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December 28, 2020

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Acknowledgements

The authors are grateful to all participants for providing the data used in the E3N cohort study. The E3N cohort study is being carried out with the financial support of the "Mutuelle Générale de l'Education Nationale", the European Community, the French League against Cancer, the Institute Gustave Roussy, and the French Institute of Health and Medical Research (Inserm). This present study was also supported by the French Research Agency (Agence Nationale de la Recherche) via an "Investissement d'Avenir" grant (investment for the future grant, ANR-10-COHO-0006) that supports the E4N study and by the Strategic Research Initiative NutriPerso from the University Paris-Saclay. The funders had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; and preparation, review, or approval of this manuscript.

We thank Gabriella Conti, Sergei Guriev, Yang Xie, two anonymous reviewers and participants at the Sciences Po Friday lunch seminar and the UC Berkeley Environment, Resource and Energy Economics Seminar for their helpful comments and suggestions. All remaining errors are ours.

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February 23, 2021

Abstract

We investigate the effects of early-life exposure to war on adult health outcomes including cancer, hypertension, angina, infarction, diabetes and obesity. We combine data from the French prospective cohort study E3N on women employed in the French National Education with historical data on World War II. To identify causal effects, we exploit exogenous spatial and temporal variation in war exposure related to the German invasion of France during the Battle of France. The number of French military casualties at the level of the postcode area serves as main measure of exposure. Our results suggest that exposure to the war during the first 5 years of life has significant adverse effects on health in adulthood. A 10 percent increase in the number of deaths per 100,000 inhabitants in the individual's postcode area of birth increases the probability of suffering from any of the health conditions considered in this study by 0.08 percentage points. This is relative to a mean of 49 percent for the sample as a whole.

Keywords: early-life exposure, developmental origins, World War II, health, human capital development

JEL codes: I10, I12, J13

1 Introduction

Wars diminish economic resources and threaten access to food, shelter and health care. Populations are subjected to acute stress related to the experience of violence and disruption of families and communities. Exposure to such hardship is likely to have devastating and potentially long-lasting effects on the health of wartime children. Although exposure to particular environments and experiences appear to influence health development at all stages, it has been suggested that exposure to environmental insults during childhood and adolescence has particularly powerful and long-lasting consequences on health due to the persistence of bio-behavioural attributes that are acquired early in life (Almond and Currie, 2011; Baird et al., 2017; Cunha and Heckman, 2007; Fall and Kumaran, 2019; Halfon and Hochstein, 2002; Hertzman, 1999). Yet, there has been only limited research exploring how early-life exposure to war affects long-term health outcomes in the civilian population. Most of the existing literature focuses on developing countries and shows that the experience of armed conflicts during the 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 (Akresh et al., 2012a,b; Alderman et al., 2006; Mansour and Rees, 2012; Minoiu and Shemyakina, 2014).

In this study, we estimate the effects of exposure to World War II (WWII) during childhood and adolescence on objectively measured adult health outcomes including cancer, infarction, diabetes, angina, hypertension and obesity. We use data from the French prospective cohort study E3N on over 28,000 women employed in the French National Education (mainly teachers) born between 1925 and 1950. We combine this demographic and health data with historical data on French military casualties, French prisoners of war (POW) and the Allied bombing of France during WWII. To establish causality, we exploit variation in the intensity of the war across time and space which is plausibly exogenous to individual and family characteristics. More precisely, we compare health outcomes for women born in postcode areas which were intensely affected by the war with women belonging to the same group of birth cohorts but who were born in less affected postcode areas, relative to women from other birth cohorts. Identification strategies of this type are often used in the literature but exploiting data at such a fine geographical level as the postcode area is less common.

Despite the scale and scope of WWII, surprisingly few economic studies exploit the events of this war to investigate the relationship between war exposure and long-term health. Using retrospective data from SHARELIFE for several European countries, Kesternich et al. (2014) find that experiencing WWII during early-life increases the probability of suffering from diabetes, depression, and heart disease in adulthood. However, health outcomes in this study are self-declared - such data are often argued to be unreliable and subject to self-reporting bias relative to other sources of information, such as medical records or laboratory measurements (Dowd and Todd, 2011; Jürges, 2007) - and treatment is defined at the aggregate (country or region) level, which potentially leads to measurement errors limiting the causal interpretation of the results. Havari and Peracchi (2017) also use SHARELIFE data and find that early-life exposure to hardship related to World War I and II is associated with worse physical and mental health, education, cognitive ability and subjective well-being later in life. Different from Kesternich et al. (2014) and our study, Havari and Peracchi (2017) provide descriptive evidence rather than attempting to uncover causal relationships through quasi-experimental methods.

Our work is closest to Akbulut-Yuksel (2017) who also uses data at a fine geographical level and a similar identification strategy to study the effects of early-life exposure to warfare on adult health. Akbulut-Yuksel (2017) considers the impact of Allied bombing in Germany, using the German Socio-Economic Panel together with data on air bombing to exploit city-by-cohort variation in the intensity of exposure. She finds that individuals exposed in-utero and during early childhood are more likely to be obese and to suffer from stroke, hypertension, diabetes, and cardiovascular disorder in adulthood. The caveat of this study is that it exploits data on the place of residence during later-life and not place of birth which means it must be assumed that individuals do not move away from their birth place. This is likely to undermine the identification strategy. In addition, the health outcomes in this study are again self-declared. We consider place of birth and not place of residence during later-life to define treatment status which should attenuate problems related to treatment miss-classification.

Schiman et al. (2019) also exploit data at a fine geographical level to study the effects of WWII. However, the focus of this study is different. They do not study the effects of warfare, but rather the war-induced rise in infant mortality in 1940–1941 in England and Wales on self-reported health, income and employment. Conti et al. (2019) examine the effects of warfare on height,

weight, BMI, IQ and mental deficiency using city-level data of monthly civilian deaths during the Dutch Hunger Winter. Different from the present study which focuses on early childhood exposure, Conti et al. (2019) focus on prenatal exposure.

In contrast to most of the existing studies which have to rely on self-reported health outcomes we use data on objectively measured health outcomes including cancer, hypertension, angina, myocardial infarction, diabetes and obesity. We thereby avoid potential bias from misreporting. The richness of our data allows us to control for a range of family and individual characteristics including, for example, the socioprofessional categories of the woman and her father, and health-affecting behaviours such as tobacco consumption, sleep duration, and diet. We are able to distinguish the effects of war-related hardship as captured by our measures of war exposure based on the historical data from the effect of war-related nutritional shortages by controlling for the level of hunger suffered during WWII as reported by the participants in our data.

We find evidence for adverse long-run consequences of exposure to WWII on wartime children's health outcomes. Women who have been exposed more intensely to WWII are more likely to suffer from any of the health conditions registered in the data but only if the exposure occurred during the first five years of their life. A 10 percent increase in the number of deaths per 100,000 inhabitants in the postcode area of birth increases the probability of suffering from any of the health problem by 0.08 percentage points for women exposed at ages 0 to 5 relative to older and younger cohorts. This is relative to a mean of 49 percent for the sample as a whole. These results are robust to the inclusion of the registered health-affecting behaviours (tobacco consumption, sleep duration, and diet) which suggests that the effects are not mediated through changes in these health behaviours. We also find some limited evidence for adverse health outcomes among women born in postcode areas which were home to an above average number of POW compared to women from postcode areas with a below average number of POW. However, this result is not very robust and should be interpreted with caution. Using Allied bombing as measure of war exposure did not yield any significant results.

War exposure as measured by the number of military casualties could potentially capture the effect of exposure to stress from experiencing or witnessing battle-related violence or stress related to fleeing the advancing troops. The number of POW could be an indirect measure for the likelihood

that the woman has grown up in the absence of a father or other male relative, which could have implied lower household resources and thus worse outcomes in adulthood. Interpreting the measures in this way, the results of this study suggest that it could have been the exposure to violence and, to a lesser extent, the absence of a father or male relative which potentially impacted later-life health outcomes. Our results remain unchanged when we control for the level of hunger suffered during WWII as reported by the participants in E3N, suggesting that the effects we capture through our measures of war exposure are distinct from the effects of war-related nutritional shortages.

Contrary to the effects of war on physical capital, which have been shown to be relatively short-lived (Bellows and Miguel, 2009; Brakman et al., 2004; Davis and Weinstein, 2002; Miguel and Roland, 2011), the results presented in this paper suggest that the effects of war on human capital are long-lasting. Our findings underline the importance of post-conflict policies primarily targeting children exposed during early childhood to mitigate, or potentially reverse, the adverse long-term health effects caused by exposure to war. This is of particular relevance in a world where the number of armed conflicts is at an all-time high (Strand et al., 2019).

2 Background and data description

2.1 The German invasion of France as exogenous shock: historical background

We consider the spatial and temporal variation in war exposure related to the German invasion of France during the Battle of France to be a shock that is exogenous to individual and family characteristics. This is not an unreasonable assumption given that the German invasion occurred suddenly and unexpectedly. The battle fronts moved quickly and were not concentrated in areas that the French expected to have to defend. The Battle of France lasted only six weeks (10 May - 25 June 1940). Germany relied on surprise *blitzkrieg* ("lightning war") techniques. Their strategy was to carry out a subsidiary attack through neutral Belgium and the Netherlands, with the main attack against France to be launched a little later through the Ardennes. This was a hilly

and forested area on the German-Belgian-French border, where the Allies did not expect an attack. In what follows we briefly summarise the main battle movements.

The German attack began on 10 May 1940, with German air raids on Belgium and the Netherlands, followed by parachute drops and attacks by ground forces. On 14 May the Dutch surrendered. As planned by the Germans, the British and French responded by pushing their forces into Belgium. On 13 May, the first German forces arrived near Sedan, on the River Meuse. With most of the Allied forces fighting in Belgium, the German forces encountered little resistance. They quickly broke the Allied supply-lines and reached the English Channel on 20 May. The German forces then advanced through Belgium and encircled the Allied forces by moving tanks up from the south and west. The Belgian army surrendered on 28 May. Between 26 May and 4 June, 338,000 Allied troops were evacuated from Dunkirk. On 5 June, the German forces started to move southwards from the River Somme and launched an offensive on Paris on 9 June. Paris was captured on 14 June, a little more than a month after the beginning of the Battle of France. The German troops crossed the River Loire in the west on 17 June and reached the Swiss frontier a few days later. The Battle of France ended with the surrender of France on 22 June (BBC, 2011).

The armistice led to the creation of a new "French state" under *Maréchal* Philippe Pétain governing from Vichy. German troops occupied three-fifths of the French territory, northern France and the Atlantic coast, leaving the south and eastern two-fifths under Vichy's control. The northern departments of the Nord and Pas-de-Calais had direct military control from Brussels, Alsace and Lorraine were reincorporated into the Reich, a forbidden zone was established in north-eastern France, and an Italian zone was created in south-eastern France in November 1942 (Mouré, 2010).

2.2 Explanatory framework for causal long-run effects on health of early-life conditions

The "Developmental Origins of Health and Disease (DOHaD)" hypothesis postulates that risk factors, protective factors, and early-life experiences affect long-term health and disease outcomes. Although exposure to particular environments and experiences appear to influence health development at all stages in life, it has been suggested that exposure to environmental insults during

childhood and adolescence has particularly powerful and long-lasting consequences on health due to the persistence of bio-behavioural attributes that are acquired early in life.

The relationship between early life exposure and health trajectories has been explained using both latency and pathway models. The latency model links early-life exposure to adult health outcomes in a direct manner independently of intervening life circumstances. It proposes that early-life exposures can program long-term or permanent changes in biological and behavioural systems (Barker, 1992; Halfon and Hochstein, 2002; Hertzman, 1999). The pathway model proposes that early-life exposure relates to adult health outcomes indirectly through changes in health-affecting behaviours and life conditions. Negative childhood experiences may lead to unhealthy behaviours such as substance abuse and poor school performance in adolescence and limited opportunities in adulthood. Inadequate resources and stressful life circumstances in adulthood, in turn, increase the risk of morbidity and mortality (see for example Ben-Shlomo and Kuh (2002)). The latency and pathway models are not competing explanations, but are thought to be intertwined in a complex manner. Chronic disease may be the long-term outcome of childhood conditions and experiences combined with cumulative exposures across adulthood (Blackwell et al., 2001).

2.3 Data description

The *Etude Epidémiologique auprès de femmes de la Mutuelle Générale de l'Education Nationale* or E3N is a French prospective cohort study which was initiated in 1990 to investigate the risk factors associated with cancer and other major non-communicable diseases in women. E3N participants were insured through a national health system that primarily covered teachers, and were enrolled in the study from 1990 onward after returning baseline self-administered questionnaires and providing informed consent. The cohort comprised nearly 100,000 women (mainly teachers) born between 1925 and 1950 and therefore aged 40 to 65 years at recruitment. Follow-up questionnaires were sent approximately every 2-3 years and addressed general and lifestyle characteristics together with medical events which include among others cancers, cardiovascular diseases, diabetes, depression, fractures and asthma. Our study includes data from the follow-up questionnaires until 2014. The follow-up questionnaire response rate remained stable at approximately 80% (Clavel-Chapelon, 2014).

The E3N data provide information on a wide range of individual characteristics including the date and place of birth, educational achievement, a measure for the level of stress experienced at work, marital status, information on early childhood conditions such as preterm birth, birth weight and height, physical activity during childhood, age of the mother and father at birth, the woman's, her husband's and father's socioprofessional status, whether the individual lived on a farm, number of siblings and information on the presence of health conditions in the family. Registered health outcomes include occurrence of any cancer, myocardial infarction, angina, diabetes, hypertension and obesity. These health outcomes have been validated using the data from the national health system. We observe a range of health behaviours including tobacco consumption, average sleep duration, and diet in terms of carbohydrate, protein, fat, and total calorie intake. In the first questionnaire, the participants were asked how much they suffered from the hardships of WWII in terms of food deprivation. The possible answers were "not born at the time", "not at all or few suffering", "moderate suffering", "a lot" (continuous hunger), "enormously" (deportation). Albeit subjective, this variable is interesting as it allows us to distinguish the effect of nutritional shortages from the effect of other war-related hardship. Figure 1 in the Appendix shows a map with the postcode area average of the self-reported level of food deprivation. Individuals seemed to have suffered from hunger all across France and there is no apparent spatial clustering.

We merge the E3N data with data we collected from several historical sources. Our most important source is the database *"Memoire des hommes"* managed by the French Ministry of Armed Forces and available at their website. The data is based on the death records of French soldiers and is said to be exhaustive. We scrape the information on the soldier's place of death and construct a measure of war exposure based on the number of military casualties or the number of military casualties per 100,000 inhabitants at the level of the postcode area. Figure 2 in the Appendix shows a map with the distribution of the French military casualties per 100,000 inhabitants at the level of the postcode area. Unsurprisingly, military deaths appear to be concentrated in the north-east along the path of the German invasion during the Battle of France. Figure 3 shows how the number of French military deaths evolved during the months of the Battle of France in the northern zones which would become the German occupied zone soon after the end of the battle, compared to the southern zone which only fell under German military administration in November 1942. We can see that the number of deaths rises rapidly during the months of May and June 1940 and only in

the north of France which coincides with the timing and location of the battles during the Battle of France (see section 2.1 on the historical background).

We construct several other measures of war exposure. We use data from digitised paper mission reports of air warfare between 1939 and 1944 to construct different measures of intensity of the Allied bombing of France: an indicator variable equal to one if any bomb was dropped in a given postcode area, the number of bombs dropped in a given postcode, the distance of any postcode area centroid to the nearest bomb, or the mean distance to any k nearest bombs. Figure 4 shows a map with the exact location of where the bombs were dropped. We further use data on the French prisoners of war from the official lists provided by the German military authority between 1940 and 1941, made available online by the National Library of France. Using optical character recognition software and merging the place of birth of the prisoners with postcodes, we obtain a data set including about 10% of the total population of French prisoners of war. We use the number of prisoners of war original from a given postcode area as a measure of war exposure in terms of the absence of a father or male breadwinner, supposing that children in areas which were home to a larger number of prisoners of war had a higher probability of having suffered from the absence of a father. Figure 5 shows a map with the number of POW at the level of the postcode area.

For the merging and for the construction of the maps, we use a France shapefile with administrative boundaries at the postcode level. We exclude observations for which we do not have full information on important covariates (lives with a partner, higher education, socioprofessional category of the individual and her father, born preterm, mother's and father's age at birth, number of siblings, physically or mentally stressful job, lived on a farm, lives in deprived area, suffered from hunger during WWII). Our results are robust to using the full data set including fewer covariates. Table 3 in the Appendix presents summary statistics for this final sample.

3 Identification strategy

To estimate causal effects of early-life exposure to WWII on later-life health, we exploit variation in the intensity of WWII across time and space, which is plausibly exogenous to individual

and family characteristics. More precisely, we compare the health of women born in postcode areas which were intensely affected by the war with the health of women belonging to the same generation but who were born in less affected postcode areas, relative to the health of women from other birth cohorts. Similar approaches to establish causality have been used in the literature (see for example Akbulut-Yuksel (2017); Kesternich et al. (2014); Schiman et al. (2019)). We write our model as follows

$$H_{ipt} = \alpha + \beta_1 Exposure_p + \beta_2 Exposure_p \cdot gen_{it} + \gamma_t + \delta_d + \rho X_{ipt} + \epsilon_{ipt} \tag{1}$$

where H_{ipt} denotes the health outcomes for individual i born in postcode area p in year t, $Exposure_p$ is the measure of war exposure at the level of the postcode area p, gen_{it} is an indicator variable equal to one if individual i born in year t is part of the generations we consider to be treated, and X_{ipt} is a vector of controls for individual and family characteristics. Year of birth and spatial (department level) fixed effects are denoted γ_t and δ_d, respectively, and ϵ_{ipt} is the standard error. Results are robust to clustering at the level of the spatial fixed effects (the department).

War exposure is constructed either based on the number of French military casualties, the number of French prisoners of war original from the postcode area or the intensity of the Allied bombing. The dependent variables are binary as they describe whether an individual is affected or not by disease. We therefore run Logit regressions and report the estimates of the average marginal effect. The results are qualitatively similar when we use linear probability models.

Including the indicator variable gen_{it} which marks individuals as belonging to the treated generations allows us to compare the effects across groups of birth cohorts. This is useful to investigate whether exposure to the war has heterogeneous effects with respect to age at exposure. Almond and Currie (2011) suggest for example that adverse shocks negatively affect individuals between conception and 5 years of age more intensely than older individuals whereas others such as Sparén et al. (2004) argue that the onset of puberty is a sensitive period as the body collects resources in anticipation of the adolescent growth spurt. Our estimate of interest is the coefficient β_2 on the interaction term between the exposure to WWII and the variable indicating the individual belongs to the treated generations. This is the differences-in-differences estimate capturing the effect of WWII on health for women who have been exposed intensely relative to women from the

same group of birth cohorts but who have been less exposed, and relative to women from other birth cohorts. We test several model specifications in which we consider different groups of birth cohorts to be treated. In our main specification, we consider as treated all individuals born from 1935 to 1939 and thus aged 0 to 5 years at the time of the invasion, while the younger and older birth cohorts serve as the control group.

Our identification strategy relies on the assumption that the geographical measure of exposure is sufficiently correlated with the actual hardship experienced at the level of the individual. This does not have to be the case. Families who are exposed to WWII in a similar way might be able to mitigate the effects of the war on the children more or less well, for example, because they belong to the ruling elite or because the parents differ in altruism or capability. We control for all observable family characteristics, such as for example the father's socioprofessional category which should capture to some extent the parent's capacity to mitigate shocks.

The vector of covariates X_{ipt} includes the individual's marital status, a measure for the level of physical and mental stress experienced by the individual at her work, her educational achievement, indicator variables for the socioprofessional category of the individual and her father, the number of siblings, information on whether the individual was born preterm, whether the individual lived on a farm, and indicators for the existence of cancer, diabetes, and hypertension in the family. Controlling for hunger allows us to distinguish the effect of exposure to the war as captured by our measures of war exposure from nutritional deprivation as another source of war-related hardship. In some regressions we add controls for the health-affecting behaviours we observe in the E3N data including tobacco consumption, average sleep duration, and diet-related measures such as fat, protein, carbohydrate and total calorie intake.

Finally, the validity of the difference-in-differences estimates relies on the presence of a parallel trend across treatment and control postcode areas. Outcomes in the affected postcode areas should be similar to outcomes in the unaffected areas had there been no exposure to the war. We test if the assumption is plausible by interacting the measure of war exposure with different sets of cohort dummies to check whether the difference in health outcomes for women born in affected versus unaffected postcode areas is significant only for cohorts that could have been exposed. We

should only find effects for the generations that were alive at the time of the Battle of France but not for the cohorts born afterwards.

4 Results

4.1 Effects of war exposure as measured by French military casualties

Table 1 presents evidence for the plausibility of the parallel trend assumption and reports our main results for the effect of exposure to WWII as measured by the logarithm of French military casualties per 100,000 inhabitants on the probability of suffering from health problems during adulthood (we use log of deaths+1 to avoid missing values for observations with 0 deaths). Column one shows results for the interaction of the measure of war exposure with 5-year birth cohort groups. We find that an increase in the intensity of war exposure affects health outcomes negatively for women born from 1935 to 1939 but not for older and younger cohorts. These women were in between 0 and 5 years old during the Battle of France at the onset of the war when most of the exposure occurred. Figure 6 in the Appendix illustrates this result graphically. Not finding any significant results for the cohorts born after the Battle of France supports the parallel trend assumption. Outcomes for women born in the affected versus the unaffected areas appear no different for the cohorts who did not experience the German invasion. Interestingly, we do not find results for the cohorts born before 1935. These cohorts experienced the Battle of France, albeit at an older age. This suggests that exposure affects in particular young children.

Estimation of our differences-in-differences specification as formalised in Equation (1) is reported in the second column of Table 1. The dependent variable here is a single binary variable indicating the presence of any health condition, equal to 1 if the individual developed any of the observed health conditions and 0 otherwise. A 10 percent increase in the number of deaths per 100,000 inhabitants in the individual's postcode area of birth increases the probability of suffering from any of the health conditions considered in this study by 0.08 percentage points for the women born from 1935 to 1939 relative to older and younger cohorts. This is significant compared to a mean of 49% for the sample as a whole. We do not find such effects for individuals from other birth cohorts.

Considering only the deaths occurring during the Battle of France (both per 100,000 inhabitants or absolute numbers) yields qualitatively similar results. Columns three and four of Table 1 report results for health outcomes on a more disaggregate level. We find that exposure to WWII increases the probability of suffering both from cancer and from "metabolic syndrome" diseases which includes hypertension, angina and diabetes. Cardiovascular diseases and diabetes, as well as obesity, are often grouped under the term "metabolic syndrome" because they are related (associated with lifestyle, e.g. diet and physical exercise habits) and often occur together. Including or excluding myocardial infarction and obesity for the construction of the 'metabolic syndrome" variable does not change the precision of our estimation or the point estimate. Considering hypertension, angina and diabetes separately does not yield statistically significant results.

We found evidence to suggest that our results may be driven by the effects in women exposed at around 2 years of age. Looking at the interaction of exposure with year of birth shows that the effects are only visible for women born in 1938 (and surprisingly not for those born in 1939). See Figure 7 in the Appendix for a graphical representation of this result. However, when we consider smaller cohorts as treatment cohorts (e.g. the 2-year cohort born in 1938-39 or the 3-year cohort born from 1937-39) relative to the younger and older cohorts in the differences-in-differences regressions, we mostly obtain results that are not significant.

Table 1: Effect of early-life exposure to WWII as measured by the number of French military casualties per 100,000 inhabitants in the individual's area of birth on adult health

	Any health condition		Cancer	Metabolic diseases
	(1)	(2)	(3)	(4)
Exposure x Born 1925-34	0.0011			
	(0.0035)			
Exposure x Born 1935-39	0.0076**	0.0080**	0.0053*	0.0056**
	(0.0036)	(0.0038)	(0.0029)	(0.0027)
Exposure x Born 1940-45	−0.0033			
	(0.0026)			
Exposure x Born 1946-50	0.0013			
	(0.0027)			
Exposure		−0.0004	−0.0004	−0.0008
		(0.0017)	(0.0013)	(0.0019)
Lives with partner	−0.0054	−0.0055	−0.0019	0.0043
	(0.0085)	(0.0085)	(0.0052)	(0.0077)
Higher education	−0.0405***	−0.0405***	0.0034	−0.0519***
	(0.0055)	(0.0055)	(0.0047)	(0.0055)
Born preterm	0.0426**	0.0425**	0.0109	0.0343**
	(0.0187)	(0.0187)	(0.0098)	(0.0172)
Mother's age at birth	−0.0003	−0.0003	−0.0007	0.0005
	(0.0007)	(0.0007)	(0.0007)	(0.0007)
Father's age at birth	−0.0006	−0.0006	0.0003	−0.0010
	(0.0008)	(0.0008)	(0.0006)	(0.0006)
Nb. of siblings	−0.0035**	−0.0034**	−0.0011	−0.0024*
	(0.0014)	(0.0014)	(0.0012)	(0.0013)
Physically stressful job	0.0080	0.0080	0.0178***	0.0026
	(0.0077)	(0.0077)	(0.0054)	(0.0067)
Mentally stressful job	0.0127	0.0128	−0.0025	0.0154*
	(0.0098)	(0.0098)	(0.0054)	(0.0088)
Lived on a farm	0.0046	0.0047	0.0085	0.0019
	(0.0072)	(0.0072)	(0.0054)	(0.0063)
Lives in deprived area	0.0053	0.0053	−0.0031	0.0088***
	(0.0036)	(0.0036)	(0.0022)	(0.0033)
Hunger (scale)	0.0340***	0.0340***	0.0144**	0.0233***
	(0.0064)	(0.0064)	(0.0058)	(0.0066)
Bombs	0.0005	0.0006	0.0041	−0.0005
	(0.0070)	(0.0070)	(0.0059)	(0.0073)
Num. obs.	28324	28324	26165	28324
Log Likelihood	−19023.9073	−19024.8850	−11624.4387	−17494.7512

$^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

Exposure is the logarithm of the number of fallen French soldiers per 100,000 inhabitants in the woman's postcode area of birth. All models include birth-year, department fixed effects and dummies for the individual's and her father's socioprofessional category. The coefficients show average marginal effects. Standard errors are clustered at the department level.

The results are robust to the inclusion of the different covariates. We show in Table 4 of the Appendix that the coefficient of interest changes only marginally when we control for a range of individual and family characteristics and remains unchanged when we add further controls for individual life circumstances. The coefficients on the control variables generally have the expected sign. For example, being born pre-term or suffering from hunger during WWII is associated with a higher probability of being sick later in life. This association between WWII-related food deprivation and health outcomes in adulthood is investigated by Mink et al. (2020). Including additional controls for health-related behaviour such as tobacco smoking, sleep duration, physical activity during childhood, and diet still yields similar results.

We further consider the possibility that the effects of war exposure on health manifest only in individuals who have been exposed with an intensity above a certain threshold. We divide individuals into groups according to their quartile of exposure and interact the exposure quartile indicator with the indicator marking whether the individual belongs to the affected generations born from 1935 to 1939. The first quartile includes postcodes areas with less than 14 deaths per 100,000 inhabitants, in the second quartile this number ranges from 15 to 67 and in the third quartile from 68 to 231, while the most exposed fourth quartile includes postcode areas with 232 or more deaths per 100,000. We find that result appear to be driven by the adverse health outcomes faced by women who were born in the most intensely exposed areas. As shown in Table 2, having been born in one of the 25 percent most affected postcode areas relative to the 25 percent least affected postcode areas raises the probability of suffering from any health condition by 4.9 percentage points, for women exposed at ages 0 to 5 relative to the other cohorts.

4.2 Other measures of war exposure

We find some evidence for adverse health outcomes among women born in postcode areas which were home to an above average number of POW compared to women from postcode areas with a below average number. The results are reported in Table 5 of the Appendix. However, this effect only manifests when we use cut-off values, here a cut-off at the average number of POW. Using the absolute number of POW or the number of POW per 100,000 inhabitants does not yield any results. The number of POW could be too imprecise a measure of exposure to war hardship

Table 2: Effect of exposure to WWII on health, any health condition - Heterogeneity with respect to intensity of exposure

	Any health condition
Second quartile exposure x Born 1935-39	0.0201
	(0.0190)
Third quartile exposure x Born 1935-39	0.0306
	(0.0212)
Fourth quartile exposure x Born 1935-39	0.0492**
	(0.0194)
Second quartile exposure	−0.0014
	(0.0084)
Third quartile exposure	0.0012
	(0.0094)
Fourth quartile exposure	−0.0007
	(0.0092)
Num. obs.	28324
Log Likelihood	−19024.2468

$^{***}p < 0.01$; $^{**}p < 0.05$; $^{*}p < 0.1$.
Women are divided into quartiles according to the intensity of exposure (number of fallen soldier) in their postcode area of birth. First quartile from zero to 14 deaths; second quartile from 15 to 67 deaths; third quartile from 68 to 231 deaths; fourth quartile from 232 deaths or more. The omitted category is the first quartile. The model includes birth-year and department fixed effects and the full range of individual and family characteristics with the exception of health related behaviors. The coefficients show average marginal effects. Standard errors are clustered at the department level.

which could explain the absence of more robust effects. We discuss this in more detail in the next section. We do not find any effects when we use Allied bombing as measure of war exposure, despite our best efforts to construct several distinct measures of exposure. The absence of effects could potentially be due to measurement error. The Allied bombing happened during an extended period (1940-1945) and the bombing was strategical, aiming industrial and transport targets which makes it possible that individuals avoided areas that were likely to be bombed.

5 Conclusion and discussion

This study provides causal evidence of the long-run consequences of war on health outcomes. We find that an increase in the intensity of exposure to WWII as measured by the number of French military casualties leads to worse health outcomes in adulthood for individuals who were 0 to 5 years old at the time of the exposure. Our findings are consistent with evidence from the literature suggesting that exposure to WWII-related hardship has important negative consequences on the health of survivors (for example Akbulut-Yuksel (2017); Havari and Peracchi (2017); Kesternich et al. (2014)). Finding effects only for individuals who were exposed during the first 5 years of their life suggests that there exists a critical or sensitive period of development during which individuals are more vulnerable to adverse experiences. This is in line with results from the literature (see for example Almond and Currie (2011); Cunha and Heckman (2007)).

In contrast to the effects of war on physical capital, which have been shown to be relatively short-lived (Bellows and Miguel, 2009; Brakman et al., 2004; Davis and Weinstein, 2002; Miguel and Roland, 2011), the results presented in this paper suggest that the effects of war on human capital are long-lasting. Our findings underline the importance of post-conflict policies primarily targeting children exposed during early childhood to mitigate, or potentially reverse, the adverse long-term health effects caused by exposure to war.

Potential channels and pathways

Our results are robust to controlling for all of the observed health-related behaviour (tobacco smoking, sleep duration, physical activity, diet) and the level of hunger suffered during WWII as reported by the study participants. This suggests that the effects that we capture through our measures of war exposure are distinct from the effects of war-related nutritional shortages and that the effects are not entirely mediated through changes in the observed health-related behaviours. However, we cannot exclude that there are other unobserved mediating factors or other aspects of the war that affect health outcomes. We therefore cannot conclude that our results are evidence for a direct link between early-life exposure and adult health or if early-life exposure relates to adult health outcomes indirectly through changes in health-affecting behaviours and life conditions (see also the discussion in section 2.2).

Our measures of war exposure are too imprecise to allow the identification of a precise channel through which exposure to the war affects later-life health outcomes. We can only make some attempts at interpretation. We obtain our main results using the number of French military casualties as a measure of exposure to the war. This measure could potentially capture the effect of stress from experiencing or witnessing battle-related violence or stress related to fleeing the advancing German troops. We also find some limited evidence for adverse health outcomes among women born in postcode areas which were home to an above average number of POW compared to women from postcode areas with a below average number of POW. The number of POW could be interpreted as an indirect measure for the likelihood of growing up in the absence of a father or other male relative, which could imply psychological distress and/or lower household resources and thus worse outcomes in adulthood. However, the results for this measure are not very robust. The effect manifests only when we use cut-off values but not when we use the absolute number of POW. The number of POW could be too imprecise a measure of exposure to war hardship. A higher number of POW could have only a modest impact on the probability of growing up without a father or male relative. This increased probability must then lead to sufficiently large adverse effects on mental or material well-being to induce discernible effects on later-life health.

If we interpret the different measures of exposure as described above, the results of this study suggest that it could have been the exposure to the violence during the Battle of France and, to a lesser extent, the absence of a father or male relative which impacted later-life health outcomes.

Sample representativeness, selective mortality and selective fertility

Our sample is not representative of the general French population. Composed of women enrolled in a national health insurance system which primarily covered teachers, the women in our sample are on average more educated. These women may come from a relatively more privileged background, which may have mitigated their exposure to the war and its effects. Women who were so intensely affected by the war that they were not able to get the necessary education to become employed in the French national education are altogether excluded from our sample. We therefore consider our estimates to be a lower bound for the effects of exposure in the general population.

In addition, there may have been a change in the composition of the population caused by differential mortality. If the least healthy have been more likely to die, the pool of survivors could on average be healthier. In case of such selective mortality, the average health of a population intensely affected by the war would be better than the average health of a less affected population, leading us to underestimate the impact of war on health. This would mean that our estimates are a lower bound for the true effects.

The outbreak of the war is also likely to have affected fertility. The cohorts conceived before 1940 should be unaffected which means that the results showing worse health outcomes for women born in the affected versus unaffected postcode areas for the cohorts 1935 to 1939 but not for the cohorts born before 1935 (Table 1, Column one) should not be subject to bias from selective fertility. However, the composition of the cohorts born at the end of 1940 or later could have been affected. For this to impact our results, there would need to exist differences in fertility not only across cohorts but also across the affected and unaffected postcode areas. We do not find worse health outcomes for women born in the affected relative to the unaffected postcode areas for the generations born after the war. If anything, a potentially different fertility pattern across postcodes

would have resulted in a population that is healthier on average in the affected areas relative to the unaffected areas in a way to offset the negative health effects.

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Appendix

Table 3: Summary statistics

	mean	sd	min	max	N
Health outcomes					
Cancer	0.17	0.37	0	1	28666
Myocardial infarction	0.01	0.1	0	1	28666
Diabetes	0.05	0.23	0	1	28666
Angina	0.02	0.14	0	1	28666
Hypertension	0.35	0.48	0	1	28666
Obesity	0.02	0.15	0	1	28666
Any health condition	0.49	0.5	0	1	28666
Health behaviors					
Sports during childhood	42.67	25.92	0	215.3	28447
Hours of sleep	7.59	1.09	4	15	25600
Tobacco usage	0.33	0.47	0	1	28641
Carb. intake	234.2	73.22	12.46	1051.35	28666
Protein intake	93.14	25.59	4.74	391.03	28666
Lipids intake	89.8	26.97	4.52	250.69	28666
Calorie intake	2193.93	561.47	110.94	6341.53	28666
Takes the pill	0.59	0.49	0	1	28666
Preventive behaviour	0.4	0.25	0	1	28666
Takes hormones	0.22	0.41	0	1	28666
Treatment variables					
Deaths per postcode 1940-45	98.07	260.31	0	1853	28666
Deaths per 100,000	220.9	487.7	0	9947.64	28324
Log(1+deaths per 100,000)	3.89	2.07	0	9.21	28324
Covariates					
Year of birth	1941.3	6.47	1925	1950	28666
Lives with partner	0.85	0.36	0	1	28666
Higher education	0.38	0.49	0	1	28666
Born preterm	0.03	0.18	0	1	28666
Number of siblings	2.14	1.87	0	22	28666
Age of mother at birth	28.23	5.65	13	57	28666
Age of father at birth	31.38	6.37	13	93	28666
Lived on a farm	0.22	0.42	0	1	28666
Population density birth city	3859.01	7502.34	1.46	46529.85	28324
Physically stressful job	0.23	0.42	0	1	28666
Mentally stressful job	0.86	0.35	0	1	28666
Deprivation index	-0.25	1.02	-4.11	2.67	28666

Continued on next page

Table 3: Summary statistics

	mean	sd	min	max	N
Relative had cancer	0.78	0.41	0	1	26482
Relative had diabetes	0.12	0.32	0	1	28666
Relative had hypertension	0.37	0.48	0	1	28666
Relative had infarct	0.21	0.41	0	1	28666
Woman of high SES	0.13	0.33	0	1	28666
Woman of middle SES	0.86	0.35	0	1	28666
Woman of low SES	0.02	0.13	0	1	28666
Father of high SES	0.22	0.41	0	1	28666
Father of middle SES	0.6	0.49	0	1	28666
Father of low SES	0.18	0.38	0	1	28666
Hunger (scale 1-5)	1.76	0.78	1	5	28666
Bombing in postcode	0.25	0.43	0	1	28666
Infant mortality 36-47	3.4	0.85	1.25	5	28666

Table 4: Effect of early-life exposure to WWII as measured by the number of French military casualties per 100,000 inhabitants in the individual's area of birth on adult health - Robustness to different model specifications

	Any health condition			
	(1)	(2)	(3)	(4)
Exposure x Born 1935-39	0.0088**	0.0083**	0.0083**	0.0076*
	(0.0037)	(0.0037)	(0.0037)	(0.0039)
Exposure	−0.0011	−0.0004	−0.0005	0.0007
	(0.0018)	(0.0018)	(0.0018)	(0.0020)
Lives with partner		−0.0062	−0.0057	−0.0056
		(0.0085)	(0.0086)	(0.0092)
Higher education		−0.0448***	−0.0421***	−0.0508***
		(0.0066)	(0.0067)	(0.0072)
Born preterm		0.0460***	0.0443***	0.0300*
		(0.0167)	(0.0167)	(0.0179)
Mother's age at birth		−0.0003	−0.0004	−0.0007
		(0.0008)	(0.0008)	(0.0009)
Father's age at birth		−0.0006	−0.0006	−0.0004
		(0.0007)	(0.0007)	(0.0008)
Nb. of siblings		−0.0034**	−0.0036**	−0.0022
		(0.0017)	(0.0017)	(0.0018)
Physically stressful job			0.0084	0.0124
			(0.0074)	(0.0079)
Mentally stressful job			0.0134	0.0105
			(0.0090)	(0.0096)
Lived on a farm			0.0049	0.0023
			(0.0077)	(0.0082)
Lives in deprived area			0.0055*	0.0051
			(0.0032)	(0.0034)
Hunger (scale)			0.0355***	0.0372***
			(0.0076)	(0.0081)
Bombs			0.0006	−0.0040
			(0.0083)	(0.0088)
Health behaviours	No	No	No	Yes
Num. obs.	28324	28324	28324	25089
Log Likelihood	-19080.2047	-19040.3675	-19023.8850	-16800.1374

$^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.
Exposure is the logarithm of the number of fallen French soldiers per 100,000 inhabitants in the woman's postcode area of birth. All models include birth-year and department fixed effects and dummies for the woman's and her father's socioprofessional category. Health-related behaviors are the hours of physical activity in a typical week during childhood, smoking status, the average number of hours slept per night, measures for diet including carbohydrate, protein, lipids, and total calorie intake. The coefficients show average marginal effects. Standard errors are clustered at the department level.

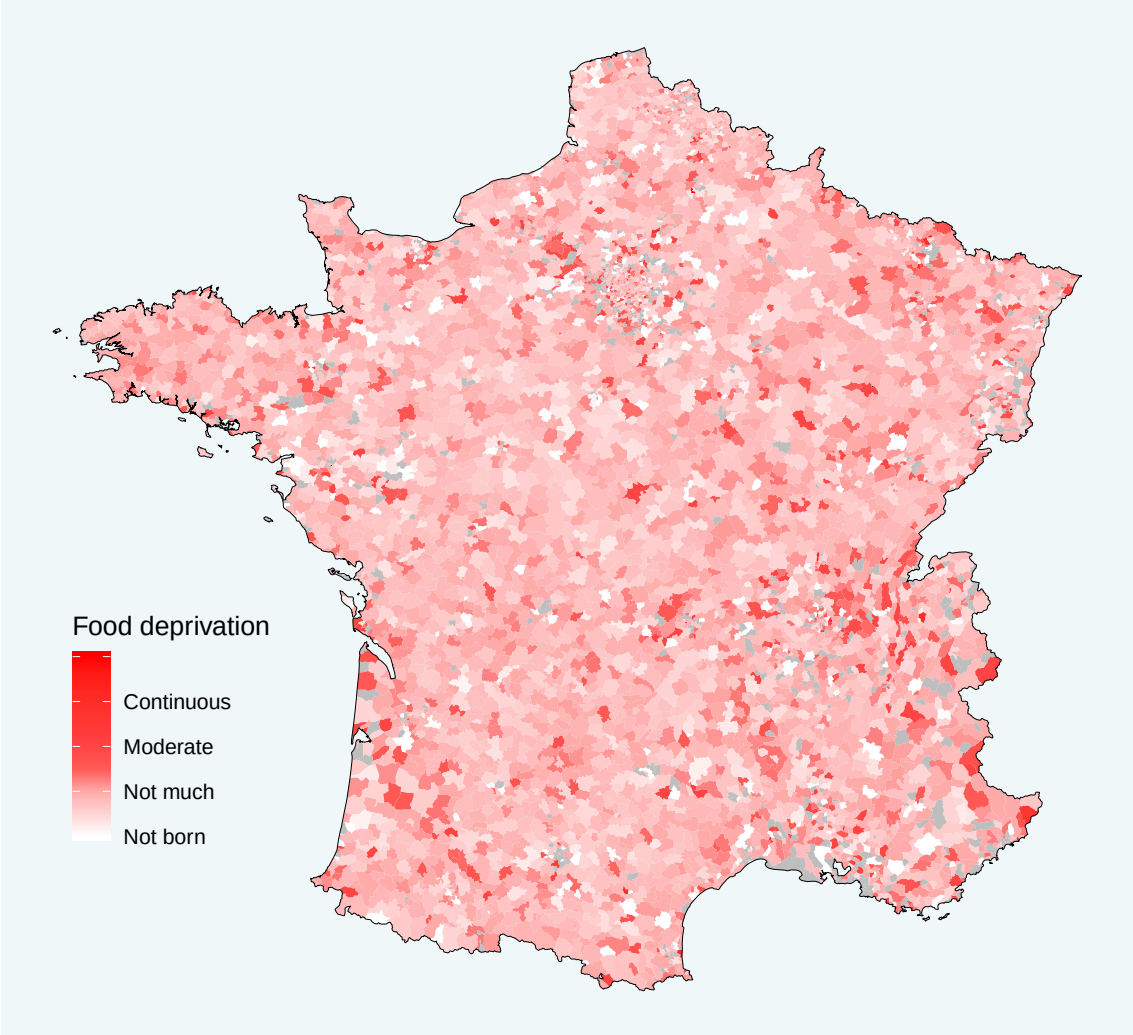

Figure 1: Post-code area average of self-reported level of food deprivation. *Source:* Own work.

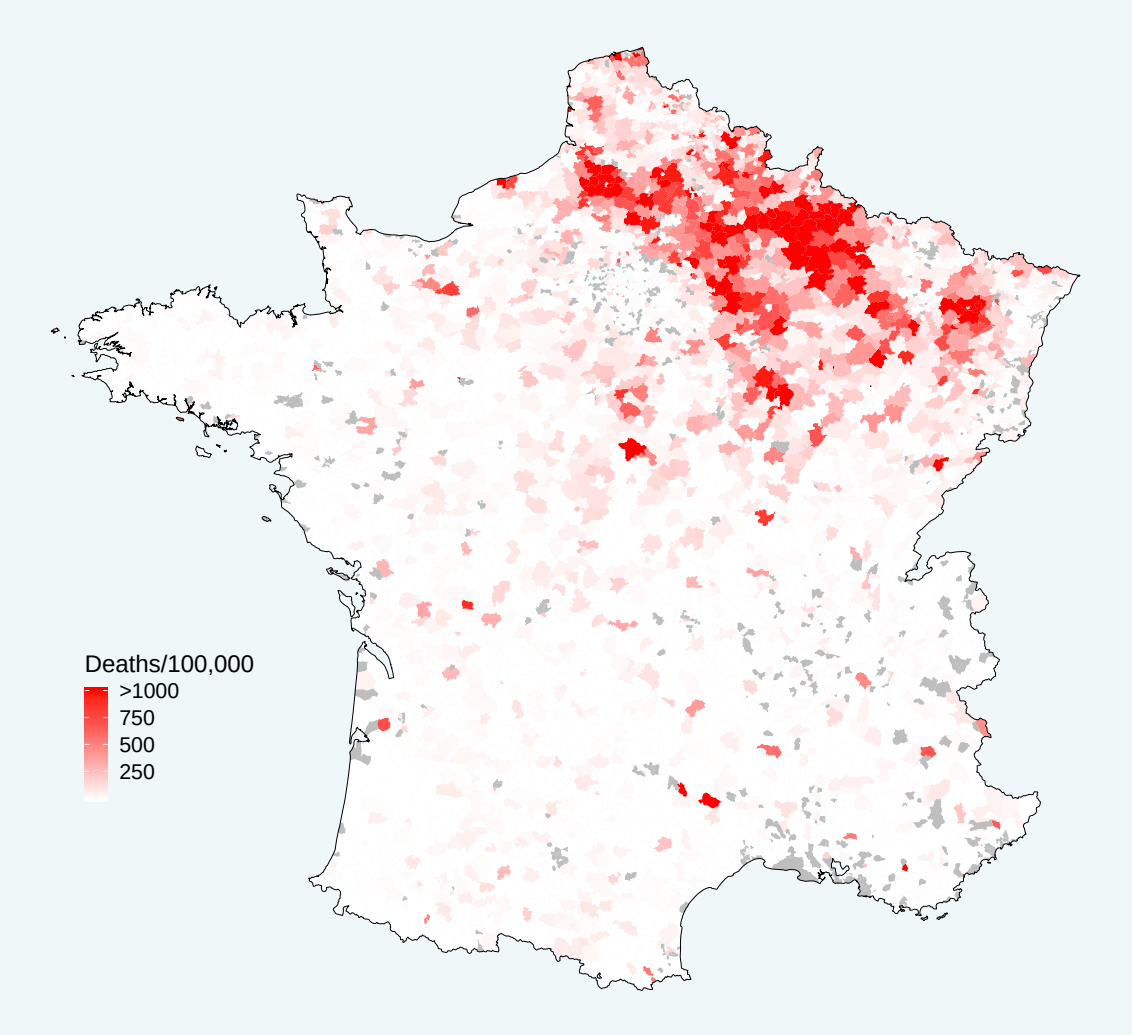

Figure 2: Number of French military deaths per 100,000 at the postcode level, 1940. *Source:* Own work.

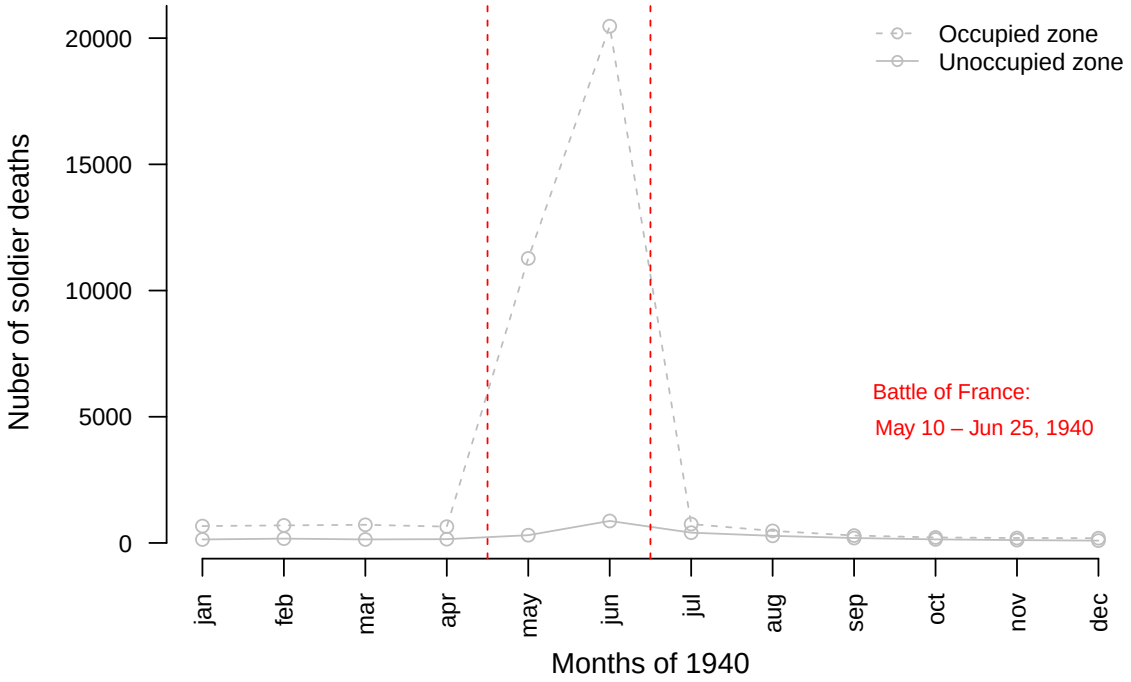

Figure 3: Time-line of the number of deaths of French soldiers, North occupied zones versus South unoccupied zone. *Source:* Own work.

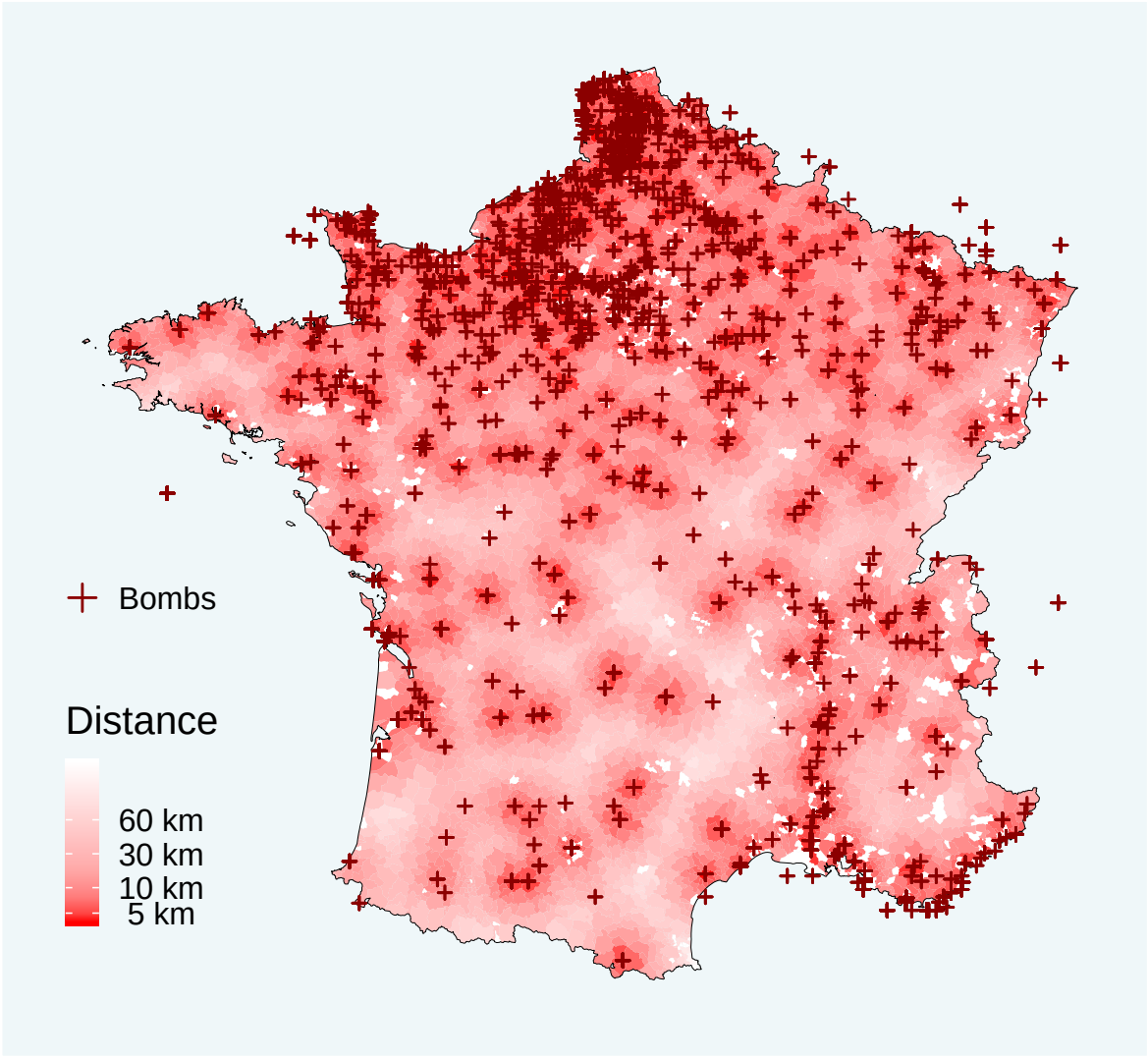

Figure 4: Allied bombing. *Source:* Own work.

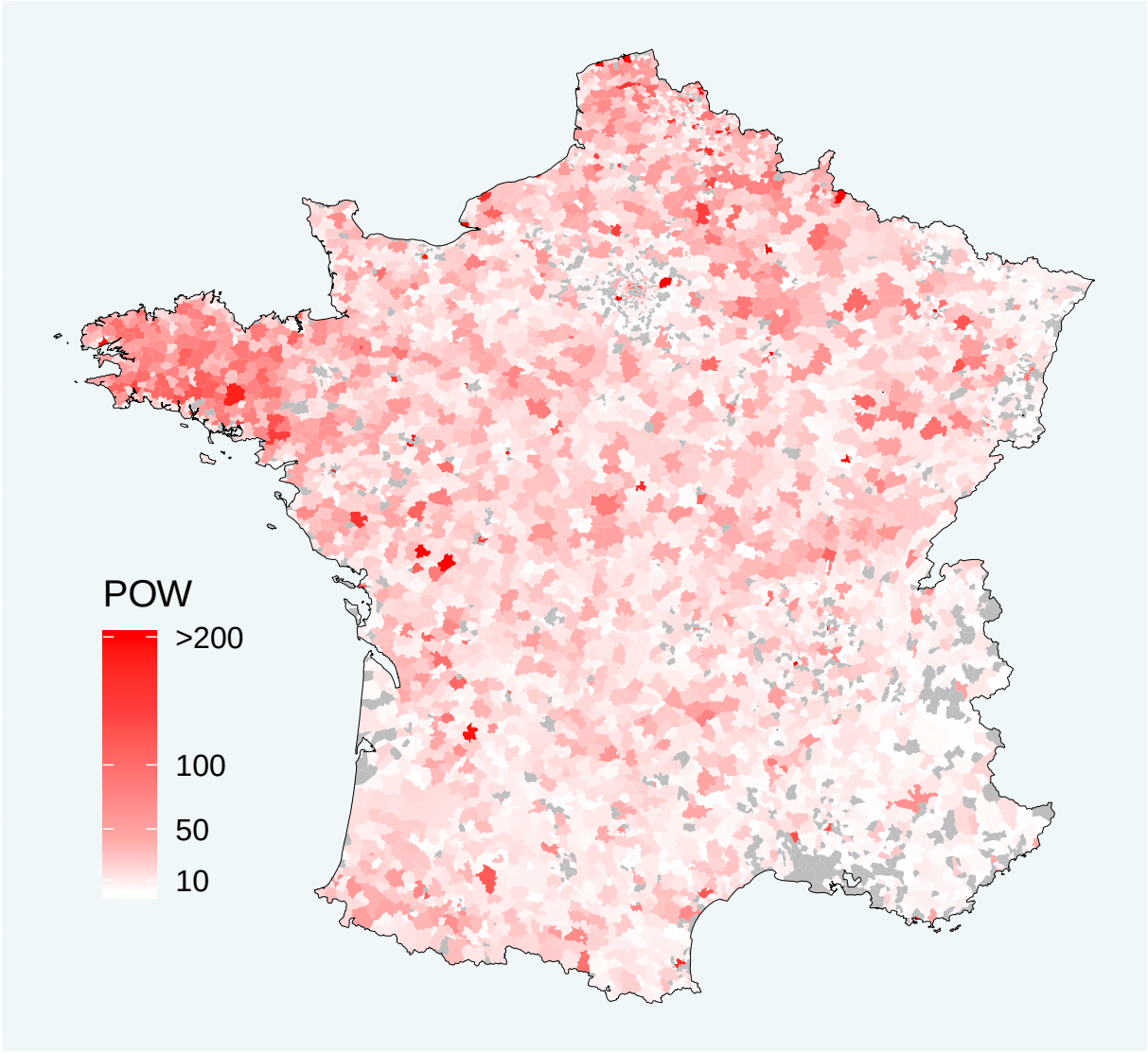

Figure 5: Origin of prisoners of war. *Source:* Own work.

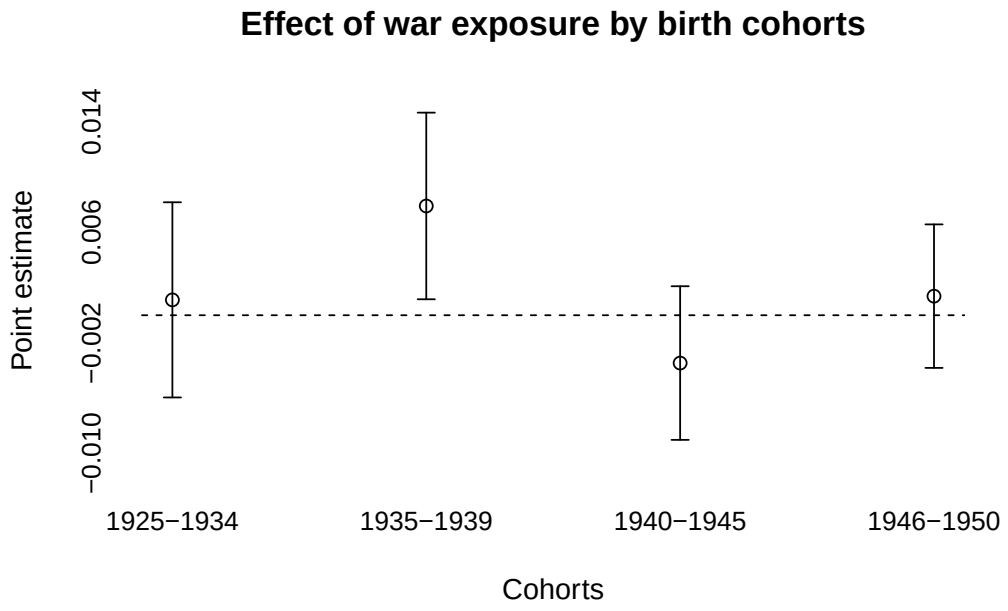

Figure 6: Effect of exposure to war as measured by the number of French military casualties in the individual's postcode area of birth on the probability of suffering from any of the reported health conditions in adulthood. Point estimates by birth cohort. Brackets show 95% confidence intervals.

Table 5: Effect of early-life exposure to WWII as measured by above average number of POW on adult health

	Any health condition	
	(1)	(2)
Exposure x Born 1925-34	0.0001	
	(0.0234)	
Exposure x Born 1935-39	0.0540**	0.0382*
	(0.0220)	(0.0229)
Exposure x Born 1940-45	0.0129	
	(0.0187)	
Exposure x Born 1946-50	0.0256	
	(0.0169)	
Exposure		0.0159
		(0.0127)
Lives with partner	−0.0064	−0.0063
	(0.0097)	(0.0097)
Higher education	−0.0428***	−0.0429***
	(0.0076)	(0.0076)
Born preterm	0.0510***	0.0511***
	(0.0188)	(0.0188)
Mother's age at birth	−0.0006	−0.0006
	(0.0009)	(0.0009)
Father's age at birth	−0.0002	−0.0002
	(0.0008)	(0.0008)
Nb. of siblings	−0.0040**	−0.0040**
	(0.0018)	(0.0018)
Physically stressful job	0.0097	0.0098
	(0.0083)	(0.0083)
Mentally stressful job	0.0165	0.0164
	(0.0101)	(0.0101)
Lived on a farm	0.0004	0.0004
	(0.0083)	(0.0083)
Lives in deprived area	0.0073**	0.0074**
	(0.0037)	(0.0037)
Hunger (scale)	0.0389***	0.0385***
	(0.0086)	(0.0085)
Bombs	−0.0037	−0.0038
	(0.0093)	(0.0093)
Num. obs.	22662	22662
Log Likelihood	-15191.1931	-15191.6729

$^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

Exposure is defined as above average number of prisoners of war in the woman's post code area of birth. All models include birth-year and department fixed effects and dummies for the woman's and her father's socioprofessional category. The coefficients show average marginal effects. Standard errors are clustered at the department level.

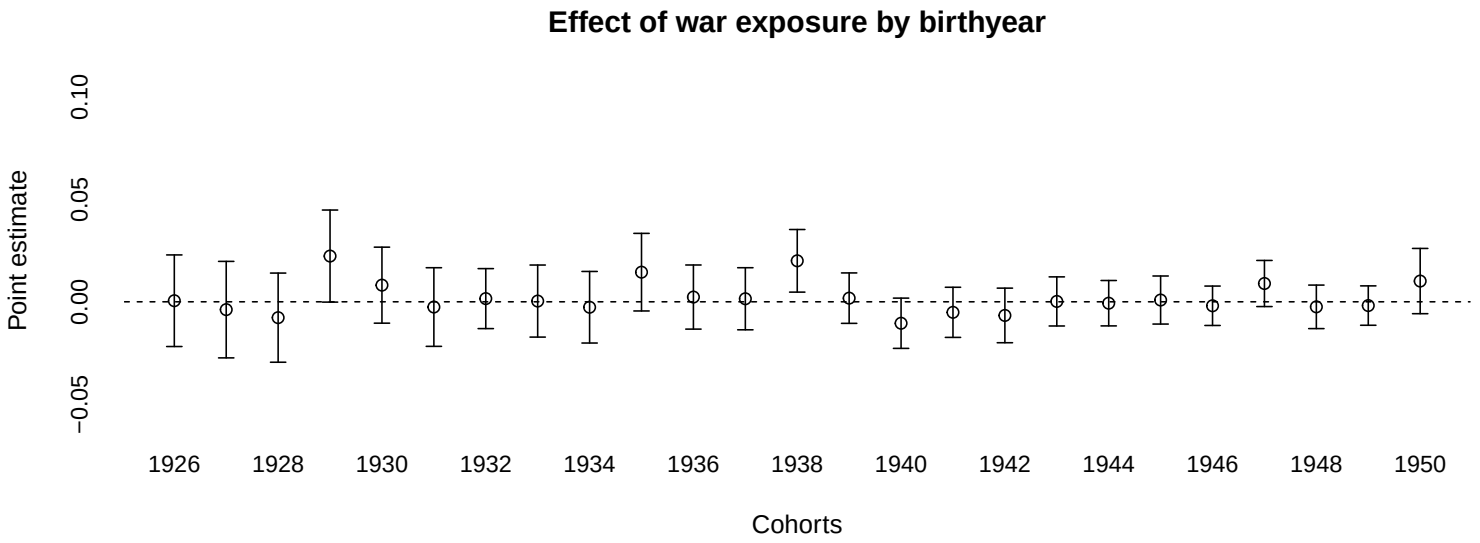

Figure 7: Effect of exposure to war as measured by the number of French military casualties in the individual's postcode area of birth on the probability of suffering from any of the reported health conditions in adulthood. Point estimates by birth-year. Brackets show 95% confidence intervals.