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Heterogenous Mechanisms in WWII Stress Transmission: Evidence from a Natural Experiment

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Heterogenous Mechanisms in WWII Stress Transmission: Evidence from a Natural Experiment

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Abstract
This paper analyses how in utero exposure to maternal stress from WWII affects long-term health and economic outcomes and describes different mechanisms at work, showing that current health conditions are heterogeneously related to the type of fetal stressor. We exploit the Italian armistice of September $8th$ 1943 as exogenous variation in the war intensity, providing WWII long-run causal effects on objectively measured health and economic outcomes. We find that in utero exposure to intense WWII events had long- lasting effects on health and that Nazi massacres predict late-onset depression, while nutritional deprivation suffered in large cities had lasting effects on diabetes. Finally, we innovate by showing that these effects increase with the age of the treated individuals.

JEL classification: I1, O1

Keywords: Fetal programming hypothesis; War exposure; Nazi massacres, Stress; Famine; Chronic diseases; Health expenditure, Long-term effects, Italy.

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1 Introduction

The "fetal origins" hypothesis developed by Barker (1990), gave origins to a prominent stream of clinical and economic literature analyzing how "intrauterine environment - and nutrition in particular - *programs* the foetus to have specific metabolic characteristics, which can lead to future disease" (Almond and Currie, 2011). This literature spans across three main dimensions, *i)* the type of treatment analyzed, ranging from disasters, such as famines, pandemics, war, to milder shocks; *ii)* the time lag between the treatment event and the outcome realization, ranging between short, medium and long term; and *iii)* the outcome studied, usually involving health, education or income.[1]

In this paper we contribute to this large body of literature by estimating the causal effects of the heterogenous mechanisms of WWII stress transmission on long run objectively measured health and economic outcomes for a large sample of the Italian population, finding further support to the "fetal origins" hypothesis.

From both a clinical and an economic perspective, investigating the long-run impact of adverse prenatal conditions is of particular importance, since many health complications emerge only later in life. In fact, several chronic diseases are typical of the elderly and are infrequent to appear until the age of 50. Similarly, a series of mental health issues, such as late-onset depression, are likely to have prenatal origins, where intrauterine exposure to adverse environment gives rise to a persistent psychological vulnerability, which in turn may be triggered in periods of accumulated psychosocial stressors typical of the elderly, such as bereavement (e.g., death of a spouse), loss of social role or disease (I. Brilman and Ormel, 2001; Fiske et al., 2009; Gatt et al., 2009; Alexopoulos, 2005). More generally, according to Petronis (2010), and following the theory of epigenetic modifications, adverse conditions experienced in utero determine a series of switches in the "epigenome" endowment, which cause various parts of the genome to be totally latent and potentially, but not necessarily, activated later in life.[2]

[1]For a comprehensive review of the studies carried out see Almond and Currie (2011), Almond et al. (2017) and Lumley et al. (2011).

[2]Epigenetics is defined as heritable changes in gene activity and expression occurring without modification of DNA.

Nevertheless, the detection of long-run causal effects hinges upon a precise measurement of both the treatment nature and the outcome realization. Health proxies such as height or body weight, especially if measured at the time of conscription Banerjee et al. (2010), may neglect specific aspects of adult disease related to fetal programming.[3] Different types of shock suffered in the prenatal era, are likely to foster the occurence of specific health issues, with the literature linking the intrauterine nutritional shortages to adult metabolic disorders, while psychological stress and violence to late-onset mental complications. In a neat representation of the stress suffered and the outcomes scrutinized, objective health measures are likely to deliver a more accurate reflection of how exposure to prenatal adverse conditions alters the phenotype of the offspring. Finally, it can also inform on the possible importance of health protection to pregnant mothers and its long-lasting effects on population health status and health inequality.

To the best of our knowledge, the first economic study of WWII in the intergenerational intrauterine transmission setting is that of Kesternich et al. (2014), who investigate its long-run effects on socioeconomic and health outcomes of the elderly in 13 European countries, employing retrospective data from SHARELIFE. While they find that several measures of war exposure at the individual level predict significant differentials in economic and health outcomes at older ages, their study relies on (retrospective) self-reported outcomes and on aggregated treatment, which potentially leads to measurement errors, thus limiting the causal interpretation of the estimates.[4] A second recent study by Akbulut-Yuksel (2017), uses German Socio Economic Panel together with data on air bombing, in order to exploit city-by-cohort variation in the intensity of WWII. He finds that individuals exposed to war destruction during the prenatal and early postnatal periods are more likely to be obese and to develop metabolic conditions defined as stroke, hypertension, diabetes, or cardiovascular disorder in adulthood. While this study uses refined data on WWII bombing destruction, it relies on the informtion on the place of residence, not birth, of the individuals, and on self-reported

[3]Similarly, mortality data may capture only the most extreme effects of early life shocks, leaving aside the problems related to morbidity.

[4]Another study based on SHARE data is that of Havari and Peracchi (2017), who investigate a similar research question, but extend the analysis to other events such as the WWI and the Spanish flu.

health outcomes, which severely undermines the identification strategy.[5] Overall, both of the studies fail to address the selective fertility issue, which is a crucial source of bias in this kind of estimates.

Compared to this literature on intrauterine transmission of WWII shocks, we innovate under several aspects providing new results, which are arguably not always in line with the exisitng findings. In our understanding, these differences stem from several refinements introduced to the identification strategy, related to the availability of better data on detailed WWII treatment and objectively measured health data.

First, our empirical analysis is based on a dataset consisting of individual electronic clinical records (ECRs) - disease diagnoses and healthcare expenditures - collected by general practitioners (GPs) in Italy between 2004 and 2010 on a large nationally representative sample of adults born in a specific time window during the war time, which have been then linked to different measures of war stressors. The objectively measured health outcomes according to Baker et al. (2004), eliminate a large amount of systematic reporting error due to false negatives.[6] Moreover, given the detailed GP data, we estimate the causal impact of WWII on health expenditure, thus quantifying for the first time in the economic literature the current health care costs of the WWII.

Second, while the disaster literature has frequently focused on single-shock treatments (extreme enough to have persistent effects), war can cause a heterogenous set of disadvantages in adult health through different mechanisms, with not all individuals suffering the same type of stress. Therefore, we innovate by identifing three different mechanisms of war shock transmission and describe how they may heterogeneously affect health outcomes: *i)* the general armed conflicts between the armies with an indirect impact on the civilians, *ii)* the Nazi massacres aimed directly at the civilian population, and *iii)* the nutritional shortages occurring in large urban areas. We measure the general stress intensity with the number of the dead due to general war causes ISTAT (1957), the stress from Nazi activities with the number of

[5] A further limitation is represented by having information only on aerial bombing, which is only one part of the stress caused during war periods.

[6] As also pointed out by Almond et al. (2017), having a large sample of individuals is more likely to provide sufficient sample sizes to detect milder effects of treatments.

victims due to Nazi massacres (Italian Institute of Nazi Massacres), and lastly, we proxy for famine episodes during WWII with the information on the city size.[7]

Third, our empirical identification strategy exploits the variation in type of the adversities of WWII in Italy on a very refined space-time scale (i.e month/province). We employ a difference-in-differences analysis, taking advantage of the unexpected war outbreak occurred after the Italian armistice on September 8^{th} 1943, representing an unambiguous discontinuity in the evolution of the prenatal era circumstances, which breaks the distribution of WWII intensity over time (months) and across space (provinces). The armistice represented the official outbreak of armed conflicts, with important political consequences originating in diffusion of Nazi massacres against the Italian civilian population. Thanks to this unique historical setting, and under very mild assumptions, the estimates obtained from the reduced-form relationship between adverse early life conditions and adult health outcomes can be interpreted as causal.

Fourth, in order to reinforce our causal claims, we carefully discuss and solve the usual problems of this experimental setup, such as possible: *i)* presence of selection due to increased likelihood of infertility, miscarriages, stillbirths; *ii)* errors in geographical attribution of the treatment exposure; *iii)* imprecise measurement of exposure period (gestation); *iv)* early childhood conditions, separating the potential confounding impact of the conditions after birth; and, most importantly, *v)* selective fertility choices. Despite being the quintessential caveat in this type of literature, the last point has never been tackled in the estimation of prenatal exposure to WWII shocks. Our study thus introduces an important novelty by providing a credible solution to this issue.

Based on this setup, we provide several innovative results. We show that individuals exposed to WWII stress in the prenatal era are more likely to bare health disadvantages and, contrary to the previous literature, on WWII, this effect is significant only if the extent of this stress is very intense. We find different health responses, according to the heterogeneity

[7]The lack of food supplies was directly proportional to city sizes, where the destruction of main economic activities, logistic infrastructures and unavailability of several basic goods opened grounds to black markets and impressive inflationary pressure Po (1974).

of the stressor suffered in the prenatal era. In particular, we find that individuals exposed to high intensity of general war stress in the prenatal era have a higher probability of developing a chronic disease and are more likely to suffer from dislipidemia. These effects turn into an overall greater health expenditure and a higher number of contacts with GPs. Moreover, we find that events of violence due to Nazi massacres suffered in utero have a relevant impact on the onset of depression and our difference-in-differences regressions show that Nazi violence is the only mechanism inducing higher depression prevalence in this experiment.

As expected, we also show that the impact of war-induced famine increases the probability of onset pf metabolic diseases as described by diabetes. Finally, we show that the differentials resulting from WWII insults are more likely to appear at older ages. We show that the age-onset pattern is heterogenous and depends on the type of health outcome analysed.

This evidence reconciles with several findings from other literature and builds on them in a unique evaluation. First of all, we confirm the relationship of famine with metabolic complications involving diabetes later in life Lumley et al. (2011). More importantly, as the effect of in utero stress on adult depression is a relatively new research topic, our analyses delivers an important contribution on these grounds. From a clinical perspective, Radtke et al. (2011) find that prenatal exposure to extreme violence, similar to the Nazi massacres, can often contribute to developing depression later in life. Also Class et al. (2014) and Persson and Rossin-Slater (ress), who focus on a sample of cohorts in their thirties, suggest that the uterine environment propagates the impact of stress to the unborn child. At the same time, they highlight that the optimal age for the evaluation of the effects of prenatal stress on psychological disorders is the late adulthood, which coincides with the age of individual analyzed in our study.

In what follows, the paper is organized in 6 sections, including this introduction. In particular, Section 2 describes specific aspects of WWII in Italy, relevant from the point of view of this study. In Section 3 we present the data and the relative sources. Section 4 discusses the identification strategy, while in Section 5 we review the core results and discuss the robustness exercises. Finally, Section 6 concludes showing that an accurate and detailed analysis of the fetal programming hypothesis, based on objective measures, can deliver important findings from a policy maker perspective, enlightening how providing health protection to pregnant mothers may have long-lasting effects on population health status and, perhaps, health inequality.

2 Spatial and temporal intensity of WWII in Italy: the armistice as a natural experiment

In this section we describe the historical events of the WWII armistice in Italy, which are crucial to understand the setting of the study and the sample selection criteria applied. The aspects and circumstances of Pre and Post armistice period let us believe that, under very mild assumptions discussed in Section 4, this scenario can be considered a natural experiment.

Italy was a non-belligerent country until June 1940, when Mussolini declared war to Britain and France. From June 1940 until the end of summer 1943, Italy moved her armed troops mostly outside its national territory in order to expand the colonies. This period was marked by a relatively scarce number of casualties, which concentrated in the surroundings of strategic bombing targets, such as military and commercial harbors, significant industrial sites (i.e. metallurgic, transport and heavy machinery industries), or important railways (Baldoli et al., 2011; Baldoli and Knapp, 2012).

On the 3rd of September the armistice was secretly signed, and made public on the radio on the 8th, with the prime minister and the Italian royal family running away from Rome on the morning of the 9th.[8] Only a few weeks before that date, the Allies landed in Sicily, in order to start advancing in September from the South of Italy towards the mainland and the North of the country against the resisting German forces (see Figure 1). Based on the time-line of these events, synthesized in Figure 2, we develop a precise identification strategy,

[8]The was secretly signed in Santa Teresa Longarini district of Syracuse, 3 km away from Cassibile, from which the armistice was named (Vitali, 1980). It is recognised as the act by which the Kingdom of Italy ceased hostilities towards the Allies during World War II and the beginning of the Italian resistance against fascism. Since this act established its entry into force from the moment of its public announcement, it is commonly referred to as "September 8th", when, at 18:30 Italian time, was announced earlier by the microphones of Radio Algiers by General Dwight Eisenhower and, just one hour later, at 19:42, confirmed by the proclamation of Marshal Pietro Badoglio via the Italian public broadcasting network (EIAR) microphones (Zangrandi, 1974).

Figure 1: WWII fronts in Italy

accounting for two periods: a pre-armistice period (May 1943 - August 1943) that involved a preparatory increase in the number of casualties due air bombing and limited conflicts in the western provinces of Sicily, and the post armistice period, representing a proper war onset, starting in September 1943.

Figure 2: Average in-utero province war victims per 100,000

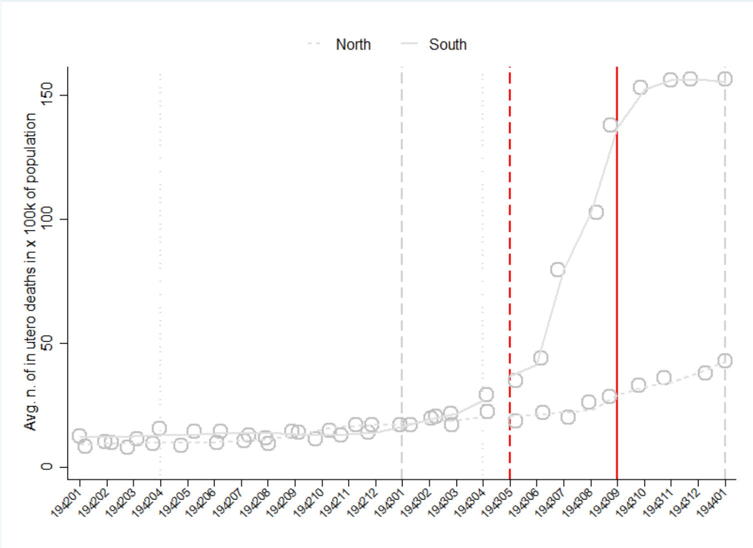
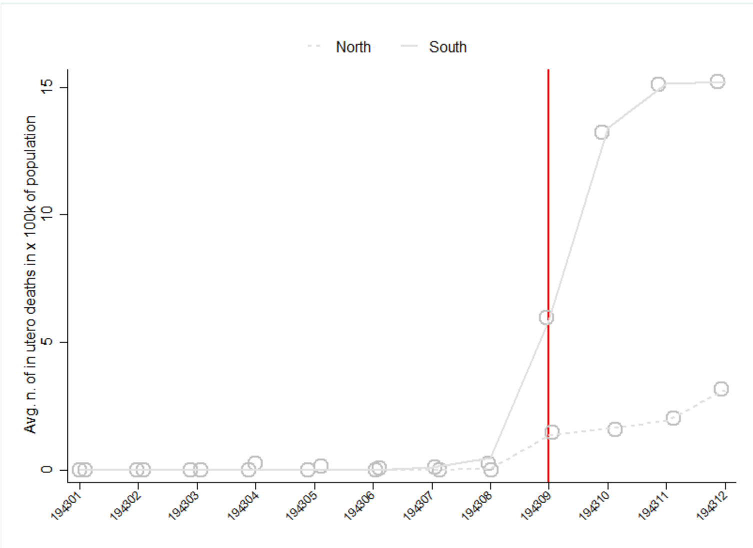


Figure 3: Average in-utero province nazi massacre victims per 100,000



The events of 1943 were very difficult to predict, and their unexpected and harsh nature had non trivial effects on civilian lives, ruling out any possibility of strategic reaction. By September 8$^{\text{th}}$1943 the Italian Army was left without instructions about the line of conduct to be taken against the German armed forces. According to Strazza (2010), very frequently the information about the arrival of military troops did not spread across neighbouring villages Strazza (2010). Moreover, according to Baldoli et al. (2011), national evacuation plans did not exist. On top of that, from a logistics point of view, moving across provinces was extremely difficult, since railroads and main transportation networks were destroyed by the Allies tactical bombing (Baldoli and Knapp, 2012).

The post September 1943 period exposed the civilians to various types of shocks, out of which we carefully track three types of adversities: *i)* general armed conflicts between the armies with an indirect impact on the civilians, *ii)* Nazi massacres aimed directly at the civilian population, and *iii)* nutritional shortages occurring in large urban areas. Concerning the general armed conflict, the post armistice period was characterized by war events, involving armed conflicts of different scale, ranging from quick victories and movements of the frontline entailing relatively limited casualties, to long stalemates associated with a sizable number of fatalities. Figure 2 reports a detailed description of the heterogeneous distribution of civilian casualties over time and space. The army movements started rapidly in the Southern Italy, and then got stuck along the Winter line, experiencing a very long stalemate, which caused huge losses among civilians.[9] Secondly, in some occasions when leaving the occupied areas, German troups organized massacres of the civilians. The massacres were less intense in terms of the number of causalities, but were characterized by pronounced violence and direct impact on the civilians. Lastly, during war time, citizens of the largest Italian cities were exposed to long periods of food insecurity and undernutrition Baldoli and Knapp (2012). The lack of food supplies was directly proportional to city sizes, where the destruction of main economic activities, logistic infrastructures and unavailability of several basic goods opened grounds to

[9] The Winter (or Gustav) Line, though ultimately broken, effectively slowed the Allied advance for months between December 1943 and June 1944. Major battles in the assault on the Winter Line at Monte Cassino and Anzio alone resulted in 98,000 Allied casualties and 60,000 Axis casualties.

black markets and impressive inflationary pressure.[10]

3 Data sources

We assembled an individual-level dataset, combining individual ECRs containing diagnoses of chronic diseases (i.e. Diabetes, Dislipidemia, Depression, Chronic diseases), obesity/BMI measures and primary health-care utilization measures, namely expenditure and number of visits, with historical data on the intensity of war faced while in utero.

3.1 Economic and health outcomes

Economic and health outcomes are derived from the Health Search/CSD Patient Database (HS), an Italian general practice registry of computer-based patient records collected by a selected group of 900 general practitioners (GPs), homogeneously distributed across all Italian regions, covering a patient population of over a 1.8 million between 2004-2010. The GPs are selected in order to guarantee the representativeness of the Italian population in terms of region, age and sex, and to meet "up-to-standard" quality criteria in terms of the levels of coding, prevalence of several diseases, mortality rates, and years of recording Fabiani et al. (2004). The HS database complies with the European Union guidelines on the use of medical data for research and has long been demonstrated to be a valid data source for scientific research.[11]

[10] After the armistice of Cassibile and with the war hereinafter, the black market grew to gigantic proportions (Po, 1974) The requisitions implemented by the Allies in the South and by the Germans in the Central-North, the evanescence of the Italian authorities and the simultaneous presence of the occupying foreign competition authorities (official or surreptitious) created areas where the black market could flourish almost without any kind of conflict. Moreover, in many cases even the Italian authorities were forced to tolerate the phenomenon, given the impossibility of otherwise replenish the population of basic necessities (Mafai, 2008; Revelli, 2014). Victims of the black market were mainly representatives of small middle class and white collar, mainly resident in urban areas, particularly affected by inflation and the inability to make bartering with foods of own production. In the South, where the Am-lira - currency of occupation allied - was subject to a strong inflation and was often rejected by smugglers, the only way to get unavailable food had become the barter of personal and family property. Entire fortunes went lost to ensure fewer meals to families La Cava (2006). City residents were used to commute to the countryside to buy, at great cost and personal risk, agricultural food products from farmers. The phenomenon was so extended and pervading to attract in 1945 the attention of the Neapolitan writer Eduardo De Filippo, who wrote his famous comedy "Naples millionaire" in which he effectively described the daily difficult lives of families living in the large cities whiles struggling with the black market economy during the war.

[11] See among others Cricelli et al. (2003), Filippi et al. (2005), Mazzaglia et al. (2009) and Atella et al. (2015).

The HS data include individual diagnoses, clinical events, prescribed tests, diagnostic results, drug prescriptions and health expenditure data.[12] The ECRs consist of daily information, while for the purposes of this study, the records have been collapsed to yearly aggregates. In the Italian healthcare system, the GP practices are chosen by individuals on the basis of geographical proximity. Each GP is allowed to have at most 1500 patients registered, with a core of individuals who are observed each year (on average 70%), and a proportion of new patients who register with a GP or old patients who are removed from the list due to a GP change or death. Thus, formally, the dataset is an unbalanced panel, with new entries and attrition. We concentrate on the 2010 wave for the main part of the study, which we complement with other wave specific ad-hoc analysis.

The precise outcome variables in this study are represented by individual clinical outcomes, excess body weight measures and health care utilization in terms of annual primary care expenditure (in euro) and number of visits. In terms of clinical outcomes, we restrict our analysis to pathologies potentially relevant to "fetal programming" hypothesis. As first proposed in Barker and Osmond (1986), the Developmental Origins of Health and Disease (DOHaD) hypothesis suggests that adverse conditions during the early life period may result in persistent changes in physiology and metabolism, which in turn alter the risk of adults to develop metabolic disorders related to pathologies such as diabetes, dislipidemia, mental health problems and, more generally, to anthropometric measures. In terms of mental health, the exposure to early life adverse conditions is frequently associated with mental disorders, depression and schizophrenia in particular.[13] Since the HS data do not contain representative information on the prevalence of schizophrenia, we concentrate on the depressive disorder.[14] Furthermore, we exploit the wide set of diagnoses related to conditions coded according to the ICD-9 (International Classification of Diseases - Version 9) classification, by including two

[12]The health expenditure data are obtained through SISSI project (Simulation of the Italian Health Care Expenditure) which evaluates individual healthcare costs by attaching prices and tariffs to HS data on healthcare utilization.

[13]Lumley et al. (2011) and Susser and Lin (1992) show an elevated risk of schizophrenia at adult ages after prenatal exposure to famine.

[14]In the Italian healthcare system schizophrenic patients are typically managed by specialist in mental healthcare units, not by GPs.

indicator variables, denoting the presence of any chronic at patient level, which delivers a synthetic measure of individual health status.[15]

Moreover, in line with the existing literature, we study individual obesity and BMI. Importantly, for the first time in this particular WWII literature, we also exploit individual health care utilization measures, such as health expenditure devoted to drugs, specialist visits and diagnostic tests, and the number of visits attended by each individual. The objective nature of the health data is a novelty in this field. In the absence of alternative data sources, the existing literature has relied on self-reported outcomes, which according to Baker et al. (2004) are subject to a large amount of systematic reporting error. The authors find that false negative reporting in the survey under study, where respondents used medical services but not reported a matched health condition, was around 50% for most chronic conditions examined, including diabetes and hypertension. The false negatives are likely to occur when patients do not to feel comfortable reporting the condition, or have memory issues. Moreover, Johnston et al. (2009) find that the probability of false negative reporting is significantly higher for individuals living in low income households. More generally, survey respondents may report their health differently depending on their expectations of their own health, their use of healthcare, and their comprehension of the actual survey questions asked Bago d'Uva et al. (2008). All the above mentioned sources of measurement error might be exacerbated in the setting of the elderly patients.

3.2 WWII treatment mechanisms

The first treatment variable is represented by the general WWII intensity in Italy and is obtained from the official publication "Morti e dispersi per cause belliche negli anni 1940-45" (The dead and the missing due to war causes between 1940-1945) ISTAT (1957), collected by

[15]International Statistical Classification of Diseases and Related Health Problems (ICD) pertains to the WHO international standard diagnostic tool for epidemiology, health management and clinical purposes, divided according to Major Diagnostic Categories (MDCs), which from the general practice point of view, are particular representative among diseases and disorders of the Nervous System, the Respiratory System, the Circulatory System, the Digestive System, the Musculoskeletal System And Connective Tissue, the Skin, Subcutaneous Tissue And Breast, and the Endocrine, Nutritional And Metabolic System.

the Italian National Institute of Statistics (ISTAT).[16] The data fit perfectly with the purposes of this study, as they refer to the number of war victims, according to province and month of death.[17] Based on the information, Figure 2 maps the intensity of WWII in Italy, as described by the in-utero toll of victims per 100,000 inhabitants in each province and month. As already described in Section 2, the data on the deaths follow closely the dynamics of the battle ground. As discussed in the historical introduction, the number of victims witnessed by the Southern residents started to swell right after the landing of Allied forces in Sicily in May 1943. From that date the pre-armistice phase of the war begun and lasted till the outbreak of the Armistice in September 1943.

The second treatment is based on the Nazi and Fascist massacres and is obtained from the "The Atlas of Nazi and Fascist massacres", a research project providing, among others, a database listing all massacres and individual murders of civilians and resistance fighters killed in Italy after September 8^{th} 1943 by both German soldiers and soldiers of the Italian Social Republic outside of the armed fights. The database ranges from the first murders in the South to the northern withdrawal massacres, all representing violent crimes perpetrated against civilians during the German occupation of Italy.[18] As before, the treatment is formulated by month/province of death of the victims, and is depicted in Figure 3.

Finally, we use the information on the famine episodes occurring in large Italian cities, due to the shortages of food supplies in urban areas. We thus define an indicator variable of individuals born in large cities.

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[16]The publication is available and accessible either at the ISTAT archives, or online at `http://lipari.istat.it/digibib/causedimorte/IST3413mortiedispersipercausebellicheanni1940_45+OCRottimizz.pdf`.

[17]The analysis is framed in the todays administrative province division of the country, while ISTAT data report the intensity of war according to the historical administrative provinces. In the recent decades some provinces changed their structure by incorporating/separating some of their municipalities. We thus account for these changes and adopt a re-weighting procedure which follows the portions of population which change their province reference.

[18]The information about the project, the database and access possibilities are available at `http://www.straginaziFasciste.it/?page_id=9&lang=en`.

3.3 Additional controls

In order to control for living conditions in the Italian provinces we use current data on average gross income from the Italian Ministry of Economics. We also account for the health conditions across Italian regions during war times by collecting ISTAT data for two cause specific mortality rates (mortality rates for pregnancy, delivery and puerperium, and mortality rate for early childhood diseases). Finally, based on the data on the toll of war victims, for each combination of province and year/month of birth, we construct an indicator of early childhood exposure to war intensity in the first year of life from birth month in each province.

3.4 Data selection

In order to build our identification strategy based on the historical events presented in Section 2, we extract information from the 2010 wave of HS data on all individuals born in the Southern (Campania, Apulia, Basilicata, Calabria, Sicily) and Northern (Piedmont, Lombardy, Trentino, Veneto, Friuli and Liguria) regions of Italy between January 1943 to December 1943. Conceptually, the 12-month period is subdivided into 3 segments, of which the first 4 months refer to the period free of any war occurence, the following 4 months describe the pre-armistice period, and the last 4 months refer to the proper war outbreak in the post armistice. This data selection process leave us with a total of 7,721 individuals, of which 3,130 refer to the South and 4,591 to the North (see Table 1). We then define as "treated" group all individuals born in the South and, conversely, as "control" group those born in the North. Limiting our analysis to this particular 12-month window enables us to capture the sole conflict in the Southern territories, the first ones to be exposed at war events, given that until January 1944 the battle front was stuck below the Winter line and the only territories directly affected by ground battles were those in the Southern territories. As a consequence, Northern residents represent individuals born in the parallel time frame, but not directly exposed to war events in their prenatal era. We intentionally exclude from the analysis the Central area of the country, which was exposed to some dose of the treatment throughout the whole WWII period in Italy due to the political and strategical importance of Rome. Most importantly, the 12-month window, with the proper treatment initiating in the last 4 months of the period, enable us to isolate the possible measurement error resulting form the endogenous fertility choices. Indeed the conception date of all individuals considered is framed in the period between April 1942 and March 1943, a window which falls before the outbreak of the Armistice, but also before the pre-armistice preparatory phase (see Figure 2). By restricting the sample to these birth-months, we ensure that the individuals considered in the analysis are all conceived in a period when parents of both the treated and the controls disposed of the exact same priors in terms of the evolution of the pregnancy.

We determine the intrauterine period for each individual by counting 9 months backwards from their month/year of birth date.[19]. In case of shorter pregnancies, the actual exposure to treatment is lower, and the resulting estimate of treatment likely to be biased, since the overestimation of exposure period is likely to be correlated with higher probability of negative health outcomes typical of the preterm babies. On the one hand, the maximum exposure period allowed in our setting amounts to 4 months prior to birth of the proper treatment and 4 months of pre-treatment phase, which is undoubtedly inferior to any duration of pregnancy concluded in a live birth. In general, preterm pregnancies in our historical setting are likely to represent a narrower issue, since premature live births were rare during the WWII times. According to Kopp and Krakow (1983) early follow up studies done between 1920 and 1949 show that child weight at birth is the main parameter to consider for survivorship. In fact, until the WWII neonatology was not able to save pre-term babies weighting less than 1.8 kg, which was usually achieved not before the 31st week of pregnancy. Moreover, for each individual we trace his/her province of birth.[20] Within the treatment data, we compute two province and year/month specific gestational exposures to treatment, one for the general war stress and the other for Nazi massacres, by summing up the relative number of victims within each province in 9 months prior to each year/month of birth. Subsequently, we transform the continuous measures into dummy variables, indicative of intensity thresholds. In case of

[19]It is well know that the hypothesis of 9-month-pregnancies induces measurement errors in case of premature births, as emphasized by Currie and Rossin-Slater (2013).

[20]Italy is currently organised on 110 provinces, which represent the second-level administrative sub-division of 20 regions.

general war, since the Southern provinces all underwent a non-zero amount of victims, we define the average number of victims as the cut-off above which individuals are deemed to have underwent intense war conditions. In terms of the Nazi massacres, the average number of victims was much narrower, and the violences had a quick and spot character. For this purpose, we construct a binary variable indicating the sole presence of massacres witnessed by the mothers during the gestation period. Finally, the third mechanism is represented by a dummy variable for individuals born in large cities ($\geqslant$ 900,000 inhabitants), proxying for intense food shortages.

3.5 Descriptive statistics

Table 1 presents descriptive statistics of the variables used in the analysis, separately for the North and the South. As we consider the 2010 wave, all individuals are same age, amounting to 67 in that year. The two samples are fairly identical in terms the proportion of females (53%). In terms of other characteristics included in the analysis, there are several differences between the two populations. The prevalence of at least one chronic conditions is very similar (95.6% vs. 92.7%), the same holds true for dislipidemia (31.6% vs. 33.1%). The prevalence of depression of the Northern residents matches the exact same share among the Southern ones (7.1%). With respect to the North, the South features slightly higher prevalence of obesity (20.9% vs. 16.4%) and of diabetic patients (23.4% vs. 18.0%), and the differential is in line with the official ISTAT data.[21] Overall, the South features higher average health expenditure (796.4 euro vs. 691.5 euro) and more contacts with GPs (19.1 vs. 14.5). Moreover the Southern GPs are more likely to display a higher price per prescription (18.2 euro vs. 13.7 euro), but similar GP-specific proportion of prevention visits (a mere average of 0.3%). Finally, the southern provinces register lower average annual income (21,904 euro vs. 23,615 euro) and a slightly higher probability of death in the post war era (1950 - 1955) (11.86% vs. 10.85%).

[21] The official ISTAT statistics on obesity prevalence point to a lower obesity rate among the Italian adults/elderly. However, it is worth mentioning that ISTAT data are underestimated due to their self-reported nature. On the contrary, HS data on obesity are in line with Progetto Cuore (`http://www.cuore.iss.it`) by the Italian National Institute of Health (ISS), a population study with objectively collected height and weight data.

Table 1: Summary statistics

Variable	Mean	Std. Dev.	Min.	Max.	N
South					
Female	0.532	0.499	0	1	3130
Age	67	0	67	67	3130
Any chronic disease	0.956	0.206	0	1	3130
Total expenditure	796.368	886.12	0	9812.08	3130
N. of contacts with GP	19.12	13.349	0	120	3130
Depression	0.071	0.257	0	1	3130
Dyslipidemia	0.316	0.465	0	1	3130
Diabetes	0.234	0.424	0	1	3130
Obesity	0.209	0.407	0	1	3130
BMI	28.351	4.966	15.6	58.333	2081
General war intensity (n x 100k)	71.296	72.237	3.659	422.521	3130
General war intensity (0-1)	0.404	0.491	0	1	3130
Nazi massacres intensity (n x 100k)	2.638	9.909	0	81.098	3130
Nazi massacres intensity (0-1)	0.171	0.377	0	1	3130
Province avg.income (1,000 euro)	21904.336	3519.567	14341.819	27576.4	3130
GPs price per prescriprion	18.213	30.309	11.275	355.335	3130
GPs preventive visits	0.004	0.009	0	0.082	3130
Probability of death (%)	11.865	4.632	5.893	19.649	3130
Deaths in I year if life (n. x 100,000)	82.693	66.148	0	330.519	3130
Mortality in pregnancy, delivery and puerperium	4.222	0.656	3.854	6.025	3130
Mortality of early childhood diseases	88.97	15.593	70.136	109.05	3130
North					
Female	0.528	0.499	0	1	4591
Age	67	0	67	67	4591
Any chronic disease	0.927	0.261	0	1	4591
Total expenditure	691.525	773.301	0	8413.83	4591
N. of contacts with GP	14.469	11.131	0	82	4591
Depression	0.071	0.257	0	1	4591
Dyslipidemia	0.331	0.471	0	1	4591
Diabetes	0.18	0.384	0	1	4591
Obesity	0.164	0.371	0	1	4591
BMI	27.221	4.716	15.157	51.455	3181
General war intensity (n x 100k)	25.53	25.842	3.116	146.063	4591
General war intensity (0-1)	0.09	0.286	0	1	4591
Nazi massacres intensity (n x 100k)	0.367	1.538	0	20.23	4591
Nazi massacres intensity (0-1)	0.09	0.286	0	1	4591
Province avg.income (1,000 euro)	23615.573	2919.491	11012.237	35750.73	4591
GPs price per prescriprion	13.74	3.724	9.987	62.706	4591
GPs preventive visits	0.003	0.008	0	0.071	4591
Probability of death (%)	10.854	3.929	5.621	20.112	4591
Deaths in I year if life (n. x 100,000)	82.998	62.96	0	314.458	4591
Mortality in pregnancy, delivery and puerperium	2.848	0.685	1.978	5.033	4591
Mortality of early childhood diseases	47.842	10.886	26.89	74.460	4591

When it comes to mortality due to pregnancy, delivery and puerperium and due to early childhood diseases at the moment of birth (1943), the statistics are almost twice higher in the South with respect to the North. Also the average number of general war deaths experienced and the Nazi massacres victims in the intrauterine period is higher in the South than in the North (72 vs. 25, and 3 vs. 0 respectively), but almost identical in the first year of life (88). The differences are apparently due to the evolution of the war events, which, as explained in the historical background, had their origin in the South, coinciding with the in-utero phase of the Southern patients.

4 Identification Strategy

4.1 Econometric specification

Our empirical identification strategy exploits the variation in type, space and time of the adversities of WWII over several months and across Italian provinces. We employ a difference-in-differences strategy, taking advantage of the unexpected war outbreak occurred after the Italian armistice on September 8^{th} 1943, which represents an unambiguous discontinuity in the evolution of the prenatal era circumstances that breaks the distribution of WWII intensity over time (months) and across space (provinces).

The quintessential caveat in the estimation of exposure to early life shocks on long term outcomes is selective fertility. As far as we know, no other paper on the effect of WWII provides a reliable solution to this issue. Individuals are likely to adjust their fertility choices to the underlying uncertainty about the future. As a consequence, war intensity, the treatment variable implemented in this study, may on its own determine the choice about fertility, distorting the treatment *vs.* control group through selection issues. In such a setting, regressing long-term health outcomes on early-life war exposure may deliver results correlated with the error term causing inconsistency in the estimated parameter. The identification strategy adopted in this study provides a razor-sharp solution to this issue, derived from subsampling individuals born in a 12-month window, between January 1943 and December 1943, all conceived before the materialization of any sign of upcoming war on the national territories, between April 1942 and March 1943. This sample selection allows us to make a reasonable claim that *foetuses* in the two subgroups were conceived in a fairly similar environment as far as the lack of war is concerned, hence the fertility choice concerning their conception was made according to similar uncertainty priors (see Figure 2).[22]

[22] This is a clear improvement with respect to Havari and Peracchi (2017) and Akbulut-Yuksel (2017), who

Also, by excluding from the analysis the central part of the country, we wash out issues related to misclassification into treatment, potentially resulting form the historical events, which saw the center of Italy involved in a limited extent of conflicts throughout all the duration of WWII. Finally, the sudden and unexpected changes in war events and the nature of war described in the historical section rule out, to a reasonable extent, the issue of endogenous maternal mobility, hence also the possible issue of misclassification in treatment.

Given the historical setting presented in Section 2, and after controlling for several confounding factors, individuals born in the Southern provinces from September 1943 onwards experienced a sudden increase in prenatal insults due to war adversities with respect to their control counterparts. The raise in war victims affected the evolution of the pregnancies, hence the resulting long-term health differential between individuals born in the South *vs.* those born in the North can be interpreted as the direct effect of stress suffered in utero. Additionally, but to a much narrower extent, from May 1943 onwards, individuals born in the South were exposed at a preparatory phase involving a sizeable extent of destablization. For this purpose, and as already described in the data section, we define three distinct groups of birth months: 4 months before any war occurrence (1943/01-, 1943/04), 4 months of the pre-armistice period (1943/05 - 1943/08) and 4 months after the armistice proclamation and the withdrawal of the Allied forces from the Southern territories (1943/09 - 1943/12).

The baseline model specification is run on data from the HS 2010 wave, when individuals are 67 years old. Additionally, we investigate to what extent the war impact can change over time with the ageing process of the individuals affected by the treatment. This represents an important innovation, since the past literature has limited to a static differential between "treated" and "control" groups, while in this setting we explore how this differential evolves in time. In order to gauge this aspect, we additionally run our model specification for waves w=2004-2009, overall obtaining 7 age specific subsamples, relative to individuals aged 61 to 67. As prevalence of chronic conditions varies with age, health differentials resulting from prenatal exposure to adverse conditions may follow heterogeneous patterns compared to "control"

cannot control for the month of birth.

group. By looking at the age specific pools, we end up with samples homogeneous in terms of age, thus reducing also part of the potential unobserved heterogeneity in terms individual health.

We thus build the following baseline reduced form, evaluating whether the war adversities resulting in maternal stress, can be shown to have affected the long-term health outcomes of the children:

$$\begin{aligned} H^w_{ipyg} = &\beta^w_1 S_p + \beta^w_2 PreArm_y + \beta^w_3 PostArm_y + \beta^w_4 S_p * PreArm_y + \beta^w_5 S_p * PostArm_y + \\ &\psi^w X_{py} + \zeta^w GP_g + \alpha^w_p + \gamma^w_y + \epsilon^w_{ipyg} \end{aligned} \tag{1}$$

where i denotes individuals, p the province, y the month-year, w the wave, g the GP, and where S_p is the South dummy indicator, equal to 1 for individuals born in Southern provinces, $PreArm_y$ represents a dummy variable equal to 1 for individuals born in the pre-armistice period, $PostArm_y$ represents a dummy variable equal to 1 for individuals born after the armistice, X_{py} is a vector of controls consisting of the province income observed at the moment of outcome realization, the number of deaths due to WWII in the first 12 months of each individual's life (expressed by 100,000) at province level, probability of death in the post war era (1950 - 1955) at the regional level, mortality due to pregnancy, delivery and puerperium and due to early childhood diseases at the moment of birth (1943) at the regional level, and GP_g controls for relevant GP characteristics, such as average drug price per prescription and average number of prevention visits. Finally, we include province fixed effects α_p to wash out the systematic time invariant differences between provinces and birth month/year effects γ_y in order to account for time varying characteristics common to individuals born in all provinces and for the effects of seasonality in birth outcomes. As customary, ϵ_{ipyg} is a province clustered standard error term.

In Equation 1 our goal is to estimate β^w_4 and β^w_5, which under the hypothesis of parallel trend in outcome, is the difference-in-differences coefficient. Equation 1 is estimated using Ordinary Least Squares (OLS) for each wave separately.

We conduce a parallel pre-trend test using the same specification, but falsifying $PreArm_y$ and $PostArm_y$, artificially imposing it two months before the actual armistice took place. The customary placebo test for our difference-in-differences estimates should show not significant results when treatment status is assigned artificially before the occurrence of the exogenous shock in war adversities. Thus we claim that if there is a significant difference in health outcomes in the two subgroups, this should be due to the additional effects of war after the armistice or by time invariant different characteristics between treatment and control groups, which, however, are ruled out by controlling for a wide set of fixed effects.

In order to understand the mechanisms through which the war shocks are at work in our setting, we exploit the data on various pathways of war shock transmission. Although the war outbreak occurred after the armistice, not all individuals witnessed the war events in the prenatal era to the same extent. We thus provide an *ad hoc* disentangling of the war outbreak, and by additionally interacting the Southern pre-armistice phase and armistice war outbreak ($S_p * PreArm_y$ and $S_p * PostArm_y$) with the high intensity war indicator dummy variable (HI_{py}). In analytic terms, equation 1 can be rewritten as a difference-in-difference-in-differences specification of the following form:

$$\begin{aligned}H^w_{ipyg} =&\beta^w_1 S_p + \beta^w_2 PreArm_y + \beta^w_3 PostArm_y + \beta^w_4 S_p * PreArm_y + \beta^w_5 S_p * PostArm_y +\\ &\beta^w_6 HI_{py} + \beta^w_7 S_p * HI_{py} + \beta^w_8 PreArm_y * HI_{py} + \beta^w_9 PostArm_y * HI_{py} +\\ &\beta^w_{10} S_p * PreArm_y * HI_{py} + \beta^w_{11} S_p * PostArm_y * HI_{py} +\\ &\psi^w X_{py} + \zeta^w GP_g + \alpha^w_p + \gamma^w_y + \epsilon^w_{ipyg}\end{aligned} \tag{2}$$

where the new parameters of interest are now β^w_{10} and β^w_{11}. In order to account for the possible differential effect of the impact of war on the onset of metabolic diseases for individuals born in large cities, hence exposed not only to high intensity of war, but also to food shortages, we augment Equation 2 with an additional interaction term with large city dummy variable.

Finally, in order to analyze the channel of Nazi massacres, we rewrite Equation 2, substituting general war intensity dummy variable HI_{py} with nazi massacres indicator variable

(NM_{py}) denoting the presence of any massacre underwent during the gestational period, with the Equation 2 taking the following form:

$$\begin{aligned} H^w_{ipyg} =& \beta^w_1 S_p + \beta^w_2 PreArm_y + \beta^w_3 PostArm_y + \beta^w_4 S_p * PreArm_y + \beta^w_5 S_p * PostArm_y + \\ & \beta^w_6 NM_{py} + \beta^w_7 S_p * NM_{py} + \beta^w_8 PostArm_y * NM_{py} + \beta^w_9 S_p * PostArm_y * NM_{py} + \\ & \psi^w X_{py} + \zeta^w GP_g + \alpha^w_p + \gamma^w_y + \epsilon^w_{ipyg} \end{aligned} \quad (3)$$

In this case, due to the non existence of any episode of Nazi violence before the armistice proclamation, the difference-indiference-in-differences term is only estimated for the armistice period (β^w_9).

4.2 Identification issues and solutions

As largely discussed in the previous pages, WWII does not provide randomization in strict causal inference terms, but delivers a setting that can be considered satisfactory for the causal interpretation of the conflict intensity parameter. The war events exploited in this study evolved in an unpredictable way, following the initial landing of Allied forces in the Southern territories and the signing of armistice, which introduced a sudden outbreak of harsh conflicts. The historical events - including the king fleeing Rome a day after the armistice proclamation - delivers an important representation of the information asymmetry about the war events that civilians were exposed to. It is thus reasonable to consider local adverse conditions as exogenously assigned to the civilians, after controlling for time invariant heterogeneity at province level, and the inclusion of time-characteristics based on month/year fixed effects. This set of controls, along with wave fixed effects, GP characteristics, at birth and early-childhood mortality rates, as well as province income observed at the moment of outcome realization, ensure that, net of other possible unobserved time varying factors, the remaining information explained by the difference-in-differences parameter estimates identify the exogenous causal impact of war stress on adult outcomes.

Even if strict exogeneity between $PreArm_{py}$, $PostArm_{py}$ and H_{ipywg} doesn't seem to be an issue in our data, in order to establish a causal relationship we carefully account for other possible unobserved factors potentially correlated with the error term ϵ_{ipyg} in our setting. Despite detailed health data and refined treatment variables, there are distinct unobserved time varying issues related to the treatment assignment, selection bias or measurement error. In particular, as also anticipated in the introduction, our causal claims hinge on the capacity of our empirical analysis to rule out, or control for, the effect of selection of survivors in the treated sample, potential endogenous movement of pregnant mothers and endogenous fertility choices induced by the the war itself. Furthermore, we need to consider potential measurement error in the exposure to treatment caused by the approximation of pregnancy duration and/or the omitted variable bias related to the potential impact of early childhood circumstances that can be closely related with war events occurred in pregnancy.

First of all, war events are likely to increment the likelihood of infertility, miscarriages, stillbirths or simply higher overall mortality, which may distort the characteristics of the individuals in the treated sample (those exposed to war stress). Moreover, by analyzing the long-term impacts of war on the elderly, we are likely to be exposed at an even greater amount of survival distortion. Notwithstanding, the direction of this bias leads to an underestimation of health differential, due to the selection process occurring both during early and adult life, where the overall sample, and the treated individuals in particular, are expected to be on average healthier.

Secondly, pregnant mothers may respond to the shock by moving. On the one hand, as discussed in Section 2, movements across provinces during WWII in Italy were very limited due to the military strategies and to lack of consistent evacuation plans Baldoli and Knapp (2012). Moreover, if any, the bias is again expected to underestimate the causal impact, given that the true control group (individuals whose mothers resided in low war adversity provinces both during pregnancy and at birth) is likely to be contaminated with treated individuals (individuals whose mothers resided in high war adversity provinces and at some point of the pregnancy moved to safer provinces). In fact, assigning individuals erroneously to a treatment according to the birthplace represents a typical case of misclassification in treatment, which leads to an underestimation (attenuation bias) of the treatment effect Lewbel (2007). In our setting this bias is likely to increase with the length of the exposure to treatment, which we approximate on the basis of the information about province and month of birth (a similar strategy is adopted in Quintana-Domeque and C. Rodenas-Serrano (2014).[23] Consequently, any statistically significant estimate of health differential found for the treated sample can be considered a lower bound of the true average treatment effect.

Third, mothers may respond to the shock by deciding not to get pregnant or having difficulties getting pregnant. It is very difficult to predict the direction of this bias. On the one hand, it is likely that mothers who decide to get pregnant in war times feature on average lower socio-economic status, which is also likely to determine worse childhood conditions, hence poorer health in the long-term and an overestimation of the causal effect. On the other hand, it might be that only families with better socio-economic status decide to have children, being able to buffer the adverse effect of war. It is important to highlight that in our setting socio-economic characteristics, for which we cannot control, affect only the choice of having a baby and not the chance to get treated, which remains exogenous. In such case fertility choices based on the socioeconomic status would entail better long-term health outcomes among the treated, and an underestimation of the causal effect. Our identification strategy deals with this issue by comparing individuals who are subjected to the same conditions at the moment of conception, hence their parents are exposed at the same prior about the development of war conflicts and the armistice consequences. Thus, under the very mild assumption that socio-economic status is not related to the armistice *per se*, our identification strategy is robust to this issue.

Finally, since war may be responsible for the deterioration of living conditions straight after the actual events take place, it is important to disentangle the gestation exposure period form the very early childhood circumstances. In order to address this issue, we include at-birth and infant mortality relative to the birth year/region and for the residual province war intensity that individuals went through in the first 12 months after birth.

[23] We measure exposure by province of birth, rather than mother's province of residence, although the latter could be a better geographic unit of analysis. However, in our historical setting we believe that residence won't be a very reliable source of information on geographical location (see Simeonova (2011).

5 Results

In this section we present the results obtained from the specification and identification strategies discussed before.[24] The outcomes examined are the occurrence any chronic disease, diabetes, dislipidemia, depression, obesity, and measurement of BMI, health expenditure (in euros) and number of visits. All difference-in-differences (DiD) estimates have been obtained accounting for the relevant DiD controls, province of birth fixed effects, birth month x year fixed effects, GP fixed characteristics (average prescription prices and preventive visits), province average income, as well as indicators of gestational, neonatal and infant mortality as explained in the previous sections.

Table 2 show the results of the DiD parameters β_4 and β_5 from the baseline model in Equation 1. In particular, the top panel reports the DiD estimates under the actual timing of the armistice, while the bottom panel shows the same estimate obtained using a placebo specification. We do not find any statistical significant results, with the sole exception of the health expenditure coefficient which is slightly statistically significant, suggesting that individuals exposed to WWII outbreak in the prenatal era are likely to spend 141 euro more on annual basis. Moreover, despite the coefficient estimates on any chronic disease and on the number of visits are positive, they are not statistically significant. The lower panel in Table 2 shows that the placebo estimates are always not statistically significant. This result confirms the causal claims about the estimates of β_5, given that before September 1943 the Southern *vs.* Northern differential outcomes can be explained only by time invariant characteristics, which we rule out through our set of controls and time invariant characteristics (i.e. fixed effects) in Equation 1.

What emerges form these results is that they are at odds with the previous literature findings, suggesting that once we control for measurement error, selective fertility and specify a detailed identification strategy the sole fact of being born in a province and month interested by WWII treatment does not provide health differentials later in life. This does not necessarily imply that WWII stress did not impact long term outcomes, conversely, it suggests that the

[24]Given the richness of our results, for the sake of brevity, we present only results relevant for our discussion.

treatment is not linear and that the effect becomes sizeable only above a certain threshold of war impact.

Table 2: Estimates for baseline.

	Any chronic	*No.visits*	*Expenditure*	*Depression*	*Dyslipidemia*	*Diabetes*	*Obesity*	*BMI*
Pre-armistice	0.017	0.609	65.819	-0.002	-0.005	0.006	0.001	-0.216
	(1.54)	(0.64)	(1.05)	(-0.12)	(-0.18)	(0.29)	(0.06)	(-0.64)
Armistice	0.026	1.420	141.151*	0.019	-0.013	-0.001	0.007	-0.102
	(1.34)	(1.15)	(2.06)	(0.77)	(-0.32)	(-0.02)	(0.23)	(-0.20)
r2	0.026	0.062	0.028	0.027	0.033	0.023	0.026	0.039
N	7721	7721	7721	7721	7721	7721	7721	5262
Placebo	0.023	-0.212	-17.273	0.007	-0.008	-0.009	-0.007	-0.383
Pre-armistice	(1.53)	(-0.25)	(-0.28)	(0.58)	(-0.28)	(-0.39)	(-0.39)	(-1.38)
Placebo Armistice	0.011	0.108	33.294	0.019	-0.013	-0.031	0.004	-0.453
	(0.56)	(0.10)	(0.46)	(1.03)	(-0.35)	(-0.72)	(0.16)	(-0.95)
r2	0.026	0.062	0.027	0.027	0.033	0.023	0.026	0.039
N	7721	7721	7721	7721	7721	7721	7721	5262
province fe	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
birth year/month fe	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
gp characteristics	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
neonatal and infant mortality	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*

Province clustered std. errors

To further investigate this possibility we estimate the model described in Equation 2, which includes the interaction with the HI_{py} indicator. Table 3 shows the parameter estimates for the coefficients β_{10} and β_{11} in Equation 2. Again, the bottom part of Table 3 reports placebo tests for this new specification and shows that our DiD specification still holds. Results in the upper part of the table are now in line with the expectations, andto some extent with the literature. The coefficient estimates point to a detrimental and statistically significant effect of war exposure on adult health. The probability of developing any chronic disease increases by 10%, and the coefficient is strongly significant. The coefficient for dislipidemia is around +0.3, implying that for the treated individuals the probability of having high lipid levels almost doubles, considering that the mean value for dislipidemia is around 0.3 (see Table 1). Furthermore, the treated patients tend to have almost 4 more contacts with their GP on annual basis, amounting to a 25% increase with respect to the average of 19 visits for the South individuals. Moreover, their per capita health expenditure is 260 euro higher, a 30% differential relatively to the respective baseline average of 790 euro. Interestingly, metabolic related disorders as described by BMI, obesity and diabetes are not statistically significant in this specification, while depression is significant only at 10% level.

Table 3: Estimates for baseline with high war intensity.

	Any chronic	*No.visits*	*Expenditure*	*Depression*	*Dyslipidemia*	*Diabetes*	*Obesity*	*BMI*
Pre-armistice H	0.025	0.325	-5.761	0.007	0.236***	-0.033	0.029	1.606
intensity	(0.87)	(0.28)	(-0.08)	(0.30)	(4.89)	(-0.81)	(1.01)	(1.71)
Armistice H	0.100***	3.618*	260.277***	0.063*	0.303***	0.044	0.091	1.524
intensity	(3.50)	(2.63)	(3.58)	(2.07)	(5.39)	(0.92)	(1.65)	(1.47)
r2	0.026	0.064	0.029	0.027	0.035	0.024	0.026	0.040
N	7721	7721	7721	7721	7721	7721	7721	5262
Placebo	-0.033	-2.352	136.620	0.000	-0.076	-0.163	-0.023	-0.609
Pre-armistice H intensity	(-0.80)	(-1.25)	(1.33)	(0.00)	(-0.78)	(-1.30)	(-0.54)	(-0.98)
Placebo Armistice H	0.042	-1.051	175.398	0.037	-0.015	-0.045	0.023	0.300
intensity	(0.84)	(-0.61)	(1.72)	(0.84)	(-0.20)	(-0.88)	(0.66)	(0.50)
r2	0.026	0.063	0.028	0.027	0.034	0.024	0.026	0.040
N	7721	7721	7721	7721	7721	7721	7721	5262
province fe	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
birth year/month fe	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
gp characteristics	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
neonatal and infant mortality	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*

Province clustered std. errors

Moreover, once we augment the model with the large city dummy interaction (Table 4) the coefficient on diabetes is significant and positive, indicating an increase of +0.2. We do not find statistically significant results for BMI and obesity, which may appear puzzling, given the existence of numerous literature findings showing that famines experienced while in utero are responsible for higher prevalence of both obesity and diabetes later in life Lumley et al. (2011). We are inclined to believe that the divergence of our results is caused by possible measurement errors of BMI in GP records. In fact, GPs produce very accurate data in terms of diagnosis which give origins to prescriptions, visits and any other expression of demand for health services, which in turn generate costs (and they are accountable for costs). Obesity *per se* does not generate costs, it constitutes a risk factor, which boosts probability of having certain conditions. As such, it is subject to reporting issues, where GPs may neglect proper monitoring of these aspects. We thus report the results for the sake of completeness and comparability with other studies, but acknowledge the potential limitations of these measures.

Table 4: Estimates for baseline with high war intensity and large cities.

	Dyslipidemia	*Diabetes*	*Obesity*	*BMI*
Pre-armistice H	0.228***	-0.034	0.013	1.452
intensity	(4.94)	(-0.87)	(0.42)	(1.41)
Pre-armistice H	0.066	0.023	0.054	0.105
intensity in large cities	(1.36)	(0.92)	(1.93)	(0.21)
Armistice H	0.302***	0.037	0.098	1.744
intensity	(5.13)	(0.81)	(1.76)	(1.75)
Armistice H	0.132	0.211***	-0.067	0.851
intensity in large cities	(1.74)	(7.34)	(-1.66)	(0.58)
r2	0.036	0.025	0.026	0.042
N	7721	7721	7721	5262
province fe	*yes*	*yes*	*yes*	*yes*
birth year/month fe	*yes*	*yes*	*yes*	*yes*
gp characteristics	*yes*	*yes*	*yes*	*yes*
neonatal and infant mortality	*yes*	*yes*	*yes*	*yes*

Province clustered std. errors

The next part of the empirical analysis focuses on the estimates of Equation 3, through which we disentangle the mechanisms of WWII stress due to Nazi violence. Table 5 reports the estimates. We find that being exposed only to Nazi massacres during the gestational period is likely to increase the probability of being diagnosed with depression, with a strong effect (with respect to the average prevalence reported in Table 1 we observe a doubling of the prevalence). It is interesting to note that while in Table 3 the coefficient on depression was only slightly statistically significant, once we account for violence-specific exposure, the statistical significance improves. Validation of this identification strategy comes from clinical evidence obtained by Radtke et al. (2011), who find that prenatal exposure to extreme violence, similar to the Nazi massacres, can often contribute to developing depression later in life (mainly after age 60). From an economic perspective, Persson and Rossin-Slater (2016), focusing on a sample of cohorts in their thirties, suggest that the uterine environment propagates the impact of stress to the unborn child, in particular in family suffering from disruptions during gestation, thus increasing the probability of mental health issues in the adulthood of the offspring (ADHD and depression).

Table 5: Estimates for baseline with Nazi massacres.

	Any chronic	*No.visits*	*Expenditure*	*Depression*	*Dyslipidemia*	*Diabetes*	*Obesity*	*BMI*
Pre-armistice	0.017	0.591	65.207	-0.001	-0.004	0.006	0.003	-0.208
	(1.52)	(0.62)	(1.05)	(-0.05)	(-0.16)	(0.27)	(0.14)	(-0.62)
Armistice	0.024	1.469	102.933	-0.010	0.019	-0.018	0.010	0.132
	(1.05)	(1.40)	(1.45)	(-0.35)	(0.51)	(-0.54)	(0.31)	(0.26)
Nazi massacres	-0.007	-0.409	58.774	0.071**	-0.045	0.026	0.025	-0.161
	(-0.32)	(-0.34)	(0.85)	(2.96)	(-0.94)	(0.69)	(0.81)	(-0.31)
r2	0.026	0.062	0.028	0.028	0.034	0.023	0.026	0.040
N	7721	7721	7721	7721	7721	7721	7721	5262
province fe	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
birth year/month fe	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
gp characteristics	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
neonatal and infant mortality	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*

Province clustered std. errors

Table 6: Estimates for baseline with high war intensity and Nazi massacres.

	Any chronic	*No.visits*	*Expenditure*	*Depression*	*Dyslipidemia*	*Diabetes*	*Obesity*	*BMI*
Pre-armistice H	0.021	0.267	-1.307	0.018	0.241***	-0.032	0.040	1.654
intensity	(0.68)	(0.23)	(-0.02)	(0.78)	(5.32)	(-0.81)	(1.44)	(1.71)
Armistice H	0.096**	3.548*	253.759**	0.065	0.312***	0.041	0.101	1.573
intensity	(3.14)	(2.46)	(3.14)	(1.93)	(6.01)	(0.89)	(1.95)	(1.63)
Nazi H intensity	-0.005	0.039	88.288	0.075**	-0.028	0.032	0.021	-0.077
	(-0.20)	(0.03)	(1.08)	(3.06)	(-0.61)	(0.91)	(0.62)	(-0.13)
r2	0.026	0.064	0.029	0.028	0.036	0.024	0.026	0.040
N	7721	7721	7721	7721	7721	7721	7721	5262
province fe	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
birth year/month fe	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
gp characteristics	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
neonatal and infant mortality	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*

Province clustered std. errors

Based on the results presented in Tables 4 and 5, we further enrich our specification where the DiD specification in equation 2 is augmented with the Nazi massacres channel as expressed in equation 3. The results are described in Table 6. We find, that estimating jointly the effect of general war intensity and Nazi massacres confirms the results presented separately in Tables 4 and 5. The health care utilization measure (number of contacts and health expenditure) is only affected by the high intensity WWII exposure, the same holds true for dislipidemia and for any chronic disease prevalence. On the other hand, diagnosis of depression differential is confirmed to be driven by those patients whose pregnant mothers experienced the violence of nazi massacres. Finally, when we also add the large city dummy, the results are in line with the previous model specification estimates. Again, the health expenditure differentials is found as a result of exposure to high war intensity in the post armistice era, and the same holds true for dislipidemia. Moreover, diabetes is significant only in the case of pregnant mothers exposed at high war intensities in large cities, reflecting the food shortages effect on the evolution of diabetes in the offspring. Also in case of depression, we reconfirm the Nazi massacres channel as the probable violence-driven stress propagation to the foetus.

Table 7: Estimates for baseline with high war intensity, large cities and nazi massacres.

	Any chronic	*No.visits*	*Expenditure*	*Depression*	*Dyslipidemia*	*Diabetes*	*Obesity*	*BMI*
Pre-armistice H	0.008	-0.383	-29.290	0.023	0.236***	-0.038	0.028	1.563
intensity	(0.23)	(-0.27)	(-0.35)	(0.86)	(5.09)	(-0.99)	(0.89)	(1.51)
Pre-armistice H	0.073**	3.929**	164.622	-0.019	0.048	0.031	0.047	-0.021
intensity in large cities	(2.98)	(3.25)	(1.96)	(-0.91)	(0.93)	(1.28)	(1.55)	(-0.04)
Armistice H	0.096**	3.116*	248.918**	0.055	0.303***	0.036	0.108*	1.755
intensity	(3.04)	(2.06)	(2.85)	(1.58)	(5.32)	(0.80)	(2.10)	(1.90)
Armistice H	0.059*	5.407	452.188	-0.008	0.150	0.206***	-0.087	0.845
intensity in large cities	(2.01)	(1.90)	(1.81)	(-0.16)	(1.71)	(5.62)	(-1.99)	(0.54)
Nazi massacres	-0.000	-0.578	62.459	0.059*	-0.036	0.010	0.036	-0.001
	(-0.01)	(-0.40)	(0.68)	(2.04)	(-0.63)	(0.28)	(0.89)	(-0.00)
r2	0.027	0.066	0.031	0.029	0.037	0.025	0.027	0.042
N	7721	7721	7721	7721	7721	7721	7721	5262
province fe	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
birth year/month fe	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
gp characteristics	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*
neonatal and infant mortality	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*	*yes*

Province clustered std. errors

Finally, some additional results emerge from the interaction of large cities and the pre-armistice war events. In particular, we find that individuals born in large cities and exposed to pronounced war events already in the preparatory phase of war are more likely to suffer from any chronic disease (+0.07), which is also reflected in a higher average of number of GP visits (+3.9). These results could be easily rationalised if we think that national food production at that time was already in shortage due to years of autarchy policy imposed by the fascist regime and by economic sanctions imposed by international community and, most notably, by shortage of manpower who was required by the ongoing war activities.

5.1 Trend effects in exposure to war stress

In the last part of the analysis, we investigate to how the effects of exposure of pregnant mothers to war stress evolve over time. Put differently, we analyze if on top of a "level" effect we can also find a "trend" effect. In fact, as prevalence of chronic conditions varies with age, health differentials resulting from prenatal exposure to adverse conditions may follow age specific patterns and the differential effect could widen over time between "treated" and "control" group. To study this phenomenon, we run model specifications discussed in the previous sections additionally on earlier HS data waves from 2004 to 2009, overall obtaining 7 age specific subsamples, relative to individuals 61- to 67-years old. This set of results is presented in a graphical form in Figures 4-8, where we plot the year-by year coefficient estimates with the relative confidence intervals.

Reading the figures, we find that the impact of war adverse events seems to increase over time, thus suggesting the presence of a trend effect. Overall, for health expenditure and for the number of GP contacts, we see that the differentials start to emerge only around the age of 65. This pattern is confirmed for depression, while for diabetes the differentials seem to be homogeneous across ages. In case of the prevalence of at least one chronic disease, we see the most pronounced differentials for the 65-year-olds, with the difference diminishing with the lag between birth and the outcomes measurement. This last result may be attributable to the fact that the prevalence of individuals with at least one chronic condition is very high,

Figure 4: Aging and war impact on number of visits

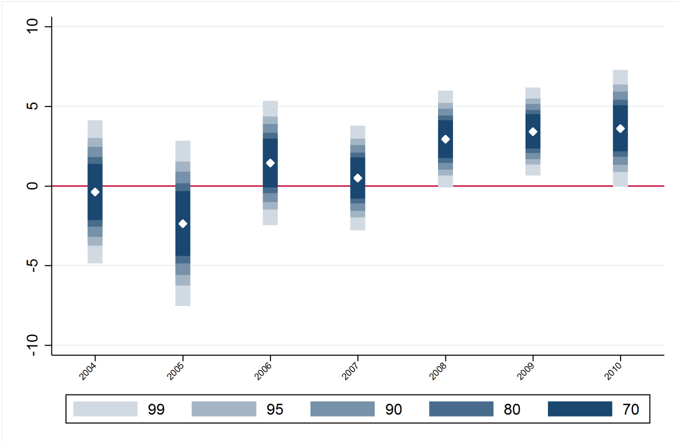
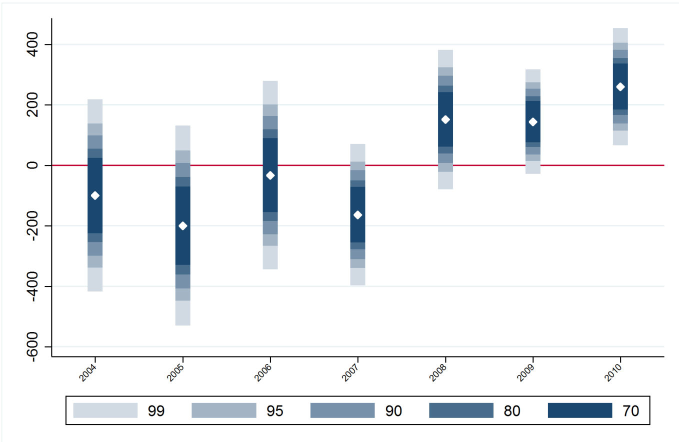


Figure 5: Aging and war impact on health expenditure



reaching 95% at the age of 67. Hence at certain age, it is very unlikely not to suffer from any chronic condition. For this reason, a convergence effect may be in place, where each additional year of age is able to account for a minor portion of the discussed differential.

To our knowledge, this is the first study to capture the dynamics of the evolution of the effect of WWII exposure. As a by product of this analysis, we show also that if measured at earlier stages, the differentials on some outcomes are unlikely to be expressed, confirming the assumptions behind Barker's fetal origins hypothesis.

Figure 6: Aging and Nazi impact on depression

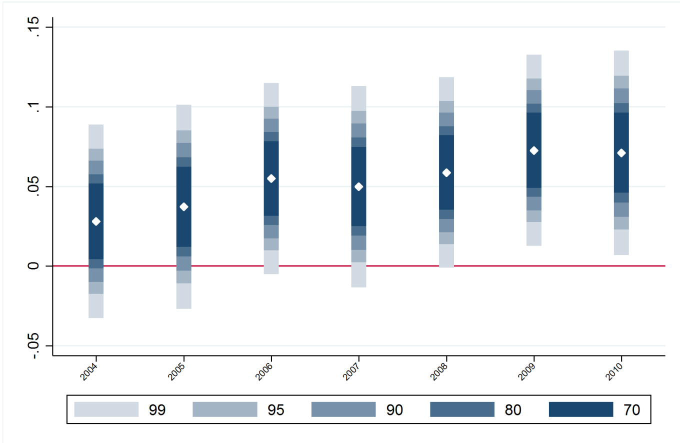


Figure 7: Aging and large city war impact on diabetes

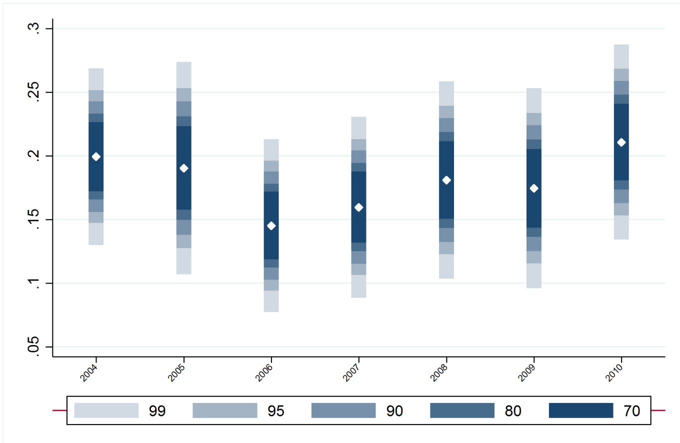
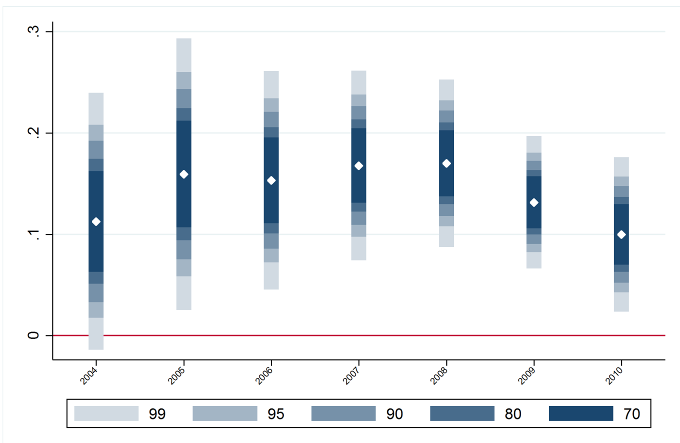


Figure 8: Aging and war impact on any chronic disease



5.2 Discussion

Compared to existing literature, our results present a new scenario on the long-run effects of prenatal exposure to WWII. Working with precisely measured health outcomes and detailed treatment effects enables us to identifying the "true" treated and control groups and, therefore, to provide an accurate estimation of the causal effect. In fact, our results differ in a substantial way with respect to other papers in this field, with magnitudes which sometime are 10-fold bigger. The range of the difference is likely to reflect a different approach offered by this study. First of all, to the best of our knowledge, this is the first work on WWII effects offering an identification strategy which is able to address the issue of selective fertility. Moreover, the differentials are also likely to result from the objective measurement of the data, where the false negatives typical of the self-reported data, are not an issue in this study. Also, due to lack of precise data, the previous studies are likely to have suffered from misclassification in treatment, which might be the cause of the underestimation (attenuation bias) of the treatment effect Lewbel (2007). Finally, and in favour of the estimates presented in this study, despite 95% of patients present in our sample have at least one chronic condition (see Table 1), the model estimates are sizeable and robust across specifications and placebo regressions.

6 Conclusions

In this paper we present new and detailed evidence on the long run impact on adult health outcomes and health expenditure of WWII adversities occurred in Italy following the 1943 armistice. More importantly, and differently from the previous literature, we are able to disentangle the role of various mechanisms at work that produce heterogenous response of certain outcomes to peculiar type of shocks suffered during war. Along this line, we have refer to three main mechanisms: *i)* general war stress, *ii)* nazi violences, and *iii)* famine in large cities.

From an econometric point of view, we exploit the exogenous variation in the war intensity resulting from the Italian armistice of September 1943, and provide WWII long-run causal effects on objectively measured health and economic outcomes. We use a difference-in-differences setup, within a unique dataset that matches GPs electronic clinical records (ECRs) with a detailed information on the intensity of WWII defined on a refined time-space scale (province/month of birth). The objectively measured and diagnosed health outcomes combined with precisely specified treatment status and the natural experiment setting of the Italian WWII historical events, allow us to provide causal estimates of the adult health differentials due to the exposure to different WWII stressors.

Our results show that the WWII events caused significant health disadvantages, and that the disadvantages are detectable after more than 60 years, in the elderly population. In virtue of different exposure of foetuses to WWII events, we find that in utero exposure to famine is more likely to cause diabetes, while Nazi violence affected the onset of depression. Exposure to intense war events had effects on the number of chronic conditions, number of GP visits, prevalence of dislipidemia and health care costs. Finally, for the first time within this literature we show that these effects increase with the age of the treated.

The evidence of a sizeable extent of health differential leads us to believe that there might be other economic margins potentially affected by these shocks. In fact, in our further research, we aim to investigate the effect of war adverse events on labour market productivity, wage and income accumulation. Overall, our paper shows that real economic consequences of WWII are sizeable and linger for decades. In view of numerous armed conflicts and terrorism pervading many countries these days, it is crucial to gauge the economic consequences of such phenomena. More importantly, our findings suggest that if the events suffered during the in-utero period are likely to propagate for so long, the protection of mothers for the sole nine months of the gestational period is likely to deliver a substantial economic payoff.

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