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Why People Born During World War II are Healthier

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Why people born during World War II are healthier¹

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Abstract

War leads civilians to suffer. This can take extreme forms, such as during periods of intense violence or famines. But also outside of such episodes, civilians' lives during wars can be harsh, as they suffer from poorer nutritional situations, stress, recessions, and sub optimally functioning health care systems. The more extreme types of suffering are proven to lead to a worse health among those prenatally exposed to them. But long-run effects of prenatal exposure to the latter circumstances have thus far largely been unexplored, even though in many wars more pregnant women are exposed to these "everyday" circumstances than to the extreme circumstances. We study the general, populationwide effects of prenatal World War II exposure in three occupied countries: France, Belgium and The Netherlands, without zooming in on specific severe episodes such as the Dutch famine. We show that - contrary to expectations - prenatal exposure to WWII does not lead to poorer health among the older population. We even find strong indications for a better health, especially among exposed females, but demonstrate that this is due to selective mortality among the war cohorts during infanthood and to selective fertility during WWII. As these selection effects are likely to be stronger during more extreme historical circumstances than the ones studied here, previous research on longterm effects of such prenatal exposures may have underestimated effects. Long-run negative population-wide health effects from prenatal WWII exposure in France, Belgium and The Netherlands are absent or at most very small.

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1. Introduction

World War II (WWII) had enormous effects on the health of the European population: battles, bombings raids, famines and persecutions exacted a giant toll on countless people. Yet during any given moment in time, most of the civilians — and particularly those in occupied countries such as France, Belgium, and The Netherlands — in Europe experienced neither extreme levels of violence and destruction, nor famines. Nevertheless, they still went through circumstances that were clearly adverse. Food was rationed, the economy experienced a sharp recession, individuals' stress levels were often high and health care systems functioned sub optimally. A similar pattern in which most civilians at any specific moment in time do suffer, but do not directly experience violence or famine, applies to a substantial share of more recent and contemporary wars, too.

We know from a growing body of research that prenatal exposure to adverse circumstances can affect human capital and health outcomes of the prenatally exposed throughout their entire lives (Almond & Currie, 2011). This has been demonstrated for extreme circumstances including famines (Chen & Zhou, 2007; Jürges, 2013; Roseboom et al., 2000) and large-scale destruction through warbombings (Akbulut-Yuksel, 2014), as well as for less extreme circumstances such as fasting (Almond & Mazumder, 2011; Van Ewijk, 2011). This means that inferior conditions such as those resulting from wars might negatively affect large parts of a population until many decades later.

This paper investigates the effects of prenatal exposure to the WWII-conditions that were experienced by the majority of women who were pregnant during the war in the occupied countries Belgium, France, and The Netherlands. We do not search for specific groups for which long-term effects exist. It has been proven by now that famines during WWII such as those in Greece, Leningrad and the western part of The Netherlands during the final war months affected the health of the prenatally exposed (Barber & Dzeniskevich, 2005; Neelsen & Stratmann, 2011; Scholte et al., 2015). Our focus, instead, is on the less extreme, but more common circumstances that have thus far largely been neglected. We investigate whether the later-life health of the cohorts born during the war was negatively affected by their exposures. Within these cohorts, we explicitly do not try to isolate specific

subgroups for which the WWII-exposure may or may not have been more extreme. Our focus is on health at older ages, and we study this using three waves of the Survey of Health, Ageing and Retirement in Europe (SHARE).

In this way, we aim to show whether and how the health of current generations of elderly Europeans is still affected by their prenatal experience of WWII. Moreover, by focusing on less extreme (but still clearly adverse) conditions than those usually studied in the fetal origins literature, we aim to find out more about the long-term effects of more commonly experienced exposures. In contemporary wars, the general audience's focus is mostly on those directly affected by violence and famines. But at any specific moment, most people – and of particular relevance here: most expectant women – do not experience such extreme conditions. Therefore, in the long run, a much larger group of people will have been exposed to milder forms of warlike circumstances. We know little about whether and how their later-life outcomes are impaired.

One other contribution of our paper is that it is, to our knowledge, the first one to carefully investigate whether and how selective fertility and selective mortality may bias estimates on the long-run effects of prenatal exposures to historical events. Selective mortality refers to the fact that some of the weakest individuals may not survive long enough to make it into a study's sample. This may either happen directly, because mortality goes up during the historical event (in this case WWII), or indirectly, because the prenatal exposure leads to increased mortality rates at older ages. Selective fertility may take place during historical events that last a longer time, as conceptions are postponed or become impossible among sub groups of women.

We show that prenatal exposure to WWII initially seems to lead to unexpected positive long-term health effects, which however is an artifact of selective mortality and selective fertility. Once these two are accounted for, sizeable population-wide long-run adverse health effects can be excluded. Selective mortality and selective fertility are likely to have led many previous studies on long-run effects of prenatal exposures to historical events to under estimate effects.

2. Previous literature

Most literature on the long-term effects of early-life exposure to warlike circumstances focused on famines during WWII. Prenatal famine exposure has been linked to a wide range of health impairments among adults (see Lumey et al., 2011 for a review). Most of this literature comes from the field of epidemiology, although a few studies by economists have looked at prenatal famine exposures during WWII, demonstrating negative effects on education and labor market outcomes (Neelsen & Stratmann, 2011; Scholte et al., 2015). Akbulut-Yuksel (2014) shows that prenatal exposure to the intense aerial bombardments that destroyed almost half of the housing stock in German cities led to a wide range of health problems in adulthood. Lee (2014) finds that people who had been in utero during the Korean War have worse human capital and health outcomes at age 40-50. In contrast to our paper, Lee explicitly focuses on the most intense types of war exposures.

Other researchers investigated the long-run effects of exposure to WWII during childhood. Ichino & Winter-Ebmer (2004) and Akbulut-Yuksel (2017) show that WWII-exposure on school-aged German children led them to receive less education and have lower incomes later in life. This is due to the reduced access to schools, mainly resulting from destruction by bombardments. Atella et al. (2017) study childhood exposure to high stress events (massacres and intense fighting) and famines in Italy in 1943 and demonstrate negative health impacts among the elderly. Van den Berg et al. (2015) demonstrate long-run health effects of famines experienced during childhood. Kesternich et al. (2015) investigate how childhood exposure to the German famine that started shortly after the war affects behavioral outcomes later in life. Some other papers looked at the effects of early-life exposure to wars in developing countries, particularly Africa. Most of these papers focused on exposure to war during childhood and on intense conflict and combat exposures (Akresh, Bhalotra et al., 2012; Akresh, Lucchetti et al., 2012; Bundervoet et al., 2009).

Kesternich et al. (2014) show that exposure to WWII in childhood increases the likelihood of suffering from diabetes, depression, or a poor self-rated health among elderly people. Their analyses include people from thirteen different countries, several of which saw extreme rates of civilian

casualties (e.g. Germany, Poland), dispossessions (e.g. Czech Republic) or famines (e.g. Greece, postwar Germany). (Note that up to 2.8% of the Greek population died during its famine (Neelsen & Strattman, 2011), compared to only 0.2% for The Netherlands, which is due to the localized nature and short period of the Dutch famine.) Their results are therefore mainly due to extreme circumstances. Like them, we also focus on outcomes at advanced ages and use data from the SHARE-survey (although they use wave 3, whereas we use waves 1, 2 and 4). But unlike their paper, we focus on the non-extreme circumstances that were experienced by the majority of the pregnant women in the occupied Western-European countries. And we focus on prenatal exposure rather than exposure during childhood.

3. Background and pathways for effects

Our focus is on three countries which experienced roughly comparable circumstances during WWII: Belgium, France and The Netherlands. Each of these countries was occupied by Germany in spring 1940 and (depending on region within the country) was liberated between June 1944 and May 1945. As described below, the civilian population in each country was exposed to several types of adverse circumstances from which long-run effects could be expected.

Medical theory predicts strong long-term effects for exposures *in utero* and relatively smaller effects for exposures during later periods in childhood. During the fetal stage, the human body goes through critical growth periods during which organs are formed or experience rapid growth. During these periods, the body is highly vulnerable to experiencing adverse circumstances (Barker, 1997). And adverse health effects may only reach their peak when the prenatally exposed person ages (Barker, 2002).

If we exclude famines and direct exposure to extreme violence and destruction, there are four channels through which prenatal exposure to warlike circumstances in the three occupied countries we study is likely to cause population-wide long-term health effects. First, even outside of famines, the nutritional situation was clearly sub optimal. In each of the three countries, food was rationed

throughout the war. The average daily ration in France in 1941-1944 was limited to 1180 kcal (Mouré, 2010). This is considerably less than the recommended 2,000 kcal for non-pregnant women of. For pregnant women, generally higher amounts are recommended. For Belgium and The Netherlands, the average daily rations were 1400 and 1800 kcal, respectively. Furthermore, the quality and variety of nutrition decreased as several types of food such as meat, dairy, fat, eggs and bread became rationed (see Egle, 1943). Malnutrition in France led to anemia, vitamin deficiencies and diseases (Mouré, 2010). Prenatal exposure to poor nutrition can hamper fetal growth and may lead to damage to fetal organs. Prenatal malnutrition has been shown to lead to a wide range of diseases — most of which only show up at older ages, including coronary heart disease, type-2 diabetes and hypertension (Roseboom et al., 2011) and it has also been shown to lead to higher mortality rates among the elderly (Lindeboom et al., 2010). Moreover, it can negatively affect the cognitive performance of the offspring (De Rooij et al., 2010).

Second, stress levels were likely to be elevated during WWII as a result of occupation and repression, as well as the fear of air raids and combat. War-related stress was strong enough to lead to long-lasting mental health problems including post-traumatic stress disorder among many civilians (Bramsen & Van der Ploeg, 1999). Experiencing stress leads to increased levels of corticotropin-releasing hormone (CRH). High CRH-levels prepare the fetus for a likely preterm birth. Maturation of the tissue is sped-up, while fetal growth is reduced (Hermann et al., 2001; Hobel & Culhane, 2003). Prenatal stress exposure increases the responsiveness of the hypothalamic-pituitary-adrenal axis, which controls the body's hormonal reactions to stress. As a result, the prenatally exposed become more likely to develop hypertension later in life (Seckl & Holmes, 2007). Prenatal exposure moreover affects neurological development and can therefore lead to cognitive, behavioral and emotional problems, and even to schizophrenia (Cotter & Pariante, 2002; Van den Bergh et al., 2005). Van Os & Selten (1998) show that schizophrenia was more common among those who were in utero during the 1940 German invasion in The Netherlands, which they attribute to stress.

Third, the occupied countries experienced a severe economic downturn during the war years. GDP in 1944 had contracted by 52%, 49% and 20% compared to 1939 for France, The Netherlands and Belgium, respectively (Maddison, 2011). Studies into early life effects of economic crises in the late 19th and early 20th centuries found negative effects on later-life health outcomes (Banerjee et al., 2010; Yeung et al., 2015). But studies on later crises found no effects (Cutler et al., 2007; Lindeboom & Van Ewijk, 2013) or even positive effects (Dehejia & Lleras-Muney, 2004). Perhaps malnutrition, stress and epidemics were the most relevant effects of economic crises that took place longer ago, whereas nowadays, parental health behavior improves in recessions as the opportunity costs of health are reduced (Dehejia & Lleras-Muney, 2004). It is hard to say which process will dominate for WWII, especially because the channels through which economic crises affect prenatal health are closely intertwined with the previously discussed channels of nutrition and stress.

Fourth, health care systems tended to function sub-optimally during WWII. Hospital staffing may have been affected by persecutions. Supply lines may have been severed and drugs, medical supplies and staff diverted to the occupying country. At the same time, demand for care may have been higher due to the war circumstances, putting extra strain on the health care system.

4. Data and Methodology

Our main data source is the Survey of Health, Ageing and Retirement in Europe (SHARE). SHARE is a longitudinal survey study among individuals aged 50 and older. The first wave of SHARE took place in 2004/05 and included a nationally representative sample from eleven European countries. Subsequent SHARE waves took place in 2006/07, 2008/09 and 2011/12. Wave 3 (SHARELIFE) had a different setup and goal than the other waves and the variables that were collected differed from the other waves. We therefore run our analyses on data from waves 1, 2 and 4 and only utilize wave 3 data to obtain some supplemental characteristics of respondents.

Our sample includes Belgium, France and The Netherlands, which were occupied during WWII, as well as Sweden and Switzerland, which are geographically and culturally close to our three exposed

countries but which remained neutral throughout the war. In some analyses, we will utilize the neutral countries in a difference-in-differences approach.

To construct our sample, we start with the sample of SHARE 1 and SHARE 2 respondents who were age 50 or older. Now our youngest respondents are born in 1956. Respondents who are new in wave 4 are only added if they were born no later than 1956, to avoid ending up with low numbers of people from cohorts that were born long after our exposed group. The exposed group consists of people for whom at least part of their mother's pregnancy fell during WWII. They are born between May 1940 and January 1946 (assuming a 9-month gestational period) and are aged 58-65 in wave 1 and 65-71 in wave 4. This means that most are in their final stages of their working life or shortly after retirement.

Our control group consists of people born before the war or conceived after the war. The latter group includes people born no later than 1956 as described. In our main analyses, we limit the prewar born group to people conceived no earlier than 1934. These people are no older than 70 in our wave 1 sample, which makes them well comparable to our exposed group.

Including older cohorts into the control group may have a few disadvantages: first, regression results may become more sensitive to the choice of the right functional form for the effect of birth cohort (or age) on health outcomes. Second, they might constitute an increasingly selective sample due to mortality and due to the fact that SHARE does not include people who are institutionalized. Third, including older people would lead to the inclusion of people who had experienced the most extreme phases of the Great Depression in utero, which might have an autonomous effect on later-life outcomes (Banerjee et al., 2010; Yeung et al., 2015).

We show that our results do not change when we include all elderly respondents available in the data, without a lower bound limit on the birth year. Moreover, using other ranges of birth years (e.g. excluding all people born before WWII, who would have experienced WWII during their childhood) does not alter our conclusions either.

Besides the SHARE data, in some analyses we use data from the Human Mortality Database (HMD). The HMD contains birth and mortality data by country, age, birth year and sex for each of the countries in our sample. The HMD-data allow us to analyze whether the circumstances in the three occupied countries indeed deteriorated substantially during the war years. Figure 1 shows the life expectancy at birth for these countries for females.² Females are less likely to die in combat than men and moreover, females are the relevant group when we study exposures during pregnancy. We run a regression separately for each country of female life expectancy at birth on birth year (which runs from 1934-56)³ and compare the actual data to the long-term trend. The strongest drop occurred in France in 1944, where female life expectancy was 12.1 years below trend. But also in e.g. 1943, French life expectancy was 7.1 years lower than expected. In each of the three countries, life expectancy dropped below its long-term trend in each of the war years – and not only in the years during which the invasion and liberation campaigns took place and which therefore saw most combat. In contrast, for the neutral countries Sweden and Switzerland, no substantial drops in life expectancy during WWII show up. These results confirm that health circumstances for civilians indeed worsened considerably throughout WWII in the three occupied countries.

In our main analyses of later-life health outcomes on prenatal exposure, we focus on five dependent variables that measure health at old age across several important dimensions. The first variable is cognitive performance. The interviewer read a list of ten words to the respondent and respondents immediately had to repeat as many of the words as they were able to remember. A few minutes later, during which other cognitive performance tasks were completed, respondents again had to repeat as many words as possible from the same list. We measure cognitive performance as the sum of both tests: learning and delayed recall. Second, self-reported health is measured using a binary variable indicating a fair or poor self-reported health. Self-reported health is known to be a good

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² Life expectancy at birth indicates how many years a newborn child would live if the age-specific mortality rates at the moment of its birth would stay constant throughout its life. Age-specific mortality rates indicate, for each age k, the probability that a k-year old person will die in a one-year time interval.

³ Lindeboom & Van Ewijk (2015) show that the pattern of results is robust against taking a broader range of cohorts (1920-1965).

predictor of mortality among the elderly (Idler & Benyamini, 1997). Third, we employ a binary variable indicating whether the respondent was hospitalized in the last 12 months. Fourth, we take respondents' numbers of visits to a general physician (GP) in the last 12 months. Fifth, physical limitations are defined as having one or more limitations with activities of daily living (ADL) or instrumental activities of daily living (IADL).⁴

Our main analyses follow the equation:

(1) $Health_{iwc} = \beta Z_{iwc} + \varphi_1 WWII_{iwc} + \varphi_2 D45_4 6_{iwc} + \mu_w + \theta_c + \xi_i + \varepsilon_{iwc}$

in which $Health_{iwc}$ indicates the health of person i from country c in wave w. θ_c are country of birth fixed effects for the three included countries and ξ_i are individual random effects. To deal with clustering of the error terms ε_{iwc} , we use panel robust standard errors. In line with most of the literature on long run effects we run our regressions separately for males and females. WWII is a dummy variable indicating whether a respondent was prenatally exposed to the war. It takes on the value 1 for people who were born no earlier than May '40 and conceived no later than April '45, and the value 0 otherwise. We additionally include a dummy variable $D45_46$ for people conceived between May 1945 and Dec. 1946 (i.e. born between Feb. '46 and Sept. '47). The reason for this is that the post-war period did not immediately see a return to pre-war conditions: food was still rationed and there were considerable flows of refugees. As our exposure variable is determined based on country, year and month of birth, it is important that none of our three countries saw major border changes after the war. This means that e.g. "born in Belgium" refers to the same thing, irrespective of whether the respondent was born in 1938, 1943 or 1950. Note that in The Netherlands, a famine occurred in the winter of 1944/45. This famine (unlike e.g. the Greek famine) was localized in only a

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⁴ ADL include dressing, including putting on shoes and socks; walking across a room; bathing or showering; eating, such as cutting up your food; getting in and out of bed; using the toilet, including getting up or down. IADL include using a map to figure out how to get around in a strange place; preparing a hot meal; shopping for groceries; making telephone calls; taking medications; doing work around the house or garden; managing money, such as paying bills and keeping track of expenses.

⁵ We do not know month of conception but assume a 9-month gestation length for each respondent.

⁶ Persons whose reported birth year, birth month or country of birth changed between waves or was missing were excluded from analysis.

part of the country (the West) and was relatively short: ca. 0.5 year. The share of the total population that perished was relatively low: during the Greek famine, this share was up to 14 times higher. Because we worry that our estimates might be driven by the Dutch famine, we run analyses in which we estimate effects separately by country and by country*birth cohort.

Z measures trends in health: health depends on age, birth year and year of measurement. We need to filter this out in order to separate effects of WWII-exposure from non-war related trends in health. In our main specification, we capture these effects using year of birth as our variable Z, as well as wave fixed effects μ_w . In alternative specifications, in addition to the wave fixed effects, we use age; age and age squared; or age and year of birth as our measures of trends in health over time. We prefer the specification using only year of birth, since the latter two specifications may lead to overfitting. Results in all three alternative specifications remain robust.

In the Results section, we first robustly demonstrate that there are no negative long-term effects, and seemingly even positive effects of prenatal exposure to WWII. After that, we turn to selective mortality and selective fertility as potential explanations for this pattern and to their respective importance.

5. Results

5.1 Main results

Table 1 shows our main results from the regressions following equation (1) of elderly people's health on prenatal WWII exposure. Columns (1) (for females) and (5) (for males) show results for our main sample, which consist of people conceived no earlier than 1934 and born no later than 1956. Columns (2) and (6) show similar results for the sample which includes respondents born in earlier years as well. Contrary to expectations, we do not find that those older people who had prenatally experienced WWII

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⁷ Generally, due to collinearity, one can only control for two out of the three variables age, time of birth, and time of measurement. We are, however, able to include age, year of birth, as well as wave dummies in one regression since age varies a bit with birth year and data collection for each SHARE wave took place in more than one calendar year.

have worse health. For females, all point estimates even suggest that prenatal exposure leads to a *better* health. For cognitive performance, number of GP visits and whether the respondent had any physical limitations, these estimates get significant, and for the sample including elderly born conceived 1934, this also applies for the variable "fair or poor self-reported health". For males, all but one point estimate suggest positive health effects as well, although none of these estimates gets significant in the main sample. In the sample including elderly conceived before 1934, three out of five estimates get significant.

It might be possible that our results of no worse health (and potentially even better health) among the exposed are biased due to specific characteristics of the post-war cohorts. During the liberation time, there might have been selective fertility. Our dummy indicating conceptions between May 1945 and December 1946, however, already dealt with this possibility. But it is also conceivable that the included 1950s cohorts are systematically different, since these people were part of the baby boom generation and moreover are relatively young – and therefore healthy – compared to the rest of the sample in a way that our model might potentially not sufficiently capture. We therefore also run regressions in which we exclude all those born after 1950. As columns (3) and (7) of Table 1 show, this does not change our results. (Note that we also ran regressions in which we excluded all those born after WWII. This considerably limits our sample size, increases our standard errors and leaves us with no control group born after the exposure period. Like our other estimates, these results show no adverse health effects of prenatal exposure.)

Another possibility that we need to investigate is that our results may be driven by effects on older cohorts. Everyone in our sample who was born before the war was exposed to WWII during childhood. Kesternich et al. (2014) and Havari & Peracchi (2017) demonstrate that childhood exposure to WWII leads to a wide range of negative health effects among elderly people. However, we believe that the absence of negative health effects of prenatal exposure that we find is not an artifact of stronger negative health effects on older cohorts. There are four reasons for this: first, we study another type of exposure than these previous papers. Their results are mainly driven by exposure to

extreme circumstances such as wide-scale destruction and famines as they also include countries such as Germany, Greece and Poland. Second, most of the body's critical growth phases occur during the fetal stage, so that biologically, one would expect stronger effects of prenatal than of postnatal exposure.⁸ Third, Figure 2 shows the average cognitive performance (the variable for which we most consistently found positive estimates) for females across cohorts. There was no dip from the long-term trend among the pre-war years. For other variables, the pattern is the same. Fourth, columns (4) and (8) of Table 1 show regressions in which people born before May 1940 have been excluded. Still no consistent pattern of a better than expected health among the prenatally exposed shows up.

Finally, we also run the regressions from Table 1 using alternative specifications in which we replace the covariate "year of birth" with age; age and age squared; or age and year of birth as our measures of trends in health over time. All regressions additionally still include wave dummies. All results remain robust. Appendix Table 1 shows the results for the 1934-56 sample.

5.2 Difference-in-differences analysis

We next run the same regressions in difference-in-differences form following equation (2).

(2)
$$Health_{iwc} = \beta Z_{iwc} + \varphi_1 WW2_{iwc} + \varphi_2 D45_46_{iwc} +$$

$$\varphi_3 occupied_c * WWII_{iwc} + \varphi_4 occupied_c * D45_46_{iwc} + \mu_w + \theta_c + \xi_i + \varepsilon_{iwc}$$

As control countries, we take Sweden and Switzerland. These are the only two neutral countries included in the SHARE waves and therefore did not experience the same wartime exposures as Belgium, France and The Netherlands. Moreover, both countries provide a good counterfactual to the occupied countries, since they are geographically and culturally close and generally experienced the same pre- and post-war trends (a declining pre-war fertility trend and a post-war baby boom). Due to their closeness to the warring countries, it is possible that some of the adverse exposures discussed for the occupied countries also occurred in the neutral countries, albeit in a weaker form. Food was

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⁸ In addition, certain measures were taken in an attempt to keep damage to children limited. These were founded in knowledge from the First World War on the damaging effects that malnutrition had on children. Eglé (1943) in her contemporary review of rationing during WWII for example lists that in The Netherlands, full-fat milk was only made available to children under 14. And in France, families with many children received additional rations (above those that were calculated per capita). Similar measures may also have somewhat alleviated the situation for pregnant and lactating women, who also received higher rations.

rationed in both Sweden and Switzerland (Egle, 1943) and there may have been an additional strain on the health care system due to refugees and severed (medical) supply lines. Therefore, the coefficient of interest in the difference-in-differences regression (φ_3) may be attenuated. We therefore prefer the estimates from equation (1). The results in Table 2 confirm that there are no negative health effects of prenatal WWII-exposure (see panel A). Most coefficients for females are no longer significant, but this seems due to the fact that most point estimates for the neutral countries (panel B) suggest a better than expected health among the war cohorts for these countries as well.

5.3 Heterogeneity in effects by country and birth cohort

In the subsequent step, we return to equation (1), but now investigate heterogeneity in effects between countries by interacting the exposure dummy (as well as the dummy for being conceived between May 1945 and December 1946) with a country dummy. For our main sample (1934-56), Table 3 shows that for none of the three occupied countries, there are negative health effects of prenatal WWII-exposure. The pattern of mostly positive point estimates that, in case of females, are in many cases significant, shows up for each of the three countries.

Since the type of war exposure is likely to differ between the war years (e.g. due to the invasions in 1940 and liberation campaigns in 1944/45), we next define the exposure dummy separately for each war year. Recall that the exposed were born between May 1940 and January 1946. We split this group up using six dummy variables: D1940-D1944 are dummy variables for being born between May of the respective year and April of the following year. D1945 is a dummy variable for being born between May 1945 and January 1946. We again include a dummy variable for people born between February 1946 and September 1947 (who were calculated to have been conceived between May 1945 and December 1946). The results are reported in Table 4. For none of the war years we find consistent evidence for negative effects of prenatal exposure. For females, we find better cognitive

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⁹ We find the same when we use the alternative sub samples: all respondents born no later than 1956; 1934-50 and 1940-56. The same applies to the other results in this section reported below.

scores, fewer limitations and fewer GP visits for people exposed throughout the various war years. For males, a few significant estimates do show up, but there is no consistent pattern in this, so that this is likely due to multiple testing and chance: there is no clear evidence for decreased health resulting from prenatal WWII-exposure.

Figure 3 shows the results from regressions in which the exposure variable is defined separately by birth cohort*country combination. The figure shows the results for females. The results for males are similar (see Appendix Figure 1). A priori, we expected that originally hypothesized adverse health effects might be driven by the Dutch 1944-45 famine. However, even for the Netherlands for these years, no adverse health effects show up. It should be noted that the Dutch famine only affected part of the country (the western part) and that our sample includes people from all parts of the country, which may explain the difference with previous literature. Moreover, the famine lasted from December 1944-April 1945, so that those who had been prenatally exposed to the Dutch famine are spread between the group captured by our 1944 dummy and the group captured by our 1945 dummy. And both these groups also include people who were not in utero during the time period of the Dutch famine. For Belgium and France as well, no patterns of adverse health effects among the exposed show up for any birth cohort. Estimates indicating significant health improvements show up for exposures in various years, with some of the strongest effects occurring for France in 1943. This was a year that was militarily among the quietest among the war years.

6. Selective mortality and selective fertility

The important question to answer now is why – contrary to expectations – no adverse health effects – and seemingly even some positive health effects occur. Two likely explanations are that this is the result from either selective mortality or selective fertility.

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¹⁰ Note that the waves of SHARE that we use in our regressions do not include information on region of birth. Moreover, it is not our goal to prove that effects of famines exist: this has been proven convincingly by many previous studies.

6.1 Selective mortality

Perhaps prenatal WWII-exposure did lead to adverse health effects but this is not visible anymore at older ages, as the least healthy people have already died before that moment as a result of their exposure to wartime conditions. If this selective mortality solely occurs during infancy, then the mortality itself is not a result of the long-run, fetal programming type of health effects we are studying here. If, however, mortality rates among those prenatally exposed to WWII are consistently higher throughout all ages, then this is an indication that prenatal exposure does lead to long-run effects. To investigate the selective mortality explanation, we use aggregate data from the Human Mortality Database to show how the probability of reaching one's birthday in 2004 (the year in which the first SHARE-wave started) develops across cohorts. We run the following regression separately by country and sex:

(3) ShareSurvive_y = $\alpha + \beta_1 Birthyear_y + \beta_2 Birthyear_y^2 + \varphi_1 D40_45_y + \varphi_2 D46_47_y + \varepsilon_y$ in which ShareSurvive_y gives the share of birth cohort y who survived till their birthday in the year 2004. The analysis includes the birth cohorts 1934-1956. $D40_45$ is a dummy for the 1940-45 birth cohorts (note that unlike in the analyses using SHARE data, we can only utilize year, and not month of birth).¹¹ In subsequent analyses, $D40_45$ is replaced by a set of dummies per birth cohort ($\sum_{j=1940}^{1945} \varphi_j Dcohort_j$). $D46_47$ is a dummy variable used to "dummy out" the cohorts born in 1946 and 1947, which, as described before, might form a sub optimal comparison group.

Figure 4 shows that for women born in the occupied countries, the probability of surviving till 2004 is lower than one would expect based on pre- and post-war trends. Belgian females born during the war have a 1.6 percentage point (pp) lower chance of living till their birthday in 2004 (ranging from 0.8 pp for those born in 1943 to 2.9 pp for the 1945 birth cohort). For France, the effect is 1.8 pp (ranging from 0.6 pp in 1941 to 3.7 pp in 1945) and for The Netherlands, the effect is 1.7 pp (ranging from 0.3 pp in 1940 to 3.2 pp in 1945). The effects for each of the war years for each of the three countries are

 $^{^{11}}$ Among the 1940 birth cohort, only those born in the final 8 months of the year were exposed to WWII at some point during gestation. Therefore, we assign $D40_{-}45$ the value 2/3 for the 1940 birth cohort.

highly significant. For males, the pattern is similar (Appendix Figure 2). For the neutral countries Sweden and Switzerland, this pattern appears for females nor for males. If we assume that it is mainly the weakest people who die, then these results suggest that some selective mortality related to prenatal exposure took place.

However, if we condition on having reached age 2, and thus focus only on mortality between age 2 and the year 2004, the pattern for the occupied countries completely disappears and is not significant anymore for any war year: people who had been exposed to WWII in utero and who survived till age 2 are no more likely to die before 2004 than one would expect based on the long-term trend. (See Figure 5 for females and Appendix Figure 3 for males.) This means that selective mortality may contribute to the unexpected sign of many of our estimates in Tables 1-4 and hence at least partially explain why the "positive" health effects reported in those tables are likely just artifacts. But it also means that all selective mortality takes place during infanthood. The increased mortality during later life stages predicted by the fetal origins hypothesis does not show up. This means that prenatal exposure to WWII leads to immediate mortality increases, but does not affect long-term mortality patterns.¹²

One might argue that this finding could be due to the fact that long-term mortality effects from prenatal exposure only become visible at later ages: the oldest exposed are no older than 64 in 2004 and the probability to have died before this age is relatively low. (E.g. a French woman born in 1941 who survived till age 2, afterwards had a 90% chance to survive till 2004.) We therefore run the same analyses using the probability to survive till one's birthday in 2014, which is currently the latest year for which such data are available in the Human Mortality Database for all three countries. As Appendix Figures A4 (for females) and A5 (for males) show, once they have survived till age 2, the chances of dying are also not higher for the prenatally exposed if one observes them till older ages. These

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¹² Similar results were shown in Lindeboom & Van Ewijk (2015). They argue that the pattern of "culling" (i.e. selective mortality of weaker individuals) in the first few years of life means that effect estimates of prenatal exposures in many studies are likely to have been downward biased.

mortality patterns are therefore another indication that prenatal WWII-exposure did not lead to negative long-run health effects.

Nevertheless, given the increased infant mortality during WWII, we need to investigate whether this (potentially selective) mortality might have masked negative long-run health effects in our analyses on the SHARE sample. I.e. the current SHARE-sample includes only people who managed to survive their first few live years. If the unhealthiest people could not be included since they had already died, then the counterfactual health of the people in our sample would have been better than assumed. Negative long-run health effects on these people would then have been masked.

Based on the previous analysis, we know the excess mortality before 2004 by country and sex for each of the birth cohorts 1940-1945. We proportionally add respondents to our sample for each birth cohort*country*sex combination.¹³ For our binary dependent variables (a fair/poor self-reported health, being hospitalized, having one or more limitations) we assign these people a bad health. For the continuous variable cognitive performance, for each country*sex combination, we take the 10th percentile value of all respondents born during the war and assign the added respondents this value. For the number of GP visits, we take the value corresponding to the 90th percentile. Next, we regress these health outcomes on prenatal WWII-exposure following equation (1), but this time including these additional respondents to compensate for selective mortality. Note that this is a very conservative approach, as we assume that all who have died before 2004 as a result of WWII would have had a poor health if they would still have been alive. To the extent that this is not the case, we

The numbers of respondents to be added are calculated as $\frac{Excess_{mort_{cjs}}}{1-Excess_{mort_{cjs}}}$, with $Excess_{mort_{cjs}}$ being the deviation from trend in the share of birth cohort j from country c that did not survive till 2004, calculated as the coefficient $\hat{\varphi}_i$ to $Dcohort_i$. (See Figure 4 and Appendix Figure 2 for the analyses that lead to the corresponding numbers.) This ratio is calculated separately for males and for females. The numbers of respondents that are added are rounded to the nearest integer for each country*birth year*sex combination.

Our sample consists of three SHARE-waves. We proportionally allocate the added respondents to each of the three waves, so that the distribution of added respondents over the three waves is the same as the distribution of the actual respondents over the three waves.

The analyses using Human Mortality Database data on which we base the numbers of respondents to be added are based on birth year, without taking into account birth month. We similarly add people based on their birth year (not birth month) and assume that all respondents that are added in this way are prenatally exposed to WWII. This means that no people are added who are classified as being born in the first months of 1940 and who were hence not exposed. This approach is based on the assumption that the increased mortality rates can only come from people who were actually exposed.

may over correct for selective mortality. Table 5 shows the results from the regressions that included these additional respondents. For males in the main sample (1934-56), there is now evidence that exposure may increase the chance of being hospitalized at older ages, but results for the other four outcomes remain insignificant, suggesting that they are not affected by prenatal WWII-exposure. For females, there is still no proof that prenatal WWII-exposure leads to a worse later-life health — even when using this conservative approach. For cognitive performance, the results still show better scores for those who had been prenatally exposed.

6.2 Selective fertility

It is possible that people conceiving during WWII differed systematically from those conceiving in earlier or later years. People may have postponed pregnancies till after the bad years were over, or may not have been able to conceive due to hardship (e.g. women may not have had their menstrual cycles) or had miscarriages. We study selective fertility during WWII using information on characteristics of respondents' parents. Our main worry is that parents conceiving during the war had "better" characteristics, which would have been transmitted to their offspring. Suppose that this were true and also suppose that prenatal WWII-exposure would have caused negative long-run health effects: then naïve estimates would show no long-run effects of prenatal WWII-exposure due to the combination of one positive and one negative effect. This might be what happened in our results and it is this possibility that we want to exclude.

Before we proceed, it is relevant to point out that in our context, selective fertility and selective mortality likely work in opposite directions. In case of high selective mortality, the exposed would have had lower probabilities to survive. In case of high selective fertility, the exposed would have had higher probabilities to survive. Figure 4 shows that at least initially, selective mortality effects were likely more of an issue than selective fertility effects. Nevertheless, one cannot exclude the possibility that

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¹⁴ Rooseboom and Van de Krol (2010) find that during the Dutch famine about half of the women did not menstruate.

selective fertility may have taken place and that this would have influenced our results. We hence investigate whether the parents of the exposed were systematically different from the parents of the non-exposed.

The SHARE survey asks respondents about the age at which their parents died or, if these were still alive, what their age was at that moment. The age people attain (their longevity) is a strong indicator for their overall health. We therefore study selective fertility by comparing the longevity of parents whose pregnancies did versus did not overlap with the adverse years of WWII. For this analysis, we first establish the age at which respondents' parents died. For parents who were still alive during the last survey wave in which a respondent was observed, we add their remaining life expectancy, which we calculate from the Human Mortality Database. (E.g. an 88-year old French male in 2011 has an expected remaining life expectancy of 4.0 years.) Next, we estimate ordinary least squares regressions of the longevity of respondents' mothers/fathers on whether respondents had been prenatally exposed to adverse WWII-conditions. We add as covariates the sex and country of the respondent, as well as the respondent's birth year in order to adjust for time trends in parental longevity. (Note that we cannot include wave fixed effects and individual random effects in these regressions, as we have only one observation from a single moment in time per respondent.) These analyses show whether fathers and mothers whose children were in utero during the adverse WWII-years had a worse or better health than parents conceiving in earlier and later years.

Other indicators for parental health and socioeconomic status come from SHARE wave 3 (which is otherwise not used in the present research). In this wave, respondents were asked about characteristics of their parents when they themselves were age 10. These included: whether the biological father was present in the household at that time and whether the parents smoked, drank heavily, or had mental health problems. These variables are taken as dependent variables in similar regressions to the ones for parental longevity. The sample size in the regressions utilizing information

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¹⁵ We do need to make the assumption that being pregnant during WWII does not directly affect parents' longevity, but this seems a reasonable assumption.

from SHARE wave 3 is smaller, since only respondents can be included who appeared both in wave 3 and in at least one other wave.

Our analyses demonstrate that the mothers of the exposed had a somewhat higher longevity than the mothers of unexposed persons. For fathers, we find no such difference (see Table 6). Also, the parents of the exposed were less likely to smoke or drink heavily. These results suggest that part of the positive association we find between prenatal WWII-exposure and good health at older ages is due to selective fertility.¹⁶

We further investigate the possibility that selective fertility is the reason why we find no negative health effects of prenatal exposure by adding the characteristics of respondents' parents as covariates to our main regressions following equation (1). We additionally add a set of dummy variables as covariates indicating the occupation of the household's main breadwinner when the respondent was 10 years old. Table 7 shows the result when adding the parental characteristics one at a time (columns (2) through (5)), or all simultaneously (column (6)). Columns (7) through (9) show the results for the alternative sub samples. Appendix Tables 2 and 3 show results similar to those from column (6) of Table 7, separately by country and year of exposure. We robustly find that the estimated effects of prenatal exposure on older people's health do not change into negative health effects. However, several coefficients do get closer to zero. This suggests that indeed some of the unexpected association between prenatal WWII-exposure and a better health at older ages may be a result of selective fertility. Yet it should be noted that the better cognitive performance among exposed females remains virtually unattenuated.

6.3 The combined importance of selective mortality and selective fertility

The results presented above suggest that both selective mortality effects and selective fertility effects may have contributed to our unexpected finding of a better health among the prenatally exposed. To

¹⁶ Alternatively, the more favorable parental characteristics among the exposed may (partially) result from selective mortality if mortality rates were highest among children from parents with unfavorable characteristics.

examine the combined importance of both channels we proceed with a rough and conservative test. Ideally, we would have liked to combine the selective fertility analyses in which we correct for parental characteristics (Table 7) with the selective mortality analysis of Table 5. However, this is not possible, because for the selective mortality analyses we added "respondents" of whom we do not observe the characteristics of their parents.

To get an indication, we consider the information from Table 1 in conjunction with that from Tables 5 and 7. Table 1 provides estimates that are both subject to selective mortality and selective fertility. Table 5 corrects for selective mortality. The difference between the coefficients in row (1) of Table 5 and those in columns (1) and (5) of Table 1 gives an indication of the effect of selective mortality for females and males, respectively. E.g. from Table 1, we estimate that prenatal exposure led to a 1.198 percentage point reduction in the probability of reporting a fair or self-reported health. Table 5 suggests an increase with 0.578 percentage points. Apparently, selective mortality led to an upward bias of 1.776 percentage points (0.578 – (-1.198)) in Table 1. Now we add this information to our Table 7 results, which correct for selective fertility. Column (6) of Table 7 suggests a 0.685 percentage point reduction in the probability of reporting a fair or self-reported health. If we assume that this result is still biased by 1.776 percentage points as calculated above, then the true effect would be a 1.091 increase in the probability of reporting a fair or self-reported health (-0.685+1.776). We can of course not calculate standard errors, but at unchanged standard errors, none of the estimates for females would significantly point to a worse health as a result of prenatal exposure to WWII. For cognitive performance, the results would even show a better health among the exposed which is significant at the 5%-level. For males, the effects on hospitalizations and on self-reported health would be significant at the 1% and at the 10%-level, respectively, in both cases pointing toward a worse health among the prenatally exposed. But the results for the other three outcomes would not be significant at conventional levels. The absence of a pattern of results indicating long-term effects of prenatal exposure holds as well when including different sets of cohorts into the sample (cohorts 1934-50, 1940-56, all elderly respondents available in the data). This is illustrated by Table 8, which shows the

results for the various specifications and samples for the outcome on which we consistently found the strongest positive health effects (cognitive performance) and for the one outcome on which we did not find any significant effects in Table 1, hospitalizations.

Note that this is a conservative approach: the Table 7 results may on the one hand not incorporate all dimensions of selective fertility, although we could use a broad range of parental characteristics. But on the other hand, the Table 7 results may partially already incorporate a correction for selective mortality (see footnote 16). And the Table 5 results may have over corrected for selective mortality if not all who died before 2004 due to WWII would have had a poor health at older ages if they would still have been alive. These results therefore provide a strong indication that if we would be able to simultaneously correct for selective fertility and selective mortality, there would still be no proof that prenatal WWII-exposure leads to a worse later-life health.

7. Conclusion

Violence during wars is what captures the headlines. But at any given time, a large share of the populations of countries at war usually does not experience violence, but still suffers substantially. Supply lines are severed, leading to reduced food quantities and qualities; stress levels are high; the economy is in a recession and health care systems may collapse. This wide-scale suffering is less likely to grab the headlines. Once the war is over, however, biological theory leads to the hypothesis that the effects of this suffering linger among those who had been prenatally exposed and lead to worse health at older ages.

During World War II, the populations of countries like Belgium, France and The Netherlands evidently suffered from the war during five years of occupation. Most of this period was not characterized by direct exposure to extreme levels of violence, nor was the food situation so bad that it can be characterized as a famine. It is now known that exposure to famines and other extremely adverse conditions, such as very large-scale destruction during WWII, led to a poorer health among those who had been prenatally exposed to them. But little is known about the long-term effect of the

less-extreme conditions that characterized most of the war period in the occupied countries and that were thus experienced by the majority of women who were pregnant during the war in these countries.

We demonstrate that prenatal exposure to WWII in Belgium, France and The Netherlands has no substantial population-wide negative effects on health at ages 50 and older. Many of our estimates even show a better health among older people who had been exposed. We demonstrate that this unexpected result is due to selective mortality shortly after birth and to selective fertility, in the sense that healthier people were more likely to become pregnant and give birth during WWII. In the same way that our initial estimates were biased toward finding a better health due to prenatal exposure, previous research on prenatal exposure to other (more extreme) historical circumstances most probably also suffered from the same bias. Selective mortality and selective fertility therefore mean that long-term effects of prenatal exposures to historical circumstances in existing research were likely often under estimated.

Even when we take selective mortality and selective fertility into account, we find no evidence that prenatal exposure to WWII leads to the expected long-term health damage. Previous research showed that prenatal exposure to the extreme circumstances (famines, destruction) that some groups of people experienced during WWII negatively affects their health at older ages. Apparently, the circumstances experienced by the majority of civilians in occupied western Europe (that were evidently adverse, although in a less extreme sense) did not lead to similar long-term health damage.

Previous research also showed that prenatal exposure to relatively mild shocks can already lead to long-run health effects (Almond & Currie, 2011). Our results raise the question why this was not the case for WWII exposure. It must be noted that most of those previously studied mild exposures, such as infections, low-level radioactivity and pollution, were qualitatively different from the exposures we study now, which means that the biological pathways leading to effects would have been very different. This makes comparisons difficult. In cases that one might argue share more similarities with the exposure we study, such as prenatal exposure to Ramadan (Almond & Mazumder, 2011; Van

Ewijk, 2011), three issues need to be considered. First, although both exposures involve nutritional restrictions, the exact form of the restrictions differs, which may lead to differences in biological impacts – without exact insights into what happens in the human body, we cannot simply assume that both are mild exposures and should therefore lead to similar effects. Second, fetal programming effects due to malnutrition may be moderated by differences between the prenatal and the postnatal environment. Gluckman & Hanson (2004) describe that the fetal body may react to potentially harmful outside circumstances with biological adaptations that prepare the body for postnatal circumstances that are "predicted" to be similar to the prenatal circumstances (predictive adaptive response). Hence, one could hypothesize that a mild exposure throughout pregnancy that continuous postnatally may cause less strong effects than a similar exposure that is constrained to only a limited period during pregnancy, as physical adaptations in the fetus are misdirected. Third, the fact that we find no longrun health effects for France, Belgium and The Netherlands, does not automatically imply that no population-wide effects of prenatal exposure to WWII occurred for any country. We study three countries that are arguably relevant to study, but circumstances in other countries have been worse across the board so that possibly, affected cohorts at large may suffer from long-run adverse health effects. From exactly what level of adverseness on negative effects arise is hard to gauge.

The findings of our study may have important implications for more recent wars, too. It is known that children born to mothers who experienced extreme wartime conditions during pregnancy tend to have more health problems throughout their life courses. So one would for example expect children born in a beleaguered city to suffer more often from health problems later in life. But in many conflicts, at any specific time point, most civilians do not suffer from extreme levels of violence and destruction, nor from famines. The suffering they do experience may drive up infant mortality rates. But children born during such war episodes and who survive infancy are apparently resilient to the adverse circumstances they experienced before birth: they tend to recover from their bad prenatal circumstances and not to suffer from negative health consequences later in their lives.

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Table 1: Prenatal exposure to WWII and health at old age

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	Mean		Females	es			Males	SS		
	(SD)	(1)	(2)	(3)	(4)	(2)	(9)	(2)	(8)	
Cognitive	9.5	0.423***	0.549***	0.381***	-0.018	0.015	0.113	-0.017	-0.207	
performance	(3.3)	(0.097)	(0.088)	(0.100)	(0.186)	(0.098)	(0.091)	(0.102)	(0.197)	
Fair / poor	26.0	-1.198	-2.221*	-0.139	0.287	-1.325	-3.505***	-1.320	0.144	
self-rep. health	(43.8)	(1.362)	(1.236)	(1.402)	(2.525)	(1.375)	(1.248)	(1.421)	(2.618)	
Hospitalized in	12.7	-0.675	-1.013	-0.421	0.193	0.534	-0.152	0.160	-2.128	
past year	(33.3)	(0.882)	(0.797)	(0.907)	(1.669)	(096.0)	(0.875)	(26.0)	(1.887)	
Number of	4.0	-0.407**	-0.514***	-0.285*	-0.086	-0.107	-0.308**	-0.161	-0.046	
GP visits	(4.9)	(0.160)	(0.145)	(0.162)	(0.274)	(0.142)	(0.133)	(0.146)	(0.266)	
Having 1 or more	14.5	-2.876**	-6.274***	-2.238*	-0.601	-0.918	-4.251***	-0.492	1.327	
limitations	(35.2)	(1.130)	(1.017)	(1.159)	(2.022)	(1.040)	(0.951)	(1.067)	(1.825)	
-										
Sample (cohorts)		1934-56	$AII \leq 1956$	1934-50	1940-56	1934-56	$AII \le 1956$	1934-50	1940-56	
Sample size		10,805	14,749	7,641	8,644	9,412	12,371	6,778	7,521	

exposure, captured using a dummy variable, on the listed health variable. The results come from regressions that additional control for year of birth, wave, country, and a dummy for respondents conceived between May '45 and Dec. '46. Sample sizes vary slightly between the regressions within each column. Reported sample sizes are for the regression with the highest sample size within each column. Other regressions in the same column may have sample sizes that are up to 2% smaller. * p < 0.10, ** p < 0.05, *** p < 0.01Means and SDs refer to the 1934-56 sample. Each cell in the columns to the right of that shows the result from a separate regression. Presented are the effects of prenatal WWII

Table 2: Prenatal exposure to WWII and health at old age - Difference-in-differences

	-	Fem				Mal	es	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
A. Occupied cour	ntries							
Cognitive	0.154	0.141	0.287*	-0.039	-0.073	-0.128	0.022	-0.223
performance	(0.148)	(0.142)	(0.163)	(0.157)	(0.152)	(0.145)	(0.167)	(0.164)
Fair / poor	1.541	-0.794	-0.593	4.047**	-0.476	-2.366	-1.087	1.507
self-rep. health	(1.960)	(1.878)	(2.154)	(2.060)	(1.918)	(1.867)	(2.153)	(2.017)
Hospitalized in	-0.151	0.758	-0.629	0.462	-1.955	-1.407	-1.179	-2.941*
last year	(1.340)	(1.296)	(1.462)	(1.414)	(1.553)	(1.505)	(1.723)	(1.620)
Number of	-0.252	-0.791***	-0.575***	0.111	0.059	-0.343*	-0.041	0.148
GP visits	(0.202)	(0.197)	(0.212)	(0.214)	(0.177)	(0.178)	(0.194)	(0.186)
Having 1 or more	-1.519	-2.856*	-3.043*	0.658	1.014	-0.374	0.572	1.830
limitations	(1.638)	(1.610)	(1.806)	(1.716)	(1.458)	(1.457)	(1.637)	(1.523)
B. Neutral count	ries							
Cognitive	0.219*	0.410***	0.095	0.014	0.054	0.239**	-0.039	0.027
performance	(0.119)	(0.112)	(0.128)	(0.176)	(0.121)	(0.113)	(0.132)	(0.192)
Fair / poor	-1.939	-1.614	0.459	-1.760	-0.331	-1.185	-0.168	-2.253
self-rep. health	(1.514)	(1.414)	(1.633)	(2.289)	(1.447)	(1.384)	(1.615)	(2.403)
Hospitalized in	-0.420	-1.751*	0.185	-0.147	2.279*	1.288	1.318	1.376
past year	(1.073)	(1.018)	(1.146)	(1.573)	(1.282)	(1.220)	(1.404)	(1.875)
Number of	-0.038	0.228*	0.291**	-0.017	-0.146	0.012	-0.120	-0.149
GP visits	(0.137)	(0.131)	(0.136)	(0.239)	(0.118)	(0.116)	(0.128)	(0.200)
Having 1 or more	-0.753	-3.556***	0.809	-0.849	-1.689	-3.906***	-1.031	-0.465
limitations	(1.282)	(1.238)	(1.384)	(1.904)	(1.116)	(1.088)	(1.238)	(1.782)
Sample (cohorts)	1934-56	All ≤ 1956	1934-50	1940-56	1934-56	All ≤ 1956	1934-50	1940-56
Sample size	15,451	20,965	11,190	12,306	13,395	17,826	9,846	10,600

Each cell in Panel A. shows the results from a regression of the respective health variable on year of birth, wave, country, as well as a dummy indicating whether the respondent had been in utero during WWII and a dummy indicating respondents conceived between May '45 and Dec. '46. Both latter dummies are interacted with a dummy indicating occupied countries. Panel A. shows the interaction effects for "in utero during WWII" * "occupied country". The cells in Panel B. show the main effects for "in utero during WWII". Occupied countries are Belgium, France and The Netherlands. Neutral countries are Sweden and Witzerland. Sample sizes vary slightly between the regressions within each column. Reported sample sizes are for the regression with the highest sample size within each column. Other regressions in the same column may have sample sizes that are up to 2% smaller.

^{*} p < 0.10, ** p < 0.05, *** p < 0.01

Table 3: Prenatal exposure to WWII and health at old age - effects by country

			Females)		Males		
	Cognitive	Fair / poor	Hospitalized in	Number of	Having ≥1	Cognitive	Fair / poor	Hospitalized in	Number of	Having ≥ 1
	performance	self-rep. health	past year	GP visits	limitations	performance	self-rep. health	past year	GP visits	limitations
	(1)	(2)	(3)	(4)	(2)	(9)	(2)	(8)	(6)	(10)
Belgium	0.360**	0.188	-0.576	-0.196	1.387	0.013	-3.121	2.146	0.117	-0.343
	(0.164)	(2.219)	(1.574)	(0.373)	(2.055)	(0.161)	(2.187)	(1.629)	(0.295)	(1.888)
France	0.530***	-0.297	-1.100	-0.401**	-4.564***	-0.091	-2.677	-1.496	-0.174	-0.813
	(0.153)	(2.248)	(1.438)	(0.192)	(1.671)	(0.167)	(2.353)	(1.697)	(0.217)	(1.703)
The Netherlands	0.365**	-3.827*	-0.276	-0.647***	-5.693***	0.138	2.219	0.793	-0.288*	-1.664
	(0.166)	(2.323)	(1.320)	(0.169)	(1.825)	(0.167)	(2.307)	(1.425)	(0.159)	(1.497)
Sample size	10700	10805	10785	10747	10798	9239	9412	9386	9357	9410
Each column shows results from a separate regression, similar to those in	s results from a	separate regressio	in, similar to those		but in which th	e dummies for be	Table 1, but in which the dummies for being exposed and for being conceived between May 1945 and	for being conceive	ed between Ma	ay 1945 and

Dec. 1946 are interacted with country dummies. Sample: 1934-56. * p < 0.10, ** p < 0.05, *** p < 0.01

Table 4: Prenatal exposure to WWII and health at old age - by year of exposure

			Females			Females		Males		
	Cognitive	Fair / poor	Hospitalized in	Number of	Having ≥1	Cognitive	Fair / poor	Hospitalized in	Number of	Having ≥1
	performance	self-rep. health	past year	GP visits	limitations	performance	self-rep. health	past year	GP visits	limitations
	(1)	(2)	(3)	(4)	(2)	(9)	(7)	(8)	(6)	(10)
1940	0.159	-1.473	-0.633	-0.371	-2.669	0.083	-5.644*	1.598	-0.576*	-2.230
	(0.211)	(3.017)	(1.976)	(0.400)	(2.496)	(0.218)	(2.999)	(2.207)	(0.320)	(2.264)
1941	0.434**	0.193	-0.522	-0.269	-2.224	-0.009	2.679	4.776**	0.091	0.449
	(0.212)	(3.057)	(1.939)	(0.275)	(2.622)	(0.229)	(3.004)	(2.122)	(0.320)	(2.288)
1942	0.405**	2.580	-2.050	-0.131	-1.406	-0.294	5.975**	1.044	0.239	2.341
	(0.197)	(2.921)	(1.692)	(0.429)	(2.397)	(0.203)	(2.934)	(1.997)	(0.260)	(2.305)
1943	0.434**	-2.642	-1.706	-0.538	-3.703*	0.288	-7.229***	-2.017	-0.338	-0.120
	(0.197)	(2.638)	(1.710)	(0.329)	(2.153)	(0.190)	(2.513)	(1.765)	(0.233)	(2.080)
1944	0.687***	-1.496	0.862	-0.420**	-2.233	0.020	-4.157	-1.838	-0.099	-2.778
	(0.184)	(2.612)	(1.750)	(0.212)	(2.117)	(0.188)	(2.561)	(1.829)	(0.354)	(1.820)
1945	0.306	-4.359	0.010	-0.706**	-5.204**	-0.000	0.766	0.740	-0.038	-3.840*
	(0.214)	(3.039)	(2.000)	(0.287)	(2.321)	(0.207)	(3.197)	(2.109)	(0.268)	(2.066)
Sample size	10700	10805	10785	10747	10798	9239	9412	9386	9357	9410
Each column	shows results fro	Each column shows results from a separate regression, similar to those in Table 1, but in which the exposure dummy is replaced by six war year dummies. 1940-1944 are dumm	ession, similar to	those in Table	1, but in which	ι the exposure d	ummy is replaced	by six war year du	ımmies. 1940-	1944 are dumn

Each column shows results from a separate regression, similar to those in Table 1, but in which the exposure dummy is replaced by six war year dummies. 1940-1944 are dummy variables for being born between May 1945 and January 1946. Sample: 1934-56. * p < 0.10, ** p < 0.05, *** p < 0.01

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Table 5: Respondents added to compensate for selective mortality

			Females					Males		
	Cognitive	Fair / poor	Hospitalized in	Number of	Having ≥1	Cognitive	Fair / poor	Hospitalized in	Number of	${Having} \geq 1$
	performance	self-rep. health	past year	GP visits	limitations	performance	self-rep. health	past year	GP visits	limitations
	(1)	(2)	(3)	(4)	(2)	(9)	(7)	(8)	(6)	(10)
A. All 3 countries										
Sample: 1934-56	0.314***	0.578	1.133	-0.248	-0.720	-0.081	0.645	2.364**	0.059	1.351
	(960.0)	(1.367)	(0.913)	(0.159)	(1.158)	(0.098)	(1.386)	(0.993)	(0.142)	(1.084)
Sample: All < 1956	0.443***	-0.492	0.721	-0.352**	-4.142***	0.016	-1.559	1.610*	-0.139	-2.002**
l -	(0.087)	(1.241)	(0.828)	(0.145)	(1.046)	(060.0)	(1.259)	(0.906)	(0.133)	(0.996)
Sample: 1934-50	0.274***	1.598	1.328	-0.127	-0.123	-0.110	0.577	1.950*	0.005	1.748
	(660.0)	(1.407)	(0.934)	(0.161)	(1.185)	(0.102)	(1.432)	(1.028)	(0.147)	(1.109)
Sample: 1940-56	-0.139	2.397	2.427	0.123	2.138	-0.344*	2.780	0.448	0.169	4.410**
	(0.185)	(2.536)	(1.712)	(0.273)	(2.063)	(0.197)	(2.638)	(1.933)	(0.266)	(1.897)
B. Per country Sample: 1934-56										
Belgium	0.290*	1.664	0.945	-0.026	3.247	-0.119	-0.798	4.316**	0.377	2.534
	(0.162)	(2.226)	(1.642)	(0.366)	(2.106)	(0.160)	(2.223)	(1.709)	(0.294)	(1.999)
France	0.367**	1.770	0.976	-0.201	-2.129	-0.162	-1.033	-0.055	-0.029	0.874
	(0.152)	(2.257)	(1.532)	(0.195)	(1.765)	(0.166)	(2.380)	(1.775)	(0.220)	(1.806)
The Netherlands	0.277*	-2.060	1.564	-0.549***	-3.525*	0.053	4.146*	2.641*	-0.205	0.538
	(0.165)	(2.340)	(1.398)	(0.169)	(1.895)	(0.166)	(2.324)	(1.503)	(0.158)	(1.601)
Sample size	10,728	10,822	10,822	10,784	10,822	9,262	9,416	9,416	9,386	9,416
In panel A, each cell shows results from a separate regression, similar to those	hows results fro	m a separate regre	ssion, similar to th		1. Presented are	the effects of p	in Table 1. Presented are the effects of prenatal WWII exposure, captured using a dummy variable,	sure, captured us	ing a dummy	variable,

on the respective health variable. In Panel B, each column shows results from a separate regression, similar to those in Table 3. Presented are the effects of prenatal WWII exposure, captured using a dummy variable that is interacted with three country dummies, on the respective health variable. For each of the war years, a number of "respondents" is added that corresponds to the excess mortality for the respective country*birth cohort*sex combination. These respondents are assigned a bad health on each of the outcome measures. Sample sizes refer to the regressions on the 1934-56 sample. * p < 0.10, ** p < 0.05, *** p < 0.01

Table 6: Prenatal exposure to WWII and parental characteristics

	Longe-	Longe-			When age 10:	
	vity	vity	Father	Parents	Parents drank	Parents had mental
	mother	father	present	smoked	heavily	health problems
	(1)	(2)	(3)	(4)	(5)	(6)
All exposed	0.799**	-0.566	-0.013	-0.031**	-0.019**	0.004
	(0.356)	(0.381)	(0.011)	(0.015)	(0.009)	(0.005)
Belgium	0.317	-0.613	-0.016	-0.038	-0.008	0.005
	(0.630)	(0.662)	(0.016)	(0.026)	(0.016)	(0.008)
France	0.999*	-0.743	-0.022	-0.034	-0.022	0.007
	(0.562)	(0.621)	(0.021)	(0.031)	(0.019)	(0.007)
Netherlands	1.006*	-0.306	-0.001	-0.023	-0.029***	0.000
	(0.581)	(0.621)	(0.016)	(0.021)	(0.010)	(0.011)
1940	1.122	-0.723	-0.026	-0.061*	-0.030*	-0.003
	(0.841)	(0.883)	(0.025)	(0.035)	(0.017)	(0.010)
1941	0.550	-0.699	-0.027	-0.009	-0.006	0.015
	(0.791)	(0.838)	(0.024)	(0.032)	(0.019)	(0.013)
1942	-0.100	-0.888	-0.036	-0.082**	-0.021	-0.006
	(0.736)	(0.790)	(0.024)	(0.034)	(0.018)	(0.009)
1943	1.503**	-0.561	-0.016	-0.068**	-0.009	-0.003
	(0.662)	(0.776)	(0.021)	(0.032)	(0.018)	(0.009)
1944	0.769	-0.834	0.007	0.017	-0.020	0.026*
	(0.702)	(0.737)	(0.018)	(0.029)	(0.017)	(0.014)
1945	0.909	0.461	0.023	0.020	-0.034*	-0.010
	(0.731)	(0.838)	(0.019)	(0.034)	(0.019)	(0.010)
Sample size	9,706	9,541	5,067	5,166	5,166	5,166

Table shows regressions of charateristics of respondents' parents on prenatal exposure status (= the presented coefficients), year of birth, wave, country, and a dummy for respondents conceived between May '45 and Dec. '46. Each column within a panel shows a separate regression. The sample for each regression includes both female and male respondents. Sample sizes are always the same for all three regressions presented in a single column. Sample: 1934-56. * p < 0.10, ** p < 0.05, *** p < 0.01

	Table 7: Prenata	l exposure t	o WWII and	l health at o	exposure to WWII and health at old age - adjusting for pare	sting for pa	rental chara	cteristics
	(1)	(2)	(3)	(4)	(2)	(9)	(2)	(8)
mance	0.423***	0.367***	0.414***	0.462***	\sim	0.430*** 0.391***	0.512*** (0.358***

A. Females	(1)	(2)	(3)	(4)	(2)	(9)	(-)	8	6)
Cognitive performance	0.423***	0.367***	0.414***	0.462***	0.430***	0.391***	0.512***	0.358***	0.184
	(0.097)	(0.106)	(0.126)	(0.129)	(0.127)	(0.131)	(0.120)	(0.136)	(0.257)
Fair/poor self-rep. health	-1.198	-0.766	0.124	-0.161	0.242	-0.685	-0.685	-1.449	0.531
	(1.362)	(1.486)	(1.823)	(1.828)	(1.803)	(1.926)	(1.926)	(1.741)	(1.976)
Hospitalized in	-0.675	-0.164	0.339	0.371	0.096	0.001	-0.158	-0.312	-0.157
past year	(0.882)	(0.971)	(1.140)	(1.142)	(1.124)	(1.220)	(0.209)	(0.202)	(0.218)
Number of	-0.407**	-0.287*	-0.169	-0.193	-0.143	-0.158	0.001	-0.308	-0.132
GP visits	(0.160)	(0.167)	(0.223)	(0.224)	(0.217)	(0.209)	(1.220)	(1.104)	(1.257)
Having 1 or more	-2.876**	-2.553**	0.032	0.081	0.142	-0.088	-0.088	-3.171**	0.767
limitations	(1.130)	(1.210)	(1.517)	(1.517)	(1.487)	(1.596)	(1.596)	(1.456)	(1.620)
Sample size Sample (cohorts)	10,805 '34-56	8,882 '34-56	6,616 '34-56	6,607 '34-56	6,752 '34-56	5,754 '34-56	7,642 All < '56	4,241 '34-50	4,615 '40-56
B. Males Cognitive performance	(10) 0.015	(11)	(12)	(13)	(14)	(15)	(16) 0.108	(17)	(18)
	(0.098)	(0.108)	(0.128)	(0.130)	(0.129)	(0.138)	(0.129)	(0.143)	(0.283)
Fair/poor self-rep. health	-1.325	-1.218	1.550	1.067	1.386	1.304	1.304	-1.416	-2.089
	(1.375)	(1.505)	(1.808)	(1.820)	(1.810)	(1.936)	(1.936)	(1.782)	(3.824)
Hospitalized in	0.534	1.093	0.826	0.803	0.965	1.686	-0.002	-0.264	-0.135
past year	(0.960)	(1.066)	(1.232)	(1.232)	(1.216)	(1.353)	(0.215)	(0.201)	(0.464)
Number of	-0.107	-0.118	0.042	0.021	0.048	-0.002	1.686	0.942	-1.275
GP visits	(0.142)	(0.151)	(0.193)	(0.192)	(0.190)	(0.215)	(1.353)	(1.250)	(2.727)
Having 1 or more	-0.918	-1.079	-0.455	-0.555	-0.142	-0.046	-0.046	-2.365*	1.445
limitations	(1.040)	(1.136)	(1.344)	(1.350)	(1.359)	(1.483)	(1.483)	(1.389)	(2.763)
Sample size	9,412	7,661	5,514	5,509	5,594	4,684	6,040	3,496	3,680
Sample (cohorts)	'34-56	'34-56	'34-56	'34-56	'34-56	'34-56	$AII \leq '56$	'34-50	'40-56
Parental longevity	No	Yes	No	No	No	Yes	Yes	Yes	Yes
Parental occupation (main breadwinner)	οN	οN	Yes	No	No	Yes	Yes	Yes	Yes
Biological father in household	οN	No	No	Yes	No	Yes	Yes	Yes	Yes
Parental smoking/drinking/mental health	Νο	Νo	No	Νo	Yes	Yes	Yes	Yes	Yes
Each cell shows the results from a separate regression, which is similar to the regressions in Table 1, but with the additional covariates listed at the bottor	regression, v	vhich is simi	lar to the re	gressions in	Table 1, but	t with the a	dditional cov	ariates liste	at the botto

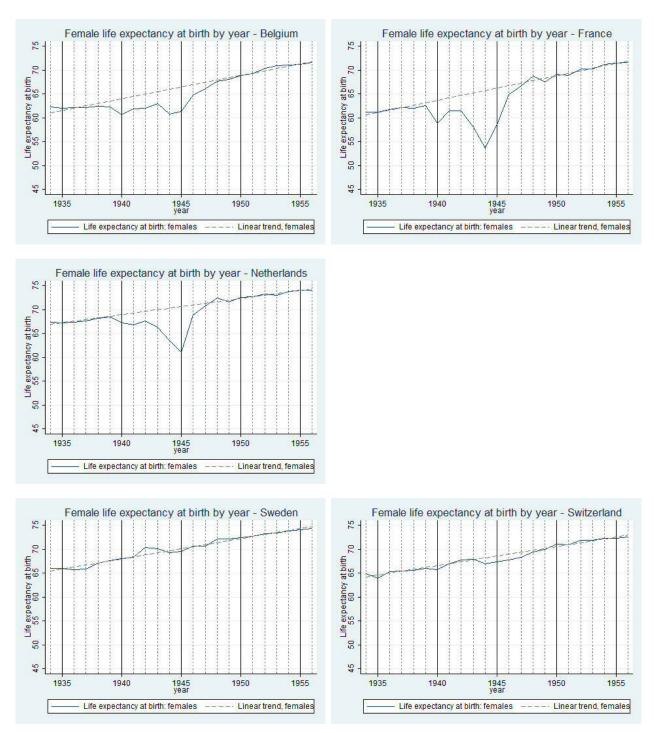
sizes vary slightly between the regressions within each column. Reported sample sizes are for the regression with the highest sample size within each column. Other regressions in the same column may have sample sizes that are up to 2% smaller. * p < 0.10, ** p < 0.05, *** p < 0.01

Table 8: Integrating the results: estimates for the various cohort ranges, adjusting for selective mortality and selective fertility

		Š	Cognitive performance	rmance				H	spitalized	Hospitalized in past year	ear	
		Females			Males			Females			Males	
	(1)	(2)	(3)	(4)	(2)	(9)	(7)	(8)	(6)	(10)	(11)	(12)
Sample: 1934-56	0.423***	0.314***	0.391***	0.015	-0.081	-0.010	-0.675	1.133	0.001	0.534	2.364**	1.686
	(0.097)	(960.0)	(0.131)	(0.098)	(0.098)	(0.138)	(0.882)	(0.913)	(1.220)	(096.0)	(0.993)	(1.353)
Sample: All ≤ 1956	0.549***	0.443***	0.512***	0.113	0.016	0.108	-1.013	0.721	-0.308	-0.152	1.610*	0.942
	(0.088)	(0.087)	(0.120)	(0.091)	(0.090)	(0.129)	(0.797)	(0.828)	(1.104)	(0.875)	(906.0)	(1.250)
Sample: 1934-50	0.381***	0.274***	0.358***	-0.017	-0.110	-0.047	-0.421	1.328	-0.132	0.160	1.950*	1.610
	(0.100)	(660.0)	(0.136)	(0.102)	(0.102)	(0.143)	(0.907)	(0.934)	(1.257)	(266.0)	(1.028)	(1.394)
Sample: 1940-56	-0.018	-0.139	0.184	-0.207	-0.344*	-0.366	0.193	2.427	0.898	-2.128	0.448	-1.275
	(0.186)	(0.185)	(0.257)	(0.197)	(0.197)	(0.283)	(1.669)	(1.712)	(2.364)	(1.887)	(1.933)	(2.727)
Added respondents to compensate	No	Yes	No	No	Yes	No	No	Yes	No	No	Yes	No
for selective mortality												
Controlling for selective fertility:												
Parental longevity	No	No	Yes	N	No	Yes	No	No	Yes	No	N	Yes
Parental occupation (main breadwinner)	No	No	Yes	N	No	Yes	No	No	Yes	No	N	Yes
Biological father in household	No	No	Yes	N	No	Yes	No	No	Yes	No	N	Yes
Parental smoking/drinking/mental health	No	No	Yes	N	No	Yes	No	No	Yes	No	N	Yes
Results as reported in Tables 1, 5 and 7, except for the non-1934-56 samples in columns (3), (6), (9) and (12), which follow from similar specifications to those rep	xcept for the	non-1934-	56 samples	in columr	ls (3), (6)), (9) and	(12), w	iich follo	v from si	imilar spe	cifications	to those re

column (6) from Table 7. Presented are the effects of prenatal WWII exposure, captured using a dummy variable, on the respective health variable. * p < 0.10, ** p < 0.05, ** p < 0.01

Figure 1: Female life expectancy at birth by birth year



Data: Human Mortality Database. Trends are calculated based on the years 1934-1939 and 1948-1956.

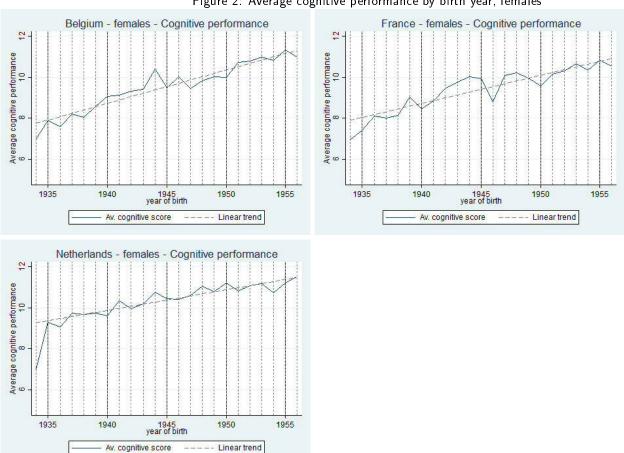
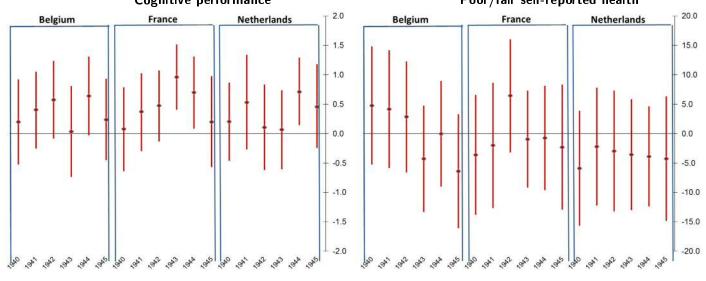


Figure 2: Average cognitive performance by birth year, females

Figures show the average cognitive performance over all observations in the three included SHARE waves for each birth cohort. Linear trends are calculated using regressions, weighted by cohort size, from the average cognitive scores on a linear in birth year.

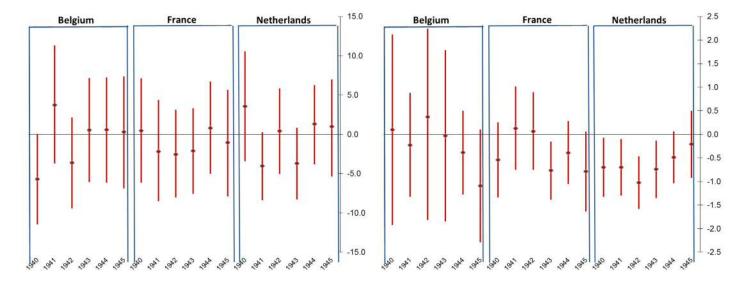
Figure 3: Effects by country and year of exposure for females: 95% confidence intervals

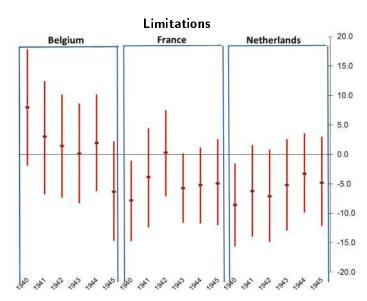
Cognitive performance Poor/fair self-reported health



Hospitalizations

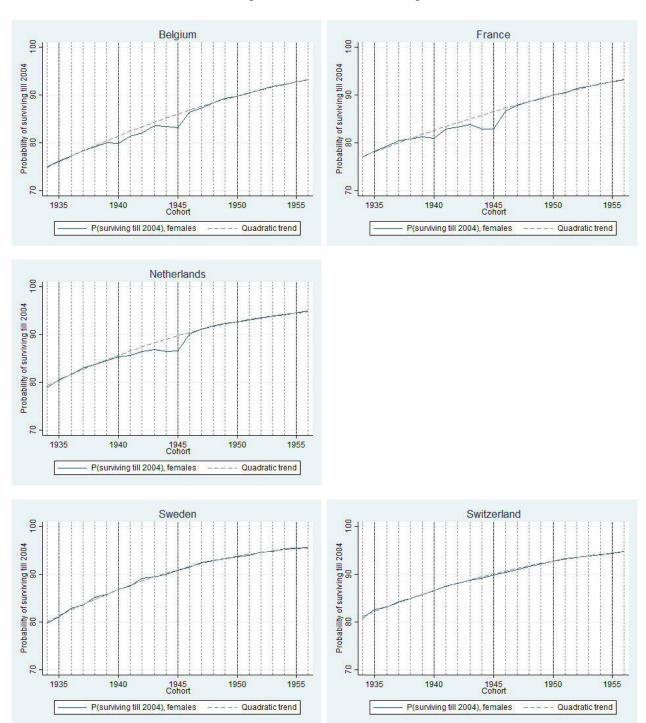
Number of GP visits





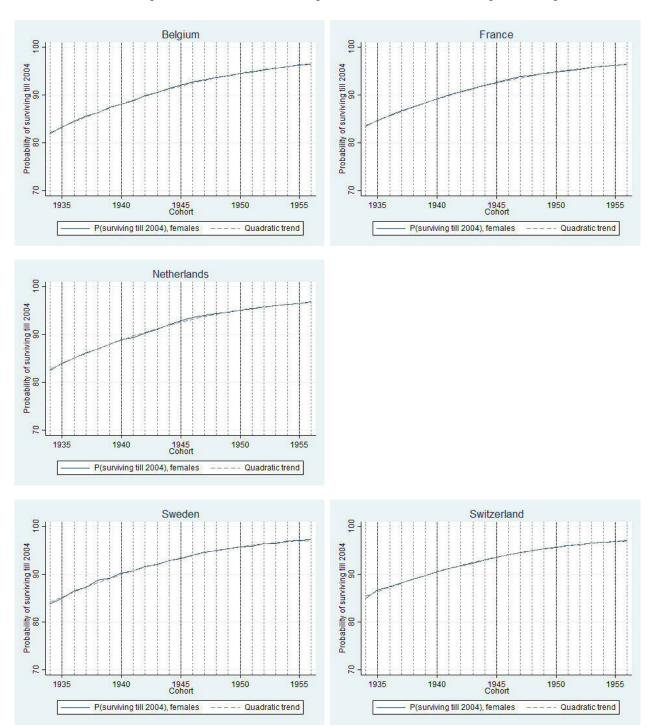
For each dependent variable, all results come from a single regression, similar to those in Table 1, but in which dummies indicating exposure per birth year are interacted with country dummies. Regressions control for wave, country and year of birth and a dummy for being conceived between May '45 and Dec. '46 which is interacted with country dummies.

Figure 4: Probability of surviving till 2004, females



Data are aggregated country*birth cohort*sex data from the Human Mortality Database. Probability of surviving till 2004 is defined as the probability that someone born in a given year survives till her birthday in 2004. Trends are calculated based on the birth cohorts 1934-1939 and 1948-1956.

Figure 5: Probability of surviving till 2004 conditional on having reached age 2, females



Data are aggregated country*birth cohort*sex data from the Human Mortality Database. Probability of surviving till 2004 is defined as the probability that someone born in a given year survives till her birthday in 2004. Trends are calculated based on the birth cohorts 1934-1939 and 1948-1956.

Appendix Table 1: Prenatal exposure to WWII and health at old age using alternative specifications

		Appel	IUIX IADIE.	I. Fielialai	exposition		שונו	וו שו סומי	Appendix Table 1. Frenatal exposure to WWII and nearth at oid age using any
		Females	ales			Ma	Males		
	(1)	(2)	(3)	(4)	(2)	(9)	(2)	(8)	
Cognitive	0.423***	0.418***	0.204**	0.422***	0.015	0.010	-0.140	0.015	
performance	(260.0)	(0.097)	(0.102)	(260.0)	(0.098)	(860.0)	(0.104)	(0.098)	
Fair / poor	-1.198	-1.139	0.275	-1.199	-1.325	-1.333	-1.344	-1.321	
self-rep. health	(1.362)	(1.361)	(1.430)	(1.362)	(1.375)	(1.373)	(1.446)	(1.375)	
Hospitalized in	-0.675	-0.695	-0.516	-0.665	0.534	0.524	0.754	0.540	
past year	(0.882)	(0.882)	(0.953)	(0.883)	(096.0)	(0.959)	(1.034)	(096.0)	
Number of	-0.407**	-0.403**	-0.304*	-0.407**	-0.107	-0.103	-0.114	-0.107	
GP visits	(0.160)	(0.160)	(0.165)	(0.160)	(0.142)	(0.141)	(0.151)	(0.142)	
Having 1 or more	-2.876**	-2.761**	-1.128	-2.885**	-0.918	-0.894	0.421	-0.919	
limitations	(1.130)	(1.129)	(1.183)	(1.129)	(1.040)	(1.039)	(1.101)	(1.040)	
Year of birth	Yes	No	No	Yes	Yes	No	8	Yes	
Age	No	Yes	Yes	Yes	No	Yes	Yes	Yes	
Age^2	N _o	No	Yes	No	S N	No	Yes	No.	

Age^2 No No Yes No In No Yes No Yes No Sample: 1934-56. Each cell shows results from a separate regression, similar to those in Table 1, but with the (additional) covariates listed at the bottom of the table. All specifications additionally include a dummy for respondents conceived between May '45 and Dec. '46 and control for year of birth, wave and country. * p < 0.10, ** p < 0.05, *** p < 0.01

Having tations -1.790 1.233 (2.586)0.280 (2.735)(2.051)4,683 (10)Yes Yes Yes Number -0.312 (0.211)4,669 of GP (0.447)(0.315)-0.292 visits 0.472 Yes Yes Yes 6) Appendix Table 2: Prenatal exposure to WWII and health at old age - adjusting for parental characteristics - effects by country oast year lized in 5.594** Hospita-(2.190)4,680 -1.809 (2.692) -0.130(1.893)Yes Yes Yes 8 Fair / poor self-rep. health 3.719 4,684 (2.794)(3.168)(3.964)-0.921 1.411 Yes ‱ Yes Cognitive mance -0.286 4,622 (0.220)(0.261)0.183 (0.225)0.017 9 Yes Yes Yes ≥1 limi-Having -4.769* tations 5,752 3.562 (2.803)0.749 (2.436)(2.629)(2) Yes Yes Yes -0.497** Number -0.1605,736 (0.224)of GP (0.296)0.160 (0.478)visits 4 Yes ¥es ¥es oast year Hospita-Females lized in -0.019-0.593(2.191)0.553 (1.601)5,747 (2.216)Yes (3) Yes Yes Fair / poor health -0.153self-rep. 5,754 (3.027)(3.126)1.620 (3.529)-3.181 Yes Yes Xes Xes (2)Cognitive 0.498** mance perfor-(0.234)5,713 (0.212)(0.218) 0.345 0.342 Yes Yes Yes Yes (1)Parental smoking/drinking/mental health Parental occupation (main breadwinner) Biological father in household Parental longevity The Netherlands Sample size Belgium France

Each column shows results from a separate regression, similar to column (6) of Table 7, but in which the dummies for being exposed and for being conceived between May 1945 and Dec. 1946 are interacted with country dummies. Sample: 1934-56. * p < 0.10, ** p < 0.05, *** p < 0.01

Appendix Table 3: Prenatal exposure to WWII and health at old age - adjusting for parental characteristics - effects by year

Appendix rable 3. Fichatal exposure to wivin and reach are on age - adjusting for parental characteristics by year Females Males	. i ciiatai cy		Females	מו מו	200	2 2 2	מומי מיים	Males	cts by year	
	Cognitive	Fair / poor	Hospita-	Number	Having		Cognitive	Fair / poor	Hospita-	Number
	perfor-	self-rep.	lized in	of GP	≥1 limi-		perfor-	self-rep.	lized in	of GP
	mance	health	past year	visits	tations		mance	health	past year	visits
1940	0.016	-3.403	-2.003	0.113	-0.766	-0.128	2.355	2.849	-0.367	-4.773*
	(0.286)	(3.940)	(2.391)	(0.691)	(3.397)	(0.286)	(4.278)	(2.964)	(0.327)	(2.670)
1941	0.352	-4.236	0.707	-0.179	-1.477	0.025	7.601*	6.435**	0.258	2.167
	(0.300)	(4.041)	(2.745)	(0.405)	(3.693)	(0.346)	(4.275)	(2.790)	(0.348)	(3.418)
1942	0.198	6.835	-0.922	0.314	0.034	0.108	9.553**	-1.633	0.092	1.997
	(0.278)	(4.443)	(2.351)	(0.620)	(3.489)	(0.294)	(4.544)	(2.539)	(0.338)	(3.293)
1943	0.394	2.039	-2.143	-0.434	1.040	0.046	-3.201	-1.285	-0.206	2.628
	(0.273)	(3.866)	(2.422)	(0.287)	(3.219)	(0.236)	(3.481)	(2.495)	(0.331)	(3.321)
1944	0.655***	-4.185	2.985	-0.073	0.202	0.103	-5.663*	2.079	0.048	-1.488
	(0.241)	(3.499)	(2.445)	(0.296)	(2.870)	(0.265)	(3.436)	(2.961)	(0.738)	(2.611)
1945	0.641**	-1.720	0.945	-0.712*	-0.093	-0.310	-1.627	2.116	0.143	-1.924
	(0.280)	(4.395)	(2.805)	(0.385)	(3.479)	(0.311)	(4.048)	(3.019)	(0.379)	(3.195)
Parental longevity	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Parental occupation (main breadwinner)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Biological father in household	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Parental smoking/drinking/mental health	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample size	5713	5754	5747	5736	5752	4622	4684	4680	4669	4683
Each column shows results from a separate regression, similar to column	regression. s	imilar to colu	mn (6) of Ta	able 7. but	in which t	le exposul	re dummy is	(6) of Table 7. but in which the exposure dummy is replaced by six war year dummies.	ix war vear	dummies. 19

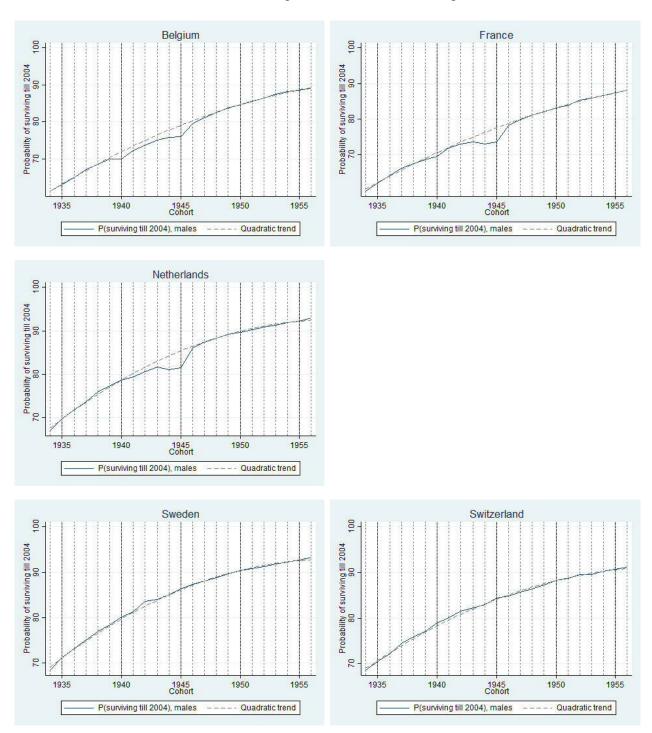
Each column shows results from a separate regression, similar to column (6) of Table 7, but in which the exposure dummy is replaced by six war year dummies. 1940-1944 are dummy variables for being born between May 1945 and January 1946. Each regression controls for all covariates listed at the bottom of the table. * p < 0.10, ** p < 0.05, *** p < 0.01

2

Appendix Figure 1: Effects by country and year of exposure for males: 95% confidence intervals Cognitive performance Poor/fair self-reported health 2.0 20.0 France France Netherlands Belgium Netherlands Belgium 1.5 15.0 1.0 10.0 0.5 5.0 0.0 0.0 -5.0 -0.5 -10.0 -1.5 -15.0 -2.0 -20.0 Hospitalizations Number of GP visits 15.0 2.5 Belgium France Netherlands Belgium France Netherlands 2.0 10.0 1.5 1.0 5.0 0.5 0.0 0.0 -5.0 -1.0 -1.5 -10.0 -2.0 -15.0 -2.5 Limitations 20.0 Belgium France Netherlands 15.0 10.0 5.0 0.0 -5.0 -10.0 -15.0 -20.0

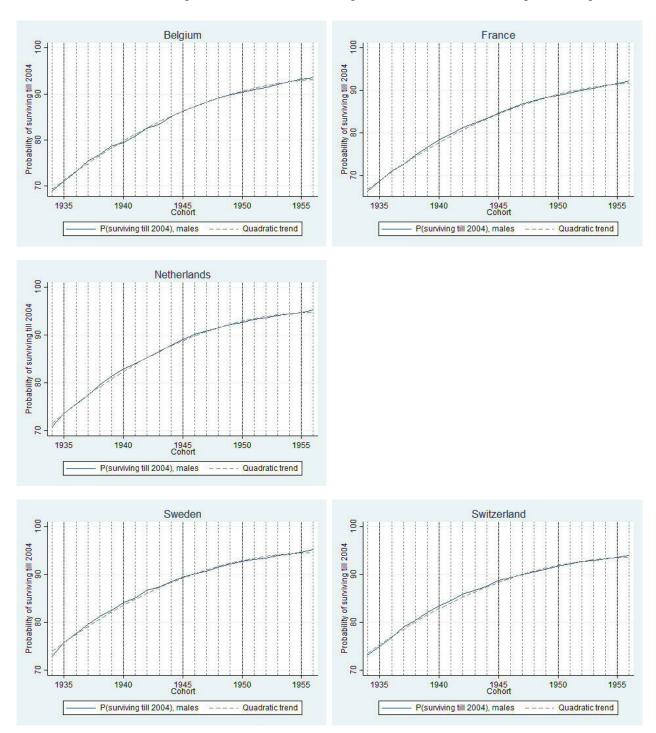
For each dependent variable, all results come from a single regression, similar to those in Table 1, but in which dummies indicating exposure per birth year are interacted with country dummies. Regressions control for wave, country and year of birth and a dummy for being conceived between May '45 and Dec. '46 which is interacted with country dummies.

Appendix Figure 2: Probability of surviving till 2004, males



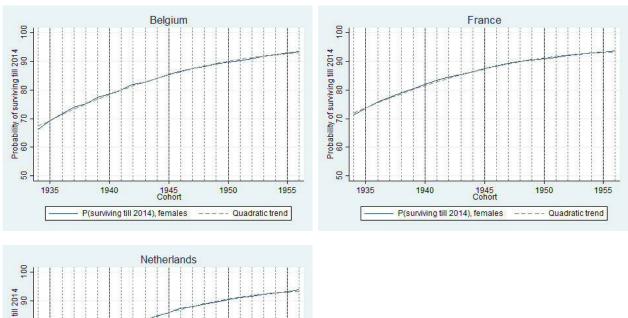
Data are aggregated country*birth cohort*sex data from the Human Mortality Database. Probability of surviving till 2004 is defined as the probability that someone born in a given year survives till his birthday in 2004. Trends are calculated based on the birth cohorts 1934-1939 and 1948-1956.

Appendix Figure 3: Probability of surviving till 2004 conditional on having reached age 2, males



Data are aggregated country*birth cohort*sex data from the Human Mortality Database. Probability of surviving till 2004 is defined as the probability that someone born in a given year survives till his birthday in 2004. Trends are calculated based on the birth cohorts 1934-1939 and 1948-1956.

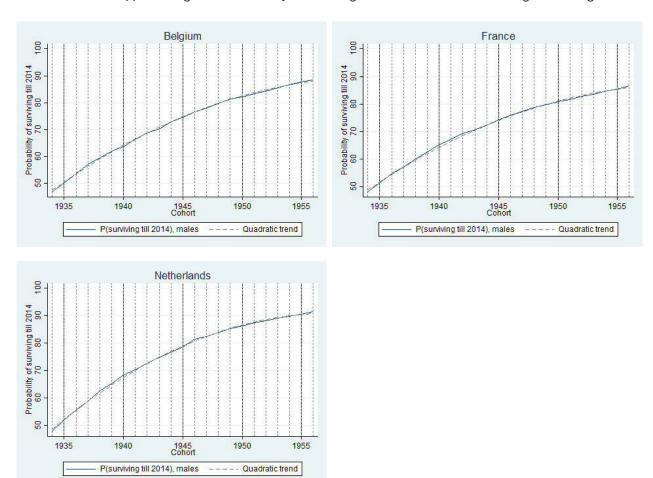
Appendix Figure 4: Probability of surviving till 2014 conditional on having reached age 2, females





Data are aggregated country*birth cohort*sex data from the Human Mortality Database. Probability of surviving till 2014 is defined as the probability that someone born in a given year survives till her birthday in 2014. Trends are calculated based on the birth cohorts 1934-1939 and 1948-1956.

Appendix Figure 5: Probability of surviving till 2014 conditional on having reached age 2, males



Data are aggregated country*birth cohort*sex data from the Human Mortality Database. Probability of surviving till 2014 is defined as the probability that someone born in a given year survives till his birthday in 2014. Trends are calculated based on the birth cohorts 1934-1939 and 1948-1956.