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War during Childhood: The Long Run Effects of Warfare on Health

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DISCUSSION PAPER SERIES

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ABSTRACT

War during Childhood: The Long Run Effects of Warfare on Health*

This paper estimates the causal long-term consequences of an exposure to war in utero and during childhood on the risk of obesity and the probability of having a chronic health condition in adulthood. Using the plausibly exogenous city-by-cohort variation in the intensity of WWII destruction as a unique quasi-experiment, I find that individuals who were exposed to WWII destruction during the prenatal and early postnatal periods have higher BMIs and are more likely to be obese as adults. I also find an elevated incidence of chronic health conditions such as stroke, hypertension, diabetes, and cardiovascular disorder in adulthood among these wartime children.

JEL Classification: I10, I12, J13

Keywords: warfare, body size, health conditions, children

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I. Introduction

Globally, child malnutrition continues to be one of the most serious problems, affecting the lives of millions of children and families. According to the UNICEF-WHO-World Bank joint Report (2012), 26% of children under five years of age worldwide, a total of some 165 million children, currently suffer from malnutrition. If current trends continue, malnutrition will affect more than 450 million children globally over the next 15 years. Child malnutrition has devastating consequences for children’s well-being, both immediate and long lasting. The thrifty phenotype hypothesis suggests that individuals’ metabolisms adapt to the dire nutritional conditions that they experience during the pre- or early post-natal period in order to survive. This leaves individuals who suffer from malnutrition during this critical period more susceptible to both obesity and chronic health conditions such as coronary heart disease, stroke, diabetes and hypertension later in life (Barker, 1992). Wars and armed conflicts pose a substantial threat to the economic resources and health care available to infants and children, and create food shortages and changes in the composition of food eaten; therefore, they may have especially enduring and devastating impacts on children’s long-term health. However, to date there has been only limited research exploring how such an exposure to armed conflict before birth and in early childhood affects children’s body size, obesity, and probability of having a chronic health condition later in life.

This paper analyzes the long-run causal effects of being born or growing up during war on an individual’s adult BMI, obesity and probability of having a stroke, hypertension, cardiovascular disorder, or diabetes. Specifically, I use the city-by-cohort variation in destruction in Germany that arose from the extensive Allied Air Forces (“AAF”) aerial attacks during WWII as a unique quasi-experiment. I employ a difference-in-differences-type strategy, where the “treatment” variable is an interaction between the city-level intensity of WWII destruction and an indicator for being born or being a young child during WWII. In this setting,

I also control for city and birth year fixed effects. The validity of difference-in-differences estimation relies on the existence of parallel trends in body size and chronic health conditions between the affected and control cohorts across cities of varying levels of intensity of wartime destruction, had WWII not occurred. I assess the plausibility of this assumption below by performing a falsification test where I repeat the analysis using the older and younger cohorts. The control experiment shows that the parallel trend assumption is satisfied, which lends credence to the difference-in-differences estimation.

This paper contributes to several areas of research. The first is the literature that examines the causal association between the early childhood environment and health outcomes later in life. In accordance with Baker’s (1992) hypothesis, this strand of the literature finds that malnutrition and poor living conditions *in-utero* and during early childhood have long-lasting adverse effects on individuals’ self-reported health status, mental health, height, schizophrenia in adulthood, and life expectancy.¹ Such research has addressed the effects of extreme weather conditions, famines, disease, natural disasters, and economic crises. This paper adds to this literature by quantifying the long-term effects of an exposure to wartime destruction following conception and during early childhood on body size, obesity and the probability of having a chronic health condition later in life.

This study also contributes in several ways to the growing body of literature examining the immediate and long-term consequences of armed conflicts on children’s health outcomes. By mainly focusing on armed conflicts in developing countries, several studies have found that exposure to armed conflicts during pre- or early post-natal period is associated with lower birth weights, lower height-for-age, height and self-reported health in teenage years and adulthood. Mansour and Ress (2012) find that Palestinian mothers’ exposure to armed conflict during pregnancy by the Israeli security forces is associated with a modest increase in the

¹For detailed information on the long-term effects of early childhood shocks, see Almond and Currie (2011a, 2011b).

probability of having a low birth weight child. Alderman, Hoddinott and Kinsey (2004), Minou and Shemyakina (2012) and Akresh, Lucchetti and Thirumurthy (2012) examine how pre-or-early post-natal exposure to armed conflict affects the height-for-age Z-scores among children in Zimbabwe, Cote d'Ivoire and Eritrea, respectively. These papers find that such exposure to armed conflict in conception or during early childhood leads to a substantially lower height-for-age among the affected children.

Similarly, Akresh et al. (2012) find that wartime girls who were 3 and younger during the Nigerian civil war are 0.75 centimeters shorter as adults relative to girls of the same cohort residing in unaffected areas. Akbulut-Yuksel (2014) shows that the school disruption caused by the extensive bombing campaign of Allied Air Forces leads to a 0.4 fewer years of schooling, and lower self-rated health satisfaction among school-age children and lower adult height. However, Almond and Currie (2011b) and Hoynes et al. (2016) suggest that *in utero* or early childhood exposure to dire conditions could have a direct effect on the individuals' long-term health outcomes such as body mass index, obesity and chronic health conditions independent of its effect through years of schooling due to the mismatch experienced between childhood and adulthood environment in the availability of nutrition. Therefore, it is imperative to investigate these latent effects of armed conflicts on very long-term health outcomes.

To the best of my knowledge, this is the first paper that studies the long-term effects of early childhood exposure to armed conflicts by focusing on adult BMI, obesity and chronic health conditions that are not readily available in many survey data. Second, this paper improves our knowledge on the pathways through which armed conflicts affect longer-term health outcomes. Such detailed formal investigation of channels and sources of heterogeneity is limited in the previous studies exploring health effects of warfare. I have collected a battery of city-level historical data from German archives such as immediate postwar birth and infant mortality rates, destruction of hospitals and postwar per capita health

expenditure for the analyses in this paper to assess the postwar conditions and health care available to wartime children. I further formally test other potential mechanisms and heterogeneity in the longer-term health effects of early childhood exposure to war by parental educational attainment, father's occupation, the loss of a parent during the war years, and the deployment of a father for war combat. Finally, by collecting very detailed data on rubble per capita for each of former West Germany's 75 cities from historical archives, this paper quantifies the realized wartime destruction and explores the spatial variation in wartime destruction intensity within Germany. Thus, it estimates the long-term effects of WWII on children's health outcomes in adulthood in a richer way than previous studies have done.

I find that an exposure to wartime destruction during the fetal period or early childhood had long-term detrimental effects on individuals' health that remained even 60 years after WWII. First, individuals who were born or were young children during WWII in a hard-hit city had about 1.5-point higher body mass index in adulthood than the wartime children in the less destroyed cities. Second, I find that these wartime children were almost 16 percentage points more likely to be obese as adults if they were residing in highly destroyed city over the course of WWII. Third, my results suggest that an exposure to armed conflict during the prenatal and early postnatal periods significantly increases the likelihood of having a chronic health condition later in life. Wartime children in the hard-hit cities are 0.23 standard deviations more likely to have a chronic health condition as adults compared to their counterparts in the less destroyed cities. The long-term health costs of WWII fell disproportionately on wartime children residing in urban areas in the most hard-hit cities, and with less well-educated parents. Furthermore, my analysis shows that a father's involvement in combat and the death of one or both parents during the war years have limited long-term effects on future body size or chronic health conditions. Therefore, malnutrition, changes in daily diet and a limited access to health care during WWII are potential mecha-

nisms for the observed long-term health effects. These results remain robust after I account for the potential changes in the composition of the population, infant and adult mortality rates, selective wartime fertility, and city-specific trends by prewar city characteristics and state-specific policies in postwar Germany.

The remainder of the paper is organized as follows. Section 2 provides a brief background of AAF bombing in Germany during WWII. Section 3 describes the city-level destruction data and individual-level survey data used in the analysis. Section 4 discusses the identification strategy. Section 5 presents the main results and robustness checks. Section 6 describes potential mechanisms and heterogeneity in the long-term health effects of WWII. Section 7 concludes.

II. Background on the Extensive Bombing Campaign of Allied Air Forces during World War II

Bomber Command’s area offensive represented “true aerial warfare”, and the bombing campaign was the only offensive action in Germany between 1940 and 1944 (Werrell, 1986). The overwhelming majority of the AAF’s aerial attacks consisted of nighttime area bombing. The objective of area bombing was to drop a bomb that would start a fire in the center of a town that might consume the whole town. The AAF’s extensive bombing campaign left more than 14 million people in Germany homeless, and killed close to 3.5 million civilians and 3.3 million soldiers (Meiners, 2011; Heineman, 1996). While most of the destroyed buildings were apartment buildings, every city also lost other kinds of public buildings, including hospitals, as well as roads, which led to food shortages and limited access to health care.

Over the course of WWII, every German city was bombed, though the number of bombs dropped and the resulting physical destruction varied substantially across cities. Figure 1 illustrates the shares of dwellings destroyed in German cities by 1945. As Figure 1 suggests, visibility from the air, not strategic significance, determined which cities AAF targeted; visibility was generally determined

by weather conditions and the presence of outstanding landmarks such as cathedrals (Grayling, 2006; Friedrich, 2002). The degree of damage and the resulting amount of rubble also depended upon the distance of each town from the English air bases (most of which were located close to London) and the technological developments at the time of the bombing. As aircraft and bomb technology and operational techniques improved over the course of the war, AAF aerial attacks deep in the heart of Germany became possible, but a town’s proximity to England continued to determine the level of destruction.

Between AAF strategy and bombing techniques at the time, the degree of destruction in each city depended on the number of bombs dropped, fixed city characteristics such as size and the presence of visible landmarks, the accuracy of the bombers, and weather conditions at the time of bombing. Therefore, based on historical accounts, I will take the cross-city variation in intensity of WWII destruction as exogenous in my analysis after controlling for city fixed effects. This suggests that the extent of the wartime destruction experienced in two similar cities such as Cologne and Munich should be random after controlling for fixed city characteristics.

III. Data and Descriptive Statistics

This analysis combines individual-level data from the German Socio-Economic Panel (GSOEP) with a novel, detailed dataset of the city-level wartime physical destruction. GSOEP is a nationally representative panel survey that provides detailed information on individual and household characteristics, including parental background, the childhood environment in which individuals grew up, and the region of residence since 1985. Moreover, the GSOEP has detailed information on whether an individual’s parent(s) died during the war years and on father’s wartime service, which enables me to quantify some of the potential mechanisms by which wartime destruction might have affected the children’s long-term health outcomes. My main analysis focuses on individuals born between 1922 and 1960,

and is restricted to former West Germany, for which wartime destruction data is available.

I utilize the 2002 survey wave for individuals' BMI and obesity, since the GSOEP has provided height and weight information since 2002. BMI is defined as the weight, measured in kilograms, divided by the square of height, measured in meters. BMI is of particular importance in the epidemiology and medical literatures, as it reflects both the height and weight of an individual, and is known as a standard measure of fatness and obesity (Oreffice and Quintana-Domeque, 2016; Cawley, 2004). Moreover, the 2009 and 2011 waves were the first to ask respondents whether they had ever been diagnosed with hypertension, diabetes or a heart condition, or had ever had a stroke. I then used this information to create a metabolic syndrome index, in order to increase the statistical power and enable the detection of effects that are consistent across specific outcomes. I constructed the metabolic syndrome index using the method of Kling, Liebman, and Katz (2007) and Hoynes, Whitmore-Schanzenbach, and Almond (2016); that is, I defined each of the health conditions as a dummy variable that takes the value 1 if an individual reports having received a diagnosis. The metabolic syndrome index is then defined as the equal weighted average of the standardized z-score measures of each component (i.e., had a stroke or heart condition or was diagnosed with high blood pressure or type 2 diabetes). The standardized z-score for each health condition is calculated by subtracting the mean and dividing by the standard deviation of the control cohorts. Therefore, a higher value of the metabolic syndrome index indicates worse health.²

As a measure of the wartime devastation in each German city, I utilize residen-

²Specifically, the metabolic syndrome index is generated as an equal weighted average of the z-score of each component as follows:

$$(1) \quad \text{MetabolicSyndromeIndex}_i = \frac{1}{J} \sum \frac{y_{ij} - \mu_j}{\sigma_j}$$

where μ_j and σ_j are the mean and standard deviations for the control cohorts, respectively. J stands for the number of components.

tial rubble in m^3 per capita accumulated by the end of WWII (Kaestner, 1949).³ In addition to wartime destruction data, I have also collected other historical data for assessing the prewar and immediate postwar conditions in each German city. The 1939 German Municipality Statistical Yearbook provides unique prewar data for each city, including the city size, population density, average income per capita, birth and infant mortality rates, and number of hospitals. I also collected city-level data on fertility and infant mortality rates between 1946 and 1949 and the number of hospitals in 1947 from the 1949 German Municipality Statistical Yearbooks. Finally, I have assembled postwar city-level data on health expenditures per capita for 1950, 1954, 1959, 1965, 1968, 1969 and 1972 to quantify the postwar health investments.⁴

The wartime destruction measure and other regional historical data that I use in my main analysis vary at the Regional Policy Region (“ROR” or “city”) level, which is the smallest representative geographical unit in GSOEP. RORs are geographical areas that encompass the aggregation of Landkreise and kreisfreie Staedte (two types of administrative districts, analogous to counties in the United States), and represent the center of the local labor market and the surrounding small towns and rural areas (Jaeger et al., 2010). The former West Germany has 75 RORs.

I obtain my historical regional variables, including the rubble in m^3 per capita, by aggregating these variables according to the 1985 German regional (ROR) boundaries. This aggregation is possible because each municipality reported in the yearbooks belongs to only one current-day region. The rubble in m^3 per capita measure was then generated by dividing the aggregate rubble in m^3 that had accumulated in a given region by the end of WWII by the population of the region in 1939. Finally, I merge this historical dataset with GSOEP using the respondents’ city of residence in 1985, which is the earliest date for which both the

³The previous papers which utilize rubble per capita as a measure of wartime destruction include Akbulut-Yuksel (2014), Buchardi and Hassan (2013), Redding and Sturm (2008) and Brakman et al. (2004).

⁴Health expenditure data are only consistently available for these years.

city of residence and individual and parental characteristics are available. None of the individual-level German datasets provide information on the place of birth or childhood place of residence for the cohorts of individuals that I focus on in this study (Pischke and von Wachter, 2008); therefore, my analysis uses the 1985 city of residence. Historically, Germany has low levels of geographic mobility relative to the United States and the United Kingdom, with childhood and early adulthood being the periods of lowest mobility (Rainer and Siedler, 2009; Hochstadt, 1999). While the urban population may have fled to the countryside at times of heavy aerial bombing, seeking shelter, food, and protection, historical accounts document that such wartime displacement was temporary. By June 1947, the urban population had reached 80 percent of its prewar levels, and it was nearly 90 percent in 1948 (Hochstadt, 1999). Therefore, the consequences of internal migration for my analysis are minor.

Table 1 displays the descriptive statistics for population-weighted historical data. We can see from the table that all cities in West Germany experienced severe destruction during the extensive AAF bombing campaign. On average, by the end of WWII, German cities had 9.2 m^3 of rubble per capita, and 38 percent of all housing units had been destroyed. Table 1 further illustrates that there was a significant level of variation in the number of bombs dropped and the intensity of destruction across cities. By the end of WWII, the German cities with above-average destruction had over five times as much rubble per capita as cities with below-average destruction.⁵ A potential complication could arise because the worst destroyed cities are larger, have higher population densities, and had higher than average per capita incomes in 1938, meaning that trends in individuals' body size and health in adulthood could actually reflect differences in city sizes and average incomes. The analysis therefore controls for city fixed characteristics. In addition, I also perform falsification tests/control experiments using data on

⁵I split the sample into above and below the mean, using rubble per capita as the measure of destruction.

older and younger cohorts, in order to assess the presence of differential trends across cities with varying intensities of wartime destruction. Likewise, to control for potential differences in prewar and postwar city-specific cohort trends, the analysis also controls for the linear state-cohort trends and the interaction between the prewar city population density and linear year of birth.

Table 2 shows the descriptive statistics for the adult health outcomes of wartime children, including BMI, obesity, and chronic health conditions, such as having a stroke, high blood pressure, diabetes or a heart condition. The average BMI in my sample is 26 points, and 17 percent of the sample are obese as adults. Table 2 also indicates that 17 percent of the respondents in my sample had been diagnosed with diabetes, and in 2009 or 2011, 26 percent reported having angina or a heart condition. This is not surprising, given that all of the participants were at least 51 years old in 2011. The health outcomes reported in Table 2 are measured six decades after WWII, and reflect the outcomes of WWII survivors who lived until at least 2002. Given that healthier individuals are more likely to survive under challenging conditions, my analysis yields the lower-bound estimates of the long-term effects of wartime destruction on children’s future health outcomes.

IV. Identification Strategy

I utilize a difference-in-differences-type strategy to causally identify the long-term health effects of wartime destruction on wartime children. In this setting, the main variable of interest is an interaction between the city-level intensity of WWII destruction and the dummy for being *in utero* or a young child during WWII.⁶ In particular, β provides the proposed estimate of the average treatment effect in the following baseline city and birth year fixed effects equation:

⁶Hence, I utilize the city-by-cohort variation in exposure to WWII destruction; this analysis may yield lower-bound estimates for the aggregate nation-wide health effects of WWII.

$$(2) Y_{irt} = \alpha + \beta \text{Destruction}_r * \text{WWII}_{it} + \delta_r + \gamma_t + \theta_s * t + \varphi \text{CB39}_r * t + \pi' \mathbf{X}_{irt} + \epsilon_{irt},$$

where Y_{irt} is the health outcome in adulthood for individual i in city r , born in year t . Destruction_r is a measure of the war damage in city r . WWII_{it} is a dummy variable that takes a value of one if individual i was born between 1934 and 1945, and zero otherwise. δ_r represents unrestricted city-specific fixed effects and γ_t is the unrestricted birth year fixed effects. $\theta_s * t$ is the interaction of state dummies with linear year of birth which controls for the potential postwar state-specific policies. $\text{CB39}_r * t$ controls for the interaction of baseline city characteristics such as the prewar population density with linear year of birth. \mathbf{X}_{irt} is a vector of individual characteristics, including gender and rural dummies, as well as parental characteristics such as parental education, father's occupation dummies, mother's age at individual i 's birth. Finally, ϵ_{irt} is a random, idiosyncratic error term. The standard errors are clustered by the individual's city.

In their seminal paper, Almond and Currie (2011b) suggest that the adverse shocks negatively affect individuals between conception and 5 years of age more often than older people. Therefore, in my empirical analysis, individuals who were born between 1934 and 1945 form the affected group, since they were five years old or younger at some point during WWII, and would all have experienced early life shock because of the war. On the other hand, the cohorts who were older at the onset of WWII (i.e., the 1922-1933 cohorts) and those born after WWII (i.e., the 1951-1960 cohorts) represent the control groups.⁷

The validity of the difference-in-differences analysis relies on the parallel trend assumption, which assumes that cities with varying intensities of wartime destruction would have exhibited parallel trends in body size and chronic health

⁷I dropped the cohorts born between 1946 and 1950 from the analysis, since they were born immediately following WWII and were exposed to the postwar reconstruction. Nevertheless, the results remain quantitatively similar but smaller in magnitude when the cohorts born between 1946 and 1950 are added to the control cohorts.

conditions had war not occurred. I assess the plausibility of this assumption below by performing a falsification test where I repeat the analysis using the older and younger cohorts. This control experiment satisfies the parallel trend assumption, lending credence to the difference-in-differences estimation.

V. Estimation Results

A. Body Mass Index and Obesity

Table 3 reports the results from estimating Equation (2), where the dependent variable is the wartime children’s BMIs in adulthood. Each column relates to a separate regression that controls for city and birth year fixed effects, along with gender and rural dummies. The difference-in-differences estimate in column (1) is 0.08. This suggests that children who resided in Cologne, a heavily destroyed city, with $25.25\text{ }m^3$ rubble per capita, have an adult BMI that is 1.5 points higher than that of children who resided in Munich, a less destroyed city, with $6.50\text{ }m^3$ rubble per capita.⁸ This suggests that an exposure to wartime destruction *in utero* or during early childhood increases the risk of being overweight as an adult. Column (1) further points to gender differences in BMI, with females having an average BMI that is one point lower than that of their male counterparts.

I include an individual’s years of schooling as an additional control in column (2). Previous studies have found that better educated individuals are more likely to have lower BMIs (Hiermeyer, 2009). Therefore, it is possible that the limitations in an individual’s educational attainments during the war years partly explain their higher BMIs. Similar to earlier studies, column (2) shows that an individual’s educational attainments are an important determinant of their BMI in adulthood, with an extra year of schooling leading to a 0.2 point decrease in an individual’s BMI. Nonetheless, the difference-in-differences estimate for wartime destruction remains economically and statistically significant after I control for

⁸These two cities were very similar in terms of their prewar characteristics, but Cologne was closer to the aerial bomber fields in England, and therefore experienced more destruction.

individuals' educational attainments, which suggests that the health effects of warfare are persistent. Column (3) of Table 3 also controls for parental educational attainments, father's occupational dummies,⁹ and mother's age at the child's birth, which might serve as a proxy for the parents' economic well-being and the resources available to the child during their childhood. The results reported here indicate that, once an individual's own education is controlled for, parental characteristics do not seem to affect the adult BMI. The last column adds state-specific linear year of birth trends and the interaction of the prewar city-level population density with linear year of birth, to account for potential postwar state-specific policies and prewar city characteristics.¹⁰ The difference-in-differences estimate for wartime destruction reported in the first row remains economically and statistically similar in this specification as well.

Having shown that wartime destruction caused the affected cohorts to have higher BMIs in adulthood, it is of interest to analyze whether WWII destruction also affects adult obesity, since obesity leads to serious health problems later in life (Hoynes, Whitmore-Schanzenbach and Almond, 2016). Table 4 presents the estimation results, where the outcome is the likelihood of wartime children being obese when over the age of 55. Individuals are coded as obese if their BMI is 30 or higher. The difference-in-differences estimate for wartime destruction reported in column (1) is 0.0087. This indicates that WWII cohorts residing in Cologne have a 16 percentage points higher probability of adulthood obesity relative to the same cohorts in Munich. Similarly to the BMI analysis, column (2) reports the specification incorporating individual's years of schooling. I find that an additional year of schooling leads to approximately a two-percentage point decrease in obesity. Furthermore, the analysis presented in columns (3)-(4) shows that the negative long-term effects of wartime destruction were borne by all wartime children, regardless of their family backgrounds and these estimated

⁹The omitted group for father's occupation is self-employed.

¹⁰I determine the prewar city-level population density by dividing the city's population in 1939 by its area in 1939.

effects are robust to different specifications.

B. Metabolic Syndrome

Table 5 estimates how war devastation following conception and in the early years of childhood affects the likelihood of having a chronic health condition later in life. The 2009 and 2011 waves of GSOEP were the first to report whether respondents had had a stroke or angina/heart condition, or had ever been diagnosed with high blood pressure or diabetes. I improve the statistical power and detect effects that are consistent across specific health conditions by using this newly available information to generate a metabolic syndrome index, following the method of Kling, Liebman, and Katz (2007) and Hoynes, Whitmore-Schanzenbach, and Almond (2016). That is, I define each health condition as a dummy variable that takes a value of one if an individual reports having received a diagnosis, and zero otherwise. Hence, all chronic conditions are presented as dummy variables, and all have the same interpretation. I then calculate the standardized z-score for each health condition by subtracting the mean and dividing by the standard deviation of the control cohorts. Finally, the metabolic syndrome index is defined as the equal weighted average across the standardized z-score of each component (i.e., had a stroke or heart condition or was diagnosed with high blood pressure or diabetes). A higher value of the metabolic syndrome index indicates worse health.

Table 5 presents the results for the long-term effects of wartime destruction on metabolic syndrome. Column (1) shows that, on average, wartime children in hard-hit cities are 0.23 standard deviations more likely to have a metabolic syndrome when they are over 65 than the same cohorts in the least destroyed cities. Similar to the BMI and obesity analyses, I find that females and individuals who are more educated are less prone to having a metabolic syndrome late in life. On the other hand, parental education, father's occupation and mother's age at the child's birth have limited influences on an individual's susceptibility to

a chronic health condition after controlling for the individual’s own educational attainments. The difference-in-differences estimates remain virtually unchanged after I control for state-cohort trends and the interaction of the prewar population density and birth year, as is summarized in column (4).

Finally, I have generated a new variable for wartime exposure by taking advantage of the variation in the number of years that an individual was potentially affected by WWII destruction. This variable is generated by assuming that an exposure to wartime destruction affected an individual’s health outcomes if they were born or were five and younger at any point over the course of WWII. Since WWII occurred between 1939 and 1945, this new variable takes a value of one if the individual was born in 1934 or 1945, two if the individual was born in 1935 or 1944, three if the individual was born in 1936 or 1943, and four if the individual was born in 1937 or 1942. Finally, the variable takes a value of five if an individual was born between 1938 and 1941, and zero otherwise. Results where the affected cohort dummy is replaced with a length of exposure are presented in Appendix Table 1. I find in Appendix Table 1 that a one-year exposure to wartime destruction leads to a 0.46 point increase in BMI and a 4.3 percentage point increase in the incidence of obesity. Similarly, metabolic syndrome increases by 0.05 standard deviations if the child was exposed to wartime destruction for a year. These coefficients are obtained by comparing wartime children in the hard-city city of Cologne to the same cohorts of children in less-destroyed Munich (the difference in rubble amount between these two cities is $18.75 m^3$ per capita). To interpret the coefficients, I multiply the difference-in-differences estimates presented in Appendix Table 1 by 18.75. These additional analyses show that the estimation results presented in Tables 3, 4 and 5 also hold when the destruction variable is interacted with a continuous measure of the number of years that a cohort was exposed to the war.

C. Sensitivity Analyses

In this subsection, I discuss whether the estimation analyses summarized in Tables 3-5 are robust to potential confounding factors, such as prewar and postwar city-specific cohort trends, differential adult mortality, selective wartime fertility and infant mortality rates, differential postwar state-specific policies, and internal migration. These sensitivity analyses are presented in Tables 6 and 7. I find that the long-term adverse health effects of WWII remain economically and statistically significant even after accounting for these potential confounding factors.

First, as has been mentioned, the results reported in Tables 3-5 rely on the parallel trend assumption, which assumes that, in the absence of WWII, the affected and control cohorts' body sizes and chronic health conditions would have been similar across cities with varying intensities of war destruction. That is, in the absence of WWII, the coefficient on the interaction between the dummy for being born in 1934-1945 and the city-level wartime destruction would be zero. I assess the validity of the identifying assumption by performing a falsification test/control experiment, the results of which are summarized in Table 6. In this control experiment, the oldest cohorts (i.e., those born between 1922 and 1933) are treated as the "placebo" affected cohorts, and the youngest cohorts (born between 1951 and 1960) are in "placebo" control cohorts. In Table 6, I find that the difference-in-differences estimates for the control experiment are statistically insignificant and close to zero for all health outcomes. This supports the parallel trend assumption, since it shows that the differences in adult BMI, obesity, and metabolic syndrome between the oldest and the youngest cohorts are similar across cities. Table 6 further shows that wartime destruction has no effect on the body sizes and health conditions of either the earlier or later birth cohorts. Therefore, it is unlikely that the differential city-specific prewar and postwar cohort trends drive the results presented in Tables 3-5.

Second, if the postwar economic and health policies in Germany had been determined at the city level, they could have had differential effects on the postwar

cohorts in the cities with higher levels of wartime destruction. However, the federal and state governments determine policies in Germany. I use a lower level of aggregation than the state for estimating the long-term effects of wartime destruction on individual’s adult health outcomes, which allows me to explore the within-state variation. This mitigates any bias that might arise from postwar state-specific policies, but I still account for the state-cohort effects formally in my analysis by controlling state-specific linear year of birth trends in the last columns of Tables 3-5. The estimation results remain robust in the specification incorporating state-cohort trends.

In addition, I have attempted to gauge the postwar health expenditure differences across cities with different levels of wartime destruction by collecting a novel dataset on the city-level postwar per capita health expenditure from the statistical yearbooks. Figure 2 illustrates the postwar per capita health expenditures across cities with high and low levels of wartime destruction in 1950, 1954, 1959, 1965, 1968, 1969 and 1972.¹¹ We see from Figure 2 that the per capita health expenditure is similar across cities with high and low levels of wartime destruction, which bolsters our confidence in the difference-in-differences estimates.

Similarly, potential policy differences between larger and smaller cities might challenge the difference-in-differences estimation. I address this by controlling for the interaction between the city-level prewar population density and the individuals’ birth year in my analysis. The results from this specification are presented in the last columns of Tables 3-5. The difference-in-differences estimates are robust to the inclusion of the interaction between the city-level prewar population density and year of birth, which suggests that the estimated long-term health effects among wartime children are not driven by the varying intensities of prewar population density.

The third potential confounding factor is related to a possible composition bias. First, children who grew up in cities with different levels of wartime destruction

¹¹Health expenditure data are only consistently available for these years.

might have experienced differential mortality in adulthood. Second, wartime destruction might also have altered the fertility and infant mortality rates in hard-hit cities, creating differential cohort sizes based on the level of wartime destruction experienced in a city. The first column of Table 7 presents the mortality results for wartime children. For this analysis, I explore the panel structure of GSOEP, which enables me to analyze the mortality of the affected cohorts between 1985 and 2011. The mortality variable refers to a dummy variable that takes the value of 1 if an individual has a recorded death sometime between the years 1985 (the beginning of my sample) and 2011 (the last wave of GSOEP used in the analysis), and zero otherwise. The difference-in-differences estimate in column (1) is statistically insignificant and close to zero, indicating that differential mortality rates across cities do not confound the results presented in Tables 3-5.

Column (2) of Table 7 provides further evidence of the lack of a composition bias. Similarly to Meng, Qian and Yared (2015), I formally test whether the birth cohort size in a given city is related to wartime destruction. As Meng, Qian and Yared (2015) suggest, the birth cohort size is a good proxy for wartime survival, since it incorporates wartime fertility, infant mortality and later life mortality, thus enabling us to assess the possibility of differential adult and infant mortality, as well as selective wartime fertility. The outcome of interest in column (2) is the birth cohort size in each city in 1985. I find that the birth cohort size is not associated with wartime destruction, meaning that it is unlikely that the estimation results are driven by differential survival rates across cities. Taken together, the analyses presented in columns (1) and (2) suggest that results are not an artefact of a mortality-induced selection bias.

Another concern that is related to composition bias is selective wartime fertility. Given that access to oral contraception was not readily available to married women until after about the late 1960s, or to single women until the 1970s (Goldin and Katz, 2002), the contemporary family planning methods were not widely available for the parents of the cohorts studied in this paper. In addition, half

of the cohorts affected (i.e., the 1934-1939 cohorts) were born before the onset of WWII, meaning that their fertility decisions were not affected by wartime destruction. Columns (3)-(7) of Table 7 further investigate whether selective wartime fertility was present among the wartime children. More specifically, columns (3)-(6) estimate whether parents that are more affluent delayed childbearing during the war years, and column (7) investigates whether wartime destruction is associated with the loss of a father during WWII. I find that mother’s age at the child’s birth, parental educational attainments, and father’s occupation are not associated with wartime destruction, suggesting that selective fertility based on parental characteristics was not present. Similarly, column (7) shows that the loss of a father during WWII was not related to the city’s wartime destruction, providing further support for the lack of selective wartime fertility.

As a final check on fertility and infant mortality, I collected city-level data on fertility and infant mortality rates before the onset of WWII in 1938 and immediately after the end of WWII between 1946 and 1949 to determine whether there are any substantial differences in prewar or postwar birth and infant mortality rates across German cities. Appendix Table 2 presents estimation results for BMI when incorporating the interaction of city-level birth rates or infant mortality rates in 1938 and between 1946 and 1949 with being in the affected cohort. Appendix Table 3 presents the same analysis for obesity, while Appendix Table 4 presents the estimation results for other health conditions.¹² The results in these appendix tables indicate that the long-term health effects of wartime destruction are still statistically and economically significant after accounting for prewar or postwar birth and infant mortality rates. This suggests that neither prewar or postwar infant mortality nor the postwar baby boom is driving the estimation results.

Non-random migration between cities might also confound the results. The

¹²The infant mortality rate is defined as the number of deaths in the first year of life per 1000 individuals.

intensity of AAF aerial attacks might have altered the population composition in highly destroyed cities. However, historically, Germany has had low levels of geographic mobility, with childhood and early adulthood being the periods of lowest mobility (Rainer and Siedler, 2009; Hochstadt, 1999). Historical accounts also document that the wartime displacement was only temporary (Hochstadt, 1999), since the destruction of postal and telephone communication during WWII meant that the only way for family members to reunite was to stay in or return to their home cities (Geo Epoche Panorama, 2014). In addition, movements between occupation zones were restricted, and individuals were not allowed to travel beyond their local areas (Allied Control Authority, 1946; Hochstadt, 2011).

I address the possibility of non-random migration by first estimating Equation (2) with the probability of moving as the dependent variable, to assess whether a city’s wartime destruction prompted individuals’ internal migration decisions. Column (8) of Table 7 provides the results. I code individuals as movers if they report in 1985 that they no longer reside in their childhood city or area.¹³ The treatment and control groups for this specification are the same as in the previous main analysis. The difference-in-differences estimates for the probability of moving are close to zero and statistically insignificant in every specification. This suggests that individuals did not choose their final destinations based on the relative destruction of the cities. Second, I drop the city-states of Berlin, Hamburg and Bremen, to which particularly large numbers of individuals might have moved, in order to test the robustness of the results to potential internal migration. The results remain statistically and quantitatively similar to the baseline specification when these city-states are excluded.¹⁴

¹³GSOEP doesn’t report the geocodes of childhood city or area.

¹⁴An additional concern related to mobility is refugees or people who fled from the former parts of the Germany and Soviet Zone/GDR. I attempt to address this potential concern by using the official 1961 city-level refugee data provided by Redding and Sturm (2008). I estimate the baseline specification separately for cities with refugee numbers that are above and below the median, and find similar effects for the two samples.

VI. Mechanisms and Heterogeneous Effects of Wartime Destruction on Health Outcomes in Adulthood

This section investigates the heterogeneity in the effects of exposure to WWII by the child’s gender, type of residence, and parental characteristics. It also analyzes the potential channels through which war destruction may have affected the children’s later life health outcomes, such as the loss of a parent during the war years, father’s involvement with war combat, and the destruction of hospitals. The results are summarized in Tables 8-10.

Table 8 reports the potential mechanisms and heterogeneous effects of wartime destruction on wartime children’s adult BMIs. The second and third columns report the difference-in-differences estimates for the female and male subsamples, respectively. These columns show that the difference-in-differences estimate is large for the female sample; however, it is not statistically significantly different from the male sample. Column (4) considers whether individuals who resided in urban areas experienced a larger war effect. The difference-in-difference estimates for the urban population in column (4) of Table 8 is larger than that for the baseline specification reported in column (1), indicating that children in urban areas suffered more from the adverse effects of wartime destruction than those in rural areas. This finding is in line with the fact that rural areas had a better capacity to feed themselves during the war years, and is therefore children residing in rural areas were less susceptible to food shortages.

In columns (5) and (6), I restrict to sample to children whose mothers and fathers had less than basic education, respectively.¹⁵ Findings presented in column (5) suggest that children whose mothers had less than basic education suffered more from adverse health effects of wartime destruction. On the other hand, the difference-in-differences estimate remain similar to the baseline specification when

¹⁵Students receive the basic school diploma (Hauptschule) after 9 years of schooling in Germany. As shown in Table 2, the majority of children have parents with basic education or less (83% of fathers and 88% of mothers in my sample completed basic education or less).

sample is restricted to children whose father had less than basic education. Thus, there is some evidence suggesting that children from well-off families probably did not experience the same mismatch between childhood and future environments in regard to nutrition availability, which leads to a higher BMI, obesity, and metabolic syndrome. Consequently, they have lower BMIs, less obesity, and a reduced incidence of metabolic syndrome later in life than their peers with less educated parents.

Columns (7) and (8) introduce war-related controls to the baseline specification, such as whether fathers fought actively in the war and whether a parent died during the war years, in order to account for a family’s firsthand experience with the consequences of warfare. Controlling for whether the father fought in WWII and for whether a parent died during WWII leaves the difference-in-differences estimates for war destruction unchanged, suggesting that direct family experience with WWII combat does not determine the effects on children’s adult BMIs.

Table 9 and Table 10 consider the potential mechanisms and the heterogeneous effects of exposure to wartime destruction on obesity and chronic health conditions in late adulthood, respectively. In both tables, the difference-in-differences estimate is larger for urban population suggesting that wartime children in urban areas were worse off relative to the same cohorts residing in rural areas. Table 9 and Table 10 further show that the adverse health effects of war exposure were stronger for children from less affluent families. Finally, the last two columns of Table 9 and Table 10 reveal no heterogeneity by the loss of parent(s) or the deployment of a father during WWII.

Taken together, Tables 8-10 suggest that maternal and infant malnutrition during the war years and the change in their daily diets probably altered German children’s biological metabolisms in the pre- or early post-natal period, leading to an increase in their body sizes in adulthood when they no longer face malnutrition. Thus, these war children are prone to chronic health problems such as cardiovascular disorder, diabetes, and high blood pressure in late adulthood,

which are correlated strongly with obesity.

A limited access to health care during early childhood might also increase the long-term health effects of warfare, given that AAF's area bombings destroyed and damaged hospitals, public buildings, and roads in every city. In addition, some doctors had to join the army, and a significant number were Jewish (Evans, 2005). Figure 3 shows the destruction of hospitals by the overall destruction intensity in the city, and indicates that cities with more rubble per capita also experienced a greater decline in the number of hospitals. Therefore, wartime children in more destroyed areas received less health care during the prenatal and early postnatal periods because the hospitals were defunct due to bombings and the departure of doctors.

VII. Conclusion

This paper provides the causal evidence on the long-run consequences of warfare and armed conflicts on wartime children's body mass index, obesity and chronic health conditions later in life. Using GSOEP, I find that an exposure to WWII destruction caused Germans who were *in utero* or in their early childhood years during WWII to have a higher body mass index and a higher probability of obesity in adulthood. Moreover, I find a higher than usual rate of high blood pressure, diabetes, and cardiovascular disorder diagnoses, as well as strokes, among these wartime children when they are over 65. Children who lived in the most-hard hit cities during bombings and in urban areas and had less educated parents disproportionately show these detrimental, long lasting effects of WWII destruction. Maternal and infant malnutrition, changes in the daily diet and limited access to health care during WWII are potential underlying mechanisms behind these estimated long-term health effects.

In recent years, armed conflicts seem to have become both more common and more physically destructive (Collier, Hoeffler and Rocher, 2009), meaning that the debate on the short- and long-term health effects of armed conflicts and

the mechanisms by which they harm children is likely to retain its place at the center of public policy. The findings of this paper shed light on the potential legacies of recent armed conflicts on the long-term health of affected children. These results suggest that even though severely-hit cities return rapidly to their prewar patterns in terms of the local population and macroeconomic indicators (Davis and Weinstein, 2002; Brakman, Garretsen and Schramm, 2004; Miguel and Roland, 2011), armed conflicts still place substantial direct and latent burdens on children's physical development that last a lifetime. These analyses therefore underline the importance of postwar policies primarily targeting infants and young children, in order to mitigate and even, if possible, reverse these adverse long-term health effects of armed conflicts around the globe.

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TABLE 1—DESCRIPTIVE STATISTICS FOR WWII DESTRUCTION

	All	RORs with Above avg. Destruction	RORs with Below avg. Destruction	Difference s.e (Difference)
	(1)	(2)	(3)	(4)
Rubble in m^3 per Capita	9.168 (6.330)	15.530 (4.293)	4.643 (2.488)	10.887*** (0.148)
Housing Units Destroyed (%)	37.521 (18.479)	49.492 (13.680)	29.007 (16.642)	20.485*** (0.682)
Total bombs dropped in tons	25,279.740 (22,201.880)	34,324.950 (21,639.400)	18,845.960 (20,277.750)	15,478.990*** (918.598)
Area in km^2 in 1938	252.339 (235.403)	342.650 (286.076)	188.102 (163.506)	154.548*** (9.813)
Population Density in 1939	2,018 (887.948)	2,249 (963.445)	1,854 (790.511)	395*** (38.171)
Income per Capita in RM in 1938	462.670 (106.979)	509.552 (53.582)	425.866 (122.890)	83.686*** (4.792)
N	2,122	882	1,240	2,122

Notes: Standard deviations are in parentheses. The sample consists of Raumordnungsregionen ("RORs" or "cities") in the former territory of West Germany. The means for destruction measures are weighted by population. The sample is divided as above and below destruction using rubble in m^3 per capita as a measure of wartime destruction.

TABLE 2—DESCRIPTIVE STATISTICS, GSOEP DATA

	All	RORs with Above avg. Destruction	RORs with Below avg. Destruction
	(1)	(2)	(3)
Body Mass Index	26.376 (4.130)	26.446 (4.010)	26.326 (4.214)
Obese	0.174 (0.379)	0.175 (0.380)	0.173 (0.379)
Had a Stroke	0.071 (0.256)	0.073 (0.261)	0.069 (0.253)
Have High Blood Pressure	0.515 (0.500)	0.525 (0.500)	0.509 (0.500)
Have Diabetes	0.177 (0.382)	0.185 (0.389)	0.172 (0.377)
Had Angina or Heart Condition	0.261 (0.439)	0.236 (0.425)	0.278 (0.448)
Years of Schooling	11.301 (2.302)	11.422 (2.368)	11.215 (2.250)
Mother with Basic Education	0.882 (0.323)	0.868 (0.338)	0.892 (0.311)
Father with Basic Education	0.830 (0.375)	0.800 (0.400)	0.852 (0.355)
Age	42.833 (11.403)	42.884 (11.175)	42.796 (11.567)
Female	0.533 (0.499)	0.515 (0.500)	0.546 (0.498)
Urban	0.571 (0.495)	0.590 (0.492)	0.557 (0.497)
N	2,122	882	1,240

Notes: Data are from the 2002, 2009 and 2011 waves of GSOEP. The sample consists of individuals born between 1922 and 1960.

TABLE 3—EFFECT OF WWII DESTRUCTION ON BODY MASS INDEX IN ADULTHOOD

	(1)	(2)	(3)	(4)
Destruction x WWII Cohort	0.0835** (0.0358)	0.0777** (0.0355)	0.0744** (0.0326)	0.0688** (0.0326)
Female	-1.0011*** (0.1957)	-1.1876*** (0.2053)	-1.1514*** (0.2311)	-1.1610*** (0.2346)
Years of Schooling		-0.2055*** (0.0518)	-0.1792** (0.0683)	-0.1930*** (0.0659)
Mother has more than Basic Education			0.0362 (0.5440)	0.0603 (0.5500)
Father has more than Basic Education			0.1897 (0.5325)	0.1667 (0.5255)
Father had a blue collar job			0.1244 (0.2685)	0.161 (0.2768)
Father had a white collar job			-0.4011 (0.3083)	-0.3676 (0.3103)
Father had a civil servant job			0.4518 (0.4283)	0.4911 (0.4268)
Mother's age at birth			-0.0184 (0.0193)	-0.0186 (0.0194)
R^2	0.095	0.106	0.112	0.12
N	2,119	2,109	1,952	1,952
Linear State-cohort Trends				Yes
Prewar Population x Linear Year of Birth				Yes

Notes: Standard errors clustered by cities are shown in parentheses. Asterisks denote significance levels (*=.10, **=.05, ***=.01). Each column controls for city and birth year fixed effects, and gender and rural dummies. Columns (4) also controls linear state-cohort trends and the interaction of prewar city-level population density with linear year of birth. The omitted category for father's occupation is self-employed.

TABLE 4—EFFECT OF WWII DESTRUCTION ON OBESITY IN ADULTHOOD

	(1)	(2)	(3)	(4)
Destruction x WWII Cohort	0.0087** (0.0037)	0.0077** (0.0036)	0.0104*** (0.0037)	0.0099*** (0.0037)
Female	-0.1248*** (0.0242)	-0.1451*** (0.0256)	-0.1425*** (0.0315)	-0.1427*** (0.0318)
Years of Schooling		-0.0207*** (0.0058)	-0.0172** (0.0071)	-0.0189*** (0.0070)
Mother has more than Basic Education			0.0047 (0.0571)	0.0002 (0.0580)
Father has more than Basic Education			0.0242 (0.0575)	0.0218 (0.0575)
Father had a blue collar job			0.033 (0.0325)	0.0352 (0.0327)
Father had a white collar job			-0.0301 (0.0352)	-0.0262 (0.0346)
Father had a civil servant job			0.0592 (0.0470)	0.0674 (0.0467)
Mother's age at birth			-0.002 (0.0025)	-0.0018 (0.0025)
R^2	0.087	0.094	0.108	0.114
N	2,119	2,109	1,952	1,952
Linear State-cohort Trends				Yes
Prewar Population x Linear Year of Birth				Yes

Notes: Standard errors clustered by cities are shown in parentheses. Asterisks denote significance levels (*=.10, **=.05, ***=.01). Each column controls for city and birth year fixed effects, and gender and rural dummies. Columns (4) also controls linear state-cohort trends and the interaction of prewar city-level population density with linear year of birth. The omitted category for father's occupation is self-employed.

TABLE 5—EFFECT OF WWII DESTRUCTION ON METABOLIC SYNDROME IN ADULTHOOD

	(1)	(2)	(3)	(4)
Destruction x WWII Cohort	0.0122** (0.0056)	0.0119** (0.0056)	0.0111** (0.0053)	0.0093* (0.0052)
Female	-0.0419 (0.0282)	-0.0582** (0.0271)	-0.0639** (0.0304)	-0.0532** (0.0301)
Years of Schooling		-0.0168* (0.0088)	-0.0119 (0.0109)	-0.0117 (0.0107)
Mother has more than Basic Education			-0.0457 (0.0740)	-0.0814 (0.0698)
Father has more than Basic Education			0.0476 (0.0788)	0.0555 (0.0759)
Father had a blue collar job			0.066 (0.0475)	0.067 (0.0463)
Father had a white collar job			0.0089 (0.0694)	0.0247 (0.0701)
Father had a civil servant job			-0.0984 (0.0606)	-0.1042 (0.0627)
Mother's age at birth			-0.0029 (0.0035)	-0.0022 (0.0035)
R^2	0.259	0.262	0.296	0.314
N	1,181	1,176	1,081	1,081
Linear State-cohort Trends				Yes
Prewar Population x Linear Year of Birth				Yes

Notes: Standard errors clustered by cities are shown in parentheses. Asterisks denote significance levels (*=.10, **=.05, ***=.01). Each column controls for city and birth year fixed effects, and gender and rural dummies. Columns (4) also controls linear state-cohort trends and the interaction of prewar city-level population density with linear year of birth. The omitted category for father's occupation is self-employed.

TABLE 6—FALSIFICATION TESTS

	Body Mass Index		Obesity		Metabolic Syndrome	
	(1)	(2)	(3)	(4)	(5)	(6)
Destruction x Born btw. 1922-1933	-0.0327 (0.0388)	0.0068 (0.0531)	-0.0042 (0.0039)	-0.0008 (0.0050)	0.001 (0.0109)	-0.0085 (0.0163)
Female	-1.2445*** (0.2472)	-1.2263*** (0.2887)	-0.1287*** (0.0295)	-0.1191*** (0.0372)	-0.0848 (0.0628)	-0.068 (0.0651)
Years of Schooling		-0.1459 (0.0893)		-0.0118 (0.0092)		-0.0194 (0.0191)
Mother has more than Basic Education		0.0086 (0.7047)		-0.0031 (0.0876)		-0.0167 (0.0844)
Father has more than Basic Education		0.5729 (0.6344)		0.0412 (0.0772)		0.0557 (0.0814)
Father had a blue collar job		0.1062 (0.3163)		0.0133 (0.0393)		0.1738 (0.1043)
Father had a white collar job		-0.6952 (0.4275)		-0.0969** (0.0474)		0.0503 (0.1253)
Father had a civil servant job		0.3233 (0.6019)		-0.0039 (0.0577)		-0.0125 (0.1075)
Mother's age at birth		-0.003 (0.0219)		0.0001 (0.0027)		0.0006 (0.0059)
R^2	0.134	0.147	0.104	0.125	0.365	0.424
N	1312	1216	1312	1216	660	610
Linear State-specific Trends		Yes		Yes		Yes
Prewar Population x Linear Year of Birth		Yes		Yes		Yes

Notes: Standard errors clustered by cities are shown in parentheses. Asterisks denote significance levels (*=.10, **=.05, ***=.01). The control group is individuals born between 1951 and 1960. The "Placebo" affected group is individuals born between 1922 and 1933. Each column controls for city and birth year fixed effects and gender and rural dummies. The omitted category for father's occupation is self-employed.

TABLE 7—VALIDITY CHECKS

	Mortality	Cohort Size	Mother's Age at Birth	Parental Educ.	Father BC	Father WC	Father died during WWII	Move
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Destruction X WWII Cohort	-0.003 (0.0018)	0.0374 (0.0469)	-0.0103 (0.0425)	-0.0020 (0.0019)	-0.0016 (0.0021)	-0.0013 (0.0015)	-0.0013 (0.0019)	0.0037 (0.0025)
Female	-0.0821*** (0.0118)	-0.0322 (0.0561)	0.235 (0.1917)	0.0345*** (0.0120)	-0.0306** (0.0145)	0.0254*** (0.0092)	-0.0129 (0.0090)	0.0595*** (0.0152)
Years of Schooling	-0.0067*** (0.0024)	-0.007 (0.0142)	0.1970*** (0.0496)	0.0683*** (0.0037)	-0.0439*** (0.0033)	0.0242*** (0.0025)	0.0000 (0.0024)	0.0303*** (0.0043)
R^2	0.161	0.548	0.061	0.228	0.1	0.092	0.071	0.116
N	4,545	4,545	4,106	4,545	4,545	4,545	4,528	4,528

Note: Notes: Standard errors clustered by cities are shown in parentheses. Asterisks denote significance levels (*=.10, **=.05, ***=.01). Each column controls for city and year of birth fixed effects and gender and rural dummies.

TABLE 8—POTENTIAL MECHANISMS AND HETEROGENEITY IN THE EFFECT OF WWII DESTRUCTION ON BMI IN ADULTHOOD

	Base Results (1)	Female Only (2)	Male Only (3)	Urban Only (4)	Mother Basic Educ (5)	Father Basic Educ (6)	Father fought (7)	Parent died (8)
Destruction X WWII Cohort	0.0777** (0.0355)	0.0939* (0.0518)	0.0567 (0.0373)	0.1082* (0.0559)	0.0928** (0.0398)	0.0770* (0.0397)	0.0810** (0.0352)	0.0786** (0.0357)
Years of Schooling	-0.2055*** (0.0518)	-0.2966*** (0.0868)	-0.1355** (0.0529)	-0.1711*** (0.0627)	-0.1995*** (0.0603)	-0.1980*** (0.0651)	-0.2086*** (0.0516)	-0.2066*** (0.0518)
Father fought in WWII							-0.2898 (0.7114)	
Mother died during WWII								-0.7290 (1.0414)
Father died during WWII								-0.2315 (0.3612)
R^2	0.106	0.160	0.135	0.141	0.118	0.138	0.106	0.106
N	2,109	1,122	987	1,200	1,780	1,656	2,104	2,109

Notes: Standard errors clustered by cities are shown in parentheses. Asterisks denote significance levels (*=.10, **=.05, ***=.01). Each column controls for city and year of birth fixed effects. Other controls are gender and rural dummies.

TABLE 9—POTENTIAL MECHANISMS AND HETEROGENEITY IN THE EFFECT OF WWII DESTRUCTION ON OBESITY IN ADULTHOOD

	Baseline Results (1)	Female Only (2)	Male Only (3)	Urban Only (4)	Mother Basic Educ (5)	Father Basic Educ (6)	Father fought in WWII (7)	Parent died during WWII (8)
Destruction X WWII Cohort	0.0077** (0.0036)	0.0096* (0.0051)	0.0041 (0.0053)	0.0101* (0.0055)	0.0103** (0.0043)	0.0100** (0.0042)	0.0081** (0.0036)	0.0078** (0.0035)
Years of Schooling	-0.0207*** (0.0058)	-0.0257*** (0.0088)	-0.0187*** (0.0065)	-0.0164** (0.0064)	-0.0195** (0.0075)	-0.0212*** (0.0078)	-0.0207*** (0.0058)	-0.0209*** (0.0058)
∞ Father fought in WWII							0.0287 (0.0832)	
Mother died during WWII								-0.0537 (0.1125)
Father died during WWII								-0.0701 (0.0480)
R^2	0.094	0.143	0.130	0.113	0.104	0.130	0.094	0.095
N	2,109	1,122	987	1,200	1,780	1,656	2,104	2,109

Notes: Standard errors clustered by cities are shown in parentheses. Asterisks denote significance levels (*=.10, **=.05, ***=.01). Each column controls for city and year of birth fixed effects. Other controls are gender and rural dummies.

TABLE 10—POTENTIAL MECHANISMS AND HETEROGENEITY IN THE EFFECT OF WWII DESTRUCTION ON THE METABOLIC SYNDROME IN ADULTHOOD

	Baseline Results (1)	Female Only (2)	Male Only (3)	Urban Only (4)	Mother Basic Educ (5)	Father Basic Educ (6)	Father fought in WWII (7)	Parent died during war (8)
Destruction X WWII Cohort	0.0119** (0.0056)	0.0048 (0.0071)	0.0197* (0.0103)	0.0212*** (0.0070)	0.0127** (0.0060)	0.0138** (0.0063)	0.0121** (0.0056)	0.0114** (0.0056)
Years of Schooling	-0.0168* (0.0088)	-0.0287*** (0.0105)	-0.0103 (0.0125)	-0.0227* (0.0117)	-0.0087 (0.0106)	-0.0108 (0.0111)	-0.0161* (0.0091)	-0.0160* (0.0089)
Father fought in WWII							0.1657 (0.1236)	
Mother died during WWII								0.1645 (0.1729)
Father died during WWII								0.0871 (0.0754)
R^2	0.262	0.352	0.331	0.337	0.286	0.298	0.266	0.264
N	1,176	631	545	666	981	925	1,172	1,176

Notes: Standard errors clustered by cities are shown in parentheses. Asterisks denote significance levels (*=.10, **=.05, ***=.01). Each column controls for city and year of birth fixed effects. Other controls are gender and rural dummies.

TABLE A-1—SPECIFICATION WITH LENGTH OF EXPOSURE

	Body Mass Index		Obesity		Health Conditions	
	(1)	(2)	(3)	(4)	(5)	(6)
Destruction x Number of Years Exposed	0.0262*** (0.0079)	0.0237*** (0.0066)	0.0026*** (0.0008)	0.0027*** (0.0007)	0.0026** (0.0013)	0.0025** (0.0012)
Female	-0.9976*** (0.1939)	-1.1525*** (0.2330)	-0.1245*** (0.0240)	-0.1421*** (0.0317)	-0.0437 (0.0282)	-0.0539* (0.0295)
Years of Schooling		-0.1912*** (0.0654)		-0.0188*** (0.0068)		-0.0115 (0.0108))
Mother has more than Basic Education		0.1292 (0.5469)		-0.0052 (0.0583)		-0.11 (0.0732))
Father has more than Basic Education		0.091 (0.5059)		0.0169 (0.0526)		0.0696 (0.0717)
Father had a blue collar job		0.1667 (0.2780)		0.0353 (0.0326)		0.0702 (0.0455)
Father had a white collar job		-0.3541 (0.3092)		-0.0236 (0.0343)		0.0328 (0.0710)
Father had a civil servant job		0.513 (0.4273)		0.0697 (0.0461)		-0.0998 (0.0629)
Mother's age at birth		-0.0182 (0.0196)		-0.0018 (0.0025)		-0.0023 (0.0035)
R^2	0.097	0.122	0.088	0.115	0.259	0.314
N	2,119	1,952	2119	1,952	1,181	1,081
Linear State-specific Trends		Yes		Yes		Yes
Prewar Population x Linear Year of Birth		Yes		Yes		Yes

Notes: Standard errors clustered by cities are shown in parentheses. Asterisks denote significance levels (*=.10, **=.05, ***=.01). Each column is from

TABLE A-2—ADULT BMI WITH BIRTH RATE AND INFANT MORTALITY CONTROLS

	(1)	(2)	(3)
Destruction x WWII Cohort	0.0864** (0.0394)	0.0904*** (0.0306)	0.0986*** (0.0294)
Female	-1.2053*** (0.2056)	-1.1557*** (0.2065)	-1.1642*** (0.2080)
Years of Schooling	-0.2047*** (0.0533)	-0.1894*** (0.0519)	-0.1879*** (0.0525)
Birth Rate in 1938 X WWII Cohort	0.1279 (0.1412)		0.0333 (0.1070)
Birth Rate in 1946 X WWII Cohort	-0.4432*** (0.1652)		-0.4043*** (0.1234)
Birth Rate in 1947 X WWII Cohort	0.5203** (0.2323)		0.5295*** (0.1751)
Birth Rate in 1948 X WWII Cohort	-0.2442 (0.1966)		-0.1291 (0.1837)
Birth Rate in 1949 X WWII Cohort	-0.0846 (0.1758)		-0.1528 (0.1641)
Infant Mortality in 1938 X WWII Cohort		0.5766** (0.2813)	0.4584** (0.2191)
Infant Mortality in 1946 X WWII Cohort		-0.2701** (0.1249)	-0.2826*** (0.1062)
Infant Mortality in 1947 X WWII Cohort		-0.0405 (0.1908)	-0.1017 (0.1368)
Infant Mortality in 1948 X WWII Cohort		-0.3532** (0.1634)	-0.2519 (0.1658)
Infant Mortality in 1949 X WWII Cohort		0.7574*** (0.2694)	0.8156*** (0.2028)
R^2	0.113	0.118	0.125
N	2,100	2,093	2,093

Notes: Standard errors clustered by cities are shown in parentheses. Asterisks denote significance levels (*=.10, **=.05, ***=.01). Each column is from a separate regression which controls for city and birth year fixed effects, and gender and rural dummies.

TABLE A-3—OBESITY WITH BIRTH RATE AND INFANT MORTALITY CONTROLS

	(1)	(2)	(3)
Destruction x WWII Cohort	0.0084** (0.0042)	0.0082** (0.0035)	0.0086** (0.0038)
Female	-0.1458*** (0.0259)	-0.1418*** (0.0258)	-0.1424*** (0.0260)
Years of Schooling	-0.0207*** (0.0059)	-0.0192*** (0.0058)	-0.0192*** (0.0059)
Birth Rate in 1938 X WWII Cohort	0.0122 (0.0160)		0.0023 (0.0135)
Birth Rate in 1946 X WWII Cohort	-0.0231 (0.0203)		-0.0205 (0.0171)
Birth Rate in 1947 X WWII Cohort	0.0342 (0.0239)		0.0323* (0.0189)
Birth Rate in 1948 X WWII Cohort	-0.0265 (0.0232)		-0.0230 (0.0219)
Birth Rate in 1949 X WWII Cohort	-0.0063 (0.0209)		-0.0028 (0.0214)
Infant Mortality in 1938 X WWII Cohort		0.0315 (0.0285)	0.0250 (0.0244)
Infant Mortality in 1946 X WWII Cohort		-0.0137 (0.0132)	-0.0136 (0.0131)
Infant Mortality in 1947 X WWII Cohort		0.0050 (0.0194)	0.0028 (0.0171)
Infant Mortality in 1948 X WWII Cohort		-0.0439** (0.0192)	-0.0405* (0.0222)
Infant Mortality in 1949 X WWII Cohort		0.0658** (0.0281)	0.0671*** (0.0241)
R^2	0.093	0.095	0.097
N	2,100	2,093	2,093

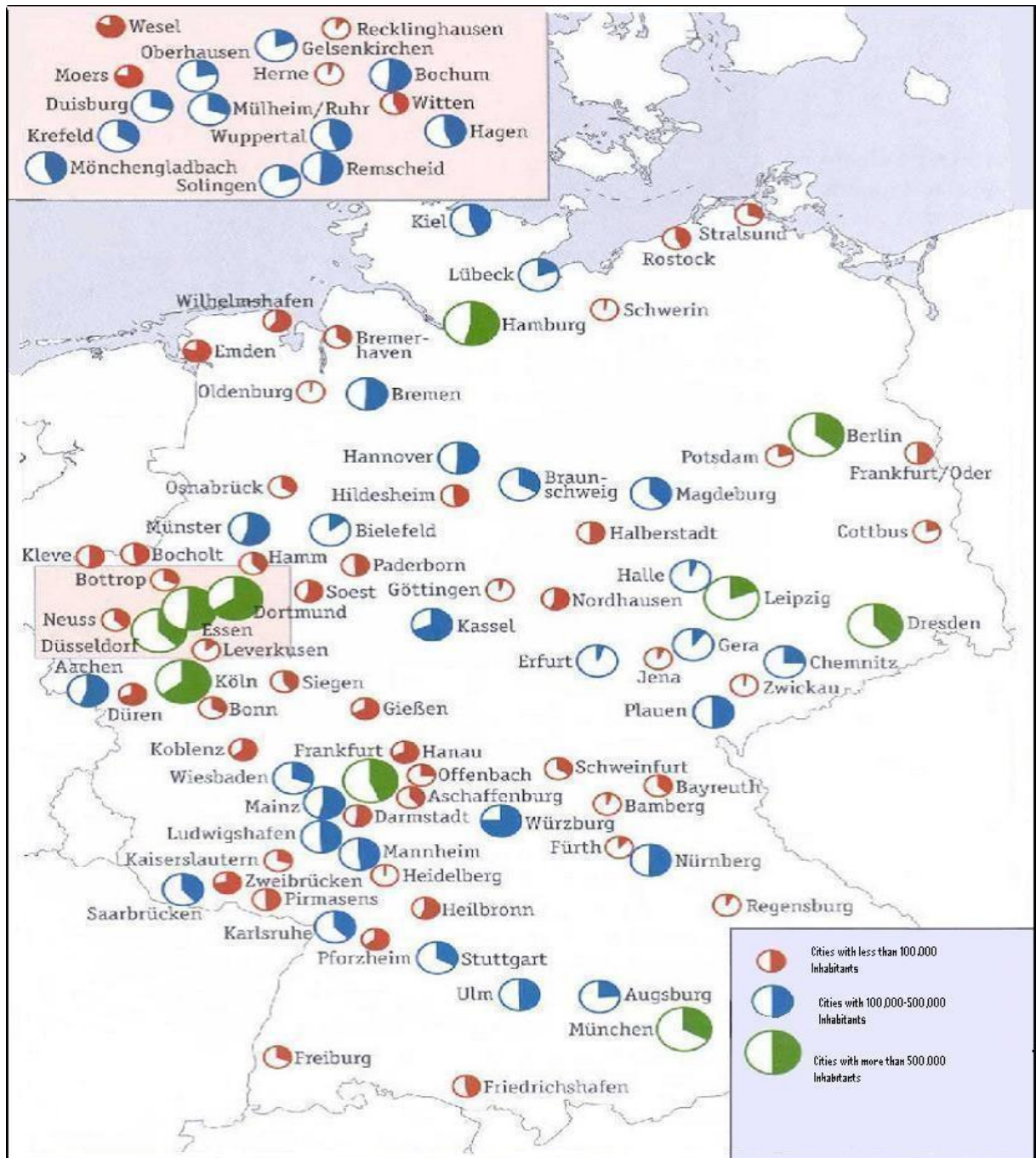
Notes: Standard errors clustered by cities are shown in parentheses. Asterisks denote significance levels (*=.10, **=.05, ***=.01). Each column is from a separate regression which controls for city and birth year fixed effects, and gender and rural dummies.

TABLE A-4—METABOLIC SYNDROME WITH BIRTH RATE AND INFANT MORTALITY CONTROLS

	(1)	(2)	(3)
Destruction x WWII Cohort	0.0110*	0.0139**	0.0135**
	(0.0058)	(0.0061)	(0.0063)
Female	-0.0564**	-0.0562**	-0.0571**
	(0.0271)	(0.0274)	(0.0276)
Years of Schooling	-0.0161*	-0.0168*	-0.0172*
	(0.0088)	(0.0086)	(0.0088)
Birth Rate in 1938 X WWII Cohort	0.0237		0.0335
	(0.0206)		(0.0228)
Birth Rate in 1946 X WWII Cohort	-0.0561**		-0.0588**
	(0.0281)		(0.0291)
Birth Rate in 1947 X WWII Cohort	0.0419		0.0440
	(0.0324)		(0.0361)
Birth Rate in 1948 X WWII Cohort	-0.0161		0.0047
	(0.0443)		(0.0440)
Birth Rate in 1949 X WWII Cohort	-0.0203		-0.0487
	(0.0368)		(0.0410)
Infant Mortality in 1938 X WWII Cohort		-0.0104	-0.0176
		(0.0553)	(0.0468)
Infant Mortality in 1946 X WWII Cohort		-0.0111	-0.0007
		(0.0239)	(0.0227)
Infant Mortality in 1947 X WWII Cohort		-0.0109	-0.0289
		(0.0386)	(0.0291)
Infant Mortality in 1948 X WWII Cohort		0.0411	0.0517*
		(0.0298)	(0.0293)
Infant Mortality in 1949 X WWII Cohort		0.0139	0.0096
		(0.0479)	(0.0370)
R^2	0.269	0.265	0.273
N	1,173	1,168	1,168

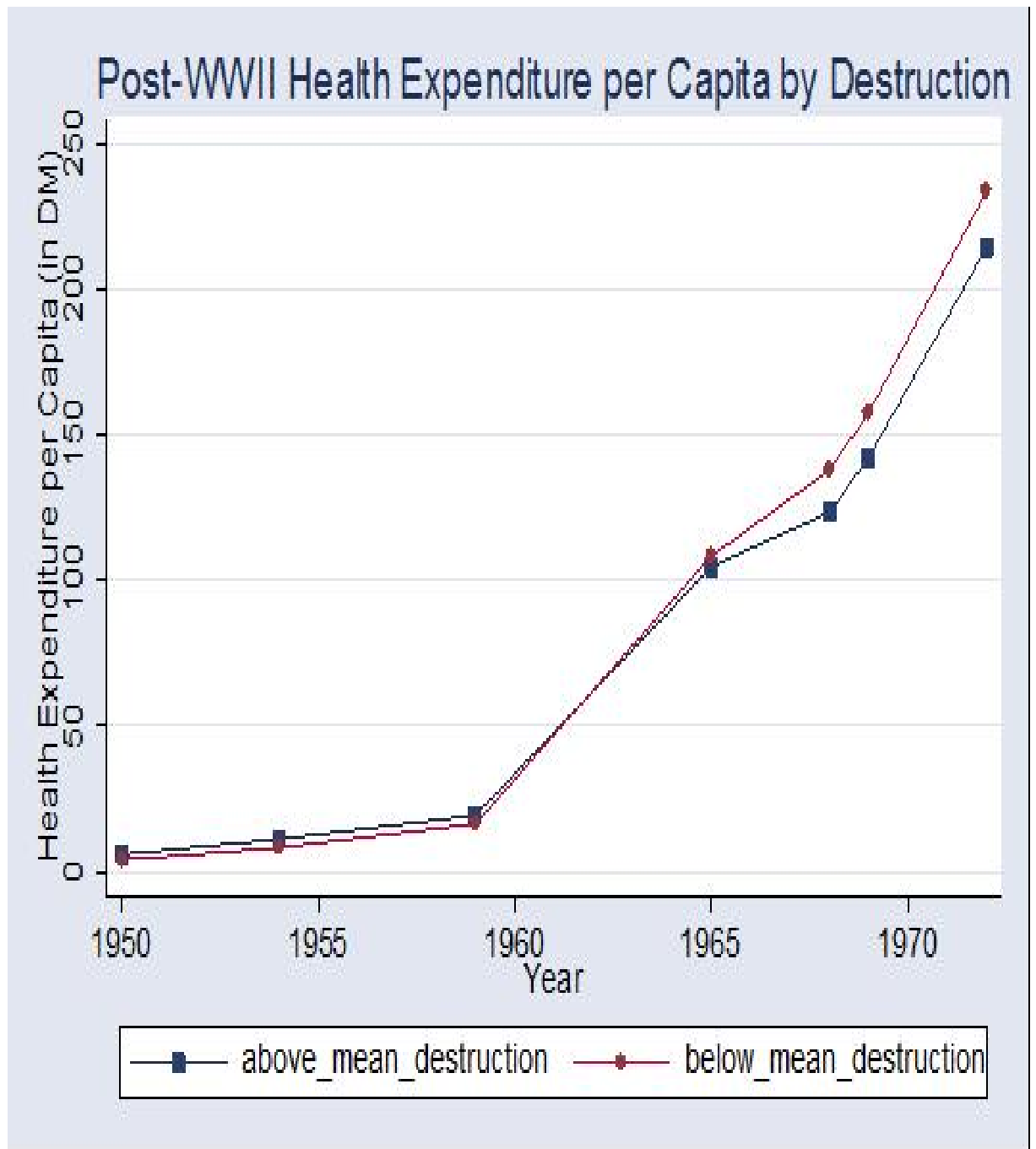
Notes: Standard errors clustered by cities are shown in parentheses. Asterisks denote significance levels (*=.10, **=.05, ***=.01). Each column is from a separate regression which controls for city and birth year fixed effects, and gender and rural dummies.

FIGURE 1. SHARE OF DWELLINGS DESTROYED IN GERMAN CITIES DURING WWII



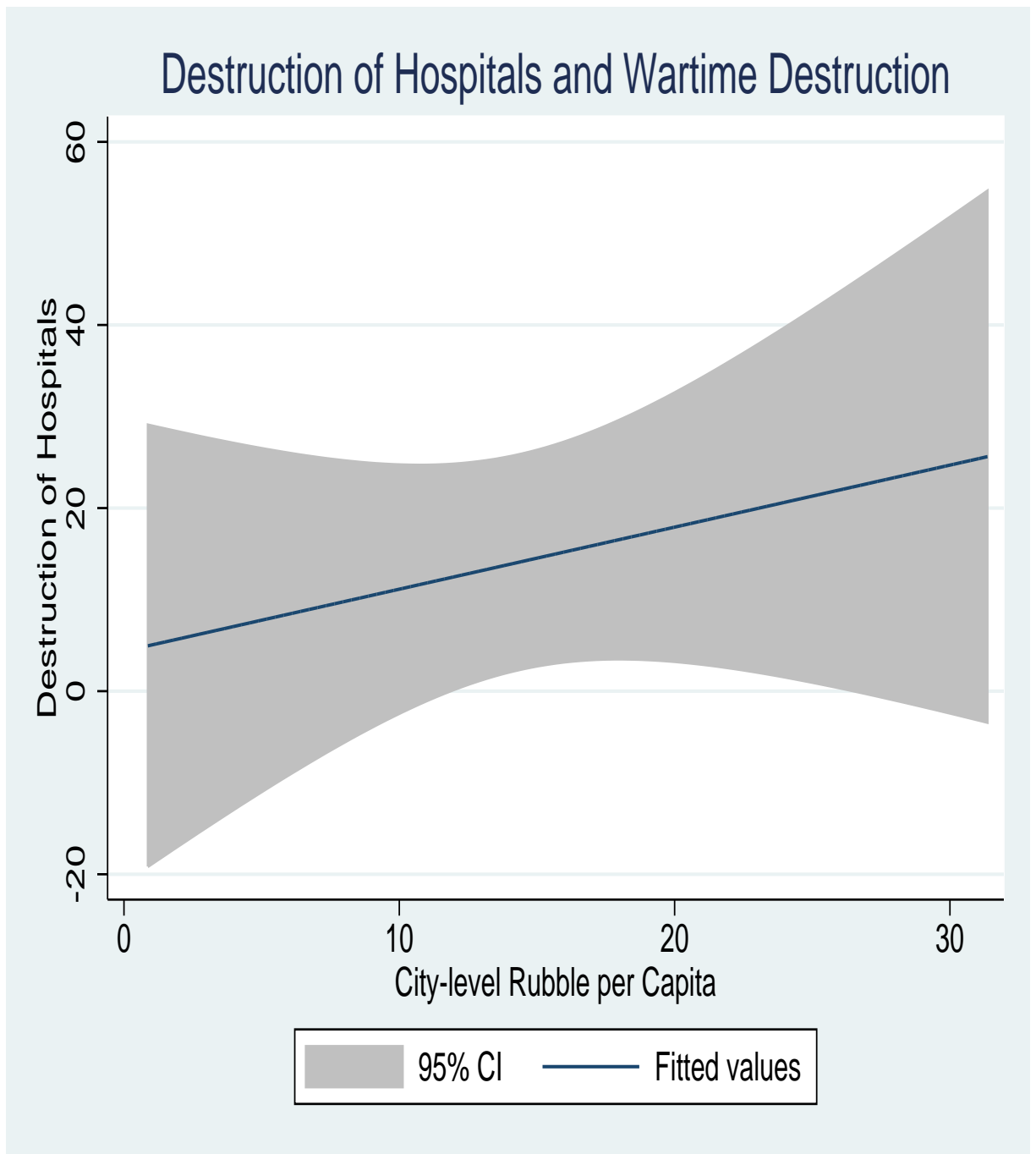
Source: Knopp (2001). The size of the circle shows the city size in 1939, where the largest circle refers to cities with more than 500,000 inhabitants; middle-size circle, cities with between 100,000 and 500,000 inhabitants and smallest circle, cities with less than 100,000 inhabitants. The shaded area in these circles is the share of the dwellings destroyed in the city by the end of WWII.

FIGURE 2. PER CAPITA POSTWAR CITY-LEVEL HEALTH EXPENDITURE



Source: Various years of German Municipalities Statistical Yearbook.

FIGURE 3. DESTRUCTION OF HOSPITALS AND WARTIME DESTRUCTION



Source: The 1939 and 1949 German Municipalities Statistical Yearbook.