infant mortality rates before age one in the residential districts in our analysis, which drastically increased in the aftermath of WWII, as a proxy of adverse childhood environment.

Applying a difference-in-differences approach, we document the negative and enduring effects of malnutrition and poor living conditions during early childhood on adult height. We find that individuals who were born during the famine years in districts with high infant mortality rates are on average almost 3 cm shorter as adults. This is a sizeable effect compared to the height increase of West German men and women by almost 2.5 cm in the second half of the 20th century (Heineck (2005)). Since we find that surviving children attain lower height as adults, our analysis suggests that the negative scarring effect of the famine dominates a positive culling and immunity effects. The results presented in this study should be generalizable to the broader context

Table 2.11: Linear treatment effect of famine

Variable	Height
Individual-level variables	
Born 1935–1940	(reference group)
Born 1946–1950	4.463***
	(1.63)
$(\text{IMR}_{1946}) \times (\text{born } 1946 - 1950)$	-0.296**
	(0.14)
Father: more than basic education	3.074***
	(0.73)
Mother: more than basic education	-0.505
	(0.92)
District-level variables	
IMR 1946	0.206*
	(0.11)
Mean IMR 1937–1938	-0.003
	(0.17)
Population 1939	0.000
	(0.00)
Birth rate 1946	-0.023
	(0.07)
American occupation zone	-0.377
	(0.52)
French occupation zone	-1.219
	(0.82)
State-level variables	
% Expellees in population 1946	0.034
	(0.03)
Constant	173.333***
	(1.71)
Birth year FE	yes
Season of birth FE	yes
Adj. R-squared	0.047
Sample size	909

Note: standard errors in parentheses are clustered at district level. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

of adverse medical or economic conditions during infancy and shed lights on the potential health consequences of these catastrophic events.

Table 2.12: Famine effect on adult height with logarithmized age

Variable	(1)	(2)	(3)
Individual-level variables			
Born 1935–1940		(reference group)	
Born 1946–1950	1.434	1.688	-0.130
	(1.31)	(1.30)	(1.84)
(IMR_{1946} high)	1.846***	2.060***	2.088***
	(0.50)	(0.56)	(0.56)
(IMR_{1946} high) $\times$ (born $1946-1950$)	-2.891***	-3.066***	-3.046***
	(0.91)	(0.94)	(0.94)
log(year of birth - 1925)	2.720	2.288	-31.933
	(1.72)	(1.73)	(20.64)
log(year of birth - 1925)^2			6.589*
			(3.98)
Father: more than basic education		2.930***	3.006***
		(0.70)	(0.71)
Mother: more than basic education		-0.473	-0.551
		(0.90)	(0.90)
District-level variables			
Mean IMR 1937–1938		-0.062	-0.062
		(0.14)	(0.14)
Population 1939		0.000	0.000
		(0.00)	(0.00)
Birth rate 1946		-0.021	-0.015
		(0.08)	(0.08)
French occupation zone		-1.341*	-1.342*
		(0.79)	(0.77)
American occupation zone		-0.331	-0.360
		(0.54)	(0.54)
State-level variables			
% Expellees in population 1946		0.029	0.028
		(0.03)	(0.03)
Constant	168.065***	169.494***	213.641***
	(4.34)	(4.76)	(26.90)
Month of birth FE	yes	yes	yes
Adj. R-squared	0.026	0.055	0.056
Sample size	909	909	909

Note: standard errors in parentheses are clustered at district level. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

3 Disentangling Stress from Nutrition as Determinants of the Long Run Effects of Adverse Conditions Around Birth on Economic and Health Outcomes Late in Life*

3.1 Introduction

Recently, social sciences have witnessed an increasing interest in the long-run effects of early life conditions on adult health and socio-economic outcomes. For a concise overview on the impact of early life conditions on later life outcomes, we refer the reader to e.g. Almond and Currie (2011a). What has started as an epidemiological strand of literature – and has most famously been theorized about in the seminal work of Barker (1994) – has triggered interdisciplinary academic work mostly for its universal relevance: If early life conditions are found to have significant impacts on late life health outcomes they may induce long-run costs for the affected individuals and a public health system as a whole. These costs may be underestimated when focusing on short term effects only.

Analysing the impact of early life conditions on later life health is a challenging empirical task. A standard approach is to use aggregate level shocks that affect a fraction of individuals during an early life period. Within this literature one can find a bulk of work studying the long run effects of early life exposure to famine as a proxy for a nutritional shortage in early life; see Lumey et al. (2011) for a recent survey. Since nutritional deprivation and food insecurity are in principle economic problems, famine studies have also

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become of special interest in the economics literature (see e.g. Lindeboom et al., 2010; Neelsen and Stratmann, 2011; Jürges, 2013; Van den Berg et al., 2016).
Outcomes found to be affected by famine range from chronic diseases and physical indicators, such as type II diabetes risk and adult height, to mental disorders, such as schizophrenia, or economic outcomes. Given the broad spectrum of outcomes affected by early life famine one question has yet to be conclusively answered: Is an individual-level nutritional shortage the only channel via which famine affects these outcomes? Or is there another component, such as famine induced psychological stress or stress evoked by factors paralleling the famine, that simultaneously influences long run health? If this is in fact the case, the interpretation of famine effects may need to be adjusted. Exogenous variation that plausibly shifts only one of these components, i.e. evokes either stress or malnutrition on an individual level, may help to answer this question.

Our study addresses this concern and uses World War II (WWII) and its aftermath in Germany[1] as a unique natural experiment to disentangle the effects of early life famine and early life stress. We combine a detailed micro data set that includes the respondents' place of birth with city-level information on the timing and intensity of Allied air raids on Germany during WWII, and time-varying regional-level information on food rations that were distributed during the German famine in the aftermath of the war. We analyse effects on adult height, life satisfaction, hypertension, and pensions as a proxy for life time earnings. Assuming that air raids evoke stress and that famine may have an effect via both, stress and malnutrition, we can use our empirical findings to trace out the distinct effect of each component on the outcomes.

Up until now, conclusive evidence on this matter is scarce. Yet, the problem we outlined above has been acknowledged in the literature on e.g. the Dutch famine. Psychological stressors may have been prevalent there also due to war-related factors paralleling the famine (e.g. Roseboom et al., 2001). Even if a specific stressor may have been identified, a solid empirical result requires a large degree of cross-sectional and temporal identifying variation in this stress inducing factor that is independent of famine exposure. Our data set provides information that is detailed in both, the regional as well as the temporal dimension.
Our findings are important for several reasons. Knowing which outcomes are influenced by a certain type of early life condition may help to determine

[1] We note that World War II in general, German aggression, and the horrendous actions taken by the Nazi regime affected many people in different nations. The focus of our paper on the effects of World War II on the German civilian population is due to the focus of our data on the German context and is not to be interpreted as a normative stance.

potential threats to late life health more narrowly and select adequate compensatory measures. Additionally, our findings aim to help researchers to interpret existing results of famine studies and to design studies in the future.
We find that exposure to bombardments in the first six months of life raises the risk of hypertension in men later in life. Famine during the last six months of pregnancy is related to lower male adult height and pensions, as a proxy for productivity throughout the working life. These results are robust to various sensitivity analyses. In light of our theoretical exposition, these reduced form effects imply that male adult height and productivity are influenced by malnutrition (and not by stress) whereas male hypertension is influenced only by stress. Moreover, the stress component under the German famine seems not to be quantitatively important. Our findings on the outcome of life satisfaction are less clear-cut.

The outline of the paper is as follows. Section 3.2 reviews theoretical and empirical literature on the effects of stress and malnutrition. Section 3.3 describes the historical context of our study. Section 3.4 presents our data sources and discusses the key variables. Section 3.5 describes our analytical framework, the empirical strategy, and discusses potential threats to identification. Section 3.6 presents the empirical findings and various robustness checks. Section 3.7 discusses the results and concludes.

3.2 Explanatory Frameworks and Previous Evidence

Infants and fetuses are generally regarded as particularly susceptible to outside influences. The fetal programming hypothesis by David Barker links these outside influences to permanent alterations in the human metabolism and long-run health risks, such as a high risk for type II diabetes and cardiovascular diseases (see e.g. Barker, 1994).[2]
The biological literature suggests that adversity in early life alters the phenotype and thereby influences health later in life: Low socio-economic status in childhood has been associated with a pro-inflammatory phenotype that might foster late life diseases in the long run (see e.g. Miller et al., 2009; Miller and Chen, 2007). Moreover, there is indication for harsh family climates to also trigger pro-inflammatory phenotypes (see e.g. Miller and Chen, 2010).
Along these lines, it is conceivable that the exposure to adversities such as stress or food shortages early in life may lead to similar epigenetic mechanisms. Thus, nutritional shortages and stress exposure could influence later

[2]See also Almond and Currie (2011b) for a comprehensive survey of fetal programming studies with economic scope.

life health, to a certain degree, through such a common channel. Additionally, in a recent review Kuzawa (2012) points towards some similar health consequences that are potentially triggered by nutritional and psychological stress *in utero*.
We are interested in the long-run consequences of prenatal and postnatal stress and nutritional deprivation on long-run health. In this section we present a brief overview of the literature on the effects of malnutrition (3.2.1) and of early life stress (3.2.2).[3]

3.2.1 The Effects of Early Life Malnutrition and Famine

Long-run health effects of malnutrition around birth are a major focus in medical as well as in economic studies, see e.g. Lindeboom et al. (2010), Neelsen and Stratmann (2011), Jürges (2013), Van den Berg et al. (2016). Barker (1990) notes that intrauterine environment and conditions very early in life explain health differences much later in life. Birth weight, breastfeeding, infant mortality rate and other indicators approximate early-life conditions, while late-life health is measured e.g. as cardiovascular mortality, risk of hypertension and current blood pressure. Other studies find a relationship between *in utero* exposure to a famine and adult height, diabetes, and schizophrenia. For an overview of the research on early-life conditions see e.g. Lumey et al. (2011), Almond and Currie (2011b) or Pollitt et al. (2005).
A potential mechanism behind these associations may be epigenetic changes during gestation which may permanently alter the phenotype:
A fetus is able to adapt to a variety of environments due to its high developmental plasticity (see Bateson et al., 2004). Especially during so-called critical periods (mostly periods of rapid organ development), the human body adaptively responds to biological or social cues (see Kuzawa and Quinn, 2009). During gestation, the mother's metabolism may transfer information about environmental conditions, e.g. nutrient availability, to her baby through the placenta (and later through breast milk composition) (see e.g. Gluckman and Hanson, 2005, p. 61; Kuzawa and Quinn, 2009). If the mother suffers from malnutrition, the baby reacts by adapting to a world with food scarcity. Hales and Barker (1992) suggest that this may involve "insulin resistance, a shift in circulation to protect the brain ('brain sparing'), and a nutrient conserving reduction in organ growth" (Bateson et al., 2004). By these modifications, the fetus gains survival advantages under the adverse situation which, however, come at a cost: If nutrient supply increases after birth, the famine-programmed individual may be more likely to suffer from cardiovascular disease, hypertension, type II diabetes, higher mortality, or obesity (see e.g. Barker et al., 1990; Cameron and Demerath, 2002; Bateson et al., 2004; Gluckman et al., 2008; Schulz, 2010).

[3] For a review on the role of psychosocial and nutritional stress for pregnancy outcomes, we refer the reader to e.g. Hobel and Culhane (2003).

Beside biological pathways, also behavioural mechanisms may explain long-run effects of early life malnutrition. Parents might try to compensate early-life food scarcity in a later stage of childhood, when the nutritional situation improves. As stated above, however, nutrient-rich food might be harmful for the famine-programmed child. A hunger period can also induce a higher stress level in the family through a fight for resources within and between households. For non-biological pathways from early life famine to later life health effects see e.g. Hamelin et al. (1999), Hadley and Patil (2006) and Whitaker et al. (2006). In the context of the German famine, Kesternich et al. (2015) present evidence for behavioural channels to be relevant for the connection between early life hunger and later life adult health.

Other studies that measure long-run effects on those born or conceived during the German famine suggest that those cohorts are worse off in terms of higher education and occupational status (Jürges, 2013). Additionally, regional variation in early life adversity during the famine is found to be associated with differences in height among men (see Chapter 2). Also Van den Berg et al. (2016) study long-run effects of the Greek, the Dutch and the German famines in the context of WWII and find undernutrition until age 4 to reduce male adult height by 3 cm.
Several other famines have also been studied to separate the effects of malnutrition from socio-economic status, which are in general strongly correlated; see Lumey et al. (2011) for a comprehensive survey of the famine literature. The 'Dutch Hungerwinter' in 1944/1945, caused by an embargo initiated by the German occupying forces, often serves as an example of adverse living conditions, see e.g. Banning (1946), Lumey et al. (2011), and Painter et al. (2005). Studies found cardiovascular diseases, lower self-rated health, obesity and diabetes to be among the consequences of malnutrition. Susser and Stein (1994) also find a decrease in adult stature after exposure to the Dutch famine shortly after birth.
Also the 'Great Famine' in China 1959-61 has been analysed in many studies with respect to long-run effects. E.g. Chen and Zhou (2007) use regional excess death rates as a proxy for famine intensity and find adult height to be lower by 3.03 cm among those severely exposed to famine. Susser and St Clair (2013) compare studies on Dutch and Chinese famine with respect to mental disorders. Increased risk of schizophrenia seems to be a common result of both famines. Li et al. (2010) find a higher risk of metabolic syndrome among those individuals exposed to the famine during fetal life or early childhood, which is aggravated by Western dietary habits or overweight during adulthood. According to Royer and Witman (2013), sex ratio, literacy, employment and marriage rate were also affected in children exposed to the famine around birth.

3.2.2 The Effects of Early Life Stress

Stress is a concept introduced by Hans Selye, see e.g. Selye (1950). As stress he defines the "interaction between damage and defence", that comes in three stages: alarm, resistance and exhaustion (Selye, 1950). Selye (1950) argues for a general pattern of biological stress reactions to diverse types of stressors. While this "general adaptation syndrome" is useful, unhealthy reactions can develop "as a result of adverse conditioning factors" (Selye, 1950). This broad understanding of stress is picked up by e.g. Birbaumer and Schmidt (2010, pp. 150f.), who describe the human stress reaction on the one hand as – at least in the short run – energy enhancing, but on the other hand potentially – mostly so in the long run – harmful mechanism.
Interestingly, as Selye suggested, different stressors might follow some similar biological mechanisms. Kelly et al. (1997) search for biological markers of chronic stress in a literature review. According to them, the biological adaptations of stress responses are consistent across various stressors. Glycosylated Hemoglobin (GHb) is associated with increased risks of e.g. atherosclerosis and strokes. GHb is found to respond similarly to very different situations of chronic stress as e.g. job dissatisfaction, rotating shifts, noise. Also the immune system, more specifically lymphocyte stimulation, is found to react in a similar way to as different sources of chronic stress as prospective bereavement, cancer disease of spouse, examinations in medical school, simulated space flight, unemployment (threat), or caring for a demented spouse. Based on these insights of Kelly et al. (1997), the exact cause or source of the elevated stress level may presumably be of minor importance in an analysis of stress effects.

Medical as well as economic studies have analysed short-, medium-, and long-run consequences of stress exposure early in life. For instance, birth outcomes have been found to be affected by large-scale man-made disruptions and natural disasters. E.g. Xiong et al. (2008) and Lederman et al. (2004) find, among other results, birth weight to be negatively associated to hurricane Katrina and the terrorist attack on September 11 of 2001, respectively. Another example is Maric et al. (2010). They show that pregnant women living in Belgrade during the 1999 bombings gave birth to significantly lighter babies, whereas no effects are found on the probability for giving pre-term birth or other complications during pregnancy. The latter findings are corroborated by the extensive literature review in Harville et al. (2010) who find that fetal growth may be reduced by stressful events but that length of gestation is typically not.

Cortisol is often considered as a biological marker for stress levels. High cortisol levels have also been found to reduce birth weight and body length at birth (e.g. Bolten et al., 2011). Bolten et al. (2011) do not find a significant re-

lation to the mother's perceived stress and emphasize contradictory findings in the existing literature.[4]

Beyond birth outcomes, stress can also have consequences for the functioning and development of the brain and thereby affect cognitive, behavioural, and emotional outcomes. Lupien et al. (2009) give a comprehensive overview of animal and human studies and provide theoretical explanations (see also Talge et al., 2007).[5]
Further evidence pointing in this direction is given in e.g. Aizer et al. (2016) and Persson and Rossin-Slater (2016). Aizer et al. (2016) find negative effects of elevated maternal cortisol levels during pregnancy on children's cognitive and educational outcomes. Persson and Rossin-Slater (2016) find detrimental effects of prenatal bereavement on birth outcomes. Moreover, Persson and Rossin-Slater (2016) find positive effects on take-up of medication against ADHD, depression, and anxiety later in life. Black et al. (2016) find only small negative birth outcome effects of stress caused by bereavement during pregnancy. In contrast to Aizer et al. (2016), outcomes later in life, such as education, cognition scores or earnings, are not found to be affected.
Moreover, several studies find an association with schizophrenia. As an example, Khashan et al. (2008) show that schizophrenia risk is increased in offspring born to mothers who experienced the death of a close relative. Also considering schizophrenia, van Os and Selten (1998) and Malaspina et al. (2008) show that this condition is more prevalent in individuals who were *in utero* during the German invasion of the Netherlands in May 1940 or during the 1967 Arab-Israeli war, respectively.
Bolten et al. (2011) state that potential mechanisms in the relations between stress *in utero* and birth outcomes are not fully understood for the case of humans. They point at Wadhwa's (2005) proposed mechanisms for linking maternal stress and the unborn child, namely "trans-placental transport of maternal hormones, reduction in intrauterine blood flow, and stress-induced suppression of maternal immune functions" (Bolten et al., 2011).

More generally, stress exposure *in utero* may lead to a 'predictive adaptive response' of the fetus' metabolism to maternal stress (i.e. an adaptation to an anticipated stressful or dangerous environment, see e.g. Gluckman et al., 2005; Gluckman and Hanson, 2005; Talge et al., 2007)[6].
To put it in a nutshell, both malnutrition and stress exposure around birth might lead to an adaptation process altering long run physiological features. Yet, a consensus on the specific mechanisms seems not yet fully reached.

[4] We refer the reader to Bolten et al. (2011) and references therein for a detailed overview and discussion.

[5] For details, we refer the reader to the reviews of Lupien et al. (2009) and Talge et al. (2007), as well as the studies cited therein.

[6] More precisely, Gluckman et al. (2005) define 'predictive adaptive response' as "a form of developmental plasticity that evolved as adaptive responses to environmental cues acting early in the life cycle, but where the advantage of the induced phenotype is primarily manifest in a later phase of the life cycle".

3.3 Historical Background

3.3.1 Allied Air Raids on Germany

In the following, we briefly sketch the Anglo-American bombing of Germany during WWII to provide some background on the variation in bombing exposure we exploit in our empirical analyses.[7] A more detailed and comprehensive description of Allied bombing in WWII can be found in e.g. Davis (2006).[8]

In May 1940, the British started their offensive against Germany focusing on infrastructure, such as oil and railway targets (see e.g. Davis, 2006, pp. 13f). Later in the war, also area and city bombing emerged. As an example, in October 1940 "the air staff ordered Bomber Command to concentrate on oil and morale – oil when visibility allowed, morale when it did not" (Davis, 2006, p. 16). A few weeks later, the first "city bombing attack" was carried out on December 16, 1940 on Mannheim (Davis, 2006, p. 16). Moreover, in early 1942, area bombing became the British Bomber Command's priority (see e.g. Davis, 2006, p. 27).[9]
Beside changes in focus and targets, the distribution of bomb load dropped over the course of the war is not uniform. As an illustration, the Bomber Command only dropped about 5 percent of their total bomb load between September 1939 and December 1941 (Davis, 2006, p. 26). Moreover, the US additionally started their heavy bomber missions in Europe in the second half of 1942 (see e.g. Davis, 2006, p. 43).[10]
Overall, the bombings on Germany show variations in intensity and focus over the course of the war. After having provided a macro-level perspective on the bombardments, we will put the focus on the civilian life in the next subsection.

[7] As pointed out in the introduction of this paper, our focus on the bombardments on Germany is due to focus of the data we use in our analysis. We note that other countries have been also hit by bombardments during WWII, and that in particular Germany was carrying out heavy bombardments on other countries.

[8] For discussions and assessments of costs and accomplishments of strategic bombing of Germany, we also refer the reader to the literature (see e.g. Werrell, 1986; Kirby and Capey, 1997; Davis, 2006).

[9] It is important to note that the selection of targets seemed not exclusively based on economic importance of a city (see, e.g., Friedrich, 2002 and Vonyó, 2012).

[10] For differences in focus and targets between the bombing forces and over time, we refer the reader to e.g. Davis (2006, pp. 565ff).

3.3.2 Civilian Life in Germany During World War II

For the purposes of the present study it is vital to shed some light on the living conditions of the civilians during the air raids on Germany. In general, bombings impacted on many facets of German civilian lives. Our exposition is mainly focused on three major sources of selection, namely selective survival, internal migration, and health effects of bombings for the survivors.
With regards to the first, it has been established that up to 600,000 people lost their lives in air raids (Werrell, 1986). According to Hewitt (1983), half of those casualties occurred in the four 'firestorms' over Hamburg, Kassel, Darmstadt, and Dresden, the first two taking place at the end of 1943, the latter in late 1944 and early 1945, respectively. Since targeted cities and areas therein were oftentimes (but not exclusively) larger in scale and more industrialized, it is conceivable that a large proportion of the casualties had been of a working-class background. In fact, Hewitt (1983) documents that the British officials explicitly declared working-class neighbourhoods as the number one civilian targets. This could lead to a positively selected sample of survivors in our analysis. Therefore, we control for socio-economic background of the parents in our analyses. Regarding excess child mortality, Enloe (1945, ch. 6) states in the United States Strategic Bombing Survey that there was no evidence for the observed increase to be attributable to bombing attacks. Still, we test robustness by excluding people potentially affected by the four 'firestorms' mentioned above (see Section 3.6.2).
With respect to the second selection issue it must be noted that large scale evacuation programs started in 1943 and a total of about 10 million people were evacuated from cities, see Heineman (1996). In total, more than two million children were evacuated by the *Reichsdienststelle KLV (Kinderlandverschickung)* during the war, about half a million below the age of six, i.e. jointly with their mothers (Kock, 1997, pp. 142f.).[11] We take care of the issue of selective migration in a robustness check (see Section 3.6.2).
The third issue deals with health effects of bombings for the survivors. Judging from historical accounts, the experience of air raids triggered psychological stress. Contemporary Allied observers find increased heart disease mortality rates as well as increases in stress-related diseases as peptic ulcer, cardio-vascular diseases, neurodermatitis, glaucoma, urticaria and excema in cities shortly after air attacks (Enloe 1945, pp. 99–149). Also subjective well-being seems to have suffered already in the first years of the war, since consumption of sleeping pills and pain killers increased substantially as well as medication-induced chronic gastric damage (Süß, 2003, p. 398). Also young children were affected, since mothers suffered psychosomatically especially from the double burden of waged work in arms manufacturing or agriculture and housework and childcare (Süß, 2003, pp. 403).

[11] For details on the evacuation of children (*Kinderlandverschickung*), we refer the reader to Kock (1997).

Apart from stress, however, Allied air attacks do not seem to have specifically affected other health risks for the surviving children – in spite of bombings drastically affecting the civilian life: 800,000 civilians were injured in area bombings, more than 2 million housing units were destroyed, making 7.5 million people homeless (Hewitt, 1983) so that many had to live in crowded emergency accommodations (Süß, 2003, p. 391). Additionally, a major part of medical care infrastructure was destroyed by the air raids (Süß, 2003, p. 391). Yet, all those factors did not lead to major epidemics of infectious diseases (see Enloe, 1945 ch. 4 and Süß, 2003, pp. 391). This does not mean that there was no increase in disease rates at all. Still, in most contemporaneous reports it is explicitly noted that infectious diseases were not directly associated with air raids (see Enloe, 1945 ch. 4).[12]

For our empirical strategy it is most important to note that health quality of toddlers and children did not suffer from adverse conditions directly associated with air raids other than through psychological stress. Namely, the adverse conditions of the war affected age groups very differently (Süß, 2003, p. 399). Whereas health status of adults decreased much earlier during the war, most health offices reported good to satisfying health quality for young children as late as 1943 and even 1944. Special health care facilities and e.g. synthetic vitamins to counteract deficiency symptoms may be responsible for their surprisingly good health (Süß, 2003, p. 400). Correspondingly, infant mortality rose very little until 1943. Only in 1944, it rose substantially to around 10% in big cities (Süß, 2003, pp. 387, 447). As noted above, this late increase cannot be attributed to air attacks alone, but rather to the cumulative adverse conditions (see Enloe, 1945 p. 126).

3.3.3 Nutrition in Germany around World War II

In this section, we briefly summarize the food situation in Germany around the Second World War. For more detailed descriptions, see also e.g. Klatt (1950), Farquharson (1985), Trittel (1990) or Van den Berg et al. (2016).
In August 1939, the German government installed a food rationing system which determined the maximum amount of food each person was eligible for (see e.g. Rothenberger, 1980, p. 59). Consumers were differentiated by intensity of physical work (see Rothenberger, 1980, p. 33), e.g. coal miners earned extra supplements, but also children got a specific composition of

[12] For instance, diphtheria, a disease mostly prevalent in children, was endemic already before the war and only exhibits a slight increase in the infection rate during the war. Apparently, most of this surge in diphtheria cases can be attributed to excess adult cases (Galazka and Dittmann, 2000). After declining between 1932 and 1938, also tuberculosis incidence rose again from 1940 onwards (Süß, 2003, pp. 382). Here, similarly, mostly young men were affected, and the rise in incidence rates was most pronounced in rural regions, which were less affected by air raids than cities.

food (see Buchheim, 2010). Before food rationing started, nutritional quality in Germany was high (see e.g. Rothenberger, 1980, p. 34). Authorities were able to hold it constant during the first war years at a sufficient level (e.g. see Rothenberger, 1980, p. 32; Schmitz, 1956).
In 1942 the food supply became slightly more difficult, until the harvest of 1943 brought a relaxation of the nutritional situation again (see Rothenberger, 1980, p. 34). In 1943 and 1944, the bombing of German cities and evacuation programs became the major challenges. Yet, the nutritional situation altogether was still not critical (see Rothenberger, 1980, p. 34). Food rations in 1943/44 amounted to about 2,500 kcal per day and capita (see Klatt, 1950, p. 46). In the beginning of 1945, food transports via train were largely impossible, the Allied forces increasingly occupied agricultural areas and nutrition became a major problem for the civilian society (see Schmitz, 1956, pp. 357f.).

By the time it surrendered to the Allied Forces, Germany had come to the end of its economic capabilities. Cities and infrastructure had been destroyed by bombardments (see, e.g. Van den Berg et al., 2016). Additionally, much of the Eastern part of the Reich was lost to the Soviet forces which implied great losses in agricultural capacities and a huge inflow of refugees into the newly established occupation zones (see, e.g., Trittel, 1990, p. 21 and Van den Berg et al., 2016). Beside transportation means, also qualified labour (see Van den Berg et al., 2016) and fertilizer (see Rothenberger, 1980) were missing for a sufficient food supply and distribution. The centrally organized food administration system was set inactive directly after the war (see Trittel, 1990, p. 28). In the following weeks, local authorities had to run the ration system independently, and throughout the famine imports from the Allies were often insufficient (see, e.g., Klatt, 1950, p. 48 and Trittel, 1990, pp. 159, 161). However, it is important to note that malnutrition was a severe problem throughout Europe after the war, so low food rations in Germany should not unanimously be interpreted as punishment or exploitation by the Allied powers (see Rothenberger, 1980, p. 36f and Klatt, 1950, p. 47 in contrast to Abelshauser, 1983, pp. 117–119). On the contrary, the Allied forces deemed the nutritional situation as essential to avoid a catastrophe, be it riots or epidemics (see Manz, 1985, p. 73 and Rothenberger, 1980, p. 92).
Nonetheless, hunger and starvation became a part of German day-to-day living. At some stages of the famine, people had to live off rations around 1,500 kcal per day and person or even less (see Cornides, 1948). Due to adverse weather conditions causing bad harvests, deficits in transportation infrastructure and a low food production worldwide the situation only improved from spring 1948 onwards (Schmitz, 1956, pp. 365–368). Especially during the harsh winter of 1946/47, the so-called 'hunger winter', the food situation deteriorated to catastrophic levels (see Van den Berg et al., 2016). Since also the crop of 1947 largely failed to meet the needs of the increasingly starving

population, rations dropped even below 1,000 kcal in some parts of Germany (see Klatt, 1950, p. 49).

Rations, however, differed across zones, since each occupational power had its own regime and its own food administration (see, e.g., Van den Berg et al., 2016). Although US and UK zone united their food administration quite quickly in late 1946 (see, e.g., Trittel, 1990, p. 85), the French and the Soviet zone had completely independent systems until the end of the famine (see e.g. Boldorf, 1998, pp. 78–82 and Rothenberger, 1980, pp. 97ff.). But also within zones, food availability varied depending e.g. on the share of agriculture in the region, which led to black market activities (see e.g. Bignon, 2007). Although the rationing system was still, at least partially, in place until April 1950 in the Western zones and even longer in the Soviet zone, nutritional quality improved a lot around the currency reform in the Federal Republic of Germany in June 1948. This can be seen in the rations data but also in contemporaneous literature for all four zones (see Klatt, 1950, p. 57), especially so for the three Western zones (see e.g. Boldorf, 1998, pp. 80–82). The currency reform is one factor that was responsible for this improvement (making it more beneficial to sell agricultural products on official instead of black markets or keeping it in stock). Other reasons given in the literature are a surprisingly good harvest in 1948 worldwide and the Marshall Plan (starting in early 1948) providing high quality food imports (see Klatt, 1950, p. 56 and Rothenberger, 1980, p. 210f.). From July 1948 onwards, the official calorie level was at an average of 1,800 kcal (see Klatt, 1950, p. 56). However, individual spending capacities increasingly determined real caloric intakes (Schmitz, 1956, p. 370), as single food types were excluded from the food ration cards in the three Western zones starting already in July 1948 (see Schmitz, 1956, p. 372 and Rothenberger, 1980, p. 59).

3.3.4 Effects of World War II and the Post-War Famine

The short-run effects of warfare on the German civilian population as surveyed by different contemporary observers have been reviewed in Section 3.3.2. Other empirical studies consider the long-run effects of WWII or similar stressful events.

Individuals who were affected by WWII are found to exhibit worse economic and health outcomes. As an example, Akbulut-Yuksel (2014) shows that war time destruction has negative effects on a broad range of outcomes (educational attainment, health, labour market earnings) of school-aged individuals during the war.[13] According to Akbulut-Yuksel, the impact on educational attainment points to school destruction and school access as important

[13] See also Ichino and Winter-Ebmer (2004) for additional evidence on the effects of WWII on educational attainment.

mechanisms. The effects on health may hint at other channels, such as malnutrition, as well. Hence, the reduced form effects measured in that paper cannot be straightforwardly interpreted since effects of war time destruction on health can be triggered by direct experience of heavy air raids or more detrimental living conditions after the war.
Another example is Kesternich et al. (2014). In their study of the effects of WWII on health outcomes and socio-economic measures, Kesternich et al. (2014) advance on this issue by using retrospective individual level reports on war time suffering and thus providing evidence on the channels via which war exposure might affect long-run outcomes.[14] In general both studies show that World War II may have evoked a whole variety of adverse factors, ranging from psychological stress to nutritional deprivation, which may all have long-run consequences and can hardly be disentangled.

In psychology, there is also a strand of literature analysing the effects of WWII trauma. For instance, Glaesmer (2013) provides a recent overview on long-run psychological effects in a German sample. She also points to a recent interest in the effects of trauma on physical health. As an example, Glaesmer et al. (2011) analyse the relationship between post-traumatic stress disorder (PTSD), traumatic events and physical health measures in a sample of elderly Germans. They find a significant relationship between health and PTSD/trauma (war-related as well as civilian). Their findings suggest a significant correlation between traumatic experiences and all 20 medical conditions under study (including hypertension), whereas 12 of 20 outcomes are also significantly related to PTSD.

The post-war famine in Germany 1945–1948 and its long-term effects have been analysed already in other studies, as pointed out in Section 3.2.1. Contemporary public health reports document that nutritional deprivation had a profound immediate and midterm impact on the German population: children as well as newborns were smaller and lighter than normal and suffered from a higher risk of infections and infant mortality (Klatt, 1950; Dean, 1951; Droese and Rominger, 1950), adults were underweight (Report of the Military Governor No. 49, 1949) and showed symptoms of psychological stress (Davis, 1951).

[14] In the context of the effects of famine on long-run health, Van den Berg et al. (2016) stress the non-causality of reduced form effects of famine exposure and use individual level reports of nutritional deprivation in childhood in order to measure a causal local average treatment effect.

3.4 Data

3.4.1 Micro Data

Our analysis is based on the Socio-economic Panel SOEP (2013, version 29) and on the information on the place of birth in the SOEP (municipality level) that was asked for the first time in 2012. In addition, we use supplementary information from the special SOEP module *Frühe Kindheit im (Nach-) Kriegskontext* (*FKM*) that deals with the early life circumstances of respondents born 1935 to 1950. This module is only used in our robustness checks regarding relocations early in life.[15] [16]
Our analysis is restricted to individuals born between 1930 and 1950 within today's borders of Germany since we have full contextual information only for birth places in Germany. Using the place of birth enables us to merge in contextual information about exposure to air raids and to famine around birth.

Our outcome variables refer to three different domains, all measured late in life: Physical health (height, hypertension), economic productivity (proxied by pensions), as well as a general measure of overall well-being (life satisfaction).
Height has been indicated to be "perhaps the single best indicator of nutritional and disease environment in childhood" (Elo and Preston, 1992) and is therefore of particular interest when studying the influence of two distinct early life adversities (stress and malnutrition). Hypertension is interesting because it is very prevalent in Germany and bears a substantial mortality risk (see e.g. Prugger et al., 2006). In the SOEP, data on health conditions like hypertension are collected by asking whether a doctor ever diagnosed a respondent with the respective disease.[17]
Second, as an economic outcome variable we use monthly pensions (in Euro) for men. We use pensions to be a proxy for overall life-time productivity. Since the entire economic system was different in East Germany until reunification, we exclude respondents born in East Germany when analysing pensions. We also drop respondents born after 1948 when analyzing this variable because they are comparatively young and thus a relatively large

[15] For more information on this module, see e.g. the description provided in the appendix of Van den Berg et al. (2014a).

[16] The data of the German Socio-Economic Panel Study (SOEP), including data on birth places and the supplementary module, are provided by the German Institute for Economic Research (DIW), Berlin. See Wagner et al. (2007) for details on the SOEP. Concerning data quality and attrition issues of the SOEP, see, e.g., Kroh (2013), Lipps (2009).

[17] Note that actual prevalence might be higher than what we see in the survey data because some individuals might not have been diagnosed despite suffering from high blood pressure. Prugger et al. (2006) point at worse awareness of hypertension in Germany compared to other countries.

Table 3.1: Descriptive statistics by gender

	Men			Women		
Variable	N	Mean	s.d.	N	Mean	s.d.
Hypertension (1/0)	1850	0.458	0.498	1889	0.429	0.495
Body height (cm)	1862	176.012	6.430	1904	163.476	5.852
Log(pension)*	1059	7.455	0.585	–	–	–
Life satisfaction	1859	7.207	1.700	1903	7.075	1.761
Less than 1500 kcal, in utero	1863	0.124	0.330	1905	0.128	0.334
Less than 1500 kcal, post-birth	1863	0.114	0.318	1905	0.117	0.322
Bombed in utero (1/0)	1863	0.052	0.222	1905	0.063	0.243
Bombs per cap. (lbs.) in utero, if > 0	97	6.370	12.673	120	11.864	29.448
Bombed post-birth (1/0)	1863	0.060	0.237	1905	0.066	0.249
Bombs per cap. (lbs.) post-birth, if > 0	111	15.507	54.087	126	9.624	19.063
Year of birth	1863	1941.566	5.704	1905	1941.767	5.639
Father's education high (1/0)	1863	0.216	0.412	1905	0.195	0.396
Place of birth: pop. $\geq$ 10,000	1863	0.585	0.493	1905	0.566	0.496
Bizone	1863	0.595	0.491	1905	0.553	0.497

Note: log(pension) only contains men born before 1947 in West Germany. Famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life.

fraction might not be retired yet. Since labour force participation of women increased tremendously in very recent decades in Germany (see e.g. Brenke, 2015), we further exclude women from our pension analysis.

Our third outcome category refers to overall well-being, as measured by self reported life satisfaction (on a scale from 0 to 10). The personal judgement of life satisfaction allows us a universal assessment of the individuals' overall subjective state. Keeping only individuals with valid information on the place (local identifier, *Gemeindekennziffer*, *GKZ*)[18] and month of birth, who were born between 1930 and 1950, we end up with a raw sample of 4140 individuals, of which 2103 are women and 2037 are men. Table 3.1 shows summary statistics on our main variables in our analysis sample. The slight drop in sample size is due to missings in some of the covariates we need for our analyses.

3.4.2 Contextual Variation: Air Raids on Germany

We supplement the micro-data set with data on air raids on Germany. The data accompany the detailed description on Allied air raids by Davis (2006). They provide detailed information on the Anglo-American strategic bombing operations in Europe, including dates, names of the cities targeted, and

[18]For some respondents the place of birth was ambiguous due to multiple cities having the same name. In this case, we assumed they were born in the largest of the candidate cities (according to population size in 2011, Statistische Ämter des Bundes und der Länder, 2014). A substantial fraction of those for whom we do not have a *GKZ* were actually born outside of todays' borders of Germany.

the tons of bombs dropped (short tons, thus a ton corresponds to 2000 lbs.).[19] For early attacks of the Royal Air Force on German facilities, the data are only crude in the sense that they do not provide information on the exact target or the exact date. Moreover, Davis (2006) points at an inferior data quality regarding early bombardments until and including 1941. As a consequence, we expect some measurement error due to these data constraints. However, as outlined earlier, bombardments became more frequent and heavier over the course of the war, in particular after 1941. Thus, we infer that incomplete information on early attacks should not affect our results very strongly.

The most important task is to identify the targeted cities and merge in the regional identifier information in order to identify survey respondents as being born in an air raid stricken city(*GKZ*, Statistische Ämter des Bundes und der Länder, 2013). This task is complicated by e.g. wrong spelling of city names in the bombings data, multiple cities having the same name, reforms that merged different localities into one, and some target names not being municipalities on their own but specific parts therein. For unmatched targets that have been bombed with a sufficiently large bomb load, we went manually through the data set and took care of them receiving the correct *GKZ*. Moreover, in case of multiple cities having the same name, we implement a routine that chooses the *GKZ* of the largest municipality (according to population size in 2012, Statistische Ämter des Bundes und der Länder, 2013).

In total, we start with data on 7261 bombing operations (observations) in Germany, whereof we immediately drop those with missing municipality name (15) and 407 observations due to missing or zero tons of bombs dropped. Moreover, we drop 19 observations because they were mining operations. Another 363 observations are dropped because the target is either not in Germany or the municipality is not exactly specified. Of the remaining 6457 observations, 467 could not be matched with a *GKZ*. Eventually, we thus succeed in matching 5990 bombings to a *GKZ*. The unmatched bombings (467) are on average much smaller than matched bombings in terms of tons of bombs dropped. Targets therein were never hit by bombings exceeding 200 tons or their name cannot be linked to a German municipality.
We aggregate the tons of bombs dropped on a municipality in a given month

[19] Davis (2006) covers in particular "bombing sorties, mining, supply missions, and special operations of all two and four-engine bombers of the RAF Bomber Command in Great Britain and the RAF 205 Group in the Mediterranean as well as all four-engine bomber (B-17 and B-24) operations of the US Eighth Air Force in Great Britain and the US Ninth, Twelfth, and Fifteenth Forces in the Mediterranean. The database contains such entries as date, total bomb load [...] for virtually every aircraft sortie credited with attacking a strategic target in Europe." (Davis, 2006, pp. xii–xiii). For more details on the data and the primary sources used by Davis, we refer the reader to Davis (2006). Unless noted differently, we stick to the observational unit as provided in the database of Davis (2006) in this section.

which will provide us with our main contextual variation. For our individual-level analyses, we scale bomb tonnage by the 1939 population size of a municipality as measured in the 1939 census (see Zeitler and Mewes, 1941). This information is available only for municipalities above 10,000 inhabitants.[20] For individuals born in municipalities for which we do not have the population size, we assume that they have not been exposed to bombardments. Hence, in a small number of cases we declare individuals who lived in a bombed municipality in one of our critical periods as not exposed to bombings because they were born in a small municipality with a number of residents below 10,000. We notice that this renders our results as conservative.

Figure 3.2 gives an overview of the geographical variation in our data, aggregated over the entire war time. For this graph we aggregate the data to the district (*(Land-)Kreis*) level, which is a coarser level than we originally have.[21] Figure 3.3 abstracts from the overall magnitude of bombs dropped and depicts the number of attacks per region.[22] Both maps show that the air raids exhibited geographical dispersion. Yet, they were mostly concentrated in some major areas, such as Berlin and the Ruhr area. Moreover, southern parts of Germany were less affected. Figure 3.1 provides some insight in the time variation in our data. The figure depicts the time pattern of bombardments aggregated over German municipalities. The basis is again the sample of bombardments that was successfully matched to a *GKZ*.[23] The figure shows an increase in bomb load and frequency over time until the end of the war. Figure 3.4 shows the variation in the data when merged into the analysis sample of SOEP respondents (here: male and female, cohorts 1939-1946 only). The figure shows that the majority of the sample has not been exposed to bombings during the age windows of interest. Pre- and post-birth bombing exposure increases over the course of the war.

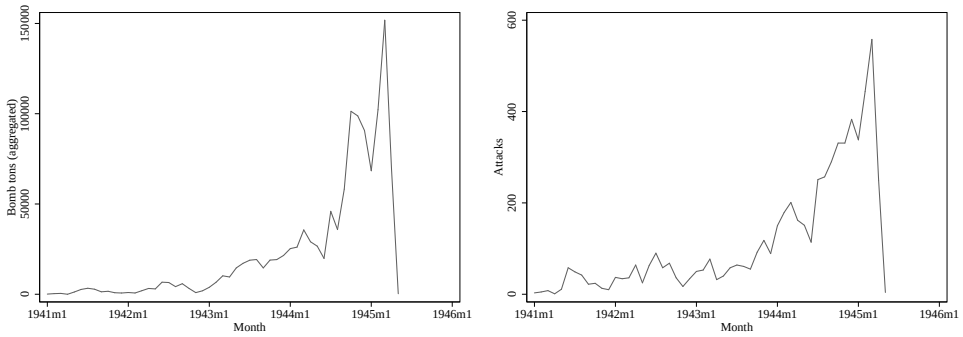
[20] This information had to be digitized first. Some cities could originally not be matched with their current local identifier (Statistische Ämter des Bundes und der Länder, 2013) because of e.g. changing city names over time, cities being merged, abbreviations. In these cases, the attempt was to find the current official name manually by online search (e.g. using Statistische Ämter des Bundes und der Länder, 2014) to enable a merge with their current local identifier. We expect some measurement error here (some identifiers having the wrong population size). For municipalities that were merged into one, we added up the population size of all entities subsumed therein for which we have information on the number of inhabitants.

[21] Note, we do not scale the overall bomb load by population size in Figure 3.2.

[22] Note, we define the number of attacks on a district as the number of observations in our (successfully *GKZ* matched) bombings database for the respective district.

[23] Note, we define the number of attacks in a month as the number of observations in our (successfully *GKZ* matched) bombings database for the respective month.

Figure 3.1: Time variation in bombardments



(a) Tons of bombs over time

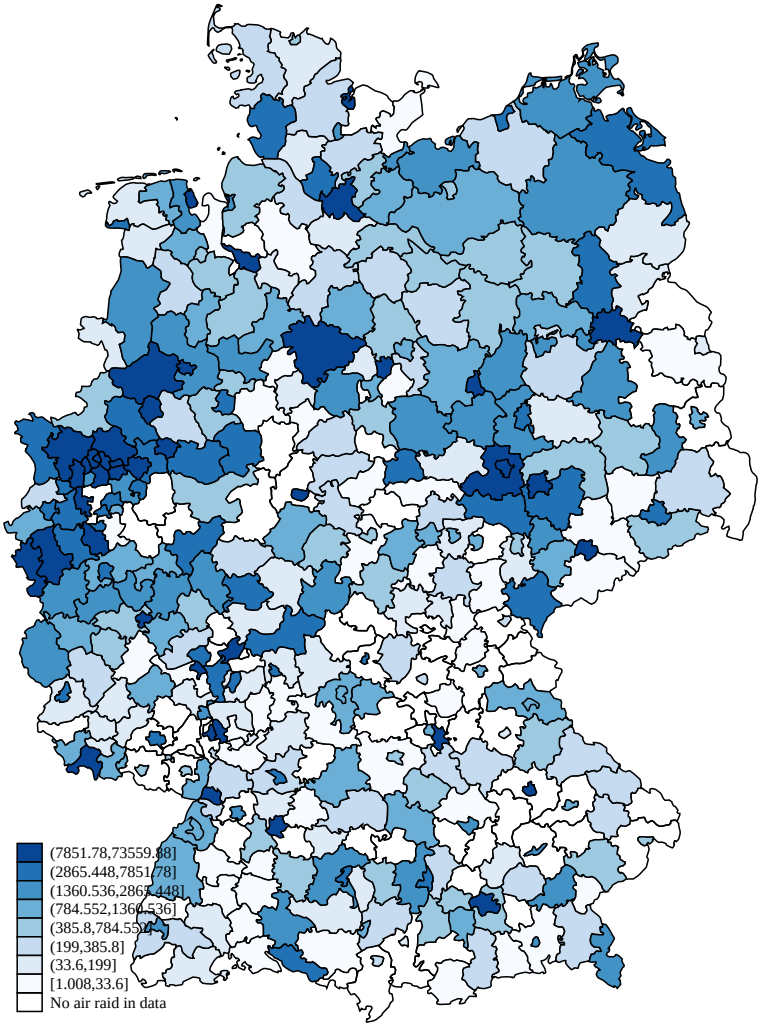
(b) Number of attacks over time

3.4.3 Contextual Variation: Regional-Level Data on Food Rations

Judging from the earlier mentioned historical accounts, the food situation can be declared as non-critical until the last months before the Nazi regime collapsed and again after the currency reform in June 1948 (see Section 3.3.3). We define a 'normal' food situation to exhibit a baseline caloric level of 2,500 kcal. To account for the deterioration in the food situation in the last months of the war, we set the ration level to 2,000 kcal for January 1945 to May 1945. In order to depict the nutritional situation in post-war Germany during the famine in June 1945 to June 1948, we use information on official food rations for the so-called normal consumer or equivalents from different historical sources. Focusing on the official normal consumer ration has the advantage that it is most widely available and that it is best comparable over space and time.[24]

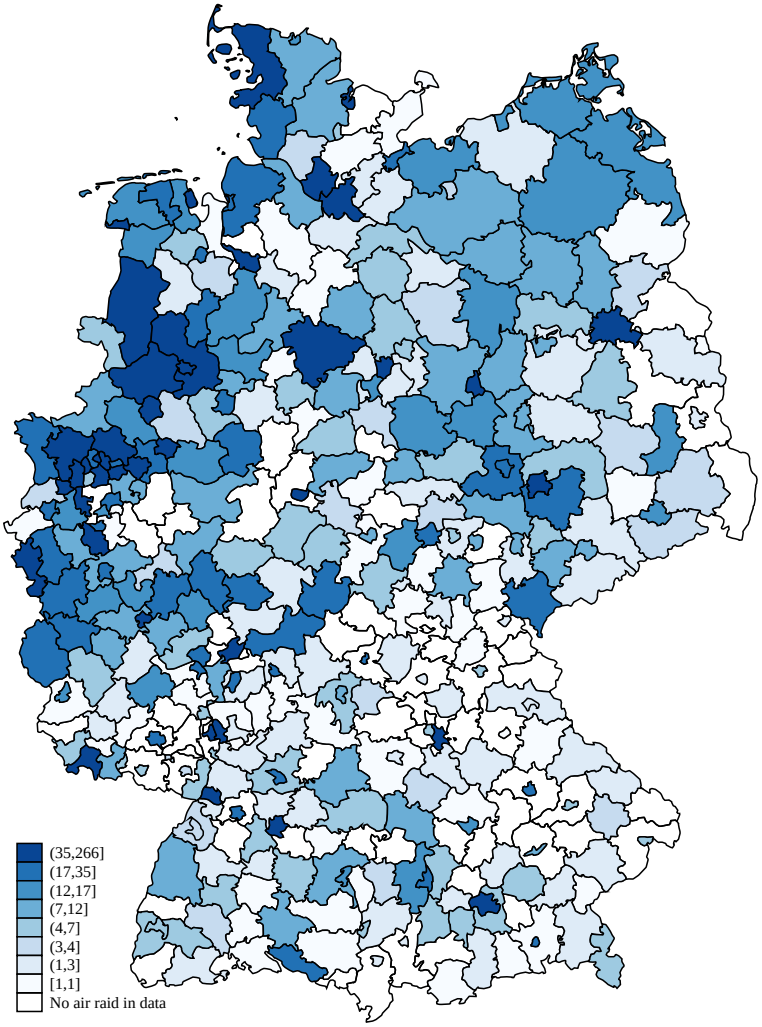
[24] To operationalize and homogenize all our information, we adopt the following conventions: (a) In US and UK zone, food rationing cards were distributed every four weeks (*Zuteilungsperiode*, abbreviated *ZP*, ration period) until December 1947, when the system changed to calendar months. If in the early rationing system, two separate rationing periods followed one another starting within the same calendar month and zone, we consider only the one covering most of the month indicated. (b) We distinguish between called-up food rations and official food rations as well as between zone-level data that gives rations for zones as whole and city-level data which refers to the specific situation in a given city. The latter does not follow from official ration policies, but from the worse nutritional situation in cities in reality. Although cities officially might have received at least as high a ration as rural areas, the data and historical accounts show an enormous penalty in terms of daily calories (due to e.g. transport deficits). We imitate this fact by adopting a so-called 'city penalty'. (c) Since some food rations are given in daily calories, whereas others describe the composition of bread, potatoes, meat etc., we standardize the latter using a contemporaneous calorie calculation table (see Schmitz, 1956, p. 469f.). The official calculation method in the Bizone changed between ration periods 100 and 101. We stick to the early system.

Figure 3.2: Tons of bombs by region



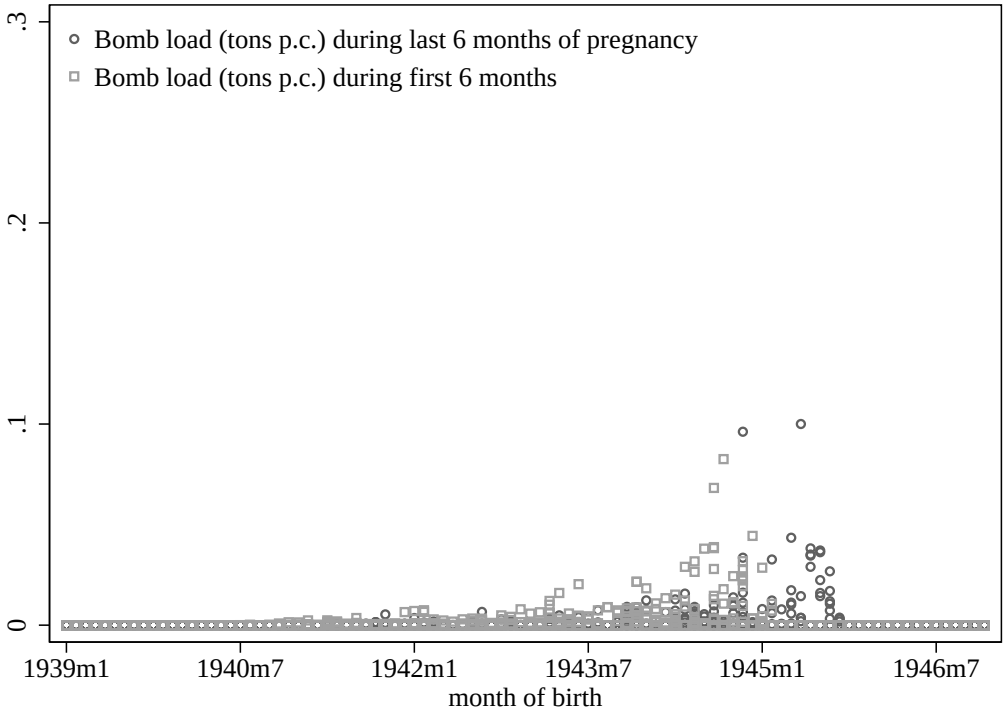
Source: own calculations, shape file: ©GeoBasis-DE / BKG 2013.

Figure 3.3: Number of attacks by region

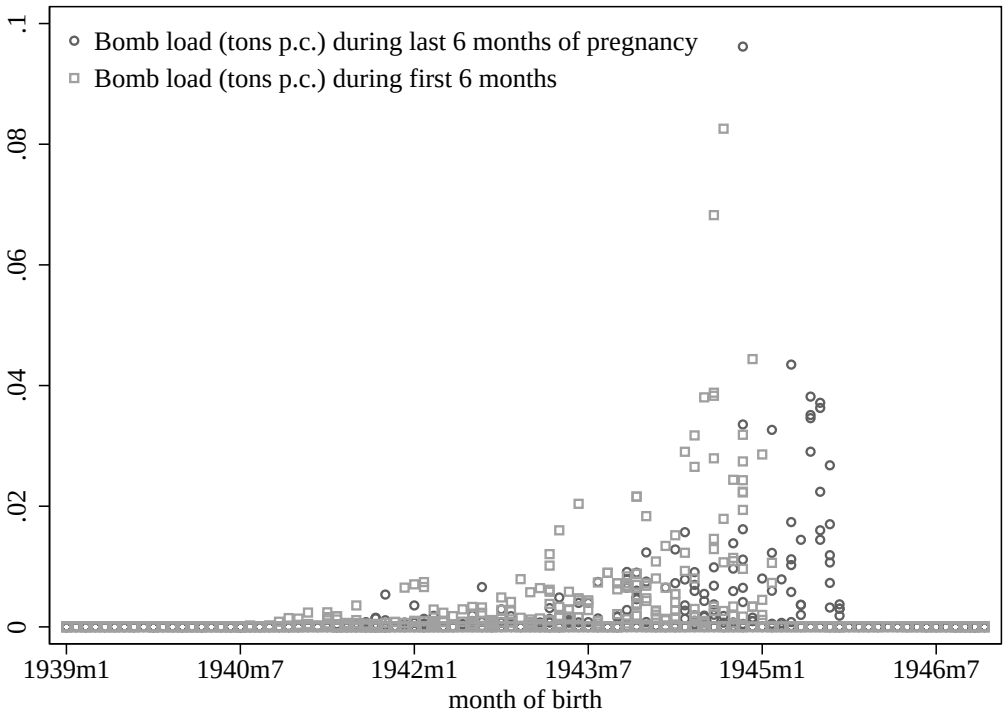


Source: own calculations, shape file: ©GeoBasis-DE / BKG 2013.

Figure 3.4: Contextual exposure of SOEP respondents to air raids



(a) All born 1939-1946 (male + female)



(b) Without large outliers (1 person $>$ .1 tons bomb load)

Using our raw data we have to tackle three challenges: First, ration time series are almost never complete. Second, the 'normal consumer' ration is oftentimes not an exact measure of the de facto food availability. Third, historical accounts show that there was variation in food availability also within zones, namely between urban and rural areas (see Section 3.3.3 on the historical background).
We address all of these challenges by applying an imputation model that exploits not only (a) official zone-level ration data for the normal consumer, but also (b) official zone-level data on actual 'call-ups', i.e. the rations that were de facto distributed, and (c) city-level rations from different sources which we also interpret to be call-up data rather than official rations.[25] We use all these data to estimate a regression model that explains variation in rations with a function of time (season and year fixed effects) and intercepts for specific data characteristics (a baseline effect for call-up-data and a baseline effect for city-level data). Moreover, we include occupational zone fixed effects. The function in time allows us to inter- and extrapolate food rations for each data source type. Furthermore, notice that an average 'call-up effect' can be identified as the average difference between the official food rations and the call-up data. The 'city effect' (recall that we define all city data to be call-up data) can be identified as the average difference between the official rations and the city data net of the already computed average call-up effect. Table 3.2 gives an overview of the imputation model.

Table 3.2: Imputation model for predicting food rations

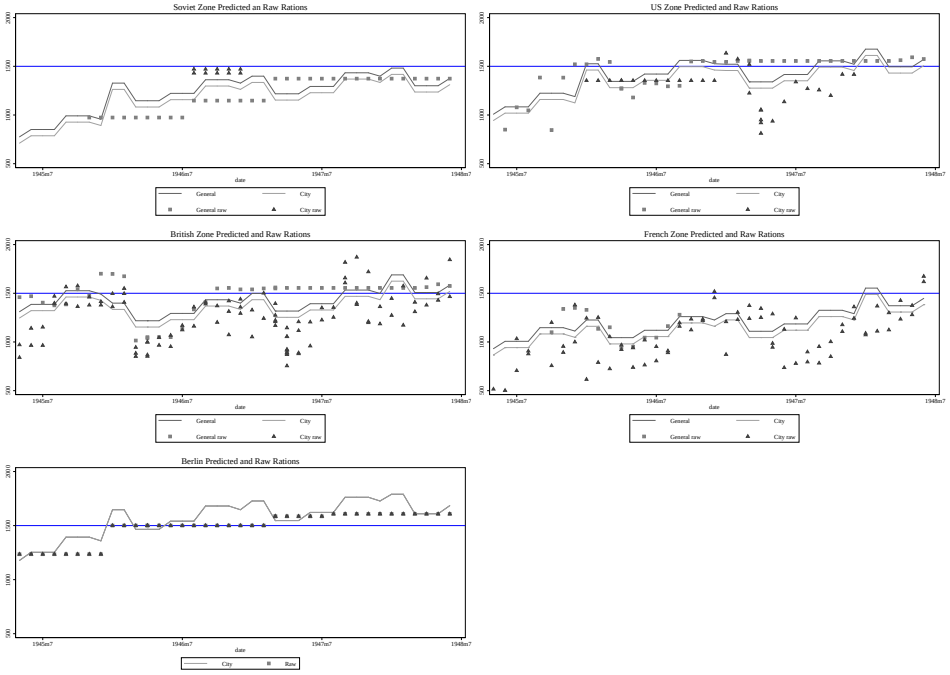
	Coef.	Std.Err.	t	p
City data (1/0)	-64.46543	28.04342	-2.298772	.0220213
Call-up data (1/0)	-74.39558	29.7957	-2.496856	.0129224
US occupational area (1/0)	-234.092	93.13908	-2.51336	.0123422
British occupational area (1/0)	68.15808	71.21363	.9570932	.3390856
French occupational area (1/0)	-311.1979	76.50223	-4.067828	.0000569
Soviet occupational area (1/0)	-466.7539	140.0921	-3.331764	.0009414
Spring season	-181.0715	23.36267	-7.750466	7.32e-14
Summer season	-105.5683	25.12212	-4.202207	.0000325
Autumn season	34.57961	25.6841	1.346343	.1789376
Year = 1946	287.8416	78.52913	3.665412	.0002795
Year = 1947	368.7339	78.52913	4.695505	3.64e-06
Year = 1948	431.4758	94.78269	4.552263	7.01e-06
R^2	0.530			
F-Stat.	19.187			
N	434			

Note: model also includes zone × year fixed effects (omitted for brevity).

Using this imputation we construct two time series per occupational zone. First, we compute a complete time series of 'official' food rations which we attribute to the entire zone except urban areas. Second, by removing the call-up effect from the city-level series predicted by the imputation model, we

[25] The rationale behind this is that the data on city-level fluctuate a lot between single ration periods and across cities within a zone which is indicative of those numbers to be call-ups.

Figure 3.5: Imputed and original rations by occupation zone



deduce a series of food rations we attribute to the urban areas. This series can be regarded as an official city ration since it is net of a call up effect but still accounting for a city-penalty inherent in our data. This makes the city ration time series best comparable to the area ration time series, while reflecting the de facto worse nutritional supply in urban areas which is not visible in the nominal/official ration data. Figure 3.5 shows the resulting data and our identifying variation. Since historical accounts, however, note substantial differences between US and UK zone with respect to the food situation in early 1948, with the US zone having more generous food rations than what our imputed rations show for March to May 1948, we improve the fit of our imputed rations data to the raw data for this short time window by slightly increasing the ration amount here to 1500 kcal.

Keeping the broad idea in mind of how to align all possible types of data on food rations, we now turn to our sources for area- and city-level ration data.

British and US Zone (*BBZ* and *USBZ*)

The Report of the Military Governor No. 39 (1948) (OMGUS) describes the nutritional quality in US and UK zone, and in the Bi-zonal area after unifi-

cation. It is the only official source available for the individual nutritional situation. Beside the official rations, also call-up data are given for parallel time spans. This is essential since food shortages in the Bi-zonal area made it impossible to maintain the official ration level from March 1947 onwards (ration periods 99 to 112). As described above, we use this information on both call-ups and official food rations to infer the average difference between them. Thereby, we can also isolate the 'city effect' when using call-up data.

To extend the time series to periods before March 1946, we refer to Schlange-Schöningen (1955). This source yields monthly averages of daily ration caloric values from April 1945 to December 1949 for Schleswig-Holstein, Essen, British zone, and American zone. Due to slightly different definitions, however, the time series are only partly comparable especially in the very first time after the breakdown. Additional imprecision might occur due to regional differences in the food quality as Schlange-Schöningen (1955) mentions. We assume that these differences are small enough not to affect our analyses.

French zone (*FBZ*)

The French military government for Germany followed a completely different strategy with respect to the German economy than US and UK (see e.g. Rothenberger, 1980 p. 93f.). Collaboration between the three powers was thus very difficult in the first years after 1945, and hence also the nutritional situation of the German population was highly unequal (see e.g. Henke, 1982, p. 519). For the food rations in the French zone, we refer to Manz (1985). The nutrition data therein stem from the "Bulletin Statistique, No. 2 - No. 9" and covers October 1945 to December 1948. Since October 1946, the official food rations were dependent on municipality size. Since Manz (1985) unfortunately only covers ration data for municipalities with less than 20,000 residents from then on, we can only make use of the data until September 1946.

Soviet zone (*SBZ*)

To the best of our knowledge, the most comprehensive source for nutrition in the Soviet zone is Boldorf (1998), who gives detailed information on the living standard in the soviet occupational zone, later GDR. He describes the official food rations between November 1, 1945 and May 28, 1958. The category of a 'normal consumer' of the Western zones did not exist in the Soviet zone. Instead, the food rationing level discriminated between six, later

five ration card groups, depending on occupation and eligibility of the single person.[26] Also the city size mattered for the food rationing level (card categories): official rations were lower in smaller municipalities due to the supposedly easier access to additional resources and higher ability to at least partly provide for their own needs. We build a weighted average over the ration level of both card categories to reflect the average nutritional situation in the Soviet zone. For this purpose, we use population numbers receiving the two card categories in 1947/48 as given in Boldorf (1998, p. 70). According to Boldorf (1998, p. 76), the population in small cities or rural regions, which did not have regular access to subsistence food production, was hit hardest by the famine. Other channels of food supply were not accessible for most consumers, also due to extremely high prices. For these two reasons, we think that the food ration level gives a quite accurate picture of the average consumption level. Still, there were periods of crisis until 1949, when food ration cards could not be fully called up.

Cities

We use information on the call-up of the official rations in several cities (i.e. with how much food the urban population was actually supplied). Officially, average food rations in the cities might have been as high or even higher as in the countryside, due to the composition of the population: the special needs of industrial workers, who were more frequent in urban areas, the low share of people growing their own food. In the French and the Soviet zone, sometimes also the official rations for the normal consumer were higher than in rural areas, reflecting the potentially long distance to the next farmer. As described in our review of the historical background before, however, the shortfall in food to be transported to the cities led to a much worse realized food supply in the cities (see Section 3.3.3). We take this shortfall into account, irrespective of the different official situation, to draw a complete picture of relative nutritional quality in Germany.
The ration data we use to identify the city effect are taken from several sources. First and foremost, we have ration data for Berlin from the Hauptamt für Statistik Berlin-Wilmersdorf (1947, p. 236). Schmitz (1956) gives a very detailed picture on the technical and practical consequences of rationing food and other consumer items for the example of the city of Essen. He collected complete time series data on rations in Essen and Düsseldorf and single data points for Hamburg, Munich, Nuremberg, Frankfurt am Main, Kiel,

[26] On the basis of Boldorf (1998, p. 63, 71), we select card type IV as the best proxy for a 'normal consumer' after February 1947, because in 1947/1948 almost half of the rations cards belonged to this type. For the ration periods before, we refer to card type VI as 'normal consumer'.

Stuttgart, Wuppertal, Münster, and Cologne (Schmitz, 1956, p. 469). Nutrition in Hamburg is also described by Boldorf (1998, p. 78). Seel (1950, p. 22) depicts the food situation at the level of single cities of the British zone, i.e. Bochum, Duisburg, Essen, and Remscheid. Stadtverwaltung Mannheim, Statistisches Amt (1950) gives ration data for the city of Mannheim in 1947. As a last source for cities in the Bizone, we consult Martini (1947, p. 31) who shows ration data for Bonn. For the French zone, city data stem from Trier, Landau, and Mainz, with the former two described in Rothenberger (1980, p. 104-188), and the latter in Schmitz (1956, p. 469). Finally, ration data on cities in the Soviet zone are taken from Liebe (1947, p.75) and describe the situation in Leipzig and Dresden as well as in the rest of *SBZ*'s 18 biggest cities.

Operationalization of Food Ration Data

In the next step, this information is combined with the micro-data.[27] Figure 3.6 shows the variation of the contextual treatment indicators (average daily calorie level in a given time window) in our micro sample. We can infer that there is a sharp drop in average rations after the war during the pregnancy and shortly after birth.
Due to the following conditions we later operationalize the food ration data as binary dummies (see Section 3.5.2 for details): the numerous data sources used, their different reliability, food rations in general not showing shortages or supplements in de facto food availability (also due to potential black market activities), the differing food ration regimes in the occupational zones, and finally the imputation which we perform to obtain complete time series. This operationalization also reflects that our goal is not to reconstruct official food rations (for pregnant women or babies) per se, but to estimate the relative nutritional situation in a given region and time period in real life.

3.5 Theoretical Exposition and Empirical Methods

3.5.1 Theoretical Exposition

To interpret the findings of our empirical analysis, it is helpful to consider the following stylized theoretical model, in which a health outcome H is a function of bombing and famine exposure:

$$H = f(Bombing, Famine) \tag{3.1}$$

[27] The information needed for matching cities with their respective occupational zone draws on Chapter 2.

Table 3.3: Contextual data sources

A) Rations	
SBZ zone rations	Boldorf (1998)
SBZ city rations	Liebe (1947, p.75)
Berlin rations	Hauptamt für Statistik Berlin-Wilmersdorf (1947, p. 236)
FBZ zone rations	Manz (1985)
FBZ city rations	Rothenberger (1980, p. 104–188, Trier, Landau), Schmitz (1956, p.469, Mainz)
Bizone zone rations	Report of the Military Governor No. 39 (1948), Schlange-Schöningen (1955)
Bizone city rations	Schmitz (1956, Essen, Duesseldorf, Hamburg, Munich, Nuremberg, Frankurt am Main, Kiel, Stuttgart, Wuppertal, Muenster, Cologne) , Boldorf (1998, p. 78, Hamburg), Seel (1950, p. 22, Bochum, Duisburg, Essen, Remscheid), Stadtverwaltung Mannheim, Statistisches Amt (1950), Martini (1947, p. 31, Bonn)
B) Bombings	
	Davis (2006)
	Middlebrook (1985) (data not included)
C) Other contextual information	
City size 1939 according to census 1939	Zeitler and Mewes (1941)
City size 2012, municipality codes (gkz)	Statistische Ämter des Bundes und der Länder (2013)
Manual corrections of municipality codes	e.g. Statistisches Bundesamt (1983), Statistische Ämter des Bundes und der Länder (2014)
Shape files	Bundesamt für Kartographie und Geodäsie (2013)

For the sake of simplicity, we restrict our exposition to one single critical period since similar considerations can be easily extended to the whole spectrum of potential critical phases of childhood. The potential causal channels between famine, air raids, stress, and malnutrition at a given critical age and later life health are depicted in Figure 3.7 for illustrative purposes.

Section 3.3.3 points out that the food situation during the war years was not critical until the last months of the war. Thus, we infer that bombing exposure is a source of stress (S_3) only. Moreover, we think of famine involving both malnutrition (M) and two possible stress components S_1 and S_2. S_1 is the stress that might be directly evoked by malnutrition. This stress component could consist of a direct physiological stress reaction[28] as well as a stress reaction to food insecurity (worries etc.).[29] Concerning the post-war famine, Allied observers state that food insecurity and insufficiency were

[28] For instance, fasting has been shown to increase cortisol concentrations in healthy men and women (see, e.g, Bergendahl et al., 1996 and Fichter et al., 1986, respectively).

[29] An empirical analysis that aims to disentangle stress from nutrition channels in famine effects might not want to detach the impact of S_1 from the nutrition effect. Being directly (physiologically or psychologically) related to the individual experience of hunger, it is actually part of the causal effect of malnutrition. However, to the extent that S_1 is an occurrence of a uniform underlying stress concept, our analysis will detach this hunger-related stress from the nutrition effect.

Figure 3.6: Average rations by critical period for SOEP respondents

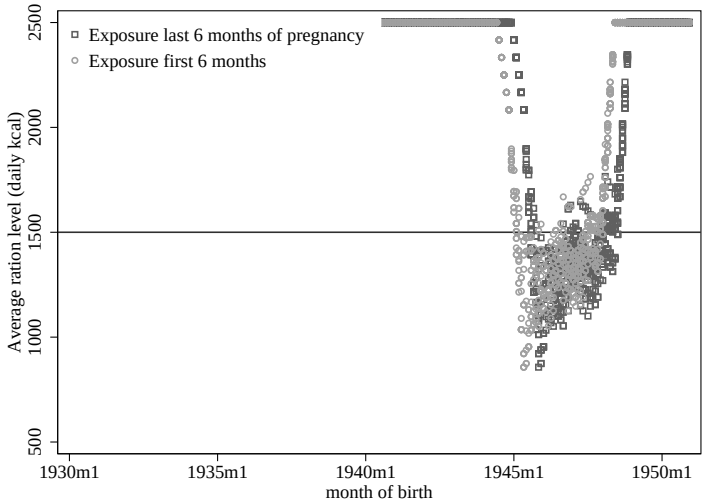
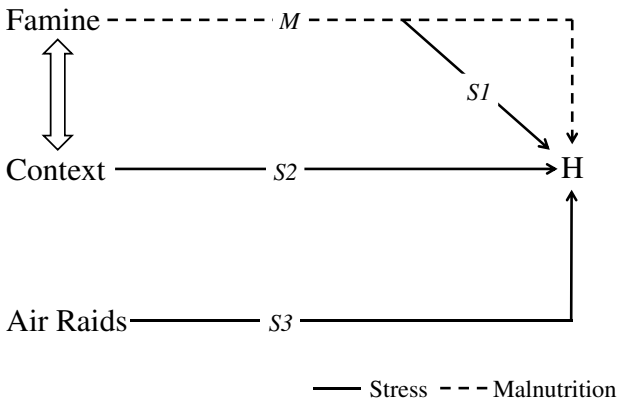


Figure 3.7: Schematic overview over possible effect components



major causes for worries and cares among the German families in the American zone, even after food rations had substantially risen to normal levels (see e.g. Merritt and Merritt, 1970, pp. 16f.). This indicates that the famine did not only affect the population physiologically, but also put a psychological burden on them. S_2 is a stress component that is evoked by contextual conditions that may parallel the famine, such as e.g. the housing situation. Assuming additivity of both famine related stress components, it follows that

$$H = f(S_3, (S_1 + S_2, M)) \tag{3.2}$$

We assume an overall linear functional form. Moreover, the different components of stress S_1, S_2, S_3 are occurrences of an overall, homogeneous stress variable S. The only dimension these stress variables are allowed to vary in is quantity, but not quality. This assumption implies that the magnitude of stress originating from different sources might vary. For a given magnitude however, all three stress channels are assumed to trigger the same physiological and psychological effects. In terms of our theoretical model, this assumption implies that the impact of S_1, S_2, S_3 on H is homogeneous for given magnitude (β_1).[30] Assuming a homogeneous impact of all stress components on the outcome, we can rewrite the model to

$$H = \beta_0 + \underbrace{\beta_1 S_3}_{\text{Bombardments}} + \underbrace{\beta_1(S_1 + S_2) + \beta_2 M}_{\text{Famine}} \tag{3.3}$$

and thus

$$H = \beta_0 + \underbrace{\beta_1(S_1 + S_2 + S_3)}_{\text{Stress}} + \underbrace{\beta_2 M}_{\text{Nutrition}} \tag{3.4}$$

Hence, the outcome is determined by potential effects of overall exposure to stress and malnutrition early in life.[31]

Model Predictions

There are four potential constellations of the effects of bombardments and of famine on later life health. In the following, we elaborate on these cases.

[30] See Section 3.2.2, and in particular the paragraph on Kelly et al. (1997) therein, for evidence on similar biological adaptations caused by different stressors.

[31] In the model with multiple critical periods, equation 3.4 can be written as

$$H = \beta_0 + \sum_{a=0}^{A} \underbrace{\beta_{1a}(S_{1a} + S_{2a} + S_{3a})}_{\text{Stress at period } a} + \sum_{a=0}^{A} \underbrace{\beta_{2a} M_a}_{\text{Nutrition at period } a}$$

where periods 0 to A are potentially critical periods in childhood within which an individual is particularly vulnerable to stress and malnutrition. All theoretical considerations can be straightforwardly extended to this extended model.

Each one has a different implication for the underlying relationships in the context of our model.
First, consider two cases, in which the effect of bombings (stress) is not different from 0 ($\beta_1 = 0$). It follows that the stress component of hunger, no matter of which magnitude it is, has no effect on H either, according to our model. Thus, the effect of famine is a pure nutrition effect: If the famine is found to have an impact (no impact) on the health outcome, only the nutritional shortage has an impact (no impact) on the outcome of interest.
Next, consider the case that famine does not have an effect, but bombings do have an impact on outcome H (i.e. $\beta_1 \neq 0$). In the light of our model, this result would imply that stress does affect H. Since the overall famine effect (nutrition component + stress components) is not different from 0, and we know that stress does have a non-zero effect on the outcome, we can infer that the magnitude of the stress components of famine ($S_1 + S_2$) must be negligibly small in this example if we can assume that the nutrition effect should go in the same direction as the stress effect.[32]
Last, if both bombardments and famine have a non-zero effect on the outcome variable of interest, we cannot draw reliable conclusions on the relevance of stress- or nutrition-related channels for the famine effect. We can only infer that the overall famine effect, i.e. combination of stress and nutrition channels, is different from zero. However, this result can potentially be caused by three candidate constellations. First, the magnitude of $S_1 + S_2$ might be small, leading to the conclusion that the famine effects are only driven by nutrition. The second possibility is that $S_1 + S_2$ are of large magnitude and $\beta_2 M = 0$ (i.e. the famine effect is only stress-driven). The last possible constellation would be a famine effect that is a combination of both, nutrition and stress components.

3.5.2 Empirical Model

To disentangle the effects of nutrition and stress exposure in early life, we run a reduced form (intent-to-treat) analysis of health and productivity related outcomes in late life on indicators of prenatal and postnatal exposure to hunger and stress through bombardments. The previous section has discussed the imposed structure and theoretical implications of the underlying model. Equation (3.3) has already hinted at the relationship between the structural health production function and the reduced form model that is the focus of our empirical analysis. In general, we want to emphasize that our empirical results only provide suggestive evidence on the structural associations of the underlying model because we estimate reduced form relations.

[32] If there is no theoretical justification for assuming this, an insignificant famine effect could also be explained by counteracting nutrition and stress components which cancel each other out.

We estimate the following equation:

$$y_i = \alpha + \theta F_i + \phi B_i + \delta X_i + \epsilon_i \tag{3.5}$$

where F_i is a vector that comprises two indicators measuring exposure to famine within six months before birth and within six months after birth, respectively. Exposure to famine is determined by the average food ration to be below 1,500 calories in the respective time frame.[33] Due to the high level of imputation and limited data quality for food rations, we use a binary famine variable to limit the impact of measurement error and noise. B_i denotes a vector that depicts the overall per capita amount of bombs (in short tons; one short ton corresponding to 2000 lbs. or roughly 907 kg) on the place of birth within the last two trimesters of pregnancy and within the first six months of life, respectively. In contrast to the food rations, here we can make use of the actual bomb load, since data quality is very high. X_i is a collection of control variables: a linear trend in the individual's birth year, an indicator for whether the individual was born in a municipality with at least 10,000 inhabitants (city), and a dummy that indicates whether the father's education is/was high.[34] Since our analysis is concerned with the very first months of life of a respondent and the prenatal period, background information referring to any period thereafter may be endogenous with respect to early life circumstances. We thus use only a limited set of individual level control variables, since any information determined after the exposure periods of interest may be an outcome of the early life experiences of interest and thus endogenous. Last, ϵ_i is an i.i.d. error component.

Using this estimation approach, we exploit temporal and regional variation in dropped bomb loads and food rations to identify the reduced form effect of hunger and stress exposure on health in late adulthood. Our identification strategy relies on the assumption that, given the covariates that are included in our model, the place and month of birth are exogenous. Including the information on being born in a city, we control for systematic differences between rural and urban areas.

3.5.3 Identification Issues

As mentioned above, our identification strategy relies on the exogeneity of the place of birth. However, this exogeneity assumption could be violated

[33] The prenatal and postnatal age window should be of the same length since we define famine exposure as a function of the average ration over those intervals. If they were of a different length it would be possible that we would define an individual as not famine exposed after birth while another individual being *in utero* at the very same instant would potentially be defined as famine exposed (and vice versa).

[34] The variable takes the value 1 if the father's education is *Realschule*, *Fachhochschule*, or *Abitur*. It takes the value 0 if the father's education is *Hauptschule* or no degree.

if parental moving decisions before the birth of their children were made in a systematic way. As an example, parents could choose their place of living according to the city's destruction level. We address these potential concerns in a sensitivity analysis.

Moreover, parental moving decisions after the birth of a child could also cause a problem for identification. If e.g. heavy bombing influenced the decision to move to a place with better living conditions (e.g. the countryside) in the first six months after the birth of a child, our estimated treatment effects would be biased. Such moves could have taken place within the framework of evacuation programs, as described in Section 3.3.2, or be privately organized. In particular, a tendency to move away from heavily bombed cities to the countryside after birth could lead to underestimated treatment effects in the study at hand. We also address this concern in a robustness check.
Furthermore, as discussed in Section 3.3.2, our sample might be positively selected since working-class neighbourhoods were common civilian bombing targets. Thus, it is likely that the survival under air raids depends on socio-economic background. Controlling for the father's education level, a proxy of socio-economic status, we also address this concern in all our analyses.
As pointed out in, e.g., Van den Berg et al. (2016), further potential threats to validity in famine studies are selective fertility and selective mortality. Van den Berg et al. (2016) argue that fertility reduction during famines may be more prevalent among poorer families. However, they do actually not find indication for selective fertility regarding the German famine. Nevertheless, controlling for a (most likely predetermined) proxy of socio-economic status during childhood (father's education), we actually already account for a potentially differential fertility reduction in our analysis. Similar to Van den Berg et al. (2016), we cannot address the general issue of selective mortality that is prevalent when using samples of elderly individuals. However, we do address the issue of war (bombardment) related selective mortality in a robustness check. In particular, we exclude individuals who were born in cities that were hit by the so-called 'firestorms'.

3.6 Regression Results

3.6.1 Main Results

For the US and UK zones (Bizone) the rationing data that we have available seem to be of better quality compared to the remaining occupational zones. Therefore, we consider the analyses on this sub-sample as most reliable and treat them as our main analyses. We show these main results in Tables 3.4 (men) and 3.6 (women). For completeness, we will also show results using

all occupational zones in Table 3.5 for males and Table 3.7 for females at the end of this section.[35] Before turning to the results, it is important to emphasize that our analysis follows a reduced form intent-to-treat design. Therefore, our primary interest is not in the magnitude, but rather in direction and significance of the estimated coefficients.

Considering the regression results for men in the Bizone, we find evidence for adult height being reduced when rations were low during pregnancy. A negative height effect of famines is common in the famine literature and with a reduced form effect of about -2 centimetres comparably large in our study. Bombings do not exert a significant impact on height even though the point estimates are relatively large. This, however, is a mechanical effect of the small scale of our bombings variables and the relatively large scale of adult height. In summary, our results imply that a reduction in height captures malnutrition rather than stress. Since height has been found to correlate positively with economic outcomes and cognitive skills (see e.g. Case and Paxson, 2008; 2010), it is conceivable that famine and thereby implied malnutrition has a profound impact on the overall economic prosperity of an individual. In fact, this hypothesis is corroborated by our findings for pension income of individuals. We find that pensions are reduced by more than 20 percent in case of prenatal exposure to famine in the US and UK occupational zones. This also hints at the importance of famine induced health conditions or deficits in other dimensions of human capital for the productivity level over the life cycle. The effects of air raids and thus stress early in life are not significant which may point at stress being of rather low importance for overall productivity. A possible explanation is that early life stress related conditions may also be prevalent only very late in life, i.e. without productivity reducing effects during the professional life of an individual.
Another striking result is that we find postnatal experience of bombings to raise the risk for hypertension among men. The experience of famine around birth is not significantly related to hypertension risk. The coefficients on our bombing measure cannot be straightforwardly interpreted. Taking the average per capita bomb load for our sample (conditional on having experienced an air raid in the first six months of life) and multiplying the coefficient found in our analysis yields an average air raid-induced excess hypertension risk of about 5 percentage points. Relative to the overall hypertension prevalence among men in our sample (45.8 percent), this amounts to an average increase of about 11 percent.
For life satisfaction, we do not find significant effects in the Bizone, neither of bombing exposure nor of famine.

[35] When turning to the sensitivity analyses in the next subsection, we will perform the robustness checks on the Bizone sub-sample. However, we turn back to all occupational zones in exceptional cases if the number of observations for the sensitivity analysis would otherwise drop too strongly.

Comparing the main results for men (using only the US and UK occupational zones) to the results when using the full sample (Table 3.5) supports our prior on data quality. Compared to the full sample, the effects of prenatal famine on height and pensions are stronger and more precisely estimated for the Bizone. Regarding post-neonatal exposure to bombardments, the effect on hypertension is twice as strong in the Bizone. However, considering life satisfaction, we find a negative and significant impact of bombing exposure only in the full sample. This result is thus entirely driven by individuals from the French and Soviet occupational zones.

Turning to the results for women, we do not find any significant associations of famine or bombing exposure around birth for the Bizone. Our results thus suggest that women are less frail to both types of adverse early life conditions. This finding regarding the gender differences in early life impacts on later life morbidity and mortality has been noted throughout the medical literature (e.g. Waldron, 1983; Kraemer, 2000). We will therefore narrow the focus on the sample of men in the remainder of the paper. For the full sample, we find a weakly significant effect of famine in the first six months of life (Table 3.7).
The directions of the estimated coefficients for year of birth and father's education are broadly in line with common expectations and literature[36]: The younger the individuals and the better educated their fathers (i.e. the higher the socio-economic status), the better off the respondents tend to be. Being born in a place with more than 10,000 inhabitants turns out to be beneficial for height, pensions, and life satisfaction in men.

3.6.2 Robustness Checks

Nine Months Pregnancy Exposure

For our main results, we choose an exposure window of one year around birth, namely 6 months before and 6 months after birth. As pregnancy is typically of nine months length, we include the first trimester of pregnancy in the exposure period in a sensitivity check. We thus alter the definition of famine during pregnancy as follows. An individual is defined to be *in utero* during famine if the 6 months running average of imputed food rations is below 1500 kcal at any time in the nine months before birth. For bombing exposure, we simply add up the tonnage over the nine months interval. The results for this exercise are shown in Table 3.8.

[36] E.g. see Currie (2009) for a review of the associations between parental socio-economic status and child health as well as between child health and future outcomes. Positive trends in height over cohorts are documented in e.g. Hatton and Bray (2010).

Table 3.4: Males – Bizone

Variable	Hypertension	Height	log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.053	-2.128**	-0.249***	-0.398
	(0.067)	(0.931)	(0.088)	(0.243)
Less than 1500 kcal, post-birth	-0.084	1.161	0.131	0.332
	(0.069)	(0.901)	(0.086)	(0.252)
Bombs dropped, in utero	-9.391	13.573	-1.373	-10.359
	(7.461)	(85.692)	(5.875)	(14.882)
Bombs dropped, post-birth	6.748**	-36.735	2.901	5.682
	(3.094)	(53.500)	(6.445)	(7.827)
Year of birth	-0.007**	0.215***	0.007	0.010
	(0.003)	(0.035)	(0.005)	(0.010)
Father's education high (1/0)	-0.013	2.671***	0.303***	0.497***
	(0.037)	(0.482)	(0.058)	(0.116)
Place of birth: pop. $\geq$ 10000	0.028	0.655*	0.165***	0.187*
	(0.031)	(0.389)	(0.041)	(0.107)
Constant	13.681**	-241.614***	-6.557	-12.335
	(5.337)	(67.342)	(9.050)	(19.742)
R^2	0.011	0.071	0.079	0.023
F-Stat.	2.374	11.328	10.046	4.412
N	1100	1108	846	1105

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

The results look qualitatively similar to our main results. However, the effect of postnatal bomb exposure on hypertension turns insignificant. Moreover, the effects of prenatal famine on height and pensions decrease in magnitude. However, note that this drop could be due to measurement error. We do not observe the exact start of pregnancy and gestational length. If gestational length suffered from prenatal famine exposure as well, we would define the exposure period too long when setting it at the full nine months before birth. In contrast, the effect of prenatal famine on life satisfaction increases slightly in magnitude and turns significant. Life satisfaction might thus be susceptible to influences particularly early in pregnancy. However, we emphasize that an analysis of sensitive periods during pregnancy is beyond the scope of this paper due to data constraints.

Deviations in the Geocode Before Birth

One of the major threats to our empirical strategy is that the place of birth might be selective. In a robustness check, we therefore examine whether our results are sensitive to omitting those who were born in a time with potentially severe exposure to any of the treatments (i.e. born 1942–1948) and might have relocated during pregnancy. This group of individuals is defined as having indicated a place of pregnancy in the *FKM* survey that is different

Table 3.5: Males – all zones

Variable	Hypertension	Height	log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.055	-1.249*	-0.195**	-0.300
	(0.055)	(0.742)	(0.086)	(0.183)
Less than 1500 kcal, post-birth	-0.007	0.498	0.145*	0.276
	(0.056)	(0.729)	(0.084)	(0.182)
Bombs dropped, in utero	-8.277	27.569	-2.194	18.817
	(7.724)	(86.747)	(5.598)	(18.884)
Bombs dropped, post-birth]	2.768***	-9.626	3.357	-7.009***
	(0.741)	(9.280)	(6.303)	(2.613)
Year of birth	-0.006***	0.222***	0.006	0.003
	(0.002)	(0.027)	(0.004)	(0.008)
Father's education high	-0.033	2.298***	0.313***	0.402***
	(0.029)	(0.365)	(0.049)	(0.095)
Place of birth: pop. $\geq$ 10,000	0.002	0.617**	0.121***	0.169**
	(0.024)	(0.302)	(0.036)	(0.082)
Constant	12.274***	-255.512***	-3.562	0.915
	(4.196)	(53.019)	(8.056)	(15.276)
R^2	0.007	0.064	0.077	0.017
F-Stat.	3.532	17.426	11.083	5.681
N	1850	1862	1059	1859

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

from the place of birth indicated in the SOEP interview.[37] There can be several possible explanations of such a deviation that might not all actually go back to a relocation. An individual could simply be born in a different municipality than the mother's place of residence without the mother having actually moved. Our robustness check is conservative in the sense that we do not only drop individuals who actually moved their residence, but also those who have differing *GKZ*s for other reasons.

Since we rely on information from the *FKM* survey, we have to restrict our analysis sample to *FKM* respondents. We do not restrict the sample to the Bizone in this sensitivity analysis because the sample size would otherwise drop even more. We further omit observations with missing information on the *GKZ* of pregnancy from the baseline *FKM* sample.[38] Both reasons explain the drop in sample size in the baseline Table 3.9 compared to the full sample in Table 3.5. Table 3.9 shows our estimation results for the (smaller) baseline sample of *FKM* respondents with non-missing *GKZ* of pregnancy. Qualitatively, the results in this smaller sample are in line with our main results using all occupational zones. However, the prenatal famine effects on pen-

[37] Note that the place of birth itself is also asked in the *FKM* survey. The information from SOEP and *FKM* are not completely consistent with each other. Thus, by using the place of birth stemming from SOEP and the place of pregnancy from the *FKM* survey, we may actually run into some measurement error problems regarding our definition of relocations. The reasons for discrepancies between the two place of birth variables are not entirely clear since there are only slight differences in question wording.

[38] Note that individuals with a missing *GKZ* of pregnancy and a valid *GKZ* of birth may actually also have moved.

Table 3.6: Females – Bizone

Variable	Hypertension	Height	Life Satisfaction
Less than 1500 kcal, in utero	-0.061	0.027	-0.252
	(0.066)	(0.672)	(0.282)
Less than 1500 kcal, post-birth	0.069	-0.755	0.313
	(0.069)	(0.714)	(0.279)
Bombs dropped, in utero	0.967	5.537	6.774
	(3.613)	(28.309)	(7.126)
Bombs dropped, post-birth	2.078	34.663	9.093
	(4.921)	(43.783)	(11.947)
Year of birth	-0.010***	0.146***	0.018*
	(0.003)	(0.033)	(0.010)
Father's education high (1/0)	-0.064*	1.017**	0.334**
	(0.038)	(0.440)	(0.133)
Place of birth: pop. $\geq$ 10000	0.040	0.079	0.168
	(0.031)	(0.364)	(0.115)
Constant	19.563***	-119.919*	-27.453
	(5.518)	(63.401)	(20.322)
R^2	0.020	0.027	0.016
F-Stat.	2.956	4.375	2.822
N	1043	1052	1052

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

sions and height loose significance, which might be due to the small sample size. Table 3.10 shows the results after we additionally drop those individuals from the sample who have differing *GKZ*s of birth and pregnancy and were born between 1942–1948. Compared to the baseline in Table 3.9, the results stay fairly similar. Therefore, we conclude that our findings are not likely to be entirely driven by selective migration before birth.

Deviations in the Geocode After Birth

Parallel to the robustness check on geocode deviations during pregnancy, we also consider the first time after birth: In this analysis, we exclude individuals from the sample, if deviations occur between place of birth and place of residence in the first 6 months after birth in times relevant for our contextual variation. This is insightful, because our treatment definition is tied to the place of birth, although the individuals can potentially have lived in another municipality with different famine or bombing exposure. This would lead to a measurement error, which theoretically could be non-random. Possible interpretations of such a deviation include e.g. a change of residence after giving birth, but also giving birth in different municipality than the place of residence.
Technically, we exclude all persons from the estimation sample, who indicate to have mainly lived in another place than their place of birth during the first 6 months of life, given the individual was born between 1942 and 1948 (the years of potential exposure to bomb attacks or famine). We infer

Table 3.7: Females – all zones

Variable	Hypertension	Height	Life Satisfaction
Less than 1500 kcal, in utero	-0.024	0.047	-0.136
	(0.051)	(0.551)	(0.189)
Less than 1500 kcal, post-birth	0.014	-0.944*	0.197
	(0.053)	(0.569)	(0.193)
Bombs dropped, in utero	0.729	6.055	8.492
	(3.310)	(27.386)	(7.786)
Bombs dropped, post-birt	1.179	52.291	13.061
	(4.515)	(40.012)	(11.561)
Year of birth	-0.010***	0.136***	0.008
	(0.002)	(0.025)	(0.008)
Father's education high	-0.082***	1.263***	0.387***
	(0.029)	(0.338)	(0.099)
Place of birth: pop. $\geq$ 10,000	0.031	-0.188	-0.062
	(0.023)	(0.275)	(0.083)
Constant	18.889***	-100.522**	-8.260
	(4.162)	(48.099)	(15.696)
R^2	0.019	0.026	0.011
F-Stat.	5.079	7.668	3.211
N	1889	1904	1903

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

the geocode of the place of first residence from the separate *FKM* questionnaire which asks the individuals whether they lived at their place of birth or in another place (and which) during this period. If they state to have lived at the place of birth, we take the geocode from the place of birth given in the *FKM* questionnaire, else the indicated place. We now infer the place of residence to deviate from the place of birth if the generated geocode of first residence differs from the normal place of birth geocode, which we stick to in our main analysis.[39] The tables show that the individuals with place of residence deviations do not drive our results: Table 3.11 shows the full available sample of individuals being asked about their main place of residence after birth (for comparison) and Table 3.12 shows the results excluding place of residence deviations as defined above, both tables excluding people with non-identified place of birth or non-identified place of first residence.

Cumulative Bombardments

More generally, our results could be confounded by the destruction level of a city caused by bombardments before pregnancy. The reasons can include

[39] Note that this definition might contain measurement error since our normal place of birth was asked in a different questionnaire than the *FKM* containing the place of first residence. Thus, both deviations or similarities in the geocodes may arise due to differing answers with respect to the place of birth in both questionnaires.

Table 3.8: Males Bizone – 9 months pregnancy, running average

Variable	Hypertension	Height	Log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero, RA	-0.007	-1.609**	-0.230***	-0.424**
	(0.060)	(0.785)	(0.087)	(0.212)
Less than 1500 kcal, post-birth	-0.042	0.767	0.113	0.335
	(0.066)	(0.827)	(0.087)	(0.232)
Bombs dropped, in utero	-0.675	7.658	-1.365	4.230
	(6.875)	(51.234)	(4.356)	(9.517)
Bombs dropped, post-birth	5.275	-36.385	2.718	2.788
	(3.311)	(53.877)	(6.329)	(8.190)
Year of birth	-0.006**	0.217***	0.008*	0.012
	(0.003)	(0.035)	(0.005)	(0.010)
Father's education high (1/0)	-0.014	2.706***	0.309***	0.500***
	(0.037)	(0.483)	(0.058)	(0.116)
Place of birth: pop. $\geq$ 10000	0.027	0.617	0.162***	0.177
	(0.031)	(0.388)	(0.041)	(0.107)
Constant	12.864**	-246.269***	-8.842	-15.781
	(5.421)	(68.757)	(9.219)	(20.028)
R^2	0.010	0.070	0.079	0.024
F-Stat.	1.760	10.936	9.825	4.773
N	1100	1108	846	1105

Note: famine exposure in utero is calculated with a 6 month running average during 9 months of pregnancy, whereas bombs dropped are calculated for the 9 months of pregnancy. Famine exposure and bombs dropped post-birth are calculated for the first 6 month of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

selective relocations but also generally worse living conditions for those who stay.

We therefore re-run our main regressions controlling for cumulative bomb load dropped on a specific place of birth until 9 months prior to a respondent's birth (scaled by 1939 population size). This variable varies over time for a given city, depending on how often and how strongly this city has been bombed. Moreover, as a cumulative measure, it can be non-zero for post-war cohorts. We use this measure as a general proxy for war-time destruction of a municipality. Controlling for this measure in our analyses, we compare individuals who exhibit similar places of birth concerning cumulative bomb loads prior conception. Table 3.13 shows the results of this exercise. We still find that hypertension risk among men is adversely affected by the postnatal experience of air raids. The effects of famine on male adult height and pension income remain robust as well.

Exposure to Air Raids and Famine at Later Childhood Ages

In another robustness check, we consider exposure to adverse circumstances also at higher childhood ages. Such exposure could be problematic since other studies have shown that later life outcomes are affected by exposure to adversities in later childhood, most importantly in the context of puberty (e.g. Van den Berg et al., 2014b; Sparén et al., 2004). There is also evidence

Table 3.9: Males *GKZ* pregnancy sample – pregnancy and birth *GKZ* known

Variable	Hypertension	Height	Log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.058	-1.009	-0.144	-0.342*
	(0.071)	(0.997)	(0.097)	(0.195)
Less than 1500 kcal, post-birth	-0.011	-0.298	0.058	0.232
	(0.071)	(0.985)	(0.092)	(0.200)
Bombs dropped, in utero	-9.101	67.041	-12.088	-1.069
	(16.626)	(243.892)	(26.167)	(81.481)
Bombs dropped, post-birth	2.772**	-13.174	1.173	-8.057*
	(1.126)	(13.922)	(5.934)	(4.737)
Year of birth	-0.007**	0.214***	0.007	-0.006
	(0.003)	(0.043)	(0.007)	(0.011)
Father's education high (1/0)	-0.039	2.183***	0.257***	0.392***
	(0.036)	(0.460)	(0.061)	(0.112)
Place of birth: pop. $\geq$ 10000	-0.029	0.617	0.104**	0.065
	(0.031)	(0.387)	(0.047)	(0.098)
Constant	13.808**	-239.461***	-5.608	19.704
	(6.621)	(83.192)	(14.295)	(21.902)
R^2	0.008	0.050	0.053	0.018
F-Stat.	2.903	7.967	4.150	6.615
N	1094	1093	616	1093

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

that nutrition around this period influences outcomes of later generations which also points at the plasticity around that age (see e.g. Kaati et al., 2002; Van den Berg and Pinger, 2016 and references therein). If air raids or famine had a sizeable impact on the later life health outcomes under study, our control group's outcomes could be contaminated and the effect measured in our analysis would be biased. This problem is aggravated by the fact that those exposed to air raids around birth were affected by the famine at a later but potentially still vulnerable age.

We construct two additional explanatory variables: the first one displaying the bomb load dropped on a respondent's place of birth in the age interval from 7 to 132 months. The second variable is an indicator for whether a respondent was exposed to a 6 months interval with average food rations below 1,500 kcal sometime in this age interval. Note that exposure to both adverse conditions is determined by the place of birth and not the residence at the specific age. We should also note that the effects of exposure to adverse conditions at later ages are not straightforwardly interpretable since non-biological channels become more relevant at later ages. For instance, famine and social unrest around puberty could limit access to education and thereby change intermediate socio-economic outcomes which then may determine health outcomes later in life. As shown in Table 3.14, including those additional variables into our models does not change our main results and conclusions.

Table 3.10: Males *GKZ* pregnancy sample – no *GKZ* change between pregnancy and birth

Variable	Hypertension	Height	Log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.075	-0.955	-0.158	-0.300
	(0.076)	(1.100)	(0.106)	(0.185)
Less than 1500 kcal, post-birth	-0.017	-0.215	0.095	0.352*
	(0.078)	(1.109)	(0.104)	(0.191)
Bombs dropped, in utero	-10.339	38.705	-2.531	-21.986
	(18.832)	(295.764)	(31.904)	(95.377)
Bombs dropped, post-birth	2.877**	-11.125	0.886	-6.691
	(1.237)	(16.662)	(6.053)	(5.599)
Year of birth	-0.007**	0.209***	0.004	-0.007
	(0.003)	(0.043)	(0.008)	(0.011)
Father's education high (1/0)	-0.021	2.249***	0.259***	0.395***
	(0.038)	(0.484)	(0.066)	(0.120)
Place of birth: pop. $\geq$ 10000	-0.034	0.545	0.119**	0.079
	(0.033)	(0.401)	(0.051)	(0.103)
Constant	14.594**	-229.715***	-0.409	20.150
	(6.707)	(83.453)	(15.076)	(22.338)
R^2	0.008	0.051	0.057	0.018
F-Stat.	2.915	7.328	4.113	6.416
N	996	996	548	995

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

Selective Mortality

As noted in Section 3.3.2, almost half of the casualties that can be attributed directly to air raids died in one of the 'firestorms' over Hamburg, Kassel, Darmstadt, and Dresden. Hence, we check whether our results change when dropping all respondents who were born in one of these cities over the whole observational period to address the issue of selective mortality. Table 3.15 shows the results of this robustness check. The results of our main analysis turn out to be robust.

Cohort, Regional, Seasonal Fixed Effects

A further concern could be that unobserved cohort-specific factors may bias our results. This may be the case if, for instance, cohort specific shocks to selective child mortality or selective fertility are determinants of late life health and on the other hand interact with the exposure to air raids. Including cohort fixed effects in our regression is very demanding on the data and on the power of our identifying variation and we thus suspect these alterations to yield at most suggestive evidence. Our measures of exposure to air raids should be less sensitive to this shortcoming since they exhibit a larger degree of cross-sectional variation within the war years. As a robustness check, we thus run another set of regressions where we change our main specification by replacing the linear trend in year of birth with cohort fixed effects. For this

Table 3.11: Males *FKM* sample – birth place and post-birth location known

Variable	Hypertension	Height	Log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.042	-1.243	-0.124	-0.296
	(0.069)	(0.987)	(0.092)	(0.187)
Less than 1500 kcal, post-birth	0.014	0.106	0.048	0.268
	(0.070)	(0.971)	(0.087)	(0.192)
Bombs dropped, in utero	-11.776*	-58.772	-12.064	6.954
	(6.739)	(128.757)	(9.572)	(33.478)
Bombs dropped, post-birth	2.494***	-7.198	9.978	-9.438***
	(0.643)	(10.450)	(9.963)	(2.082)
Year of birth	-0.007**	0.216***	0.005	-0.005
	(0.003)	(0.043)	(0.007)	(0.011)
Father's education high (1/0)	-0.040	2.107***	0.260***	0.383***
	(0.036)	(0.458)	(0.060)	(0.112)
Place of birth: pop. $\geq$ 10000	-0.026	0.597	0.094**	0.077
	(0.031)	(0.385)	(0.046)	(0.097)
Constant	13.455**	-244.075***	-2.366	16.613
	(6.605)	(83.014)	(14.229)	(22.081)
R^2	0.008	0.047	0.054	0.017
F-Stat.	3.075	7.700	4.267	9.594
N	1106	1105	622	1105

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

change in model specification our main results remain qualitatively similar (see Table 3.16). The effect of prenatal famine on height turns insignificant. It is likely that this is due to a loss in identifying variation.

Moreover, we also ran our analysis additionally controlling for regional (*Bundesland*; i.e. the 16 regions in today's borders of Germany) fixed effects to control for regional differences that might be correlated with famine or bombing exposure and could therefore bias our results, such as e.g. economic development after war and famine. The results are shown in Table 3.17 and are very similar to our main results.

Another problem of omitted variable bias could emerge from the fact that season of birth has been found to be related to health and longevity (e.g. Doblhammer and Vaupel, 2001). Both the nutritional situation as well as exposure to bombings could exhibit seasonality. We examine this issue by adding season of birth dummies to our regressions.[40] The results are shown in Table 3.18. The findings are very similar to our main results. Moreover, the season indicators are not found to be statistically significant predictors of our outcomes.

[40]Spring: March until May, Summer: June until August, Fall: September until November, Winter: December until February.

Table 3.12: Males *FKM* sample – no post-birth changes in location

Variable	Hypertension	Height	Log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.039	-1.485	-0.171*	-0.298
	(0.076)	(1.078)	(0.100)	(0.182)
Less than 1500 kcal, post-birth	0.010	0.389	0.089	0.343*
	(0.078)	(1.064)	(0.098)	(0.188)
Bombs dropped, in utero	-6.027	69.609	-26.068	-16.077
	(19.230)	(292.991)	(32.592)	(95.554)
Bombs dropped, post-birth	2.282*	-16.083	13.165	-7.521
	(1.231)	(18.429)	(10.987)	(5.728)
Year of birth	-0.006*	0.208***	0.002	-0.006
	(0.003)	(0.042)	(0.007)	(0.011)
Father's education high (1/0)	-0.029	2.252***	0.272***	0.372***
	(0.037)	(0.467)	(0.062)	(0.116)
Place of birth: pop. $\geq$ 10000	-0.031	0.475	0.103**	0.078
	(0.032)	(0.395)	(0.048)	(0.100)
Constant	12.858*	-228.298***	2.768	17.956
	(6.566)	(81.512)	(14.538)	(22.005)
R^2	0.006	0.051	0.063	0.016
F-Stat.	2.907	7.909	4.889	11.491
N	1042	1042	580	1041

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

Breastfeeding

Another concern in our main results could be caused by differential breastfeeding behaviour of women in treatment and control groups. If e.g. stressed mothers are less likely to breastfeed their newborns, then our estimated effects for stress exposure might actually originate from nutritional pathways. To analyse this concern, we use again the aforementioned supplementary SOEP module on early childhood in the war and postwar period (*FKM*). This survey asks respondents whether they have been breastfed. Having this information at hand, we can run our analyses without individuals who have not been breastfed.[41] Note that due to the smaller sample size (restricting the sample to *FKM* respondents), we use again the sample of all occupational zones here. Table 3.19 shows the baseline results for the sub-sample of *FKM* respondents and Table 3.20 show the results when we drop those who were not breastfed. Our results do not seem to be driven by breastfeeding behaviour. Compared to our main results for all occupational zones (Table 3.5), there are no major differences. The effect of prenatal famine on height turns insignificant. However, this is likely to be due to the smaller sample size when restricting the sample to *FKM* respondents only. The baseline results restricted to *FKM* respondents in Table 3.19 also show this insignificance.

[41] This question is probably prone to recall bias and measurement error. Yet, in a setting with elderly people it is impossible to collect more valid information on breastfeeding.

Table 3.13: Males Bizone – cumulative bomb load

Variable	Hypertension	Height	Log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.058	-2.065**	-0.256***	-0.386
	(0.067)	(0.944)	(0.091)	(0.242)
Less than 1500 kcal, post-birth	-0.085	1.149	0.128	0.330
	(0.068)	(0.902)	(0.086)	(0.251)
Bombs dropped, in utero	-9.057	17.670	-1.849	-9.586
	(7.433)	(84.924)	(5.997)	(15.053)
Bombs dropped, post-birth	6.577**	-39.000	2.957	5.253
	(3.114)	(53.655)	(6.464)	(7.793)
Overall bombs before pregnancy	-0.505	-6.776	0.567	-1.282
	(0.816)	(12.458)	(1.314)	(3.100)
Year of birth	-0.006**	0.224***	0.007	0.012
	(0.003)	(0.037)	(0.005)	(0.011)
Father's education high (1/0)	-0.013	2.670***	0.305***	0.497***
	(0.037)	(0.482)	(0.058)	(0.116)
Place of birth: pop. $\geq$ 10000	0.034	0.740*	0.162***	0.204*
	(0.033)	(0.409)	(0.042)	(0.114)
Constant	12.428**	-258.486***	-6.031	-15.528
	(5.734)	(71.931)	(9.107)	(21.172)
R^2	0.012	0.072	0.080	0.023
F-Stat.	2.124	10.009	8.786	3.858
N	1100	1108	846	1105

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

Other Adverse Conditions

For some cohorts under study, other adverse conditions may have been present in infancy or childhood that may potentially confound our analyses. Examples could e.g. be war related trauma or the absence of the father. Using information of the *FKM* study, we investigate the relationship between being in any of our treatment groups and different adverse conditions. We study in particular father's absence for cohorts with potential treatment exposure (1940 – 1948), as well as seeing dead or injured people and battle exposure for war cohorts with potential treatment exposure (1940 – 1945). Using linear probability models, we relate father absence to four variables indicating our treatment groups (whether or not the individual was exposed to famine 6 month before/after birth, and whether or not the individual was born in a city that has been bombed 6 month before/after birth). The latter two adverse conditions, namely seeing dead or injured people and battle exposure, are directly related to war time. Thus we only relate them to bombing exposure for cohorts born before 1945. Moreover, we additionally include the control variables of our main analysis. Note that we use all occupational zones here due to the small sample size. The results of this analysis (Table 3.21) show no significant relations between on the one hand father's absence, having seen dead or injured people, or battle exposure and on the other hand the indicators of our treatment groups.

Table 3.14: Males Bizone – exposure to air raids and famine in months 7 – 132

Variable	Hypertension	Height	Log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.047	-2.096**	-0.244***	-0.335
	(0.067)	(0.933)	(0.089)	(0.245)
Less than 1500 kcal, post-birth	-0.072	1.277	0.140	0.205
	(0.070)	(0.920)	(0.086)	(0.259)
Famine childhood	-0.027	-0.190	-0.012	0.286**
	(0.034)	(0.440)	(0.047)	(0.122)
Bombs dropped, in utero	-8.820	19.557	-0.789	-13.555
	(7.580)	(85.413)	(5.962)	(15.338)
Bombs dropped, post-birth	7.078**	-32.122	3.320	1.746
	(3.113)	(53.339)	(6.511)	(7.683)
Bombs childhood	-0.021	8.296	0.679	0.088
	(0.532)	(8.215)	(0.643)	(1.371)
Year of birth	-0.008***	0.218***	0.007	0.018
	(0.003)	(0.037)	(0.005)	(0.011)
Father's education high (1/0)	-0.013	2.653***	0.301***	0.497***
	(0.037)	(0.483)	(0.058)	(0.116)
Place of birth: pop. $\geq$ 10000	0.029	0.464	0.145***	0.180
	(0.033)	(0.432)	(0.045)	(0.112)
Constant	15.161***	-247.132***	-6.729	-27.685
	(5.650)	(71.768)	(9.167)	(21.272)
R^2	0.012	0.073	0.081	0.029
F-Stat.	1.919	9.078	7.995	3.904
N	1100	1108	846	1105

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy, for the first 6 months of life, and for months 7–132. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

Table 3.15: Males Bizone – without individuals born in a 'firestorm city'

Variable	Hypertension	Height	Log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.045	-2.522***	-0.249***	-0.429*
	(0.068)	(0.913)	(0.091)	(0.253)
Less than 1500 kcal, post-birth	-0.079	1.652*	0.112	0.348
	(0.070)	(0.896)	(0.089)	(0.261)
Bombs dropped, in utero	-9.044	-0.710	1.009	-5.549
	(7.628)	(84.690)	(6.578)	(14.519)
Bombs dropped, post-birth	6.561**	-36.797	2.877	4.995
	(3.104)	(53.328)	(6.563)	(7.728)
Year of birth	-0.006**	0.199***	0.007	0.009
	(0.003)	(0.035)	(0.005)	(0.010)
Father's education high (1/0)	-0.020	2.766***	0.312***	0.481***
	(0.038)	(0.501)	(0.060)	(0.121)
Place of birth: pop. $\geq$ 10000	0.027	0.689*	0.174***	0.200*
	(0.032)	(0.392)	(0.042)	(0.109)
Constant	12.571**	-210.947***	-6.598	-11.231
	(5.445)	(68.445)	(9.112)	(20.182)
R^2	0.010	0.072	0.084	0.023
F-Stat.	2.072	10.644	10.207	4.092
N	1053	1061	807	1058

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

Table 3.16: Males Bizone – cohort fixed effects

Variable	Hypertension	Height	Log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.046	-1.565	-0.294***	0.136
	(0.111)	(1.544)	(0.108)	(0.386)
Less than 1500 kcal, post-birth	-0.082	1.529	-0.008	0.158
	(0.099)	(1.436)	(0.121)	(0.351)
Bombs dropped, in utero	-8.740	6.621	-6.119	-8.780
	(7.880)	(80.785)	(5.224)	(15.198)
Bombs dropped, post-birth	7.482**	-26.940	-1.978	8.305
	(3.271)	(55.211)	(5.868)	(8.173)
Father's education high (1/0)	-0.015	2.694***	0.302***	0.532***
	(0.038)	(0.485)	(0.056)	(0.118)
Place of birth: pop. $\geq$ 10000	0.028	0.699*	0.185***	0.158
	(0.032)	(0.402)	(0.043)	(0.109)
R^2	0.026	0.085	0.104	0.050
F-Stat.	1.261	4.113	4.171	2.247
N	1100	1108	846	1105

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively. Cohort fixed effects are included, but not displayed for brevity.

Table 3.17: Males Bizone – *Bundesland* fixed effects

Variable	Hypertension	Height	Log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.045	-2.163**	-0.232***	-0.395
	(0.066)	(0.925)	(0.085)	(0.243)
Less than 1500 kcal, post-birth	-0.070	1.298	0.126	0.316
	(0.068)	(0.896)	(0.085)	(0.252)
Bombs dropped, in utero	-10.696	13.293	-1.190	-8.975
	(7.453)	(85.190)	(5.935)	(14.846)
Bombs dropped, post-birth	5.878*	-45.029	2.194	6.972
	(3.022)	(54.129)	(6.496)	(7.787)
Year of birth	-0.007**	0.215***	0.008*	0.010
	(0.003)	(0.035)	(0.005)	(0.010)
Father's education high (1/0)	-0.025	2.639***	0.314***	0.508***
	(0.037)	(0.483)	(0.058)	(0.116)
Place of birth: pop. $\geq$ 10000	0.029	0.424	0.154***	0.178
	(0.033)	(0.411)	(0.044)	(0.118)
Hamburg	-0.044	-0.913	0.138	0.062
	(0.103)	(1.451)	(0.128)	(0.316)
Niedersachsen	-0.063	-0.263	0.176*	-0.011
	(0.073)	(0.965)	(0.099)	(0.236)
Bremen	0.075	-2.607*	0.351**	0.223
	(0.139)	(1.481)	(0.170)	(0.672)
Nordrhein-Westfalen	-0.079	0.285	0.199**	0.012
	(0.069)	(0.898)	(0.094)	(0.227)
Hessen	0.067	0.430	0.235**	-0.166
	(0.080)	(1.015)	(0.113)	(0.270)
Baden Wuerttemberg	-0.174**	-0.661	0.247**	-0.022
	(0.081)	(1.039)	(0.115)	(0.290)
Bayern	-0.137**	-1.613*	0.194**	0.077
	(0.069)	(0.888)	(0.094)	(0.222)
Constant	13.966***	-240.708***	-8.166	-12.913
	(5.310)	(67.176)	(9.028)	(19.707)
R^2	0.028	0.087	0.088	0.025
F-Stat.	2.750	7.475	5.646	2.455
N	1100	1108	846	1105

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

Table 3.18: Males Bizone – season fixed effects

Variable	Hypertension	Height	Log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.056	-1.985**	-0.256***	-0.412*
	(0.067)	(0.932)	(0.093)	(0.243)
Less than 1500 kcal, post-birth	-0.087	1.065	0.139	0.324
	(0.069)	(0.899)	(0.093)	(0.250)
Bombs dropped, in utero	-9.213	14.250	-1.152	-11.420
	(7.511)	(81.945)	(9.587)	(14.798)
Bombs dropped, post-birth	6.804**	-32.967	2.702	4.855
	(3.083)	(53.919)	(5.352)	(8.040)
Summer	-0.022	-0.416	0.027	-0.237
	(0.043)	(0.534)	(0.058)	(0.153)
Fall	-0.013	-0.760	0.054	0.143
	(0.043)	(0.511)	(0.057)	(0.136)
Winter	0.003	-0.188	0.062	-0.092
	(0.043)	(0.527)	(0.057)	(0.146)
Year of birth	-0.007**	0.213***	0.007	0.011
	(0.003)	(0.035)	(0.005)	(0.010)
Father's education high (1/0)	-0.013	2.667***	0.306***	0.498***
	(0.038)	(0.482)	(0.050)	(0.116)
Place of birth: pop. $\geq$ 10000	0.028	0.680*	0.161***	0.187*
	(0.031)	(0.390)	(0.042)	(0.107)
Constant	13.773**	-237.923***	-6.384	-14.270
	(5.349)	(67.443)	(9.186)	(19.747)
R^2	0.012	0.073	0.081	0.030
F-Stat.	1.723	8.181	7.361	3.875
N	1100	1108	846	1105

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

Table 3.19: Males *FKM* sample

Variable	Hypertension	Height	Log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.072	-1.396	-0.178*	-0.183
	(0.066)	(0.923)	(0.096)	(0.193)
Less than 1500 kcal, post-birth	-0.019	0.415	0.088	0.135
	(0.067)	(0.908)	(0.093)	(0.202)
Bombs dropped, in utero	-12.217*	-64.092	-6.566	5.288
	(6.697)	(126.573)	(8.783)	(33.326)
Bombs dropped, post-birth	2.952***	-4.580	0.682	-8.510***
	(0.691)	(9.066)	(5.733)	(2.212)
Year of birth	-0.007**	0.210***	0.007	-0.006
	(0.003)	(0.042)	(0.007)	(0.011)
Father's education high (1/0)	-0.034	2.176***	0.269***	0.397***
	(0.035)	(0.442)	(0.058)	(0.107)
Place of birth: pop. $\geq$ 10000	-0.025	0.568	0.091**	0.102
	(0.030)	(0.378)	(0.045)	(0.095)
Constant	13.166**	-232.028***	-5.648	18.418
	(6.466)	(80.771)	(13.842)	(21.529)
R^2	0.008	0.047	0.057	0.017
F-Stat.	3.497	8.049	4.814	6.692
N	1161	1160	655	1160

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

Table 3.20: Males *FKM* sample – only if breastfed as infants

Variable	Hypertension	Height	Log(Pension)	Life Satisfaction
Less than 1500 kcal, in utero	0.112	-1.037	-0.252**	-0.114
	(0.072)	(1.065)	(0.115)	(0.232)
Less than 1500 kcal, post-birth	-0.112	0.423	0.148	-0.024
	(0.073)	(1.068)	(0.115)	(0.253)
Bombs dropped, in utero	-7.550	126.500	-21.105	24.004
	(15.747)	(221.529)	(27.098)	(77.408)
Bombs dropped, post-birth	2.744**	-16.074	1.864	-9.771**
	(1.080)	(13.132)	(6.465)	(4.472)
Year of birth	-0.008**	0.207***	0.009	-0.009
	(0.004)	(0.045)	(0.008)	(0.012)
Father's education high (1/0)	-0.016	1.954***	0.273***	0.381***
	(0.039)	(0.488)	(0.064)	(0.122)
Place of birth: pop. $\geq$ 10000	-0.067**	0.593	0.112**	0.041
	(0.034)	(0.419)	(0.052)	(0.107)
Constant	15.156**	-226.343***	-10.424	23.825
	(7.071)	(86.935)	(15.019)	(23.891)
R^2	0.013	0.044	0.069	0.016
F-Stat.	3.475	6.404	4.592	6.054
N	936	935	523	935

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

Table 3.21: Males *FKM* sample – other adverse conditions

Variable	Father Absence	Seen Injured/Dead	Battlefield
Less than 1500 kcal, in utero	-0.040		
	(0.059)		
Less than 1500 kcal, post-birth	-0.010		
	(0.058)		
Bombed in utero, (1/0)	-0.010	0.023	0.046
	(0.090)	(0.049)	(0.075)
Bombed post-birth, (1/0)	0.044	0.013	-0.055
	(0.088)	(0.048)	(0.073)
Year of birth	-0.067***	-0.041***	-0.063***
	(0.006)	(0.012)	(0.016)
Place of birth: pop. $\geq$ 10000	-0.007	0.039	-0.041
	(0.036)	(0.038)	(0.053)
Father's education high (1/0)	0.131***	0.032	-0.006
	(0.041)	(0.044)	(0.054)
Constant	131.419***	78.951***	122.661***
	(12.535)	(22.401)	(31.704)
R^2	0.185	0.035	0.050
F-Stat.	26.186	3.076	3.942
N	656	359	352

Note: famine exposure and bombs dropped are calculated for the last 6 months of pregnancy and for the first 6 months of life. Robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

3.7 Conclusion

The present empirical study sheds light on the components of famine effects. Famines are aggregate disruptions and arguably provide exogenous variation in the nutritional situation of a fraction of a population under study. Hence, famines have been used to empirically test theoretical predictions of fetal programming of humans by nutritional shortages early in life that may manifest as worse health outcomes later in life. The commonly used study design is to measure a reduced form effect of famine exposure on later life health. However, it is possible that a suchlike measured effect picks up factors different from food shortages, most importantly psychological stress. Such a stress component could be either directly evoked by individual hunger or factors paralleling the famine. It is thus questionable whether famine studies are an adequate study design to measure the long run effects of malnutrition.

Using a unique combination of German survey data and information on both food rations as a measure of famine exposure within an early life age window and the exposure to Allied air raids in World War II, we gain new insights on this matter. As argued in our paper, air raids may be assumed to solely evoke a stress reaction while exposure to famine may imply both individual malnutrition and stress. This means that if we find air raid exposure to have an effect on a given outcome, early life stress is likely to be a determinant of this dimension of health. If the same health outcome is not affected by exposure to famine, we may conclude that the stress component of early life famine is relatively small or non-existent or that malnutrition and stress under famine cancel each other out. On the other hand, if air raids do not have an effect on an outcome but famine does, malnutrition should be the driving force.
Our results show that researchers should be cautious with the conclusions drawn from famine studies and that effect components may have differential impacts depending on the outcome of interest. The main findings imply, first, the prevalence of hypertension in male respondents is higher when exposed to air raids in the first months after birth, but does not react to famine. Under the assumption that malnutrition does not have a beneficial effect on hypertension risk but most likely an adverse one (as found in experimental animal studies; see Tonkiss et al., 1998; Ojeda et al., 2008 and references therein), this means that we do not have evidence for the stress component in this famine to be quantitatively relevant. Second, we find that male adult height is significantly affected by famine exposure during gestation but not by air raids. This implies that a famine effect on height is in fact attributable to malnutrition. Since air raids affect hypertension among men, we know air raids to be an informative measure, capturing stress in the population sufficiently precisely. Would height we sensitive to, it would also be significantly associated with air raids. Since we also find evidence for prenatal famine

to have significant effects on pensions of male respondents (a proxy for life cycle productivity), our findings hint at the importance of nutritional shocks for correlates of height, namely dimensions of human capital and thus overall productivity. In general, the effects of stress under the German famine seem to be relatively small. Our findings on the outcome of life satisfaction are less clear-cut.

Our study faces some drawbacks such as measurement issues concerning the food ration data. Moreover, factors inherent in the historical background such as selective mortality and selectivity of the place of birth during the war may pose threats to identification. Conducting a range of robustness checks shows that those issues are unlikely to drive our results. However, we have to note that our results, though internally valid, are context dependent and that other famines may differ in terms of the size of the effect components we try to disentangle. Our work could thus be used as a point of departure for further research in this direction.

4 Unlucky in Life, Lucky in love? - Family Formation after Shocks Early in Life*

4.1 Introduction

Starting a family is an essential and important step in one's life, which people decide about carefully. As important as the consequences on the individual level is the relevance for the whole economy: marital stability and fertility play a major role in determining not only population growth, long-term health and life satisfaction, but also the next generation's basis for a healthy and productive life.

Germany's fertility rate (1.5 in 2015 according to Statistisches Bundesamt (2017)) is far below the replacement rate of around 2.1 children per woman already for decades (Eggen and Rupp, 2007). The long decline of fertility in Germany has shown a few plateaus of higher fertility rates, one of these taking place in the post-Second World War (from now on: WWII) era (Eggen and Rupp, 2007), specifically between 1955 and 1969. The common explanation for this phenomenon is that women delayed child-bearing during WWII ('baby bust') and caught up child-bearing after the end of the war ('baby boom') primarily due to socio-economic insecurity and the scarcity of men. Parallel to these difficult circumstances in child-bearing ages, potential mothers had been exposed to changing societal, economic and psychological conditions already very early in life. Can adverse experiences in the women's childhood explain subsequent patterns in fertility? Not only women, but also potential fathers suffered from war cruelties and famine during their childhood. There is plenty of evidence that men are even more sensitive to adverse conditions early in life. Still, male fertility is often paid less attention to. Yet, this general male sensibility towards adverse childhood experiences could translate into family formation as well.

The onset of fertility and marriage are closely related as conception-out-of-wedlock often bears socio-economic disadvantages for several generations,

*For very useful comments I thank Gerard J. van den Berg, Anna Busse, Hanno Förster, and Laura Pohlan.

see Modin et al. (2009). Plus, hormone based methods of contraception were only introduced to the market in the 1960s. Thus, timing of fertility onset and marriage often highly correlate. But also apart from fertility, the marital status is substantial for individual well-being. Many empirical studies show that marital status is associated with health, longevity and happiness, see e.g. Modin (2003), Waite (1995), and Waite and Lehrer (2003). For men, marriage is known to have a protective effect for their health which decreases mortality (Van den Berg and Gupta, 2015).

It is well-known that adverse early-life conditions decrease health, longevity and economic prospects, see e.g. Case and Paxson (2009) and Van den Berg et al. (2006). There are several – biological as well as behavioural – channels how early life conditions can also exert influence on fertility and marital status, e.g. via attractiveness and fitness, a change in opportunity costs, mortality, impairment of organs which are responsible for reproductive hormones, or altered preferences (see e.g. Boberg-Fazlic et al. (2017) and the review study by Lummaa (2003)). This makes the direction of the effects an empirical question. The analyses following in the next sections primarily focus on the existence of changes in the family formation frontier, whereas I can only speculate on the underlying causes by shedding some light on potential motives and mechanisms.

In the present study, family formation, i.e. marital and fertility biography are associated with experiences in early childhood, caused by WWII. Based on the analysis in Chapter 3, I study whether stress or famine exposure in early life delay, decrease or otherwise alter marriage or fertility. I analyse whether early life stress and early life malnutrition affect marital and reproductive success. I exploit the era around WWII in Germany, with birth cohorts 1930 to 1950, who suffered from Allied bombing and post-war famine. Individual data from SOEP is merged with contextual data on municipal living conditions, i.e. with the intensity and dates of bomb attacks and with food ration card values. Marital and reproductive behaviour is measured late in life, when fertility is completed, by various measures of marriage market success, marital stability and fertility.

Not only completed fertility, but also age at first childbirth and different family sizes are observed concerning reproductive behaviour. And not only age at marriage is analysed for the marital biography, but also divorces and current marital status. An innovative feature in the present analysis is that I additionally evaluate partner quality to check for success on the marriage market beyond the mere fact of marriage and divorce. For this purpose, I observe social class, age and health characteristics from the current partners' SOEP questionnaires. To the best of my knowledge, early life conditions like stress or malnutrition have not been connected yet with such aspects of marital biography.

The data suggest that men and women react differently to early life shocks with regard to family formation later in life. The key finding in this study is that male stress in early life affects the marital biography later in life, at various parameters. *First*, stress exposed men marry younger than their unaffected counterparts. Even more surprisingly, *second*, they are more likely to ever marry and, *third*, also show a higher marital stability. Concerning their partners, the early life stress does not show in the age gap between spouses, but, *fourth* it does improve partners' health: The men's partners are less likely to suffer from hypertension and obesity, as they are substantially more likely to have a healthy body mass index. With regards to malnutrition, instead, the men's success on the marriage market is decreased: The lower the average daily caloric intake, the higher is the chance to marry a woman of lower social status. While stressed boys seem to be somewhat compensated later in life with marital stability, women react differently to adverse experiences in childhood: Those women who experienced a famine in early life, choose a rather older partner for marriage. Also women seem to draw something positive from stress exposure in childhood, as they are substantially less likely to stay childless.

The sections are structured as follows: Section 4.2 shortly describes the historic living conditions for children born around WWII. Section 4.3 gives an overview of the mechanisms found in the literature concerned with family formation. In Section 4.4 and 4.5, I provide an overview of the data at hand and the empirical strategy. Results are presented in Section 4.6, while Section 4.7 concludes.

4.2 Children's Living Conditions 1930–1950 in Germany

In this section, I provide a short summary of the living conditions in Germany around WWII, with a focus on the children's basic needs[1]. Detailed overviews are given in e.g. Chapter 2 and Chapter 3.

The living conditions for children in Germany during the 1930s were satisfactory. Not only nutrition was of good quality according to historic sources, as described in Chapter 3, but also the infant mortality rate was in constant decline, see e.g. Chapter 2 and the references therein. The onset of the war by Germany started a negative development for the civilian society. In the very first years of the war, attacks by the Allied forces were very limited, and nutritional quality could be secured at a high level due to food imports from occupied countries. But in the longer run, the complete focus of resources on

[1]Note: I focus on children born in the area of the current German borders and who live in Germany in 2012 due to data availability. The horrible experiences of millions of other children due to German cruelties cannot be reflected here.

the war industry as well as the fading success in the war led to substantial shortfalls in nutrition and other materials needed for a living as oil and coal, while the air attacks by the Allied forces additionally brought stress and fear to the civilian society. After losing the war, in the period from 1945 to 1950, the civilians faced a time of enormous changes in society and living conditions. The air attacks had ended but new worries took their place for the German civilian society: Food supply was chaotic and extremely low, especially in the cities. Political insecurity, changes in the economic system, and the reception of around 12 million refugees from former German areas led to highly unstable living conditions.

In this paper, I focus on the fear and stress brought by the Allied air attacks on cities during the war as well as the negative consequences for living conditions due to the famine. As described in detail in Chapter 3 and the references therein, there is evidence that the bomb attacks mostly led to stress and stress-related health conditions, since only relatively few people died during the attacks and no other wide-spread consequences, like epidemics or riots, were observed. During famine times, additional to the potential stress mostly malnutrition and its consequences have to be expected. In Chapter 3, we do not find any evidence for a substantial stress component in the German famine.

In sum, it can be assumed that the German civilian society faced rising levels of stress between 1939 and 1945 due to the intensifying war. Starting around 1944, food supply became a severe problem, which could not be solved before June 1948. It is noteworthy that both kinds of adverse condition – hunger and stress – followed sharp limitations not only in temporal perspective, but also in the regional dimension.

4.3 Determinants of Family Formation

This section provides an overview on the family formation frontier in the literature. The focus here lies on roughly describing the fundamental mechanisms which might relate early live experiences of stress and hunger with family outcomes later in life.

First, I show the general pattern of family formation in the last decades in Germany. Second, I summarize different approaches in the literature which analyse the determinants, mainly from population economics and family economics. This I do separately for fertility and marital status. For more detail, please refer to e.g. Becker (1974) for marital status and to the overview study of e.g. Sloboda et al. (2011) for fertility.

There are several potential mechanisms at work that may connect early life conditions and family formation parameters later in life in general.
(1) If environmental conditions early in life are extremely bad, early *mortality* can lead to a selection effect, see Doblhammer et al. (2013) for the example of the 'Great Finnish Famine'. This could translate into advantages among the survivors through relative scarcity (Brandt et al. (2016)) or through immunity effects or 'survival of the fittest'. As described in detail in Chapter 2, infant mortality was very high in Germany during the post-war famine, with maximum values above 20%. Such levels of mortality make selection effects concerning long-term outcomes theoretically possible, see Bozzoli et al. (2009).
(2) Physiologically or psychologically, *scarring* might be a relevant factor: Scarring implies a weakening of the affected individual, e.g. in terms of bodily growth or strength, illnesses, or psychological burdens. Such biological effects were often found in empirical studies, e.g. Bertoni (2015) finds lower subjective well-being after childhood hunger. With respect to fertility or marriage market success, scarring could imply a decrease in attractiveness for the other sex and/or reduced ability to get/raise children among the affected individuals e.g. due to morbidity.
(3) *Opportunity Costs*: Scarring can, however, also work in the opposite direction and lead to higher fertility, earlier marriages or higher marital stability. If the adverse early life experiences decrease productivity, the opportunity costs of child-raising decrease as well. And if outside options are expected to be low within a partnership, one can as well marry early and stay together longer.
(4) Finally, affected individuals could also build altered *preferences* towards marriage and fertility. To sum it up, several mechanisms are conceivable, with different outcomes to be observed.

4.3.1 Fertility Patterns

Fertility levels in many European countries have been very low now for roughly a century (Eggen and Rupp, 2007). A huge branch of literature is engaged in analysing population growth, specifically, the downturn in mortality and following decline in fertility in Europe starting around 1800 (e.g. Omran (1971)). Also the last hundred years show interesting patterns in reproduction levels: Sobotka (2012) studies the fertility decline in Switzerland, Austria and Germany and observes low fertility rates in all three countries already for cohorts born around 1900. According to Frejka and Calot (2001) and Sardon and Robertson (2006), however, the total completed fertility rate in Germany has been around 2.0-2.4 for female birth cohorts of the 1930s, 1.8-2.0 for cohorts born in the mid-1940s and 1.5-1.8 children among cohorts

born in the early 1960s. In the post-WWII era, i.e. the female cohorts around 1935, halted this decline for a short time before continuing downwards.

The baby bust around the Great Depression and World War II is often interpreted as consequence of the traumatic events, missing men and adverse economic conditions, while the following booming economy and men returning home are held responsible for the baby boom in the mid 1960s (Greenwood et al., 2005). Boberg-Fazlic et al. (2017) describe fertility patterns observed around epidemics, specifically for the 1918 'Influenza Pandemic' in Sweden, where a similar reaction in birth rates can be observed. During the epidemic, fertility is *postponed* due to insecurity with regards to mortality and morbidity of adults and child. After the epidemic ends, fertility shows a positive effect, since in addition to the couples who would have conceived normally at that time, the postponed children are born. Boberg-Fazlic et al. (2017) point out that also *replacement* fertility is a common observation after times of increased mortality, where the loss of children or adults motivates couples to replace the lost family or community member by a new child. Please see also the review by Hill et al. (2004), Lindstrom and Berhanu (1999) and Agadjanian and Prata (2002) on fertility effects of wars.

4.3.2 Biological Determinants and Mechanisms of Fertility

Before diving deeper into these specific patterns and potential mechanisms, the all-time determinants of fertility, i.e. the biological determinants, are of interest: Bongaarts, Frank, and Lesthaeghe (1984) argue that proximate determinants of fertility have to be identified to understand the translation of background determinants as socio-demographic characteristics into fertility patterns. Following Bongaarts et al. (1984), the list of proximate determinants of fertility contains the frequency of sexual intercourse, lactational amenorrhoea, contraception, abortion, spontaneous intrauterine mortality, and sterility. For Sub-Saharan Africa, the authors find especially lactational amenorrhoea, post-partum abstinence and pathological sterility to play a major role in reducing fertility.

Jacobsen (1996) argues in a similar direction and identifies marital age, celibacy due to economic conditions and the status of women as significant forces which alter the fertility rate in a society. *Age at first marriage* is found to decrease the number of children, since in traditional societies it often goes hand in hand with a delay of starting sexual intercourse after menarche (see (Jacobsen, 1996, p. 14)). *Economic conditions* in historical Belgium forced 15–18% percent of the society to stay live-long servant and thus single (see (Jacobsen, 1996, p. 14)), which also mechanically depreciates fertility in the population.

Finally, Jacobsen (1996) finds high *social status of women* to statistically be correlated with low fertility, as the opportunity costs of time investment in child rearing rise with social status and wage.

Also the economic literature offers very vigorous models of fertility, see e.g. Becker (1960). Becker (1960) offers an economic model of fertility, where income, child costs, uncertainty, taste and knowledge are considered. Becker (1985) explains the changes in fertility in the post-WWII era by opportunity costs. Costs of producing and rearing children rise with labour market opportunities of women, and the rising costs reduce the demand for children. A rise in higher education in women may also have a delaying effect on fertility, see Blossfeld and Jaenichen (1992).[2]

To put it in a nutshell, fertility is effectively altered by diverse natural changes of behaviour or biological conditions. In the context of WWII, the proportion of women in sexual union, the frequency of sexual intercourse or marital age might be obvious mechanisms shaping the fertility patterns described above. Lactational amenorrhoea might also play a role during famine times as breast-feeding might be intensified and prolonged to secure the nutrition of the babies. Yet, these biological determinants do not give any obvious insights into how fertility behaviour might change in response to early child hood events. Both stress and malnutrition can affect the fertility of the analysed cohorts through the aforementioned mechanisms. Both can influence fertility via mainly behavioural changes, e.g. marital age, celibacy due to economic conditions, or frequency of sexual intercourse, or via mainly biological channels as the pathological sterility rate or spontaneous intrauterine mortality. In addition to the determinants from the literature, the use of modern contraceptive methods is certainly also relevant for the fertility among cohorts born in 1930 to 1950 in Germany. If fertility is observed to change due to early life events, marital biography might help to identify major pathways, as marital biography is likely to go hand in hand with sexual behaviour and thus gives insights into the described behavioural pathways.

4.3.3 Early-Life Determinants of Fertility

In addition to the summarized literature on biological fertility mechanisms which are often applied in developing countries, there is also ongoing research to analyse individual determinants of fertility in industrialized countries. In the present study, I analyse whether – so to say in the shadow of major historical events – individual experiences made in early childhood or even during pregnancy can explain fertility differences later in life. Early life

[2] These economic approaches can be translated into "proportion of women in sexual union", i.e. frequency of sexual intercourse of Bongaarts et al. (1984) or "status of women" of Jacobsen (1996).

experiences can be thought of as underlying sources of the before-mentioned biological fertility mechanisms.

Sloboda et al. (2011) review studies of early-life environment, especially nutrition, and its effect on female reproduction – called *nutritional reproductive programming*. They find nutrition during the whole developmental lifespan to play an "integral role" for reproduction (Sloboda et al., 2011, p. 210). E.g. nutrition during the prenatal time window can affect female reproduction via age at menarche (see Sloboda et al. (2007)), age at menopause (see Elias et al. (2003)) or reproductive functioning and health (see Elias et al. (2004); Cooper et al. (1996)). Furthermore, there is evidence of early malnutrition in general leading to a change in age at menarche and to early menopause (see Osteria (1983); Riley (1994); Kirchengast and Winkler (1996); Lindstrom and Berhanu (1999)). Through the pathway of age at menarche, nutritional supply can also affect age at marriage and age at first birth (see Riley (1994)).

Still, there are also studies in which no or limited effects of malnutrition early in life are found: Lumey (1998) finds changed reproductive function but not long-term fertility to be associated with early life nutritional problems. Van den Berg and Gupta (2015) and Lumey and Stein (1997) find adverse economic conditions or famine in the time around birth not to significantly affect age at first delivery. Also Lumey and Stein (1997) do not find an effect of maternal nutritional deprivation on family size, age at first delivery, the proportion staying childless, or on age at menarche.

4.3.4 Importance and Contemporary Patterns of Marital Biography

Parallel to fertility, I also analyse marriage market success of the cohorts born around WWII. This is interesting, since marriage – or more broadly: relationships – are highly informative about the individual 'marriage market power' but also its plans for the future. For instance, the marital status of an individual gives insight into the person's attractiveness as a partner as well as into his or her social abilities and preferences. Adverse psychological and physical conditions in early childhood affect those characteristics, see Chapter 3. Marriage both highly depends on the economic situation of the two spouses and determines their later wealth, see e.g. Becker (1974), Laslett (1977) and Dennison and Ogilvie (2014). There is plenty of evidence that marriages additionally affect later life health, happiness, and of course, fertility, see e.g. Waite (1995), Waite and Lehrer (2003), Frey and Stutzer (2010), Kohler et al. (2005), and Layard (2005). This makes the marital biography a highly relevant outcome variable.

Similar to baby boom and baby bust, several countries faced also boom and bust in marriage numbers during the 20th century, see Caldwell et al. (1988):

After WWII, age at marriage decreased and the trend went towards universal marriage – 'marriage boom'. Starting in 1960, the time between marriage and birth of the first child lengthened, while age at marriage started to rise again and marriage became less frequent in early ages. Plus, divorce rates rose steeply - 'marriage bust'.[3] In numbers, the Federal Institute for Population Research calculates an average age at first marriage among women in 1960 of 23.5, which decreases to the minimum of 22.3 years in 1975, and increases again afterwards to 22.9 years in 1980 and 25.2 years in 1990, (see Bundesinstitut für Bevölkerungsforschung (2017)).

4.3.5 Insights into Marriage and Divorce Determinants from the Literature

The literature knows several general determinants of marriage timing and marital stability. As noted before, Gary Becker for sure introduced one of the most prominent models among economists with his "Theory of Marriage", see Becker (1974). Following this theory, the rise in women's labour market opportunities after WWII led to a decrease in incentives to marry, as the traditional sex-specific division of labour loses ground, see Becker (1981). Based on Becker's theory of marriage, Caldwell et al. (1988) empirically find a delay in marriage due to rising opportunity costs of the women because of increasing female wages during the 1960's; another big chunk of the increase in age at marriage can be explained by an unfavourable sex ratio for women: missing men made the search for a partner more difficult and lengthy, leading to an additional delay. During the 1950's instead, the female wage ratio was low, and also the sex ratio was more favourable, causing earlier marriages. Another phenomenon Caldwell et al. (1988) identify is the later marriage among educated women. Also military service is found to matter on the men's side. But apart from these rather hard facts, big differences are found in age at marriage between groups of women with different aspirations – planning to become housewife by the age of 35 is associated with a lower age at marriage. Also social phenomena can play a role, according to Caldwell et al. (1988): While in the first time after WWII, people married earlier to demonstrate independence from the parents, somewhat later marriages were delayed due to a risen importance of individual goal attainment, which needed to be bargained between men and women before marriage. Becker et al. (1977) furthermore name incidental premarital conception, high costs of time, optimism about the present marriage offers (or pessimism about future offers) as possible determinants of early marriage versus late marriage. These determinants are also relevant for the divorce

[3] It is worth noting that 'marriage boom' and 'bust' do not completely coincide with 'baby boom' and 'bust'.

risk, as Becker et al. (1977) point out[4]: If individuals paid a lower cost of search and married early, the risk of marital dissolution is higher, since the gains from staying married may be lower than the gains from dissolution in this case, due to partner mismatch. This also holds true, if partners were overly optimistic about partner's quality in the beginning, e.g. due to their young age. At the same time, marrying late does not guarantee staying together: the expected number of years within marriage decreases and thus also less favourable matches become acceptable. By this, however, future gains from staying married decrease and dissolutions get more attractive.

4.3.6 Early-Life Determinants of Marriage

With his popular theory of marriage, Becker (1974) brought attention to selection into marriage, which many researchers added evidence to; selection criteria range from social background, race, ethnic origin, to health, age, education, attractiveness, and occupation. Especially health, education, and attractiveness, could be diminished by scarring adverse conditions early in life.

There is also evidence of a *protective* marriage effect. This means, a spouse can offer stress-reducing comfort, care when ill and encourage a healthier lifestyle, which translates into better health and higher productivity later in life (see Murray (2000)). Van den Berg and Gupta (2015) find this protective effect against e.g. cardiovascular diseases to be greater for men who suffered from adversities early in life.

Furthermore, Phillips, Handelsman, Eriksson, Forsen, Osmond, and Barker (2001) find marriage rates to be correlated with early life conditions measured by birth weight. Phillips et al. (2001) hypothesize that early life conditions (intra-uterine growth restrictions, lower social class) partly determine the marriage rate later in life through effects on personality and socialization.

Van den Berg and Gupta (2015) and the references therein indicate socioeconomic status of the parents – measured by occupational class, age, as well as region of origin – to be important determinants of marital status for birth cohorts in the Netherlands from the early 20th century. They also note that conception-out-of-wedlock could determine age at marriage as well, since out-of-wedlock births bore negative consequences for both mother and child.

[4] In general, Becker et al. (1977) state that divorces should not be seen as marital failure but as a response to new – favourable or unfavourable – information. The possibility to divorce enables individuals to react to new information and thus increase future utility.

The phenomenon of early life famine has been studied before, regarding later life marital status and fertility, e.g. by Brandt et al. (2016) considering the 'Great Famine' in China: They find three effects of early life famine exposure for marriage market success:
(1) The marriage market attractiveness of the famine-born cohorts suffers due to negative health effects. This reduces the demand for famine-born spouses.
(2) Due to their relative scarcity on the marriage market (the fertility during famine-times was substantially reduced), their marriage market attractiveness goes up.
(3) Due to their relative scarcity on the labour market, the famine-born cohorts earned relatively high wages, which also increased their attractiveness on the marriage market. In sum, the negative health effects weighed more than the gains from their relative scarcity, leading to a small decline in marriage rates for the cohorts affected by the Great Famine. Brandt et al. (2016) identify the age gap between spouses as a way to respond to the relative scarcity. Also in Germany, birth rates declined a lot during the war and the following famine. Thus, relative scarcity effects are likely to occur, too.

Altogether, age at marriage can thus be influenced both positively and negatively with respect to adverse early life conditions. On the one hand, selection via health and social status or altered personality or socialization may reduce the likelihood or lead to a delay in the timing of marriage. On the other hand, a fragile health can also increase benefits from marriage and thus enhance early marriage, especially among men. The general equilibrium effects of the underlying shock – which led to the adverse early life conditions – can partially offset the negative marriage market consequences due to relative scarcity of the affected cohorts.

4.4 Data and Descriptive Statistics

The data set has been used in Chapter 3 before, thus the description here is kept short: Individual data on birth place and year, and also well-being later in life stem from the Socio-Economic Panel SOEP (2013) and are combined with contextual information on the municipal level capturing early life conditions, collected from historical sources. Section 3.4 describes the preparation and combination of data sources in detail.

4.4.1 Individual-Level Data

The outcome variables of interest from the SOEP are collected in 2012 mainly, since the place of birth was newly introduced to the questionnaire in that year, which is essential for the connection with early life shocks. Adding

information from other years bears the disadvantage of losing respondents selectively. The outcome information cover on the one side relationship parameters from the *marital biography* section and the personal section, and on the other side information on fertility, mainly from *biokid*.

To measure fertility, different approaches are possible, mostly age at first birth and number of children are used. As Kohler et al. (2006) point out, completed fertility (as contained in number of children) is not only a measure of biological fertility, but also mirrors behavioural and cultural norms of the individual and society as a whole. Early fertility instead, as pointed out by Rodgers et al. (2001), may be more affected by genetic influences than completed fertility. Thus, *age at first birth* complements *number of children* in this study to shed light on two different aspects of fertility. It is possible that different social norms exist about "optimal" family size for different social classes, and thus fertility effects may be non-linear. Plus, several types of households may exist. Staying *childless* is a completely different lifestyle than having a single child, two children, or being a child-rich family. If only some of these specific family outcomes are affected by early life shocks, this would not necessarily be visible when only looking at the number of children. Thus, I also test whether the shares of the described types vary when hit by an early life shock. Another interesting outcome, sex composition of children, can not reasonably be analysed due to the relatively small sample size.

Age at first birth is calculated using the information from the children's questionnaire, indicating birth year, birth month, and sex of the child, ordered by age and marking the person identifier. The data offer a trade-off between preciseness and completeness, since only a subset of the sample includes birth month of the child. In this study, I calculate the age at birth in months whenever available, but include the other incidences in complete years, too. *Number of children* is taken from the variable $sumkids$ in the birth biography section of the SOEP, giving the total number of births. For the different family sizes, the same source is applied.

For the couple and marriage history of an individual, mostly age at marriage is applied in the literature: To analyse long-run effects of early life shocks on family formation, age at marriage is interesting, since social norms often propagate a customary age window for marriage. Deviations from this 'optimal marriage age' in both directions can be telling. As described in section 4.3.1, age at marriage is also relevant to explain fertility, since it is a proxy for time in sexual union. If early life shocks affect the age at marriage, they can indirectly also exert influence on fertility. Also the other direction is possible, though. Additionally, age at first marriage determines the starting point of the potential protective effect of marriage for later life health.

Age at first marriage is defined as the age, when the first marital spell begins. Information for this is taken from the SOEP biography questionnaire. This

data set covers spells of marital status like 'unmarried', 'married', 'divorced', 'married and separated', 'divorced' or 'gap' for unknown spells. Every respondent is asked for the last three marriage spells, their beginning and end dates are contained. Thus, I do not always observe the first marriage for a given person if more than three marriages took place. Furthermore, spell begin and end are not always known. Due to these data issues, I exclude everybody from this analysis, where I can not be sure to observe the first marriage or the age at first marriage (i.e. the change from 'unmarried' to 'married' is not available in the data, or the age is missing, or a 'gap' spell lies between the stage 'unmarried' to 'married') This excludes roughly 50 people from the specific analysis and thus should not drive the results. Since a spell of 'unmarried' is always generated to lie before the first 'married' spell, all second spells with known age at marriage can be taken as first marriages. Age at marriage is skewed and has a longer tail at the older ages. Thus, I drop observations which differ more than two standard deviations from the mean. For males and females, mean and standard deviation are calculated separately.

Another interesting aspect of couple and marriage history is marital stability. The observed increase in marital instability can be associated with a different family behaviour, as divorce became a realistic scenario for marrying couples, which may e.g. reduce fertility, see Becker et al. (1977). Furthermore, while marriages have a positive effect on life satisfaction, divorces are found to have significant negative effects, see Zimmermann and Easterlin (2006). To measure marital stability, a dummy is generated that indicates having *been divorced* at least once. Another indicator for marital stability is, whether the individual *lives in a marriage today* and cohabits with his/her partner. Additionally, a dummy is generated indicating whether an individual has *ever married* or stayed single (until the cut-off age that identifies and excludes outliers). Finally, I include a 'softer' measure for marital success: self-reported satisfaction with the family life, where the respondents can report between "0" (not satisfied at all) and "10" (completely satisfied).

4.4.2 Contextual Information on Early-Life Events

This study largely draws on the data set-up in Chapter 3. Therefore, I only shortly describe the early-life events and the instrumentation in the present study. A detailed description of data sources and preparation is given in Section 3.4.2. The birth cohorts between 1930 and 1950 lived in very changing times, with partly abrupt changes in quality of life, see Section 4.2. I use two distinct external influences on family life and thereby early life experiences, which together give insight into the basic living conditions at given place and time: nutritional supply and bomb attacks, both at the regional level of

the municipality of birth of the respondent. Nutritional supply is measured by daily caloric intake for a normal consumer, according to the monthly allowance card in a given city. Since data availability is not complete, we apply some calculation to estimate the missing time and region slots in Chapter 3. Bomb attacks are measured by short tons of bombs dropped by the Allied aircraft on a given city in a given month. These two dimensions shed light on the deprivation of some of the most basic needs: nutrition and safety, see Maslow (1943). To cover the whole period of early life time as sensible age, a broader age window for potential harmful events is taken account of in contrast to Chapter 3. Additionally, early-life events are not divided in prenatal and post-natal, but both periods are taken as one. Namely, all adverse early-life events of the described types are taken into account which happen in the first 1,000 days of life, i.e. between conception and the second birthday. The reason for this approach is that e.g. brain growth and plasticity are found to be highest during this age span, see Cusick and Georgieff (2016). Additionally, a longer single age window makes it easier to deal with the timing of historical events as famine or bombs, since specific birthdays are unknown and I thus can circumvent the risk of assigning a historical event to the wrong age window. *Daily average caloric intake* thus gives the food supply in 1000 calories for a normal consumer, averaged over 33 months: nine months of pregnancy and 24 months of childhood. *Bombs per capita* gives the bomb load in short tons dropped on the home municipality, divided by the number of residents, summed up over the same 33 months. As described before, the long-term effects of the bombs for individuals can be simplified to mostly stress and anxiety, see Section 4.2 and for more details Chapter 3.

4.4.3 Characteristics of the Data Set

The descriptive statistics for the observed sample are given in Table 4.1.[5] The sampled average man is born in 1941, with 21.6% having a father with more than basic school education, and 58.5% being born in municipalities with at least 10,000 residents. 12.9% experienced bombing and 30.3% at least one month under famine conditions during the first thousand days of their lives. Almost 97% of the men married at least once in their lives, on average at the age of 25.5 years. 19.8% have been divorced at least once, but in 2012 87.6% are married and cohabiting with the partner. In the mean, men report their first child to be born at the age of 28.5, while in total they report 1.4 children. 28.3% of the men stayed childless according to the data.

[5]The Table of descriptive statistics is restricted to the sub-sample of individuals who are represented by Tables 4.2 and 4.3, i.e. who have non-missings in the variables of at least one main estimation.

The women in the sample show some relevant differences. During childhood, however, the similarities are striking: They were also on average born in 1941, and a similar share of 19.5% has fathers with higher school education. Also 56.6% were born in cities, 15.3% experienced bombing and 31.4% at least one month with less than 1500 kcal a day in their first thousand days of life. In contrast to men, women marry younger, at on average 22.5 years. Similar to men, however, almost 97% of the women marry eventually, 19.2% divorced at least once and 83.6% are married and cohabiting when asked in 2012. Fertility-wise, women substantially differ from men: They bear their first child at the age of 24.2 years and get on average 1.94 children. Only 10.2% of the women are recorded as childless.

There are several possible reasons why male and female statistics on marriage and fertility do not perfectly match. First, the data show the widespread fact that men marry later than women, see e.g. Bergstrom and Bagnoli (1993). The consequence for the sampled individuals is that the average aged man and the average aged woman in the sample do normally not form a couple and thus do not show the same characteristics in their marital and fertility biography, since they belong to different marriage cohorts. Second, it is conceivable that the data quality for male fertility is worse than for women in the way that SOEP questionnaires do not identify all children on the male side but have better success on the women's side (Goebel, 2017, p. 120 f). Thus, the data set can falsely end up with fewer children for men than for women.

4.5 Estimation Strategy

The present study uses both temporal and spatial variation of the two adverse influences – bomb attacks and deficient food supply – to identify individuals with negative early life experiences. Chapter 3 describes in great detail, how the individual is matched with the regional information on bomb attacks and famine intensity. There, also the differentiation between effects of stress and effects of malnutrition is outlined.

Similar to Chapter 3, the main regressions are based on the following equation:

$$y_i = \alpha + \theta N_i + \phi B_i + \delta X_i + \epsilon_i. \tag{4.1}$$

y_i is the outcome of interest – various measures of family formation. N_i gives the average nutritional quality for a normal consumer within the first 1000 days of life in the specific region. The estimated daily calorie level separates between city and rural area. B_i shows the amount of bomb material dropped on the specific city during the first 1000 days of life, measured in short tons

Table 4.1: Descriptive statistics by gender

	Men			Women		
Variable	N	Mean	s.d.	N	Mean	s.d.
Age at 1st birth	1333	28.476	5.667	1705	24.174	4.396
# children	1864	1.443	1.213	1904	1.943	1.174
0 children	1864	0.283	0.451	1904	0.102	0.303
1 child	1864	0.230	0.421	1904	0.225	0.418
2 children	1864	0.316	0.465	1904	0.425	0.494
3+ children	1864	0.171	0.376	1904	0.248	0.432
Marrying age	1708	25.522	3.891	1732	22.460	3.171
Married 2012	1696	0.876	0.330	1455	0.836	0.370
Divorced	1804	0.198	0.399	1830	0.192	0.394
Never married	1856	0.033	0.178	1894	0.037	0.189
Satisf. family life	1820	8.224	1.726	1857	7.989	1.921
Bombs p.c. (lbs)	1864	4.826	33.633	1904	5.036	24.012
Bombs $> 0(1/0)$	1864	0.129	0.336	1904	0.153	0.360
≥ 1 mo < 1500 kcal$(1/0)$	1864	0.303	0.460	1904	0.314	0.464
Daily avg kcal	1864	2318.944	334.889	1904	2312.783	336.183
Year of birth	1864	1941.560	5.708	1904	1941.763	5.638
Father high educ.$(1/0)$	1864	0.216	0.412	1904	0.195	0.396
Birth place: city$(1/0)$	1864	0.585	0.493	1904	0.566	0.496

Source: own calculation based on Socio-Economic Panel SOEP (2013).

(one short ton equaling 2000 lbs or about 907 kg). Both N_i and B_i are taken at their nominal level to make use of the maximum of available information. X_i depicts the control variables: birth year as a linear trend, size of home town as dummy for at least 10,000 residents in 1939, social background as measured by father's education level being higher or equal to *Realschule* – the medium track qualifying for apprenticeships. ϵ_i captures the i.i.d. error.

In a first step, I analyse the effect of early life shocks on family formation later in life by regarding the standard measures for family formation depicted before: fertility in the form of age difference to the oldest child and the number of children ever born; marriage success measured by age at marriage in years and months, and measured by satisfaction with the family life.

In a second step, I broaden the focus and search for more subtle changes, which may serve as underlying explanations for the results in standard measures, and also may allow additional insights into the family formation. Here, shares of family types, current marital status, divorces and singles are considered. Brandt et al. (2016), Phillips et al. (2001) and Scholte et al. (2015) argue that early life conditions shape the marriage rate later in life due to effects on attractiveness to the other sex, e.g. due to socialization and personality changes or poor health. Also, the variable *age at marriage* is broken down in two additional measures: A dummy, which indicates whether the respondent married within the 'normal' marrying age range, i.e. the marrying age lies between the 25% and 75% percentile; the absolute deviation

from the average marrying age. Both are calculated separately for the two sexes as the average marrying age range differs. These two variables are thought of indicating whether a respondent fulfilled the social norms with his or her marrying age, as deviations from the customary marrying age indicate a lower ranking on the marriage market, see also Scholte et al. (2015) and Brandt et al. (2016).

Finally, in a third step, I shed light on another dimension of marriage success: partner's 'quality'. Until now, only the existence of a partner is studied. To the best of my knowledge there are no previous studies focussing on the relationship between early life conditions like stress or hunger exposure and partner quality in later life. However, quality differences can appear, be it a better or worse matching quality or differences in the general attractiveness and ability of the partner, see e.g. Becker et al. (1977). The idea is that the partner's quality (e.g. health, social status, or age) reveals an additional sphere of the respondent's marriage success. Looking at the spouse's characteristics adds another aspect which may show effects of early-life shocks and may potentially explain relations observed in the earlier steps. This information is also taken from the SOEP, since it tries and covers all persons in a given household. Starting at the respondents personal questionnaire, the partner's ID is given for all respondents that cohabit. Thus, I can match some relevant information from the spouse to the original respondent's early-life experiences: Birth year and month are highly interesting, since age differences between spouses are known to vary with the marriage market success, see Neelakantan and Tertilt (2008). Also the social origin of the spouse is highly informative since the social status of a newly formed family can largely depend on the spouses' social origins, here measured by the educational level of both the parents. A treatment effect in this sphere would hint at a step up or down in the social classes via marriage, caused by early life experiences. The last dimension of partner quality observed here is partner's health: As in Chapter 3, high blood pressure, life satisfaction and height are taken as indicators of late life health. Especially height and life satisfaction are well known for their predictive power with respect to future well-being, whereas high blood pressure is relevant for its high prevalence rate in older people. Additionally, I analyse three slightly different measures of being overweight or underweight: the body mass index (BMI) itself, as well as a dummy generated to mark an acceptable BMI range of the partner. The BMI is often used in the medical literature to predict health quality. It may also be interesting as a very rough scale of physical attractiveness to the other sex, see Tovée et al. (1998). Since these health parameters are reported after starting the marriage, they may well be affected by experiences during marriage. In this case, these health measures would not be an indicator for marriage success of the partner. At most, they could hint at a certain health behaviour during marriage life.

Of course, the partners can be influenced by the same adverse events – bombs and famines – in their childhood. This can be problematic to the analysis since spouses, who were exposed to bad conditions themselves, can suffer from decreased height, from an increased risk of hypertension or from decreased productivity, and this could affect their marital or reproductive behaviour. It would be beneficial to consider this in the analysis. However, *first*, the data source on early life conditions is limited to births between 1930 and 1950 within the current German borders. Since the spouses of the respondents can be older or younger or be born outside Germany, the sample size would be reduced. *Second*, this additional selection criterion (availability of contextual information on the spouse) is non-random. *Third*, other dependencies would make the interpretation cumbersome: The partners' early life conditions as additional *explanatory* variables would not necessarily be independent from the respondent's own early life conditions (with dependent variables: e.g. fertility, and explanatory variables: own famine exposure, partner's famine exposure, own bomb exposure, partner's famine exposure, year of birth, father's education, size of home town). Taking the partner's early life conditions as *dependent* variable as additional signal of his/her quality, Y (partner's early life conditions) could also depend on X (the respondent's early life conditions) through other mechanisms than marriage success – e.g. regional dependence in partner search, or desired marital age gap – and thus not reveal that much about the respondent's marriage success. Nevertheless, the potential early life exposure to adverse events might have shaped their long-run health and productivity as well. So, the presented outcomes late in life can be influenced by their (unobserved) childhood events.

4.6 Estimation Results

4.6.1 Main analyses

Table 4.2 shows the estimation results for equation 4.1 for men, and Table 4.3 for women, with outcome variables age at marriage, number of children and age at first birth. Bomb exposure in the city of birth during the first 1,000 days of life plays a significant role for the age at which men marry on average. The more bombs are dropped on the specific city during this time frame, the more stress the children might have felt. Interestingly, this stress exposure in early life did not affect women and men similarly, but only men show a difference significant at 10%. As described before, there are different explanations possible for this male behaviour. Infant mortality might work in favour of marriage market or labour market prospects of the surviving men, which could lead to early success on the marriage market due to the marital age gap. Also scarring could be an explanation, as the affected men

Table 4.2: Main regression results – males

Variable	Marrying Age	# Children	Age at 1st Birth
Daily avg kcal in 1000	-0.249	-0.001	-0.421
	(0.304)	(0.085)	(0.487)
Bombs p.c.(t)	-4.936*	-0.656	-8.037
	(2.625)	(1.355)	(8.500)
Year of birth	-0.042**	-0.007	0.051*
	(0.019)	(0.005)	(0.028)
Father high educ.(1/0)	1.222***	0.141**	1.551***
	(0.242)	(0.068)	(0.385)
Birth place: city(1/0)	0.012	-0.142**	0.011
	(0.191)	(0.058)	(0.317)
Constant	107.115***	15.532	-69.168
	(37.288)	(10.673)	(54.766)
R^2	0.020	0.006	0.018
F-Stat.	6.932	2.147	4.694
N	1708	1864	1333

Note: robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively. Source: Own calculations based on Socio-Economic Panel SOEP (2013).

Table 4.3: Main regression results – females

Variable	Marrying Age	# Children	Age at 1st Birth
Daily avg kcal in 1000	0.345	0.085	-0.028
	(0.234)	(0.072)	(0.357)
Bombs p.c.(t)	-0.439	2.716	6.776
	(4.977)	(2.035)	(7.831)
Year of birth	-0.073***	-0.022***	-0.082***
	(0.015)	(0.006)	(0.020)
Father high educ.(1/0)	1.667***	-0.148**	2.218***
	(0.203)	(0.066)	(0.283)
Birth place: city(1/0)	-0.081	-0.159***	-0.027
	(0.153)	(0.056)	(0.210)
Constant	163.840***	44.267***	183.483***
	(28.885)	(10.829)	(39.458)
R^2	0.059	0.021	0.048
F-Stat.	20.570	7.102	16.454
N	1732	1904	1705

Note: robust standard errors in parentheses. *,**,*** indicates significance at the 10%, 5%, and 1% level, respectively. Source: Own calculations based on Socio-Economic Panel SOEP (2013).

might expect lower outside options to a certain spouse than unaffected individuals. Scarring could also work in favour of young marriages via shorter educational attainment, since having finished the education and earning own money could be a prerequisite for marrying. Finally, the utility drawn from marriage may be especially high for the affected men, see Van den Berg and Gupta (2015), making a young marriage especially desirable. For women, there is no evidence that stress affects age at marriage. The estimates for nutritional shortcomings are insignificant for both sexes.

Column (2) and (3) show that neither the nutritional situation nor stress exposure in early childhood significantly affect fertility in later life, neither for men nor for women. The number of children as well as the age at first childbirth are only insignificantly altered. This is especially interesting for men, since often-times age at marriage determines the onset of fertility, see Jacobsen (1996). In this sample, however, marriage and fertility seem to be less connected at first glance. Yet, the effect of bombs on the age difference to the first child is still sizeable for men and in the same direction as for the marrying age. Additionally to the estimates shown in tables, I considered the temporal distance between marriage and onset of fertility as depending variable. The relation between marriage-to-fertility distance and early life conditions is insignificant. The direction of the coefficients indicates: the worse the childhood conditions, the longer the time between marriage and birth of a first child. Taken together, it seems that early life stress leads men to marry earlier but to get children only insignificantly earlier than unaffected individuals. Becker (1985) states premarital pregnancy as one possible condition favouring early marriage. Having only insignificant effects of early life conditions on the marriage-to-fertility distance, the data does not support this explanation. Data quality issues might play a role here, since fertility is less well reported among men than among women in the SOEP.

4.6.2 Education and Satisfaction with Family Life

Table 4.4 gives the estimation results of educational attainment as outcome variable. The idea is that age at marriage often depends on the ability to generate own income. Prolongued education, for example high school or university attainment, delay entry into the labour market and thus can also delay marriage. The estimates show that neither the time spent in education nor the educational level can explain the early marriage of men who were stressed early in life. *High Educ.* is a dummy variable indicating whether the respondent reports a degree higher or equal to *Realschule* – the medium track qualifying for apprenticeships – similar to *Father high educ.(1/0)*.
Years in education gives the number of years the respondent reports to have attained school, university etc. Among the male respondents, neither stress

Table 4.4: Educational attainment

	Men		Women	
Variable	High Educ.(1/0)	Years in Education	High Educ.(1/0)	Years in Education
Daily avg kcal in 1000	0.041	0.035	-0.063**	-0.403**
	(0.032)	(0.196)	(0.028)	(0.185)
Bombs p.c.(t)	-0.014	2.594	-0.263	1.979
	(0.414)	(3.219)	(0.829)	(5.841)
Year of birth	0.002	0.030**	0.002	0.056***
	(0.002)	(0.012)	(0.001)	(0.010)
Father high educ.(1/0)	0.408***	2.809***	0.335***	2.409***
	(0.027)	(0.168)	(0.027)	(0.172)
Birth place: city(1/0)	0.038*	0.378***	0.032**	0.320***
	(0.020)	(0.125)	(0.016)	(0.107)
Constant	-3.172	-47.278**	-3.935	-96.460***
	(3.685)	(23.326)	(2.874)	(19.580)
R^2	0.146	0.177	0.149	0.187
F-Stat.	50.486	65.936	37.428	60.595
N	1830	1847	1853	1878

nor nutrition significantly affect educational decisions. Thus, the relation between early life stress and age at marriage cannot be explained by a shorter time in school.

Interestingly, women do react to early life conditions in their schooling decision: Nutritional deficits in early childhood can be associated with both longer time in education and higher schooling degrees. It would be fruitful to dig deeper into this surprising relationship in future work to search for explanations for these findings.

Table 4.5 gives a 'softer' measure of success at the marriage market: It shows the satisfaction with the family life as reported by the respondents. This measure is likely determined during marriage, not before. Due to the association between younger marriage and higher divorce rate, one would probably expect people with adverse early life conditions to be less satisfied or even disappointed with their chosen partner. The contrary is the case: Stress insignificantly increases the satisfaction level. Looking at famine exposure, this positive effect is even highly significant. The more intense the famine exposure in the first 1,000 days of life, the higher is the reported satisfaction with family life among the men. For women, the coefficients go in the same direction, but are not significant.

4.6.3 Additional Analyses on Marital and Fertility Biography

To analyse potential underlying mechanisms of the decrease in marriage age among men and to search for further effects of early life experiences on marital and fertility biographies, I observe additional variables from the spheres of marriage and fertility. Tables 4.6 and 4.7 show the effects of early-life stress

Table 4.5: Satisfaction with family life (0-10)

Variable	Men: Satisfaction Family Life	Women: Satisfaction Family Life
Daily avg kcal in 1000	-0.370***	-0.200
	(0.113)	(0.134)
Bombs p.c.(t)	0.222	0.273
	(1.848)	(3.364)
Year of birth	-0.016**	0.005
	(0.008)	(0.009)
Father high educ.(1/0)	0.060	-0.023
	(0.103)	(0.119)
Birth place: city(1/0)	0.043	-0.054
	(0.083)	(0.092)
Constant	40.611**	-1.754
	(16.137)	(17.416)
R^2	0.006	0.002
F-Stat.	2.447	0.839
N	1820	1857

Note: robust standard errors in parentheses.
*,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

and nutrition on specific variables of marriage, and Tables 4.8 and 4.9 go deeper in analysing fertility effects.

Marital Biography

Table 4.6: Marital biography – males

Variable	Marrying Age 25%-75%	Deviation Marrying Age	Married in 2012	Never Married	Divorced
Daily avg kcal in 1000	-0.032	0.446**	0.006	-0.007	-0.042
	(0.037)	(0.186)	(0.027)	(0.014)	(0.033)
Bombs p.c.(t)	0.722	-1.156	0.457*	-0.173**	-0.673*
	(0.623)	(1.832)	(0.234)	(0.085)	(0.367)
Year of birth	-0.002	0.034***	-0.005***	0.000	0.010***
	(0.002)	(0.011)	(0.001)	(0.001)	(0.002)
Father high educ.(1/0)	0.024	-0.050	-0.003	0.001	0.038
	(0.028)	(0.152)	(0.020)	(0.010)	(0.024)
Birth Place: city(1/0)	0.002	0.160	-0.038**	0.006	0.047**
	(0.023)	(0.117)	(0.016)	(0.009)	(0.019)
Constant	5.229	-64.785***	10.638***	-0.311	-18.238***
	(4.117)	(22.337)	(2.910)	(1.398)	(3.452)
R^2	0.002	0.008	0.010	0.001	0.028
F-Stat.	0.760	2.556	3.883	0.910	11.222
N	1864	1708	1696	1856	1804

Note: robust standard errors in parentheses.
*,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

Tables 4.6 and 4.7 give insights into additional aspects of the marital biography of the observed individuals. Column (1) shows the probability of having married in a common age window, where common means the age between 25th and 75th age percentile. Column (2) gives the absolute deviation from

Table 4.7: Marital biography – females

Variable	Marrying Age 25%-75%	Deviation Marrying Age	Married in 2012	Never Married	Divorced
Daily avg kcal in 1000	0.012	0.085	-0.026	0.012	0.010
	(0.037)	(0.144)	(0.028)	(0.011)	(0.031)
Bombs p.c.(t)	-0.131	-6.458**	0.723	-0.183	-0.904
	(0.948)	(2.860)	(0.665)	(0.166)	(0.690)
Year of birth	-0.002	0.017*	0.002	-0.001	0.008***
	(0.002)	(0.009)	(0.002)	(0.001)	(0.002)
Father high educ.(1/0)	0.011	-0.165	-0.049*	0.017	0.053**
	(0.029)	(0.131)	(0.025)	(0.013)	(0.025)
Birth place: city(1/0)	-0.021	0.147	-0.000	0.001	0.038**
	(0.024)	(0.093)	(0.020)	(0.009)	(0.019)
Constant	4.138	-30.627*	-3.928	2.140	-16.196***
	(4.275)	(17.294)	(3.877)	(1.523)	(3.437)
R^2	0.001	0.006	0.006	0.003	0.019
F-Stat.	0.410	2.396	1.578	1.419	7.513
N	1904	1732	1455	1894	1830

Note: robust standard errors in parentheses.
*,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

the mean age of marriage. Both columns analyse whether, apart from the average age at marriage, the deviation from the customary age when marrying is determined by early-life experiences. There is a slight indication that adverse conditions in the first 1,000 days of life make people deviate less from social norms, as a low caloric intake among men and a high stress load among women are significantly associated with smaller absolute deviations from the average age at marriage.

Columns (3),(4) and (5) all belong to the sphere of 'successful' marriage life, assuming that most people would prefer to live in a stable and long-lived marriage. According to Becker et al. (1977), marrying early is often associated with a higher risk of marital dissolutions. One possible explanation is a primarily higher searching cost, which leads to both earlier marriage and a higher risk of divorce due to disappointment. In Column (3), the dependent variable is a dummy for being married and cohabit in 2012. Column (4) is a dummy for never having married, and Column (5) for having been divorced at least once. For women, there is no significant effect of early life experiences on marriage life later. For men, however, all three columns show evidence that stress and malnutrition matter for their social behaviour: Stress in the critical age under two years leads to a slightly significant increase in the likelihood of being married currently, and at the same time to a lower frequency of being unmarried or having had a divorce. In other words, men who had a stressful early childhood seem to be luckier when it comes to marriage. Thus, there is no support for the hypothesis that stressed men marry earlier because they have too low standards to pick a mate. Instead, they not only marry earlier but are also more likely marry at all and to stay together. These findings are in line with the estimates from Table 4.5, indicating higher satis-

faction with the family life if exposed to adverse conditions much earlier in life. Again, it can only be speculated about the underlying mechanisms. As Becker et al. (1977) implies, they could have had lower expectations about the marriage beforehand (and thus were not disappointed), or they face lower outside options to their spouse. Also, a higher benefit from being (and staying) married can explain this finding, as Van den Berg and Gupta (2015) point out.

Fertility

Tables 4.8 and 4.9 confirm the picture shown in the main estimations above: Early-life stress and nutrition have very limited effect on fertility in the present sample. While the total number of children could hypothetically hide effects among subgroups, here separate groups of family types are observed one by one – being childless, having a single child, having two children, and more than two children. Only for one of these groups, a significant effect of early life experiences is visible: Stress-exposed women are significantly less likely to stay childless.

Table 4.8: Fertility types: number of children – males

	0	1	2	3+
Daily avg kcal in 1000	0.024	-0.040	0.004	0.012
	(0.033)	(0.034)	(0.036)	(0.026)
Bombs p.c.(t)	0.146	0.310	-0.347	-0.109
	(0.704)	(0.683)	(0.432)	(0.327)
Year of birth	-0.005**	0.005***	0.006***	-0.006***
	(0.002)	(0.002)	(0.002)	(0.002)
Father high educ.(1/0)	-0.043*	-0.019	0.018	0.044*
	(0.025)	(0.024)	(0.027)	(0.022)
Birth Place: city(1/0)	0.039*	-0.003	-0.002	-0.033*
	(0.021)	(0.021)	(0.022)	(0.018)
Constant	9.887***	-9.627***	-10.967***	11.707***
	(3.830)	(3.640)	(3.961)	(3.321)
R^2	0.009	0.008	0.005	0.012
F-Stat.	3.386	3.061	2.187	4.271
N	1864	1864	1864	1864

Note: robust standard errors in parentheses.
*,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

This estimate fits together with Columns (3) and (4), indicating higher probabilities of two or more children among stress-exposed women, although insignificant, as well as the insignificant but positive effect of bombing on the number of children for women in Table 4.3. As Jacobsen (1996), Boberg-Fazlic et al. (2017) and Becker (1985) point out, (female) fertility is increased by a loss in female social status and labour market prospects due to decreased opportunity costs of raising children. The observed increase in education when famine-exposed does not fit into this picture, though. Still, it makes sense to observe the connection of adverse early-life conditions with increase

of fertility for the female respondents rather than for the male respondents, not only due to data limitations on the men's side. The traditional division of labour between the sexes makes female opportunity costs of raising children much more relevant for fertility than the men's.

Table 4.9: Fertility types: number of children – females

	0	1	2	3+
Daily avg kcal in 1000	-0.010	0.021	-0.054	0.043
	(0.021)	(0.031)	(0.037)	(0.028)
Bombs p.c.(t)	-0.858***	-0.081	0.504	0.435
	(0.300)	(0.754)	(1.029)	(0.940)
Year of birth	-0.001	0.004*	0.006***	-0.008***
	(0.001)	(0.002)	(0.002)	(0.002)
Father high educ.(1/0)	0.053***	-0.029	0.004	-0.029
	(0.020)	(0.024)	(0.029)	(0.024)
Birth Place: city(1/0)	0.013	0.028	0.004	-0.046**
	(0.014)	(0.020)	(0.023)	(0.021)
Constant	1.919	-6.907*	-10.407**	16.396***
	(2.476)	(3.643)	(4.262)	(3.717)
R^2	0.007	0.003	0.008	0.019
F-Stat.	3.001	1.230	2.946	7.041
N	1904	1904	1904	1904

Note: robust standard errors in parentheses.
*,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

4.6.4 Analyses on Partner Quality

Table 4.10 shows the descriptive statistics for the partner quality analysis. In Tables 4.11 to 4.15 the current partner's 'quality' is observed as potentially determined by one's own early life experiences.

These analyses look deeper into the current marriage, i.e. the partners with whom the individuals cohabit in 2012. The question here is, whether early-life experiences make people choose different partners, e.g. due to different search criteria on the marriage market. The outcomes observed contain partner's age, partner's life satisfaction, high blood pressure, height and BMI;

Table 4.10: Descriptive statistics of partners by respondent's gender

	Men's Partners			**Women's Partners**		
Variable	N	Mean	S.D.	N	Mean	S.D.
Partner: year of birth	1456	1945.247	7.116	1226	1940.229	6.299
Partner: HBP	1444	0.396	0.489	1214	0.483	0.500
Partner: life satisfaction(1/0)	1454	7.252	1.709	1224	7.223	1.631
Partner: height in cm	1456	164.203	6.026	1226	175.692	6.328
Partner: BMI	1435	26.466	4.809	1225	27.383	3.873
Partner: father's education high	1331	0.226	0.418	1131	0.219	0.414
Partner: mother's education high	1373	0.176	0.381	1152	0.139	0.346

Source: own calculation based on Socio-Economic Panel SOEP (2013).

Table 4.11: Partner's age – male respondents

Variable	Year of Birth	Age Gap	Absolute Age Gap
Daily avg kcal in 1000	-0.562	0.552	-0.539*
	(0.368)	(0.368)	(0.310)
Bombs p.c.(t)	-4.460	4.535	2.120
	(6.203)	(6.406)	(3.293)
Year of birth	0.953***	0.045**	-0.032
	(0.023)	(0.022)	(0.020)
Father: high educ.(1/0)	-0.162	0.223	-0.008
	(0.300)	(0.301)	(0.258)
Birth place: city(1/0)	-0.370	0.392	-0.471**
	(0.252)	(0.252)	(0.219)
Constant	96.224**	-92.281**	68.523*
	(44.208)	(43.973)	(38.699)
R^2	0.592	0.006	0.006
F-Stat.	428.818	1.653	1.733
N	1456	1456	1456

Note: robust standard errors in parentheses.
*,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

additionally, the social class of the partner's family of origin, measured by father's and mother's school degree.

Tables 4.11 and 4.12 look at age differences between the marriage partners of men and women, respectively. The idea is that there seems to be a preferred age gap between men and women entering marriage in almost every culture. Additionally to the own age at marriage, the chosen partner's age could be affected by early life programming, due to socio-economic or psychological mechanisms, as Neelakantan and Tertilt (2008) find the spouse's age gap as dependent on the marriage market situation.

Column (1) shows the partner's year of birth as endogenous variable, Column (2) gives the age difference and Column (3) the absolute age difference. While Column (1) only considers year of birth for both spouses, column (2) reflects the age difference in months whenever the spouse's month of birth is available, else it is identical to Column (1). In Column (3), the age difference is taken as absolute value, such that positive and negative age gaps do not cancel out.

Among the observed men, famine exposure as a child leads to an increase in deviations from the average age gap, significant at the 10% level. Columns (1) and (2) indicate that – probably, though the estimate is insignificant – the marriage age gap is increased, i.e. men marry relatively older in comparison to their wives. Table 4.12 shows that female early-life conditions affect the marriage age gap more substantially. All three measures show substantial and significant effects of famine conditions. On average, female famine exposure is associated with relatively older partners.

Apart from a cohort effect among the observed women towards larger age gaps, other explanatory variables are not systematically involved. One possi-

Table 4.12: Partner's age – female respondents

Variable	Year of Birth	Age Gap	Absolute Age Gap
Daily avg kcal in 1000	0.799**	-0.831**	-0.593**
	(0.370)	(0.371)	(0.273)
Bombs p.c.(t)	-5.116	5.089	-5.581
	(6.041)	(6.058)	(4.589)
Year of birth	0.914***	0.087***	0.065***
	(0.025)	(0.024)	(0.019)
Father: high educ.(1/0)	-0.310	0.293	0.360
	(0.319)	(0.319)	(0.251)
Birth Place: city(1/0)	0.289	-0.273	-0.030
	(0.239)	(0.239)	(0.184)
Constant	163.133***	-164.544***	-121.909***
	(48.021)	(47.947)	(37.341)
R^2	0.572	0.026	0.024
F-Stat.	323.493	7.345	6.559
N	1226	1226	1226

Note: robust standard errors in parentheses.
*,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

Table 4.13: Partner's social class: high parental education (1/0)

	Males' Partners		**Females' Partners**	
	Father	Mother	Father	Mother
Daily avg kcal in 1000	0.076**	0.003	-0.026	-0.044
	(0.033)	(0.031)	(0.036)	(0.032)
Bombs p.c.(t)	0.017	0.262	-0.044	0.479
	(0.356)	(0.443)	(1.054)	(0.923)
Year of birth	0.003	0.000	0.001	0.000
	(0.002)	(0.002)	(0.002)	(0.002)
Father: high educ.(1/0)	0.361***	0.312***	0.319***	0.254***
	(0.032)	(0.031)	(0.035)	(0.033)
Birth Place: city(1/0)	0.024	0.041**	0.076***	0.052***
	(0.022)	(0.019)	(0.023)	(0.019)
Constant	-6.322	-0.567	-2.198	-0.358
	(4.138)	(3.719)	(4.271)	(3.391)
R^2	0.130	0.120	0.120	0.107
F-Stat.	27.870	22.909	22.295	16.700
N	1331	1373	1131	1152

Note: robust standard errors in parentheses.
*,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

Table 4.14: Partner's health – male respondents

Variable	Hypertension	Life Satisfaction	Height	BMI	BMI 18.5-27
Daily avg kcal in 1000	-0.024	-0.091	-0.367	-0.154	-0.036
	(0.040)	(0.131)	(0.504)	(0.405)	(0.040)
Bombs p.c.(t)	-0.812*	2.450	6.238	-16.072***	1.031***
	(0.414)	(2.055)	(6.031)	(2.971)	(0.382)
Year of birth	-0.008***	-0.007	0.085***	-0.004	0.002
	(0.002)	(0.009)	(0.031)	(0.023)	(0.002)
Father: high educ.(1/0)	-0.132***	0.378***	1.867***	-2.140***	0.175***
	(0.031)	(0.105)	(0.402)	(0.277)	(0.030)
Birth Place: city(1/0)	0.017	-0.012	-0.027	-0.315	0.040
	(0.027)	(0.092)	(0.326)	(0.258)	(0.027)
Constant	16.538***	21.687	-1.058	34.544	-2.634
	(4.722)	(16.804)	(59.931)	(44.704)	(4.790)
R^2	0.021	0.009	0.025	0.040	0.028
F-Stat.	7.306	3.151	7.710	19.609	10.019
N	1444	1454	1456	1435	1435

Note: robust standard errors in parentheses.
*,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

ble explanation for this behaviour could be a relative scarcity on the marriage market, which might bring the less attractive of a birth cohort to deviate from the preferred, customary age gap and marry older men and younger women instead, as Brandt et al. (2016) point out.

Also the spouse's social background is associated with social norms and more or less success at the marriage market. Adverse early-life conditions could decrease one's own prospects in life and attractiveness on the marriage market which can be reflected in the social class of the spouse. Since social class of the family of origin is a predominant predictor of own social class in Germany, such a matching effect could have long-run consequences over generations. Table 4.13 shows the estimates for men in Columns (1) and (2) and for women in Columns (3) and (4). Men with a higher caloric intake as child tend to be married to women with higher social class in terms of her father's education. This means, experiencing a famine makes men rather pick a mate from a lower social class. Since famine exposure decreases men's socio-economic prospects, as Chapters 2 and 3 indicate, this finding fits to *positive assortative mating* as it is empirically most common according to Becker (1974). As expected, the explanatory variable for own social background is highly relevant for the spouse's social background for both sexes, as well as the home town's size. Exposure to bomb attacks instead does not show a significant effect on the partner's social class. In addition to parental education, the partner's own education is analysed as dependent variable (table not shown). There are no significant effects of own early life conditions on the partner's educational attainment or pension level.

Tables 4.14, and 4.15 look into the partner's health and life satisfaction, separately for men's and women's marriage partners. These parameters are

Table 4.15: Partner's health – female respondents

Variable	Hypertension	Life Satisfaction	Height	BMI	BMI 18.5-27
Daily avg kcal in 1000	-0.075*	-0.078	0.242	-0.159	0.050
	(0.044)	(0.134)	(0.515)	(0.315)	(0.044)
Bombs p.c.(t)	-1.864*	6.138**	19.528	-2.403	0.711
	(1.016)	(3.027)	(12.989)	(6.392)	(0.990)
Year of birth	-0.005	0.006	0.175***	0.046**	-0.002
	(0.003)	(0.009)	(0.035)	(0.022)	(0.003)
Father: high educ.(1/0)	0.070**	0.366***	1.849***	-0.926***	0.076**
	(0.036)	(0.112)	(0.455)	(0.261)	(0.036)
Birth Place: city(1/0)	-0.000	-0.014	0.378	-0.039	-0.002
	(0.030)	(0.097)	(0.362)	(0.227)	(0.029)
Constant	9.408*	-3.954	-166.419**	-61.841	4.408
	(5.597)	(18.141)	(68.428)	(42.011)	(5.608)
R^2	0.008	0.013	0.040	0.014	0.006
F-Stat.	2.054	3.668	9.979	3.774	1.526
N	1214	1224	1226	1225	1225

Note: robust standard errors in parentheses.
*,**,*** indicates significance at the 10%, 5%, and 1% level, respectively.

meant as additional insight into partner quality and thus success at the marriage market. While social status in the family of origin and age are clearly predetermined before marriage, some of the health parameters can additionally reflect the lifestyle during marriage.

Column (1) shows whether the partner was ever diagnosed with high blood pressure (HBP) (yes=1, no=0), Column (2) whether the partner reports high life satisfaction (yes=1, no=0). Column (3) gives partner's height in cm, which is largely predetermined before marriage and is well known as a general marker for health and earning prospects, see Chapters 2 and 3. Columns (1) to (3) are the health indicators used before in Chapter 3, so it is natural to also check for effects on the spouse's health in this way. Additionally, Column (4) gives the partner's body mass index (BMI), and thus indicates whether the weight is rather high or low. Since the BMI has an area which is known to be healthier than the extremes to the left or right, in Column (5) I check additionally whether the partner's BMI lies at an acceptable level (18.5 – 27).[6]

The spouse's health depicted in Table 4.14 is clearly associated with the men's early life experiences, namely with the early exposure to stress: Bomb load is negatively associated with the partner's high blood pressure, significant at 10%. Columns (4) and (5) show a highly significant relationship between male early-life stress and the spouse's later-life body mass index. Wives of stress-exposed men are not only lighter for their height, but also significantly more likely to have a healthy BMI. Since a high BMI is associated

[6]The range is taken from the incentive system of the German health insurer BIG direkt gesund.

with higher mortality and higher risk for certain cancers (see Berrington de Gonzalez et al. (2010) and Global BMI Mortality Collaboration et al. (2016)), this association and the reduced frequency of HBP indicate truly healthier partners.

Among women, the partner's health shows another pattern, see Table 4.15. Here, the effect on HBP is mixed, as famine exposure increases HBP and bomb load decreases HBP, where the latter confirms the finding of Table 4.14. Height and BMI do not show an effect of the respondent's early life conditions. Thus, affected women are likely to marry men as healthy as their unaffected counterparts. Life satisfaction is significantly higher if the female partner was exposed to stress in early childhood.

Secular trends in education as well as age trends in the health parameters could hypothetically drive the findings concerning partner quality, since the marital age gap changes with adverse conditions, too. Thus, I rerun Tables 4.13 to 4.15 adding partner's age to the explanatory variables. The results (Tables not shown) are almost identical, with very similar estimates and p-values. Only the famine effect on female partners' hypertension loses significance.

Taken together, for both men and women early life stress in form of bomb attacks on the home town is associated with healthier partners later in life, although in different aspects: both male and female partners have lower blood pressure, women additionally weigh less and men are happier with their lives. That means, stress-exposed individuals select partners who are substantially healthier than the average (or are chosen by them). This speaks against the hypothesis that early life suffering makes one match with less healthy partners as the findings by Becker (1974) would imply. Apart from a selection mechanism in mating, also a compensatory healthy lifestyle – induced by the early-life experiences of the partner – could be at work.

4.7 Conclusion

The literature of the last years has shown that war does not only destroy physical wealth but also endangers human capital development. Health effects translate into lower work force productivity and higher costs for the social system. Beside these negative effects for the work force, however, also psychological costs are possible that can last much longer than a lifetime, since it can materialize in reproductive success and in the upbringing of the next generation.

The estimations show large and partly surprising effects of early life experiences. Stress in early childhood seems to have a rather *positive* effect for

later life marital success and stability: Stressed boys marry earlier, they are less likely to stay single or to get divorced, and they have a higher chance to be married and cohabiting at the time of the interview in 2012. Stressed girls are less likely to stay childless. The reduced proportion of childlessness among stress-exposed women could reflect lower earnings prospects, i.e. lower opportunity costs of raising children. The education of stressed women is, however, not lower than average and thus does not support this hypothesis. The findings on male marital success fit to evasive movements on the marriage markets: The affected men might expect their market value on the marriage market to be lower. As a result, they have *lower expectations* towards their spouse, or *search shorter*. This story would be in line with the observed lower frequency of divorces. Both explanations would show in a reduced 'quality' of the spouse. Early life stress, however, does not explain the social class of the partner's family of origin. Only famine exposure translates into the social class of the spouse as expected: men who suffered from hunger as children rather marry women of lower social status. These findings might as well support the idea expressed in Van den Berg and Gupta (2015): the higher protective effect from marriage. An increased benefit from marriage could not only explain the young male marrying age, but also the lower male divorce numbers. Accordingly, the levels of satisfaction with family life indicate that adverse early conditions do not reduce happiness with the relationship. It would be fruitful to dig deeper into the spouses' health and satisfaction paths following marriage in future research.

Also the marital age gap is influenced by early-life conditions: Famine exposure tends to increase the age gap between the sexes substantially, for both affected women and men. This again is likely to reflect evasive movements on the marriage market, bringing impaired people to deviate from the preferred age gap to find an acceptable partner, see Brandt et al. (2016).

One question stays open, after all: How do those who suffered from stress and hunger early in life catch the marriage partners, who stay healthier, happier and more attractive in higher age? Is it success on the marriage market, explainable by scarcities? Is it a behavioural effect during married life? Or is it a lucky draw compensating for the earlier suffering?

The data show stressed boys and girls to have the healthier partners later in life than the unaffected counterparts: with a slightly lower risk of high blood pressure, higher life satisfaction, and a substantially lower risk of obesity, also when controlling for partner's age. Since these parameters can likely be determined also after marrying, the observed associations may reflect not only a 'good draw' on the marriage market, but as well a behavioural response to the adverse experiences made in early childhood: a healthy lifestyle, chosen by the couples to compensate for the health impairments of one of them.

Other measures of fertility (total completed fertility, age at first birth) are not found to be affected, which could partly be due to the lower data quality on male fertility here. More research and potentially different data sources are necessary to analyse fertility effects of male adverse early childhood experiences. In future work, it would also be fruitful to consider children's quality to be influenced by the said early life conditions. This could be done using the extensive data on children's health and schooling in SOEP. The present analysis does not indicate a relation between early life conditions of the parents and the fluctuating birth rate in Germany. Possibly, children's quality could serve as a missing link.

Bibliography

ABELSHAUSER, W. (1983): "Wirtschaft und Besatzungspolitik in der Französischen Zone 1945 – 1949," in *Die Deutschlandpolitik Frankreichs und die Französische Zone 1945 – 1949*, ed. by C. Scharf and H.-J. Schröder, Wiesbaden: Steiner, 111–140.

AGADJANIAN, V. AND N. PRATA (2002): "War, Peace, and Fertility in Angola," *Demography*, 39, 215–231.

AIZER, A., L. STROUD, AND S. BUKA (2016): "Maternal Stress and Child Outcomes," *Journal of Human Resources*, 51.

AKBULUT-YUKSEL, M. (2014): "Children of War: The Long-Run Effects of Large-Scale Physical Destruction and Warfare on Children," *The Journal of Human Resources*, 49, 634–662.

AKBULUT-YUKSEL, M. AND A. KUGLER (2016): "Intergenerational Persistence of Health: Do Immigrants Get Healthier as They Remain in the U.S. for More Generations?" *Economics and Human Biology*, 23, 136 – 148.

ALLIED CONTROL AUTHORITY (1946): *Enactments and Approved Papers*, vol. 2, Washington D.C.: Army Library.

ALMOND, D. AND J. CURRIE (2011a): "Human Capital Development before Age Five," in *Handbook of Labor Economics*, ed. by D. Card and O. Ashenfelter, Elsevier, vol. 4b, chap. 15, 1315–1486.

——— (2011b): "Killing Me Softly: The Fetal Origins Hypothesis," *The Journal of Economic Perspectives*, 25, 153–172.

BANNING, C. (1946): "Food Shortage and Public Health, First Half of 1945," *Annals of the American Academy of Political and Social Science*, 245, 93–110.

BARKER, D. J. P. (1990): "The Fetal and Infant Origins of Adult Disease," *BMJ: British Medical Journal*, 301(6761), 1111.

——— (1994): *Mothers, babies, and disease in later life*, London: BMJ Publishing Group.

BARKER, D. J. P., C. OSMOND, AND J. GOLDING (1990): "Height and Mortality in the Counties of England and Wales," *Annals of Human Biology*, 17, 1–6.

BATESON, P., D. J. P. BARKER, T. CLUTTON-BROCK, D. DEB, B. D'UDINE, R. A. FOLEY, P. GLUCKMAN, K. GODFREY, T. KIRKWOOD, M. M. LAHR, J. MCNAMARA, N. B. METCALFE, P. MONAGHAN, H. G. SPENCER, AND S. E. SULTAN (2004): "Developmental plasticity and human health," *Nature*, 430, 419–421.

BECKER, G. S. (1960): "An Economic Analysis of Fertility," in *Demographic and economic change in developed countries*, Columbia University Press, 209–240.

——— (1974): "A Theory of Marriage," in *Economics of the Family: Marriage, Children, and Human Capital*, University of Chicago Press, 299–351.

——— (1981): "Altruism in the Family and Selfishness in the Market Place," *Economica*, 48, 1–15.

——— (1985): "Human Capital, Effort, and the Sexual Division of Labor," *Journal of labor economics*, 3, S33–S58.

BECKER, G. S., E. M. LANDES, AND R. T. MICHAEL (1977): "An economic analysis of marital instability," *Journal of political Economy*, 85, 1141–1187.

BEIL, H. AND H. WAGNER (1951): "Die Sterblichkeit im zweiten Weltkrieg und in den Nachkriegsjahren bis 1947 in Hessen und Stuttgart," *Medical Microbiology and Immunology*, 132, 368–383.

BELOT, M. AND J. FIDRMUC (2010): "Anthropometry of Love: Height and Gender Asymmetries in Interethnic Marriages," *Economics and Human Biology*, 8, 361–372.

BERGENDAHL, M., M. L. VANCE, A. IRANMANESH, M. O. THORNER, AND J. D. VELDHUIS (1996): "Fasting as a metabolic stress paradigm selectively amplifies cortisol secretory burst mass and delays the time of maximal nyctohemeral cortisol concentrations in healthy men," *Journal of Clinical Endocrinology and Metabolism*, 81, 692–699.

BERGSTROM, T. C. AND M. BAGNOLI (1993): "Courtship as a waiting game," *Journal of political economy*, 101, 185–202.

BERRINGTON DE GONZALEZ, A., P. HARTGE, J. R. CERHAN, A. J. FLINT, L. HANNAN, R. J. MACINNIS, S. C. MOORE, G. S. TOBIAS, H. ANTON-CULVER, L. B. FREEMAN, W. L. BEESON, S. L. CLIPP, D. R. ENGLISH, A. R. FOLSOM, D. M. FREEDMAN, G. GILES, N. HAKANSSON, K. D. HENDERSON, J. HOFFMAN-BOLTON, J. A. HOPPIN, K. L. KOENIG, I.-M.

LEE, M. S. LINET, Y. PARK, G. POCOBELLI, A. SCHATZKIN, H. D. SESSO, E. WEIDERPASS, B. J. WILLCOX, A. WOLK, A. ZELENIUCH-JACQUOTTE, W. C. WILLETT, AND M. J. THUN (2010): "Body-Mass Index and Mortality among 1.46 Million White Adults," *New England Journal of Medicine*, 363, 2211–2219, pMID: 21121834.

BERTONI, M. (2015): "Hungry today, unhappy tomorrow? Childhood hunger and subjective wellbeing later in life," *Journal of health economics*, 40, 40–53.

BESSER, C. (2007): "Die deutschen Vertriebenen in der Bundesrepublik Deutschland von 1945 bis 1969," *GESIS Datenarchiv, Köln*, ZA8217 Datenfile Version 1.0.0.

——— (2008): "Die deutschen Vertriebenen in der Bundesrepublik Deutschland von 1939 bis 1990," *GESIS Datenarchiv, Köln*, ZA8217 Datenfile Version 1.0.0, doi:10.4232/1.8217.

BIGNON, V. (2007): "Black and grey markets for illegal exchanges in post WW II Germany," *manuscript*.

BIRBAUMER, N. AND R. F. SCHMIDT (2010): *Biologische Psychologie*, Heidelberg: Springer, 7th revised and extended ed.

BLACK, S., P. DEVEREUX, AND K. SALVANES (2016): "Does grief transfer across generations? Bereavements during pregnancy and child outcomes," *American Economic Journal: Applied Economics*, 8, 193–223.

BLOSSFELD, H.-P. AND U. JAENICHEN (1992): "Educational expansion and changes in women's entry into marriage and motherhood in the Federal Republic of Germany," *Journal of Marriage and the Family*, 302–315.

BOBERG-FAZLIC, N., M. IVETS, M. KARLSSON, T. NILSSON, ET AL. (2017): "Disease and Fertility: Evidence from the 1918 Influenza Pandemic in Sweden," *IZA Discussion Paper Series*, 10834.

BOLDORF, M. (1998): *Sozialfürsorge in der SBZ, DDR 1945 – 1953: Ursachen, Ausmass und Bewältigung der Nachkriegsarmut*, Stuttgart: Steiner.

BOLTEN, M. I., H. WURMSER, A. BUSKE-KIRSCHBAUM, M. PAPOUSEK, K.-M. PIRKE, AND D. HELLHAMMER (2011): "Cortisol levels in pregnancy as a psychobiological predictor for birth weight," *Archives of women's mental health*, 14, 33–41.

BONGAARTS, J., O. FRANK, AND R. LESTHAEGHE (1984): "The Proximate Determinants of Fertility in Sub-Saharan Africa," *Population and Development Review*, 511–537.

BOZZOLI, C., A. DEATON, AND C. QUINTANA-DOMEQUE (2009): "Adult height and childhood disease," *Demography*, 46, 647–669.

BRANDT, L., A. SIOW, AND C. VOGEL (2016): "Large demographic shocks and small changes in the marriage market," *Journal of the European Economic Association*, 14, 1437–1468.

BRENKE, K. (2015): "Growing Importance of Women in the German Labor Market," *DIW Economic Bulletin*, 5/2015, 51 – 61.

BUCHHEIM, C. (2010): "Der Mythos vom Wohlleben. Der Lebensstandard der deutschen Zivilbevölkerung im Zweiten Weltkrieg," *Vierteljahrshefte für Zeitgeschichte Das zentrale Forum der Zeitgeschichtsforschung*, 58, 299–328.

BUNDESAMT FÜR KARTOGRAPHIE UND GEODÄSIE (2013): "GeoBasis-DE – Geodaten der Deutschen Landesvermessung VG2500," `http://www.geodatenzentrum.de/geodaten/gdz?l=down_opendata`, last accessed June 2013.

BUNDESINSTITUT FÜR BEVÖLKERUNGSFORSCHUNG (2017): "Average Age at First Marriage of Women in Germany, Western Germany and Eastern Germany, 1960 to 2015," `http://www.bib-demografie.de/EN/Facts_Figures/Marriages/Figures/a_04_15_durchschnittl_erstheiratsalter_frauen_d_w_o_ab1960.html`, last accessed August 2017.

CALDWELL, J., P. CALDWELL, M. BRACHER, AND G. SANTOW (1988): "The Contemporary Marriage and Fertility Revolutions in the West the Explanations Provided by Australian Participants," *Journal of Population Research*, 5, 113–145.

CAMERON, N. AND E. W. DEMERATH (2002): "Critical Periods in Human Growth and their Relationship to Diseases of Aging," *American Journal of Physical Anthropology*, 119, 159–184.

CASE, A. AND C. PAXSON (2008): "Stature and status: Height, ability, and labor market outcomes," *Journal of Political Economy*, 116, 499–532.

——— (2009): "Early Life Health and Cognitive Function in Old Age," *The American Economic Review*, 99, 104–109.

——— (2010): "Causes and consequences of early-life health," *Demography*, 47, S65–S85.

CHEN, Y. AND L.-A. ZHOU (2007): "The long-term health and economic consequences of the 1959–1961 famine in China," *Journal of Health Economics*, 26, 659–681.

CONEUS, K. AND K. SPIESS (2012): "The Intergenerational Transmission of Health in Early Childhood: Evidence from the German Socio-Economic Panel Study," *Economics and Human Biology*, 10, 89–97.

COOPER, C., D. KUH, P. EGGER, M. WADSWORTH, AND D. J. P. BARKER (1996): "Childhood growth and age at menarche," *BJOG: An International Journal of Obstetrics & Gynaecology*, 103, 814–817.

CORNIDES, W. (1948): *Wirtschaftsstatistik der deutschen Besatzungszonen 1945–1948 in Verbindung mit der deutschen Produktionsstatistik der Vorkriegszeit*, vol. 3, Oberursel Taunus: Verlag Europa Archiv.

CURRIE, J. (2009): "Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development," *Journal of Economic Literature*, 47, 87–122.

CUSICK, S. E. AND M. K. GEORGIEFF (2016): "The Role of Nutrition in Brain Development: The Golden Opportunity of the 'First 1000 Days'," *Journal of Pediatrics*.

DAVIS, D. R. (1951): "Emotional disturbances and behavioural reactions," in *Medical Research Council Special Report No 275: Studies of Undernutrition, Wuppertal 1946-9*, His Majesty's Stationary Office, chap. VIII, 147–164.

DAVIS, R. G. (2006): *Bombing the European Axis powers. A historical digest of the combined bomber offensive 1939—1945*, Air University Press.

DEAN, R. (1951): "The size of the baby at birth and the yield of breast milk," in *Medical Research Council Special Report Series No. 275: Studies of Undernutrition, Wuppertal 1946-9, by Members of the Department of Experimental Medicine, Cambridge, and Associated Workers*, His Majesty's Stationary Office, chap. XXVIII, 346–378.

DEATON, A. (2007): "Height, health, and development," *Proceedings of the National Academy of Sciences*, 104, 13232–13237.

DENNISON, T. AND S. OGILVIE (2014): "Does the European marriage pattern explain economic growth?" *The journal of economic history*, 74, 651–693.

DOBLHAMMER, G., G. J. VAN DEN BERG, AND L. H. LUMEY (2013): "A reanalysis of the long-term effects on life expectancy of the Great Finnish Famine of 1866–68," *Population studies*, 67, 309–322.

DOBLHAMMER, G. AND J. W. VAUPEL (2001): "Lifespan depends on month of birth," *Proceedings of the National Academy of Sciences*, 98, 2934–2939.

DROESE, W. AND E. ROMINGER (1950): "Die Auswirkungen der Mangelernährung auf Körpergewicht und Körpergröße schleswigholsteinischer Kinder in der Nachkriegszeit," *Zeitschrift für Kinderheilkunde*, 67, 615–638.

EGGEN, B. AND M. RUPP (2007): "Kinderreichtum: eine Ausnahme in der neueren Geschichte?" *Statistisches Monatsheft Baden-Württemberg*, 6–14.

ELIAS, S. G., P. H. PEETERS, D. E. GROBBEE, AND P. A. VAN NOORD (2004): "Breast cancer risk after caloric restriction during the 1944–1945 Dutch famine," *Journal of the National Cancer Institute*, 96, 539–546.

ELIAS, S. G., P. A. VAN NOORD, P. H. PEETERS, I. DEN TONKELAAR, AND D. E. GROBBEE (2003): "Caloric restriction reduces age at menopause: the effect of the 1944–1945 Dutch famine," *Menopause*, 10, 399–405.

ELO, I. T. AND S. H. PRESTON (1992): "Effects of early-life conditions on adult mortality: A review," *Population Index*, 58, 186–212.

ENLOE, C. F. J., ed. (1945): *Morale Division, United States Strategic Bombing Survey. Medical Branch Report. The effect of bombing on health and medical care in Germany*, Washington, D.C. : War Department, October 30, 1945, `http://resource.nlm.nih.gov/31520560R`.

FARQUHARSON, J. E. (1985): *The Western Allies and the Politics of Food: Agrarian Management in Postwar Germany*, Berg Publishers.

FICHTER, M. M., K.-M. PIRKE, AND F. HOLSBOER (1986): "Weight loss causes neuroendocrine disturbances: Experimental study in healthy starving subjects," *Psychiatry Research*, 17, 61–72.

FREEDMAN, D. G. (1979): *Human Sociobiology*, New York: Free Press.

FREJKA, T. AND G. CALOT (2001): "Cohort Reproductive Patterns in Low-Fertility Countries," *Population and Development Review*, 27, 103–132.

FREY, B. S. AND A. STUTZER (2010): *Happiness and economics: How the economy and institutions affect human well-being*, Princeton University Press.

FRIEDRICH, J. (2002): *Der Brand – Deutschland im Bombenkrieg 1940–1945*, München: Propyläen.

GALAZKA, A. AND S. DITTMANN (2000): "The changing epidemiology of diphtheria in the vaccine era," *Journal of Infectious Diseases*, 181, S2–S9.

GEO EPOCHE PANORAMA (2014): *Trümmerzeit und Wiederaufbau: Deutschland 1945-1955*, Gruner + Jahr AG Co KG, Druck- und Verlagshaus.

GLAESMER, H. (2013): "Die Schatten des Zweiten Weltkrieges Folgen der traumatischen Erfahrungen in der älteren deutschen Bevölkerung," *Wissenschaft und Frieden*, 35–38.

GLAESMER, H., E. BÄHLER, H. GÜNDEL, AND S. RIEDEL-HELLER (2011): "The association of traumatic experiences and posttraumatic stress disorder with physical morbidity in old age: A German population-based study," *Psychosomatic Medicine*, 73, 401–406.

GLOBAL BMI MORTALITY COLLABORATION ET AL. (2016): "Body-mass index and all-cause mortality: individual-participant-data meta-analysis of 239 prospective studies in four continents," *The Lancet*, 388, 776–786.

GLUCKMAN, P. D. AND M. A. HANSON (2005): *The fetal matrix: Evolution, development and disease*, Cambridge University Press.

GLUCKMAN, P. D., M. A. HANSON, C. COOPER, AND K. L. THORNBURG (2008): "Effect of in utero and early-life conditions on adult health and disease," *New England Journal of Medicine*, 359, 61–73.

GLUCKMAN, P. D., M. A. HANSON, AND H. G. SPENCER (2005): "Predictive adaptive responses and human evolution," *Trends in Ecology & Evolution*, 20, 527–533.

GOEBEL, J., ed. (2017): *SOEP-Core v32 – Documentation on Biography and Life History Data*, vol. 418, SOEP Survey Papers 418: Series D, Berlin: DIW/SOEP.

GREENWOOD, J., A. SESHADRI, AND G. VANDENBROUCKE (2005): "The baby boom and baby bust," *The American Economic Review*, 95, 183–207.

HADLEY, C. AND C. L. PATIL (2006): "Food insecurity in rural Tanzania is associated with maternal anxiety and depression," *American Journal of Human Biology*, 18, 359–368.

HALES, C. N. AND D. J. P. BARKER (1992): "Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis," *Diabetologia*, 35, 595–601.

HAMELIN, A.-M., J.-P. HABICHT, AND M. BEAUDRY (1999): "Food insecurity: consequences for the household and broader social implications," *The Journal of Nutrition*, 129, 525S–528S.

HARRINGTON, C. (2010): *Politicization of sexual violence: From abolitionism to peacekeeping*, Ashgate Publishing.

HARVILLE, E., X. XIONG, AND P. BUEKENS (2010): "Disasters and perinatal health: a systematic review," *Obstetrical & Gynecological Survey*, 65, 713–728.

HATTON, T. J. AND B. E. BRAY (2010): "Long run trends in the heights of European men, 19th–20th centuries," *Economics and Human Biology*, 8, 405–413.

HAUPTAMT FÜR STATISTIK BERLIN-WILMERSDORF (1947): *Berlin in Zahlen 1947*, Berliner Kulturbuch-Verlag G.m.b.H.

HEBERT, P., J. RICH-EDWARDS, J. MANSON, P. RIDKER, N. COOK, G. O'CONNOR, J. BURING, AND C. HENNEKENS (1993): "Height and incidence of cardiovascular disease in male physicians," *Circulation*, 88, 1437–1443.

HEINECK, G. (2005): "Up in the skies? The relationship between body height and earnings in Germany," *Labour*, 19, 469–489.

HEINEMAN, E. (1996): "The hour of the woman: Memories of Germany's 'crisis years' and West German national identity," *The American Historical Review*, 101, 354–395.

HENKE, K.-D. (1982): "Politik der Widersprüche. Zur Charakteristik der französischen Militärregierung in Deutschland nach dem Zweiten Weltkrieg," *Vierteljahrshefte für Zeitgeschichte*, 30, 500–537.

HENSLEY, W. (1993): "Height as a Measure of Success in Academe," *Psychology: A Journal of Human Behavior*, 30, 40–46.

HEWITT, K. (1983): "Place annihilation: Area bombing and the fate of urban places," *Annals of the Association of American Geographers*, 73, 257–284.

HILL, K., N. R. COUNCIL, C. ON POPULATION, ET AL. (2004): *War, humanitarian crises, population displacement, and fertility: A review of evidence*, National Academies Press.

HOBEL, C. AND J. CULHANE (2003): "Role of Psychosocial and Nutritional Stress on Poor Pregnancy Outcome," *The Journal of Nutrition*, 133, 1709–1717.

HOCHSTADT, S. (1999): *Mobility and Modernity: Migration in Germany 1820-1989*, Ann Arbor: University of Michigan Press.

——— (2011): *Wir haben wieder aufgebaut: Frauen der Stunde Null erzählen*, Elisabeth Sandmann Verlag GmbH. München.

ICHINO, A. AND R. WINTER-EBMER (2004): "The long-run educational cost of World War II," *Journal of Labor Economics*, 22, 57–87.

JACOBSEN, J. E. (1996): *Population Growth*, University Science Books, Sausalito, California.

JÜRGES, H. (2013): "Collateral Damage: The German Food Crisis, Educational Attainment and Labor Market Outcomes of German Post-War Cohorts," *Journal of Health Economics*, 32, 286–303.

JÜRGES, H., S. REINHOLD, AND M. SALM (2011): "Does Schooling Affect Health Behavior? Evidence from the Educational Expansion in Western Germany," *Economics of Education Review*, 30, 862–872.

KAATI, G., L. O. BYGREN, AND S. EDVINSSON (2002): "Cardiovascular and diabetes mortality determined by nutrition during parents' and grandparents' slow growth period," *European Journal of Human Genetics*, 10, 682–688.

KELLY, S., C. HERTZMAN, AND M. DANIELS (1997): "Searching for the biological pathways between stress and health," *Annual review of public health*, 18, 437–462.

KESTERNICH, I., B. SIFLINGER, J. SMITH, AND J. WINTER (2014): "The Effects of World War II on Economic and Health Outcomes across Europe," *Review of Economics and Statistics*, 96, 103–118.

——— (2015): "Individual behaviour as a pathway between early-life shocks and adult health: Evidence from hunger episodes in post-war Germany," *The Economic Journal*, 125, F372–F393.

KHASHAN, A. S., K. M. ABEL, R. MCNAMEE, M. G. PEDERSEN, R. T. WEBB, P. N. BAKER, L. C. KENNY, AND P. B. MORTENSEN (2008): "Higher risk of offspring schizophrenia following antenatal maternal exposure to severe adverse life events," *Archives of General Psychiatry*, 65, 146–152.

KIRBY, M. AND R. CAPEY (1997): "The area bombing of Germany in World War II: An operational research perspective," *Journal of the Operational Research Society*, 48, 661–677.

KIRCHENGAST, S. AND E.-M. WINKLER (1996): "Nutritional status as indicator for reprocuctive success in !Kung San and Kavango females from Namibia," *Anthropologischer Anzeiger*, 267–276.

KLATT, W. (1950): "Food and Farming in Germany: I. Food and Nutrition," *International Affairs*, 26, 45–58.

KOCK, G. (1997): *"Der Führer sorgt für unsere Kinder..." Die Kinderlandverschickung im Zweiten Weltkrieg*, Schöningh.

KOHLER, H.-P., J. R. BEHRMAN, AND A. SKYTTHE (2005): "Partner + Children = Happiness? The Effects of Partnerships and Fertility on Well-Being," *Population and Development Review*, 31, 407–445.

KOHLER, H.-P., J. L. RODGERS, W. B. MILLER, A. SKYTTHE, AND K. CHRISTENSEN (2006): "Bio-social determinants of fertility," *International Journal of Andrology*, 29, 46 – 53.

KRAEMER, S. (2000): "The fragile male," *British Medical Journal*, 321, 1609–1612.

KROH, M. (2013): "Documentation of sample sizes and panel attrition in the German Socio Economic Panel (SOEP) (1984 until 2012)," *DIW Data Documentation*, 71.

KUZAWA, C. W. (2012): "Early environments, developmental plasticity and chronic degenerative disease," in *Human Growth and Development*, ed. by N. Cameron and B. Bogin, Elsevier, chap. 12, 325–341, second ed.

KUZAWA, C. W. AND E. A. QUINN (2009): "Developmental origins of adult function and health: evolutionary hypotheses," *Annual Review of Anthropology*, 38, 131–147.

LASLETT, P. (1977): "Characteristics of the Western family considered over time," *Journal of family history*, 2, 89–115.

LAWLOR, D. (2008): "The developmental origins of health and disease: where do we go from here?" *Epidemiology*, 19, 206–208.

LAYARD, R. (2005): *Happiness: Lessons from a New Science*, Allen Lane, London.

LEDERMAN, S. A., V. RAUH, L. WEISS, J. L. STEIN, L. A. HOEPNER, M. BECKER, AND F. P. PERERA (2004): "The effects of the World Trade Center event on birth outcomes among term deliveries at three lower Manhattan hospitals," *Environmental Health Perspectives*, 112, 1772–1778.

LI, Y., Y. HE, L. QI, V. W. JADDOE, E. J. FESKENS, X. YANG, G. MA, AND F. B. HU (2010): "Exposure to the Chinese famine in early life and the risk of hyperglycemia and type 2 diabetes in adulthood," *Diabetes*, 59, 2400–2406.

LIEBE, H. (1947): "Der Ernährungsbedarf und seine heutige Deckung," in *Die Deutsche Wirtschaft zwei Jahre nach dem Zusammenbruch – Tatsachen und Probleme*, Berlin: Albert Nauck & Co., 72–90.

LINDEBOOM, M., F. PORTRAIT, AND G. J. VAN DEN BERG (2010): "Long-run effects on longevity of a nutritional shock early in life: The Dutch Potato Famine of 1846–1847," *Journal of Health Economics*, 29, 617–629.

LINDSTROM, D. P. AND B. BERHANU (1999): "The impact of war, famine, and economic decline on marital fertility in Ethiopia," *Demography*, 36, 247–261.

LIPPS, O. (2009): "Attrition of Households and Individuals in Panel Surveys," *SOEP papers on Multidisciplinary Panel Data Research*, 164.

LUMEY, L. (1998): "Reproductive outcomes in women prenatally exposed to undernutrition: a review of findings from the Dutch famine birth cohort," *Proceedings of the Nutrition Society*, 57, 129–135.

LUMEY, L., A. STEIN, AND E. SUSSER (2011): "Prenatal Famine and Adult Health," *Annual Review of Public Health*, 32, 237–262.

LUMEY, L. AND A. D. STEIN (1997): "In utero exposure to famine and subsequent fertility: The Dutch Famine Birth Cohort Study," *American journal of public health*, 87, 1962–1966.

LUMMAA, V. (2003): "Early Developmental conditions and reproductive success in humans: Downstream effects of prenatal famine, birthweight, and timing of birth," *American Journal of Human Biology*, 15, 370–379.

LUPIEN, S. J., B. S. MCEWEN, M. R. GUNNAR, AND C. HEIM (2009): "Effects of stress throughout the lifespan on the brain, behaviour and cognition," *Nature Reviews Neuroscience*, 10, 434–445.

MACKENBACH, J., M. BOUVIER-COLLE, AND E. JOUGLA (1990): "'Avoidable' mortality and health services: a review of aggregate data studies," *Journal of Epidemiology and Community Health*, 44, 106–111.

MALASPINA, D., C. CORCORAN, K. KLEINHAUS, M. PERRIN, S. FENNIG, D. NAHON, Y. FRIEDLANDER, AND S. HARLAP (2008): "Acute maternal stress in pregnancy and schizophrenia in offspring: a cohort prospective study," *BMC psychiatry*, 8, 1–9.

MANZ, M. (1985): "Stagnation und Aufschwung in der französischen Besatzungszone von 1945 bis 1948," Dissertation, Universität Mannheim.

MARIC, N. P., B. DUNJIC, D. J. STOJILJKOVIC, D. BRITVIC, AND M. JASOVIC-GASIC (2010): "Prenatal stress during the 1999 bombing associated with lower birth weight – a study of 3,815 births from Belgrade," *Archives of Women's Mental Health*, 13, 83–89.

MARTINI, P. (1947): "Über die gesundheitliche Lage in Deutschland," in *Die soziale und gesundheitliche Lage in Deutschland - Vorträge und Entschliessung aus der Tagung des Hauptausschusses des Deutschen Städtetages zu Goslar am 18. Juli 1947*, ed. by W. Polligkeit, München: Urban und Schwarzenberg, vol. 8, 28–47.

MASLOW, A. H. (1943): "A theory of human motivation," *Psychological review*, 50, 370.

MEIER, E. (1951): "Das Untergewicht der Kinder von 9 bis 13 Jahren," *Zeitschrift für Hygiene*, 133, 1–19.

MERRITT, A. J. AND R. L. MERRITT, eds. (1970): *Public opinion in occupied Germany*, Urbana: University of Illinois Press.

MIDDLEBROOK, M. AND C. EVERITT (1985): *The Bomber Command war diaries: An operational reference book, 1939 – 1945*, London: Viking.

MILLER, G. E. AND E. CHEN (2007): "Unfavorable socioeconomic conditions in early life presage expression of proinflammatory phenotype in adolescence," *Psychosomatic Medicine*, 69, 402–409.

——— (2010): "Harsh family climate in early life presages the emergence of a proinflammatory phenotype in adolescence," *Psychological science*, 21, 848–856.

MILLER, G. E., E. CHEN, A. K. FOK, H. WALKER, A. LIM, E. F. NICHOLLS, S. COLE, AND M. S. KOBOR (2009): "Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling," *Proceedings of the National Academy of Sciences*, 106, 14716–14721.

MODIN, B. (2003): "Born out of wedlock and never married – it breaks a man's heart," *Social Science & Medicine*, 57, 487 – 501.

MODIN, B., I. KOUPIL, AND D. VÅGERÖ (2009): "The Impact of Early Twentieth Century Illegitimacy Across Three Generations. Longevity and Intergenerational Health Correlates," *Social Science & Medicine*, 68, 1633 – 1640.

MURRAY, J. E. (2000): "Marital protection and marital selection: Evidence from a historical-prospective sample of American men," *Demography*, 37, 511–521.

MYRSKYLÄ, M. (2010): "The effects of shocks in early life mortality on later life expectancy and mortality compression: A cohort analysis," *Demographic Research*, 22, 289–320.

NAIMARK, N. M. (1995): *The Russians in Germany: a history of the Soviet Zone of occupation, 1945-1949*, Harvard University Press.

NEELAKANTAN, U. AND M. TERTILT (2008): "A note on marriage market clearing," *Economics Letters*, 101, 103–105.

NEELSEN, S. AND T. STRATMANN (2011): "Effects of prenatal and early life malnutrition: Evidence from the Greek famine," *Journal of Health Economics*, 30, 479–488.

OJEDA, N. B., D. GRIGORE, AND B. T. ALEXANDER (2008): "Developmental programming of hypertension insight from animal models of nutritional manipulation," *Hypertension*, 52, 44–50.

OMRAN, A. R. (1971): "The Epidemiologic Transition: A Theory of the Epidemiology of Population Change," *The Milbank Memorial Fund Quarterly*, 49, 509–538.

OSTERIA, T. S. (1983): "Nutritional status and menarche in a rural community in the Philippines," *Philippine journal of nutrition*, 36, 150.

PAINTER, R. C., T. J. ROSEBOOM, AND O. P. BLEKER (2005): "Prenatal exposure to the Dutch famine and disease in later life: an overview," *Reproductive toxicology*, 20, 345–352.

PAMUK, E. (1985): "Social class inequality in mortality from 1921 to 1972 in England and Wales," *Population Studies*, 39, 17–31.

PEARSON, K. (1912): "The intensity of natural selection in man," *Proceedings of the Royal Society of London. Series B, Containing Papers of a Biological Character*, 85, 469–476.

PERSSON, P. AND M. ROSSIN-SLATER (2018): "Family Ruptures, Stress, and the Mental Health of the Next Generation," *American Economic Review*.

PHILLIPS, D. I. W., D. J. HANDELSMAN, J. G. ERIKSSON, T. FORSEN, C. OSMOND, AND D. J. P. BARKER (2001): "Prenatal growth and subsequent marital status: longitudinal study," *British Medical Journal*, 322, 771–771.

PISCHKE, J. AND T. VON WACHTER (2008): "Zero Returns to Compulsory Schooling in Germany: Evidence and Interpretation," *Review of Economics and Statistics*, 90, 592–598.

POLLITT, R. A., K. M. ROSE, AND J. S. KAUFMAN (2005): "Evaluating the Evidence for Models of Life Course Socioeconomic Factors and Cardiovascular Outcomes: a Systematic Review," *BMC public health*, 5, 7.

PRUGGER, C., P. U. HEUSCHMANN, AND U. KEIL (2006): "Epidemiologie der Hypertonie in Deutschland und weltweit," *Herz*, 31, 287–293.

RAINER, H. AND T. SIEDLER (2009): "O Brother, Where Art Thou? The Effects of Having a Sibling on Geographic Mobility and Labor Market Outcomes," *Economica*, 76, 528–556.

REDEKER, F. (1946): "Entwicklung der Allgemeinen Sterblichkeit in Berlin nach dem Kriege," *Ärztliche Wochenschrift*, 1, 26–30.

REICHARDT, S. AND M. ZIERENBERG (2010): *Damals nach dem Krieg: eine Geschichte Deutschlands 1945 bis 1949*, München: Goldmann.

REIDPATH, D. AND P. ALLOTEY (2003): "Infant mortality rate as an indicator of population health," *Journal of Epidemiology and Community Health*, 57, 344–346.

REPORT OF THE MILITARY GOVERNOR NO. 39 (1948): *Statistical Annex, Issue No. XIX - Report of the Military Governor*, Office of Military Government for Germany.

REPORT OF THE MILITARY GOVERNOR NO. 49 (1949): *Statistical Annex, Issue No. XXIX*, Office of Military Government for Germany.

RILEY, A. P. (1994): "Determinants of Adolescent Fertility and Its Consequences for Maternal Health, with Special Reference to Rural Bangladesha," *Annals of the New York Academy of Sciences*, 709, 86–100.

RODGERS, J. L., H.-P. KOHLER, K. O. KYVIK, AND K. CHRISTENSEN (2001): "Behavior Genetic Modeling of Human Fertility: Findings from a Contemporary Danish Twin Study," *Demography*, 38, 29–42.

ROSEBOOM, T. J., J. H. VAN DER MEULEN, A. C. RAVELLI, C. OSMOND, D. J. P. BARKER, AND O. P. BLEKER (2001): "Effects of prenatal exposure to the Dutch famine on adult disease in later life: an overview," *Molecular and cellular endocrinology*, 185, 93–98.

ROTHENBERGER, K.-H. (1980): *Die Hungerjahre nach dem Zweiten Weltkrieg: Ernährungs- und Landwirtschaft in Rheinland-Pfalz 1945-1950*, Boppard am Rhein: Harald Boldt Verlag.

ROYER, H. AND A. WITMAN (2013): "Intergenerational effects on health in utero and early life," *Encyclopedia of Health Economics*, 2, 83–90.

SARDON, J.-P. AND G. ROBERTSON (2006): "Recent demographic trends in the developed countries," *Population*, 61, 227–300.

SCHLANGE-SCHÖNINGEN, H. (1955): *Im Schatten des Hungers: Dokumentarisches zur Ernährungspolitik und Ernährungswirtschaft in den Jahren 1945-1949*, P. Parey.

SCHMITZ, H. (1956): *Die Bewirtschaftung der Nahrungsmittel und Verbrauchsgüter 1939-1950: Dargestellt an dem Beispiel der Stadt Essen*, Stadtverwaltung Essen.

SCHOLTE, R. S., G. J. VAN DEN BERG, AND M. LINDEBOOM (2015): "Long-run effects of gestation during the Dutch hunger winter famine on labor market and hospitalization outcomes," *Journal of health economics*, 39, 17–30.

SCHULZ, L. C. (2010): "The Dutch Hunger Winter and the developmental origins of health and disease," *Proceedings of the National Academy of Sciences*, 107, 16757–16758.

SEEL, G. (1950): *Ernährung und Ernährungszustand der Bevölkerung der britischen Besatzungszone Deutschlands in den Jahren 1947-1948*, Dissertation, Medizinische Akademie Düsseldorf.

SELYE, H. (1950): "Stress and the general adaptation syndrome," *British medical journal*, 1, 1383–1392.

SILVENTOINEN, K. (2003): "Determinants of variation in adult body height," *Journal of Biosocial Science*, 35, 263–285.

SLOBODA, D. M., R. HART, D. A. DOHERTY, C. E. PENNELL, AND M. HICKEY (2007): "Age at menarche: influences of prenatal and postnatal growth," *The Journal of Clinical Endocrinology & Metabolism*, 92, 46–50.

SLOBODA, D. M., M. HICKEY, AND R. HART (2011): "Reproduction in females: the role of the early life environment," *Human reproduction update*, 17, 210–227.

SOBOTKA, T. (2012): "Fertility in Austria, Germany and Switzerland: Is there a common pattern?" *Comparative Population Studies*, 36.

SOCIO-ECONOMIC PANEL SOEP (2013): "Data for years 1984-2012," *SOEP Version 29*.

SPARÉN, P., D. VÅGERÖ, D. B. SHESTOV, S. PLAVINSKAJA, N. PARFENOVA, V. HOPTIAR, D. PATUROT, AND M. R. GALANTI (2004): "Long term mortality after severe starvation during the siege of Leningrad: prospective cohort study," *BMJ: British Medical Journal*, 328, 11.

STADTVERWALTUNG MANNHEIM, STATISTISCHES AMT, ed. (1950): *Statistischer Jahresbericht der Stadt Mannheim 1947*, Mannheim: Mannheimer Zeitungsverlag.

STATISTISCHE ÄMTER DES BUNDES UND DER LÄNDER (2011): "Gemeindeverzeichnis Online Schnellabfrage," `http://www.statistik-portal.de/Statistik-Portal/gemeindeverz.asp?G=`, last accessed June 2016.

STATISTISCHE ÄMTER DES BUNDES UND DER LÄNDER, ed. (2013): *Gemeindeverzeichnis. Alle politisch selbständigen Gemeinden (mit Gemeindeverband) in Deutschland nach Fläche, Bevölkerung (auf Grundlage des Zensus 2011), Bevölkerungsdichte und der Postleitzahl des Verwaltungssitzes der Gemeinde. Ergänzt um die geografischen Mittelpunktkoordinaten, Reisegebiete und EU-Stadt/Landgliederung*, Gebietsstand 31.12.2012, Statistisches Bundesamt Wiesbaden.

STATISTISCHES BUNDESAMT (1983): *Historisches Gemeindeverzeichnis für die Bundesrepublik Deutschland: Namens-, Grenz- u. Schlüsselnummernänderungen bei Gemeinden, Kreisen u. Regierungsbezirken*, Stuttgart/Mainz: Kohlhammer.

STATISTISCHES BUNDESAMT, D. (2017): "Average number of children per woman," `https://www.destatis.de/EN/FactsFigures/SocietyState/Population/Births/Tables/BirthRate.html`, last accessed August 2017.

SÜSS, W. (2003): *Der "Volkskörper" im Krieg - Gesundheitspolitik, Gesundheitsverhältnisse und Krankenmord im nationalsozialistischen Deutschland 1939-1945*, Institut für Zeitgeschichte, Munich: Oldenbourg Verlag.

SUSSER, E. AND D. ST CLAIR (2013): "Prenatal Famine and Adult Mental Illness: Interpreting Concordant and Discordant Results from the Dutch and Chinese Famines," *Social Science & Medicine*, 97, 325–330.

SUSSER, M. AND Z. STEIN (1994): "Timing in Prenatal Nutrition: a Reprise of the Dutch Famine Study," *Nutrition Reviews*, 52, 84–94.

TALGE, N. M., C. NEAL, AND V. GLOVER (2007): "Antenatal maternal stress and long-term effects on child neurodevelopment: how and why?" *Journal of Child Psychology and Psychiatry*, 48, 245–261.

TONKISS, J., M. TRZCIŃSKA, J. R. GALLER, N. RUIZ-OPAZO, AND V. L. HERRERA (1998): "Prenatal malnutrition-induced changes in blood pressure dissociation of stress and nonstress responses using radiotelemetry," *Hypertension*, 32, 108–114.

TOVÉE, M. J., S. REINHARDT, J. L. EMERY, AND P. L. CORNELISSEN (1998): "Optimum body-mass index and maximum sexual attractiveness," *The Lancet*, 352, 548.

TRITTEL, G. J. (1990): *Hunger und Politik – Die Ernährungskrise in der Bizone (1945 – 1949)*, Frankfurt/New York: Campus Verlag.

VALENTE, C. (2014): "Civil conflict, gender-specific fetal loss, and selection: A new test of the Trivers-Willard hypothesis," *Journal of Health Economics*, 39, 31–50.

VAN DEN BERG, G. J., G. DOBLHAMMER, AND K. CHRISTENSEN (2009): "Exogenous determinants of early-life conditions, and mortality later in life," *Social Science & Medicine*, 68, 1591–1598.

VAN DEN BERG, G. J., G. DOBLHAMMER-REITER, AND K. CHRISTENSEN (2011): "Being born under adverse economic conditions leads to a higher cardiovascular mortality rate later in life: Evidence based on individuals born at different stages of the business cycle," *Demography*, 48, 507–530.

VAN DEN BERG, G. J. AND S. GUPTA (2015): "The role of marriage in the causal pathway from economic conditions early in life to mortality," *Journal of health economics*, 40, 141–158.

VAN DEN BERG, G. J., A. HAMMERSCHMID, AND M. A. WEYNANDT (2014a): "Bereavement effects and early life circumstances," *mimeo*.

VAN DEN BERG, G. J., M. LINDEBOOM, AND F. PORTRAIT (2006): "Economic conditions early in life and individual mortality," *The American Economic Review*, 96, 290–302.

VAN DEN BERG, G. J., P. LUNDBORG, P. NYSTEDT, AND D.-O. ROOTH (2014b): "Critical periods during childhood and adolescence: A study of adult height among immigrant siblings," *Journal of the European Economic Association*, 12, 1521–1557.

VAN DEN BERG, G. J. AND P. R. PINGER (2016): "Transgenerational effects of childhood conditions on third generation health and education outcomes," *Economics & Human Biology*, 23, 103–120.

VAN DEN BERG, G. J., P. R. PINGER, AND J. SCHOCH (2016): "Instrumental Variable Estimation of the Causal Effect of Hunger Early in Life on Health Later in Life," *The Economic Journal*, 126, 465–506.

VAN OS, J. AND J.-P. SELTEN (1998): "Prenatal exposure to maternal stress and subsequent schizophrenia. The May 1940 invasion of The Netherlands," *The British Journal of Psychiatry*, 172, 324–326.

VONYÓ, T. (2012): "The bombing of Germany: the economic geography of war-induced dislocation in West German industry," *European Review of Economic History*, 16, 97–118.

WADHWA, P. D. (2005): "Psychoneuroendocrine processes in human pregnancy influence fetal development and health," *Psychoneuroendocrinology*, 30, 724–743.

WAGNER, G. G., J. R. FRICK, AND J. SCHUPP (2007): "The German Socio-Economic Panel Study (SOEP) – Scope, evolution and enhancements," *Schmollers Jahrbuch*, 127, 139–169.

WAITE, L. J. (1995): "Does marriage matter?" *Demography*, 32, 483–507.

WAITE, L. J. AND E. L. LEHRER (2003): "The benefits from marriage and religion in the United States: A comparative analysis," *Population and Development Review*, 29, 255–275.

WALDRON, I. (1983): "The role of genetic and biological factors in sex differences in mortality," in *Sex differentials in mortality: trends, determinants and consequences*, ed. by A. Lopez and L. Ruzicka, Canberra: Australian National University.

WERRELL, K. P. (1986): "The strategic bombing of Germany in World War II: Costs and accomplishments," *The Journal of American History*, 73, 702–713.

WHITAKER, R. C., S. M. PHILLIPS, AND S. M. ORZOL (2006): "Food insecurity and the risks of depression and anxiety in mothers and behavior problems in their preschool-aged children," *Pediatrics*, 118, e859–e868.

XIONG, X., E. W. HARVILLE, D. R. MATTISON, K. ELKIND-HIRSCH, G. PRIDJIAN, AND P. BUEKENS (2008): "Exposure to hurricane Katrina, post-traumatic stress disorder and birth outcomes," *The American Journal of the Medical Sciences*, 336, 111–115.

ZEITLER, R., B. MEWES, ET AL., eds. (1936-1952): *Statistisches Jahrbuch Deutscher Gemeinden*, vol. 31-40, Deutscher Städtetag, Gustav Fischer Jena.

ZIMMERMANN, A. C. AND R. A. EASTERLIN (2006): "Happily ever after? Cohabitation, marriage, divorce, and happiness in Germany," *Population and Development Review*, 32, 511–528.

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