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WORKING PAPER 2012:15

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ISSN 1651-1166

# Long-run effects of gestation during the Dutch hunger winter famine on labor market and hospitalization outcomes

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July 3, 2012

**Abstract**

The Dutch Hunger Winter (1944/45) is the most-studied famine in the literature on long-run effects of malnutrition in utero. Its temporal and spatial demarcations are clear, it was severe, it was not anticipated, and nutritional conditions in society were favorable and stable before and after the famine. This is the first study to analyze effects of in utero exposure on labor market outcomes and hospitalization, and the first to use register data covering the full Dutch population to examine long-run effects of this famine. We provide results of famine exposure by sub-interval of gestation. We find a significantly negative effect of exposure during the first trimester of gestation on employment outcomes 53 or more years after birth. Hospitalization rates in the years before retirement are higher after middle or late gestational exposure.

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Keywords: nutrition, ageing, developmental origins, morbidity, income, health, employment.
We thank Bertie Lumey, Hendrik Jürges, Aliaksandr Amialchuk, Rob Euwals, Rob Alessie, Felix Weinhardt and participants at presentations in the TI and VU Amsterdam, Mannheim, Tilburg, Naples, Louvain, Autonoma, Buch am Ammersee, and Netspar Pension Day, ASHE, EEA, SMYE and iHEA Toronto conferences, for useful comments. Thanks to Netspar for financial support and to Statistics Netherlands for the use of the micro data registers, including those of the municipality of birth. First version: January 2012. JEL-codes: I10, I12, J01, J10, J13, J24.

# 1 Introduction

The Dutch Hunger Winter famine (December 1944-April 1945) is by far the most- studied famine in the epidemiological literature on long-run effects of in utero malnutrition (see Lumey, Stein and Susser, 2011, for an excellent survey). The reason for this is that this famine is uniquely suited as an instrument to assess the presence of such causal long-run effects. As has been well-documented (see Section 2), it is sharply defined in time and space, it was severe, it was not anticipated, it was embedded within an era with affluent nutritional conditions, and it occurred in a society with reliable data registration.

By now, long-run effects of exposure to the Dutch Hunger Winter famine have been found for a number of health outcomes later in life (see again Lumey, Stein and Susser, 2011). These results contribute to the overwhelming evidence on long-run health effects of early-life conditions in general (see e.g. the overviews in Pollitt, Rose and Kaufman, 2005, Barker, 2007, and Lawlor, 2008). These effects are typically explained by reference to Barker's fetal origins or fetal programming hypothesis (see e.g. Barker, 1994). In particular, effects of fetal undernutrition on metabolic adaptation in utero may affect the phenotype such that the risk of diseases later in life is increased (Hales and Barker, 1992, Bateson, 2001, Gluckman and Hanson, 2004), notably cardiovascular diseases, diabetes and hypertension. Underlying this model is the idea that several critical periods in utero influence the development of humans. During these periods, developing systems modify their settings in response to social and biological cues (Kuzawa and Quinn, 2009). This includes durable epigenetic changes that modify gene expressions. Of course, severely adverse nutritional conditions may also directly affect the build-up of organs and other body parts. All these biological causal pathways may potentially be influenced by events and decisions throughout life.

This paper is the first study to analyze effects of in utero exposure to the Hunger Winter famine on individual economic outcomes late in life. We consider three such outcomes. Two of these (annual labor earnings and employment) are labor market outcomes. The third, hospitalization, is an indicator of costly health care usage and as such combines information on health with information on its costs. In fact, we examine hospitalization occurrences by type of disease, notably for cardiovascular diseases and for cancer. Studying long-run effects on labor market outcomes is informative on the determinants of well-being of elderly workers and on the economic inequality among them.[1] Moreover, in the light of the current plans to increase the mandatory retirement

[1]Long-run effects of hunger early in life on late-life health and labor market outcomes are of obvious relevance for developing countries. But in developed countries poor children may also face hunger spells, and households may rely on programs like the "Women, Infants and Children program"

ages across Europe, it is interesting to know to what extent the elderly can be expected to remain productive at high ages or whether adverse conditions in utero on average cause adverse economic conditions later in life. The Hunger Winter famine enables us to address the latter while filtering out the effects of other systematic determinants of late-life conditions.[2]

We connect our results to those in the literature on long-run health effects of conditions in utero, and to those in the Hunger Winter famine literature in particular. To shape thoughts, one could postulate the following chain of events: [ in utero exposure to the famine ] → [ in utero malnutrition ] → [ adverse health later in life ] → [ adverse economic outcomes later in life ]. This chain takes long-run health effects to translate into effects on individual economic outcome variables. In particular, concerning the third arrow, notice that adverse health outcomes may negatively affect productivity, for example through cognitive ability or physical strength or through spells of sickness absence, while productivity in turn affects labor market outcomes. It is also possible that in utero malnutrition causes disadvantages in childhood, for example at school, and this in itself may hurt economic and health outcomes later in life as well.

There are two additional reasons why our analysis is novel. First, we provide results of famine exposure by sub-interval of gestation. This allows us to examine the presence of critical periods in utero for economic outcomes late in life. Secondly, we are the first to use register data covering the entire Dutch population to examine long-run effects of this famine. These data contain the month and municipality of birth as well as the above- mentioned economic outcomes.

The identification strategy exploits variation in the moment and place of birth. Those exposed to the famine in utero may be compared to two "control groups": those born in the famine-stricken area before and/or after the famine, and those born in similar but non-stricken areas during the famine. We distinguish between famine exposures by pregnancy trimester, and we assign trimester treatment statuses in accordance to the epidemiological literature of the famine. As the famine mostly affected urban areas in the Western part of the country, we restrict our sample to individuals born in cities. We also restrict attention to males, which is in line with Stein et al. (1975) and takes into account that only a small subpopulation of the females of this cohort is active in the labor market at advanced ages.

for additional nutritional aid. This WIC-program provides aid to approximately 9.1 million individuals in the US in 2009 and has a budget of 6.9 billion dollars (see Food and Nutrition Service, 2009). In addition, qualitative hunger, i.e. lack of appropriate nutrients, may have adverse effects on the fetus.

[2]The existing evidence on the long-run socioeconomic effects of malnutrition early in life is hampered by methodological concerns and data limitations. Notably, independent variation in initial nutrition and a sufficiently long observation window are required (see e.g. Van den Berg and Lindeboom, 2012).

A few famine-based studies exist that focus on economic outcomes as well. Neelsen and Stratmann (2011) use the Greek 1941-1942 famine to examine long-run effects on economic outcomes at high ages. The other existing studies consider economic outcomes at prime ages (up to 45) rather than high ages. Specifically, Almond et al. (2010), Chen and Zhou (2007) and Meng and Qian (2009) use the Chinese 1959-1961 famine, while Jürges (2011) relates the German 1945-1948 famine to outcomes in 1970 and 1987. These studies provide important insights into long-run effects of major and prolonged disruptive time periods in society (below we summarize results that are particularly relevant for our purposes). However, the study of these famines also poses a number of challenges in terms of empirical implementation. First, the famines are not as sharply defined in time and space as the Dutch Hunger Winter famine. Therefore, it is harder to distinguish between exposure in different intervals in gestation, and hence to identify critical periods in utero. It is also more difficult to distinguish between exposure in utero and exposure after birth. Secondly, these famines took place in less stable societies. Notably, the Chinese famine was followed some years later by the so-called Cultural Revolution (1966-1976). This potentially increases the risk of confounding and cohort effects.[3] Thirdly, the data are often less detailed. In many cases, the month of birth is not observed, exacerbating the complications with exposure mapping. In some cases, the birth place is not observed, and the city or area of residence in adulthood is used as an indicator of location at birth. Fourthly, obviously, studies where the number of years between famine and outcomes is less than 50 can not address long-run effects and hence the connection to the epidemiological literature on long-run effects is more complex. In terms of findings, the studies of the Chinese famine generally demonstrate adverse effects on labor market status, wealth and marital outcomes. Jürges (2011) finds effects on the level of education and on annual income, for a subset of the cohorts born in the famine (namely, those whose gestation started in the first part of the first winter in the famine). Neelsen and Stratmann (2011) do not find substantial effects of exposure to the Greek famine.

The remainder of this paper is structured as follows. Section 2 describes the famine and mentions epidemiological studies on health outcomes due to exposure to the famine. Section 3 describes the data. Section 4 presents the estimation results. We also provide a range of sensitivity analyses and we address the often-discussed issue that birth cohorts of individuals exposed to a famine in utero may on average be less frail than other birth cohorts, leading to possible selection biases. Section 5 discusses the results. We relate them to the existing literatures on long-run effects of early-life conditions and famines and on health effects by trimester of exposure to the Dutch Hunger Winter

[3]Long-run effects of reduced nutrition in utero are known to be stronger if the affected individuals are exposed to a much more favorable environment in childhood (Schulz, 2010).

famine. Finally, Section 6 concludes.

# 2 The Dutch Hunger Winter famine

## 2.1 Historical context

In the fall of 1944, towards the end of World War II, parts of the South of the Netherlands had been liberated by Allied forces, whereas the other parts were still occupied by German forces. The London-based "Dutch government in exile" called out a railroad strike in the occupied parts in order to support Allied military advances and in order to display its authority over the nation. As a reaction, the occupying forces initiated an embargo that prohibited food transports to the densely populated western part of the country, i.e. the provinces of North and South Holland and Utrecht. The decision by Allied Command to postpone the liberalization of the Netherlands and instead move east towards the German mainland caused a military stalemate in the western part of the Netherlands that lasted until the end of the war (European Theater) in May 1945. The effects of the food transport embargo were exacerbated by the early onset of a harsh winter, the freezing of the waterways, the generally bad state of transport infrastructure, and a naval trade blockade. As a result, the western part of the country was closed-off from any imports of food, fuel, medication, etc. This caused a famine in the western part that was particularly severe in the cities (Stein et al., 1975). The situation lasted until the end of the occupation which coincided with the end of World War II in Europe (early May 1945).

To study long-run effects, a famine must be an exogenous event with no long-run impact *apart* from the effect running through household-specific nutritional conditions. This prerequisite is more likely to apply if the famine is due to an external intervention in society and if the famine is short and is not anticipated. From the above it follows that the Dutch Hunger Winter famine satisfies these requirements.[4]

As noted above, we are in the fortunate position that a consensus has been reached concerning the temporal and spatial bounds of the famine. Figure 1 displays the area where the famine took place. Recall that we restrict attention to the cities. The North-east was sparsely populated and contained a large agricultural sector (see e.g. Stein et al., 1972). It was not subject to the food embargo. In accordance to the literature, we add its cities to those in the South in order to obtain the set of cities that were not

[4]Since a famine is not equivalent to exposure to hunger, the causal long-run effects of malnutrition in utero will exceed the reduced-form long-run effects of exposure to the famine in utero (see Van den Berg, Pinger and Schoch, 2011, for evidence in the context of the Dutch Hunger Winter famine). Selective fertility provides an additional cause of under-estimation; see Section 4.

exposed to the famine.

We illustrate the above description with some evidence on food rations and mortality. Figure 2 depicts the daily caloric averages of the official food rations by month in the western part of the Netherlands. The average food rations in the West dropped below 1000 kcal in December 1944 and they varied between 500 and 800 kcal from December 1944 to April 1945.[5] Immediately after the end of the occupation, rations rose to 2,400 kcal per day. During the famine, the official rations were supplemented by food from alternative sources, notably the black market. However, variation in the rations does reflect variation in the availability of food. Pregnant women received additional supplies until the start of the famine, whereas the rations of children below 1 year of age never dropped below 1000 kcal (De Rooij et al., 2010).

Figure 3 shows that a mortality plateau was reached in the months January-April 1945. The excess death rate in the first half of 1945 over the rate in 1944 amounts to 269 percent for men and 173 percent for women (Dols and Van Arcken, 1946). Banning (1946) reports a higher incidence of tuberculosis and hunger edema in the famine-stricken cities. Figure 4 indicates that mortality rates in the pre-famine war years are of a similar order of magnitude as in the years after 1945. Annual infant mortality peaks in 1945 (Figure 5). Figure 6 depicts age-specific mortality across years. Clearly, the bulk of excess mortality in 1945 is caused by the very young and the elderly.

Figure 7 depicts regional mortality rates. Mortality in the famine-stricken area clearly peaks in 1945, whereas this increase is only modest in the other areas. This figure also shows that the regional developments of mortality are similar in the years before and after the famine and that the levels in the years 1944 and 1946 are similar. This is important for our analyses later, where we rely on differences between areas over time.

## 2.2 Literature on long-run health effects of the Dutch Hunger Winter famine

As noted above, Lumey, Stein and Susser (2011) include a detailed survey of studies of long-run health effects of in utero exposure to the Dutch Hunger Winter famine. These studies restrict attention to outcomes up to age 60, simply because the number of years elapsed since the famine is insufficient for analyses of outcomes beyond that. High-age mortality, morbidity and death causes have not been examined yet. In particular, cardiovascular diseases (and hypertension) above age 60 have not been examined yet.

[5]The official recommendation by the Oxford Nutritional Surveys is 2500 kcal for males (Ravelli, 1999). During the famine, it was not unusual that food ration stamps could not be exchanged for food, due to a lack of food supply. Moreover, the composition of the food was subject to manipulation.

Because of the scope of our own study, we merely summarize results for men, and we ignore intergenerational effects and effects due to exposure after birth. We first discuss the evidence for physical outcomes and then turn to mental outcomes. Both physical and mental performance can be expected to affect labor productivity and hence labor income and employment.

The existing evidence of physical effects due to exposure to the Dutch Hunger Winter famine is strongest for outcomes related to diabetes. Glucose problems and diabetes are more common among individuals in their fifties who were exposed, for any gestational stage of exposure. High blood pressure is not more common at age 50 among those exposed, but evidence has been found for effects on individuals when they are in their late fifties, independent of the gestational stage of exposure. There is mixed evidence of an effect of exposure early in gestation on cholesterol problems and fat intake among individuals in their fifties. Severe obesity is more common at age 18 among those who were exposed, but this association is absent at age 50. No effects have been found for cancer among men, whereas there is not enough evidence yet for other potentially important outcomes such as cardiovascular diseases and self-reported health.

In a landmark study, Heijmans et al. (2008) show that individuals who were exposed to the famine in the early stages of pregnancy had, 60 years later, less DNA methylation of a certain imprinted gene, compared to unexposed same-sex siblings. The gene is the insulin-like growth factor II, which is a key factor in human growth and development. Their study provides strong empirical evidence for the epigenetic pathway mentioned in Section 1.

Cognitive ability and mental performance later in life are potentially important determinants of labor market outcomes in a modern economy. At age 18 there is no association between exposure and cognitive ability measures. At age 59 the evidence is mixed. De Groot et al. (2011) find no effects whereas De Rooij et al. (2010) find an effect on a selective attention task but not on a few other measures. There is no ambiguity about the elevated risk of schizophrenia and neural tube defects at adult ages after exposure early in gestation. Antisocial personality disorder and mood disorder are also more common after exposure, in the first and second trimester for the former, and in the second and third trimester for the latter disorder.

In sum, these studies provide reasons to expect effects on labor market outcomes and hospitalization. Diabetes and other physical impairments may reduce productivity and give rise to adverse labor market outcomes, and the same applies to mental impairments and disorders. Mental health in particular seems to be sensitive to exposure in early gestation. In Section 5 (where we discuss our empirical results) we return to these findings, and we also consider evidence from studies of other famines and studies

using other measures of adverse early-life conditions, in particular concerning cognitive ability.

# 3 Data

## 3.1 Population registers

In accordance to Stein et al. (1972), we distinguish between the Western part of the country and the "rest" (non-Western) part comprising the Northeast and the South. To determine whether the municipality of birth was a city or not, and in accordance to Stein et al. (1975), we use a threshold municipality size equal to 40,000 inhabitants in 1944. This results in 7 cities in the West (Amsterdam, Delft, Haarlem, Leiden, Rotterdam, The Hague and Utrecht) and 10 cities in the rest of the country (Breda, Eindhoven, Enschede, Heerlen, Groningen, Hengelo, Leeuwarden, Maastricht, Tilburg and Zwolle).[6]

From the Municipal Personal Records Database from Statistics Netherlands, we extract all individuals with the following characteristics: (*i*) male, (*ii*) born in one the 17 above-mentioned cities in the period August 1944-April 1946, and (*iii*) living in the Netherlands in January 1999 until 2004. The Municipal Personal Records Database is the longitudinal demographic register of the population. We do not have access to all variables in the register. Notably, the register includes the exact date of birth whereas we only observe the month of birth. Moreover, we observe the place of birth only for those still alive in 2004. The choice of the birth cohort interval (August 1944-April 1946) is motivated by Lumey et al. (1993).

The resulting sample size is 47627. These individual births are linked to the corresponding individual records from the national tax and hospital registration databases of Statistics Netherlands over the period 1999-2005. The tax files contain individual labor market information. The so-called LMR registration of hospitals includes over 9 million hospital records in 1999-2005. These data cover 95% of all hospitalizations in the Netherlands in the corresponding period. Both the date and the main reason for hospitalization according to the ICD-9-CM classification are recorded.

We use the month of birth to define the period in gestation during which exposure to the famine occurred. Specifically, we distinguish between 5 groups: the pre-famine born, the post-famine conceived, and the 3 treatment groups, i.e. exposed to the famine during the first, second or third trimester of gestation. Exposure in a particular trimester

[6]The cities of Arnhem and Nijmegen are left out of the analysis because of disruptions due to Operation Market Garden. Delft was left out by Stein et al. (1975) because of missing data in their sample.

is defined by the average official daily ration being less than 1000 kcal in that trimester. This results in the following five birth cohort subintervals and groups: (1) pre-famine birth: birth in August 1944-January 1945, (2) exposed in the third trimester of gestation: birth in February 1945-May 1945, (3) exposed in the second trimester of gestation: birth in June 1945-August 1945, (4) exposed in the first trimester of gestation: birth in September 1945-December 1945, and (5) conceived post-famine: birth in January 1946-April 1946. This classification follows Lumey et al. (1993) with one exception: we do not allow for overlapping trimesters.[7] For the non-West area we define five birth cohorts using the same five calendar time intervals. The moment of conception is determined by the month of birth, assuming a gestational period of 9 months. Sindram (1945) finds a famine-related reduction of the gestational period of only 4 days, so this seems a reasonable assumption. Groups 1 and 5 have not been prenatally exposed to the famine. However, note that Group 1 is exposed to the famine in the first year of life, which may have had adverse effects on long-run outcomes as well.

## 3.2 Outcome variables

Table 1 provides an overview of the outcome variables. Summary statistics are in Table 2. "Employment" records whether an individual has been in at least some paid employment in 1999. The cohorts considered in our analyses have access to generous Early Retirement (ER) schemes. Eligibility conditions depend on age. We therefore restrict ourselves in the analyses to employment in 1999, when the oldest cohorts are at most 56 years old, and the ER schemes are not yet effective. Men from the cities in the West are slightly less likely to work than men from the other cities.

The variable "labor income" is defined as the logarithm of total wage income earned in paid labor in 1999. It is only observed in case of employment. Table 2 shows that labor income is slightly higher among those from the Western cities.

"Hospital" is an ordered variable capturing whether the number of hospital visits for all diagnoses in the period[8] 1999-2005 equals 0, 1 or more than 1. In fact, most of the time we use a simple binary indicator, merging the two latter categories. It is needless to point out that bad health is not equivalent to a high number of hospital visits.

[7] According to the rationing criterion, individuals born in the months May-June 1945 and August-September 1945 are exposed in two trimesters. We assign exposure of individuals born in May to the third, in June and August 1945 to the second, and in September 1945 to the first trimester of gestation.

[8] Notice that individuals in the sample may die in 2004 or 2005, which mechanically reduces the time interval for the number of visits. However, such mortality is low, and the results are not affected if we omit those who died. We could define hospitalization based on 1999-2003 but this reduces the variation in the outcome and hence increases the imprecision of the estimation results.

Hospital visits may also be part of preventive health care. Moreover, visiting a hospital involves psychological costs. However, in the Dutch health care system, physicians act as first-line caregivers and as gate-keepers. They regulate access to care in hospitals and they provide most of the preventive care. This is also evident from our data: on average around 90% of the individuals (aged between 53 and 61) do not go to a hospital in any given year. Visits to physicians are much more common. We therefore feel that our outcome variable captures the health dimension that involves most costs.

"CVD" is analogously defined as "hospital", but it is restricted to hospitalizations for cardiovascular reasons (ICD-codes 390-459). Cancer is a binary indicator indicating a cancer-related hospital record (ICD-codes 140-208). Notice that the seven-year averages of the cause- specific hospitalization variables are low. The annual averages are about 0.02 for CVD and 0.005 for cancer. A priori we do not expect large effects of prenatal famine exposure on cancer (Van den Berg, Doblhammer and Christensen, 2011).

Individuals born in the Western cities are slightly less likely to visit a hospital and are also slightly less likely to do so for cardiovascular reasons. They are slightly more likely to visit the hospital for cancer-related diagnoses.

# 4 Estimation results

## 4.1 Results based on between-cohort variation in the famine-stricken cities

In this subsection, we use the pre-famine and post-famine birth cohorts in the cities in the West as "controls" when estimating the long-run effects of famine exposure in utero. Thus, we only use the subpopulation of individuals born in those cities. We perform the following regression,

$$y_i = \beta_0 + \beta_1 Q_{1,i} + \beta_2 Q_{2,i} + \beta_3 Q_{3,i} + \beta_4 P_i + \varepsilon_i \tag{1}$$

where $y_i$ is an outcome value of individual $i$ and $Q_{j,i}$ are binary indicators for birth in the cohort that was exposed to the famine in gestational trimester $j$. The binary indicator $P_i$ equals one iff the individual is born in the pre-famine cohort. Together, $Q_{1,i}, Q_{2,i}, Q_{3,i}$ and $P_i$ characterize the 5 birth-cohort groups, where the post-famine born cohort represents the baseline level. For example, a man $i$ born in Amsterdam in March 1945 (implying famine exposure in the 3rd trimester) has $Q_{3,i} = 1$ and $Q_{2,i} = Q_{1,i} = P_i = 0$.

Table 5 presents the results. Not surprisingly, they are in broad agreement to the

sample statistics by group, presented in Table 3. We do not discuss the results in these tables separately.[9] Individuals exposed to the famine in the first or second trimester of gestation have significantly lower rates of being employed in 1999, compared to the pre- and post-famine cohorts. The three in-utero exposed cohorts have lower labor income in 1999 than the pre- and post-famine groups, but these differences are insignificant.

All 3 prenatally-exposed cohorts face higher hospitalization rates in 1999-2005 in comparison to the surrounding cohorts. These differences are highly significant. The prenatally-exposed cohorts also face significantly higher CVD rates compared to the surrounding cohorts in these famine-stricken cities. There seems to be no effect of the famine on cancer-specific hospitalization rates in the famine- stricken cities.

To proceed, we also estimate the regression (1) separately for the subpopulation of individuals born in the cities in the (non-West) *rest* of the country. In this area there was no famine. We define $Q_{j,i}$ as a binary indicator for the calendar time interval in which birth took place, as follows: if the man would instead have been born in the West in this interval then this would entail famine exposure in gestational trimester $j$. $P_i$ is defined analogously. As an example, consider a man $i$ born in Eindhoven in March 1945. If he were born in the West then he would be exposed to the famine in the 3rd gestational trimester; hence, $Q_{3,i} = 1$ and $Q_{2,i} = Q_{1,i} = P_i = 0$. The regressions for those born in the non-West cities are informative on the extent to which conditions in the famine period were affected by nationwide cohort-specific events that are not due to the famine. If present, such events may affect the interpretation of the results in regression (1) for the famine-stricken cities, in the sense that such differences may be partly explained by such events and not by the famine.

Table 6 presents the results. Again, not surprisingly, they are in broad agreement to the sample statistics by group, presented in Table 4. Significant differences indicate that those born in the non- famine area have unusually low or high outcomes later in life if they were in utero in the period when the famine struck the West. Note that these are not effects due to the famine.

We find some evidence that in the non-West area, employment in 1999 among those born in June 1945-August 1945 (i.e., in the so-called second-trimester interval) is lower than in the cohorts corresponding to the pre- and post-famine period. The same applies to income in 1999 among those born in February 1945-May 1945 (i.e. in the third- trimester interval) although here the regression coefficient is not significant. There are also some between-cohort differences in hospitalization rates and CVD rates in 1999-2005. Notably, those born in the so-called pre-famine interval and in the first-

[9]The main difference between the statistical tests reported in these two tables is that the tests in Table 3 lump the pre- famine and post-famine cohorts together into one control cohort whereas the tests in Table 5 are with respect to the post-famine cohort.

trimester interval have significantly larger regression coefficients than those born in the post-famine interval. The pattern for the cancer rate in 1999-2005 seems to be driven by age effects: it is monotonically decreasing as a function of the birth date.

Thus, it seems that in terms of the long-run effects we are after, the time interval in which the Dutch Hunger Winter famine took place in the West may have been unusual not only because of the famine but also in some other respects, for those in gestation. Some additional evidence for unusual circumstances is provided by the facts that in the non-West part of the country, stillbirth rates and infant mortality rates were higher in the cohorts that were in gestation around the time interval of the famine, and over-all mortality was higher in 1945, than in surrounding years (see Stein et al., 1975, Figure 7, and archives of Statistics Netherlands).

We may distinguish between different explanations for this. First, the winter of 1944/45 was unusually harsh, and this may have had adverse effects on those in famine-stricken and non-stricken areas alike, for example due to exposure to infectious agents. This motivates a correction of the results for the cities in the West with the results for those in the non-West. Second, the non-West may have been subject to food shortages as well. In that case, a correction of the results for the West with the results for the non-West would entail results of the effect of the famine in the West relative to the effect of food shortages in the non-West. It would then be more informative to restrict attention to the results using cohorts born in the West only.

There is some evidence of food shortages in the East. There were no serious shortages in the South. However, stillbirth rates and infant mortality were higher in the cohorts that were in gestation around the time interval of the famine than in surrounding cohorts (Stein et al., 1975, and archives of Statistics Netherlands). Annual over-all mortality in the cities in the South was elevated in 1944 and 1945. The maximum level was attained in 1944, but this may be due to the fact that the front line of World War II was in the South in the second half of 1944.

The results in this subsection motivate the joint analysis of all available cohorts and regions. This should also take into account (i) that the five different groups we define cover different seasons of the year, (ii) that the outcomes are measured at ages that depend on the membership of the group, and (iii) that there may be a systematic time-invariant difference between outcomes for those born in the West and those born in the non-West.

## 4.2 Results based on variation within and between cohorts

In this subsection we simultaneously use all available information, by estimating the following regression model,

$$y_i = \beta_0 + \beta_1 Q_{1,i} F + \beta_2 Q_{2,i} F + \beta_3 Q_{3,i} F_i + \beta_4 Q_{1,i} + \beta_5 Q_{2,i} + \beta_6 Q_{3,i} + \beta_7 F_i + \beta_8 X_i + \varepsilon_i \quad (2)$$

where $F_i$ is an indicator of a birth in a city in the West. Since the sample size is larger than in the regressions of the previous subsection, we have some additional flexibility at our disposal. We therefore replace the pre-famine indicator $P_i$ by a vector $X$ that contains a linear function of the birth in calendar time (where the latter is measured in reverse chronological order, from the value 21 in August 1944 to the value 1 in April 1946). This controls for smooth secular cohort effects over time. Moreover, since the outcomes are measured at a common point in calendar time, it also controls for age effects on the outcome. The vector $X$ also contains indicators of the month of birth, in order to control for season-of-birth effects (Doblhammer and Vaupel, 2001).

The interaction terms $Q_{j,i}F_i$ capture exposure to the famine in gestational trimester $j$. Hence, the corresponding coefficients $\beta_1, \beta_2, \beta_3$ are the parameters of interest. They represent the effect of exposure over and above the effect of birth in a city in the West, and over and above the effect of being born at a particular moment in time (the latter effect abstracting from famine exposure). The effect of birth in a city in the West is represented by $\beta_7$ and the effect of being born at a particular moment in time is represented by $\beta_4, \beta_5, \beta_6$ and $\beta_8$. Intuitively, the effect of being born at a particular moment in time is identified by the outcomes of those born in non-West cities, and the effect of birth in a city in the West is subsequently identified by the outcomes of those in the pre- and post-famine cohorts in the cities in the West. In sum, we use the variation between cohorts and between areas to identify the effects of famine exposure by gestational trimester.

Table 7 shows the estimation results. In comparison to those in the previous subsection, we find, as expected, that the estimated famine effects are qualitatively similar but somewhat less strong. The only significant effect on labor market outcomes later in life is the effect of famine exposure in the first gestational trimester on employment. All 3 prenatally-exposed cohorts face significantly higher hospitalization rates in 1999-2005. These cohorts also face higher CVD rates, but the effect is only significant for exposure in the second trimester. We do not find significant effects of the famine on cancer-specific hospitalization rates. The linear effect of the (reverse-measured) month of birth is significantly positive for labor income. This means that income in 1999 is positively correlated to age in 1999. Similarly, all hospitalization rates in 1999 are positively related to age in 1999, but the latter relations are not significant.

In the light of the large sample sizes, one might have expected more coefficients to be significantly different from zero. The explanation of this apparent paradox is that the magnitude of the estimated long-run effects is small. The latter is in line with

the small differences between mean outcomes per area and birth group, as reported in Subsections 3.2 and 4.1. For example, the estimated effect of exposure in the first gestational trimester on the employment probability is slightly less than 3 percentage points, implying that the employment rate changes from say 73% to 76% in a cohort.

In Section 5 we discuss the results presented here, and we relate them to other studies.

## 4.3 Sensitivity analyses

We perform a range of additional analyses to assess the robustness of the results. In this subsection first examine alternative model specifications and then turn to alternative variable definitions and sample selections. In each case, the parameter estimates are available upon request.

First, we divide the single "non-West" area into separate North/East and South areas. Specifically, we include a separate indicator for North/East versus South. The results are not sensitive to this. As an alternative, we include binary indicators for the city of birth to control for within-city differences in conditions that are constant over time. The estimates of interest are virtually identical to those reported. We also consider alternatives for the linear functional form for the function capturing the effects of smooth secular changes in society on mean outcomes. It turns out that adopting higher-degree polynomials, or adopting a binary "pre- famine" indicator for birth before the onset of the famine, does not affect the coefficients of interest. The same applies if we omit the 11 binary indicators for the month of birth.

So far we have not used the information on the number of hospital visits in 1999-2005 beyond the binary indicator for the number being positive. To proceed, we estimate ordered probit models, distinguishing between 0, 1 and more than 1 visits. The estimation results are virtually identical to those reported in the previous subsections. In yet another specification, we estimate probits for the hospitalization outcomes in the year 1999 and in the year 2005 separately. Clearly, this reduces the variation in outcomes considerably, and as a result all coefficients are insignificantly different from zero, for 1999 as well as for 2005.

We also examine whether the results depend on the choice of the birth cohort observation window which is rather small. We re-estimate the models with the windows July 1944-May 1946 and June 1944-June 1946. The coefficients of interest are not out of line with those reported in the paper. The same applies if we omit all observations born before December 1944, i.e., all those born before the famine.

So far we have not exploited the data on labor market outcomes in 2000-2005. Mechanical re-estimation of our models with data from one of these years is not very

interesting because, as explained in Section 3, the results are confounded by early-retirement schemes. We may estimate a dynamic model for the age at which the individual leaves employment conditional on being employed in 1999, and make the transition rate dependent on famine exposure. This model takes the early-retirement schemes into account. However, the employment requirement for 1999 creates a selection problem. It turns out that the results are dominated by the effect of current age, reflecting early-retirement decisions.

## 4.4 Cohort size and selection effects

As in any study of long-run effects of in-utero exposure to famines, the survivors who were exposed to the famine may be systematically different in terms of unobserved characteristics than the survivors who were not exposed to the famine. This selectivity may lead to biased inference. In our setting one may distinguish between three phases of selection: at conception, in utero, and between birth and survival at high ages. First, the conception rate may systematically differ between an exposed cohort and the corresponding control cohorts. Specifically, families living in poor conditions may experience a particularly strong fertility reduction during the famine. This is relevant for the evaluation of the effect of famine exposure in the first gestational trimester. The evaluation of the effects of famine exposure in the second or third trimester is not affected by this first selection phase, because the exposed individuals were conceived before the famine, and the famine was not anticipated. Secondly, death in utero may occur more frequently during the famine, especially among frail fetuses (where frailty refers to individual characteristics that are not driven by the famine and that are not observed in our data). The same applies to spontaneous abortions and stillbirths. Thirdly, it is conceivable that mortality in between birth and 2004 depends on famine exposure. Such mortality may disproportionally affect the frailer individuals in the cohort.

As a result of all this, the birth cohorts of those exposed in the first, second or third gestational trimester may on average have less frail characteristics than the corresponding control cohorts. This difference may be exacerbated at high ages. All this tends to reduce the observed difference in outcomes between the exposed cohorts and the corresponding control cohorts. In sum, the estimated effect underestimates the causal effect of exposure in absolute value.

Our data are not suitable to study the extent of selectivity, since they only contain the cohort members who are actually conceived, born alive, and still alive in 2004. However, other data and other studies provide some insights. We focus on selectivity in terms of the cohort composition at (i.e. immediately after) birth.

As a starting point, we examine the size of the birth cohorts. Figure 8 indicates that the average national fertility fell in 1945 and increased strongly in 1946. From some simple interpolations of the sizes of cohorts that were not exposed in utero, we infer that the exposed cohorts are about 20%, 30% and 55% smaller than in the absence of the famine, for those exposed in the third, second and first trimester in gestation, respectively. It is plausible that the reduction is strongest for the cohort exposed in the first trimester, as this cohort suffers both from a reduced conception rate and from an increased in utero mortality rate, whereas the cohorts exposed in the second or third trimester only suffer from the latter. Roseboom and Van de Krol (2010) show that during the famine itself, about half of the women in the exposed areas did not menstruate. This is in line with the 55% reduction in the size of the cohort of those exposed in the first trimester of gestation.

A reduction in cohort size does not automatically entail a change in composition. Unfortunately, we have no information on parental characteristics to capture the parents' socioeconomic class. Stein et al. (1975) observe parental occupations, using military conscription data of the cohorts born in and around the famine. They find that the higher the occupational category of the father, the lower the reduction of the birth rate of the cohorts exposed in utero, as compared to the non-exposed cohorts. Painter, Roseboom and Bleker (2005) show that this finding does not change when adjusting for maternal characteristics such as age, parity, weight and weight gain during gestation. This suggests that the exposed cohorts consist on average of less frail individuals, and this may lead us to underestimate the magnitudes of the average causal effects of exposure.[10]

The analysis could in principle also be confounded by educational reforms after the famine that took place in such a way that exposed and (some of the) non- exposed cohorts faced different educational systems. However, there were no major educational changes until 1968 (see Dodde, 1983). The latter reform occurred too late to impact the schooling outcomes of the birth cohorts we use.

# 5 Discussion

Concerning labor market outcomes at ages around 55, the main finding is that the probability of employment is significantly lower if the individual was exposed to the famine during the first trimester of gestation. The quantitative effect is small, but this

[10]The Trivers and Willard (1973) hypothesis states that the gender composition or sex ratio of the offspring under adverse conditions is biased towards girls. Stein et al. (2004) and Cramer and Lumey (2010) study sex ratio effects of the famine. They find no evidence of an effect. This provides an argument against severe selectivity of birth cohorts.

may be due to selectivity of those who are conceived during the famine and subsequently survive gestation. The qualitative result can be connected to the literature on the long-run effects of adverse conditions early in life on cognitive ability later in life. Notably, as we have seen in Subsection 2.2, there is evidence for a lower cognitive ability at similar ages among those who were exposed in their first gestational trimester to the Dutch Hunger Winter famine. (The latter evidence is not uniform across ability measures, but that may be a result of selection effects as well.) Plausibly, the lower cognitive ability has led to a lower productivity and hence a lower employment probability. Since there does not seem to be any cognitive effects at age 18, the cognitive ability reduction at age 55 would be the result of a decline somewhere in between. An additional role may be played by mental disorders that reduce productivity in the labor market, notably schizophrenia, neural tube defects, and antisocial personality disorder. After all, these are also more common upon exposure in the first trimester of gestation. The prevalence of these disorders is relatively low, but their productivity effects may be severe.

The above argument fits in with results from studies that examine adverse nutritional conditions early in life that are different from famines. The occurrence of Ramadan during the first month of pregnancy increases the risk of mental/learning disabilities, among prime-aged men in Muslim populations in Uganda and Iraq (Almond and Mazumder, 2011). This confirms the importance of early gestation for cognitive and mental outcomes. Notice that Ramadan is fully anticipated and may involve a temporary adoption of a different lifestyle, which makes the exposure effects potentially different in magnitude from famine exposure effects. Doblhammer, Van den Berg and Fritze (2011) relate cognitive functioning beyond age 60 to exogenous and unanticipated macro-economic fluctuations in the year(s) of birth and gestation, using data from 11 countries. It turns out that a recession around birth significantly decreases cognitive ability in most dimensions (including numerical, verbal, and over- all). In terms of the adversity of nutritional conditions, recessions are mild in comparison to famines, so selective fertility is less of an issue. However, business cycle fluctuations are hard to use for separate analyses by gestational stage, since they are typically only measured annually. In any case, the results demonstrate that cognitive ability at higher ages is responsive to early-life economic conditions. This is further confirmed by Van den Berg et al. (2010) who find that among individuals born in the Netherlands in recessions, the decline in mental fitness after experiencing a negative life event at high ages, such as stroke, surgery, illness or death of a family member, is worse. If such life events are more common or more intrusive at high ages than at prime ages, their result may explain why adverse cognitive effects are found more often among the elderly. This in turn may mean that employment effects increase as individuals approach retirement.

The individual productivity may also be affected by adverse physical health condi-

tions among those exposed. However, the hospitalization effects are weaker for those exposed in the first trimester than for those exposed in late gestation. This suggests that physical effects are not the major driving force behind the employment effect.

The effects on labor income are not significant. Labor income and the sample for which it is observed are truncated from below by minimum wages. In fact, the effective truncation point may often be much higher than that, because elderly individuals in the Netherlands in the 1990s had access to generous disability and UI schemes in the case of job loss. This may weaken the extent to which income reflects the mean productivity within the cohort.

The cause-specific hospitalization results provide a major confirmation of the epidemiological literature on the developmental origins of late-life health. Specifically, this literature points at an impact on cardiovascular conditions but not on cancer (see the overviews mentioned in Section 1 and Van den Berg et al., 2010). Variation in the overall hospitalization variable is partly driven by variation in CVD and, to a lesser extent, cancer. Moreover, it is driven by other health problems that are known to be responsive to famine exposure in utero. In this sense it is not surprising that hospitalization depends on exposure in any gestational trimester.

The CVD effects are larger for exposure in the second gestational trimester than for exposure in the third (although the difference between the two coefficients is not significantly different from zero). The epidemiological literature typically finds that nutritional conditions during the third trimester are most important (see the overviews cited in Section 1 and references therein). Much of this literature considers effects on cardiovascular mortality or morbidity at higher ages (see also Van den Berg, Doblhammer and Christensen, 2011, who find that the cardiovascular mortality rate depends more strongly on the business cycle in the third trimester than in the second trimester, although, again, the differences are not significant). For most individuals, mortality occurs beyond the ages at which we observe our outcome variables. It is therefore not inconceivable that the effect on cardiovascular mortality and morbidity at higher ages is driven by conditions in the third trimester whereas the CVD effects at ages around 55 are also driven by conditions earlier in gestation. This is a topic for further research.

We found that the linear effect of the month of birth is significantly negative for labor income, meaning that the age in 1999 is positively correlated to the income among employed individuals in 1999. This probably just reflects increasing age-income career profiles. An alternative explanation is that individuals from older cohorts with a low labor income exited employment just before 1999 more frequently than individuals from younger cohorts with a low labor income while the exit rate was identical for high-income individuals. Such an exit pattern could again be due to the generous benefits schemes for elderly workers in the late 1990s. However, in that case we would have

expected a negative relation between age in 1999 and employment in 1999, and this is not what we find.

# 6 Conclusions

This paper establishes a connection between individual in-utero exposure to nutritional deficiencies on the one hand, and labor market outcomes and hospitalization indicators of the same individuals more than 50 years later on the other hand. The most striking result is that the probability of being employed is significantly lower in case of exposure during the first trimester of gestation. This result can be connected to the existing evidence on long-run effects of adverse conditions early in life on cognitive ability later in life. We conclude that the employment effect is most likely at least partially the result of a reduced cognitive ability but not a reduced physical ability. Mental illnesses and disorders may contribute to the ability pathway although their quantitative importance is probably small. As predicted by the epidemiological literature on the developmental origins of late-life health, we find significant effects of exposure on cardiovascular conditions but not on cancer.

The results include effects on hospitalization rates that provide inputs for assessments of the costs of adverse conditions in utero. The results of the paper are also useful in the light of current plans to increase mandatory retirement ages. We find that adverse conditions in utero restrict the productivity of the elderly. The extent to which individuals from poor countries or from countries that used to be poor in the 1940s or 1950s can be expected to continue working at a minimum necessary productivity level beyond the current retirement ages may be limited.

The paper uses data that are unprecedented in the literature on long-run effects of in-utero exposure to nutritional deficiencies. They cover the full population of the Netherlands in 2004, including records from the income tax and hospitalization registers. Observation of the month and municipality of birth allows us to exploit the fact that the Dutch Hunger Winter famine is sharply defined in time and space. Moreover, the famine's duration was so short that it enables inference by gestational stage. Nevertheless, the variation in individual circumstances around birth and in childhood is not recorded beyond the date and place of birth. Sibling pairs can not be identified. It is therefore a challenge for future research to widen the setting by including additional registers and/or survey data on individual conditions early in life. This might allow us to control for variation in those conditions and to examine to what extent decisions and events after birth are able to mitigate the effects of adverse nutritional conditions in utero. Similarly, it would be interesting to obtain data on outcomes at ages be-

yond retirement. This should enable a more precise assessment of the relevant causal pathways. The retirement literature has documented strong effects of health on labor market withdrawal. Therefore, it may be interesting to take the retirement patterns of different cohorts into account. Since the famine cohorts have only just passed the mandatory retirement age, we leave this to future work.

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# Appendix

Figure 1: The Netherlands by inhabited region, 1945

Figure 2: Daily kcal averages of the official food rations by month, Western Netherlands, 1941-1945 (source: National Bureau of Food Distribution in Wartime)

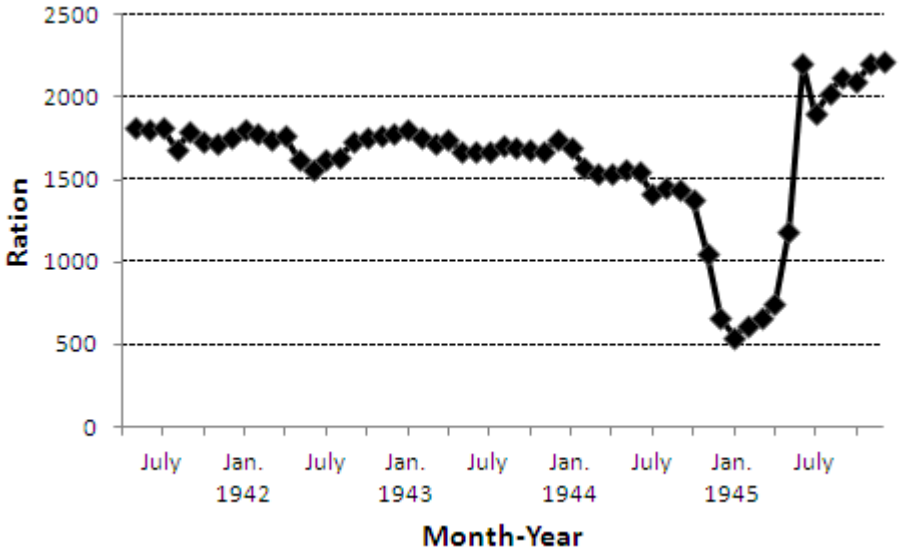


Figure 3: Monthly mortality per 1,000 inhabitants, the Netherlands, 1944-1945 (source: Statistics Netherlands Archive)

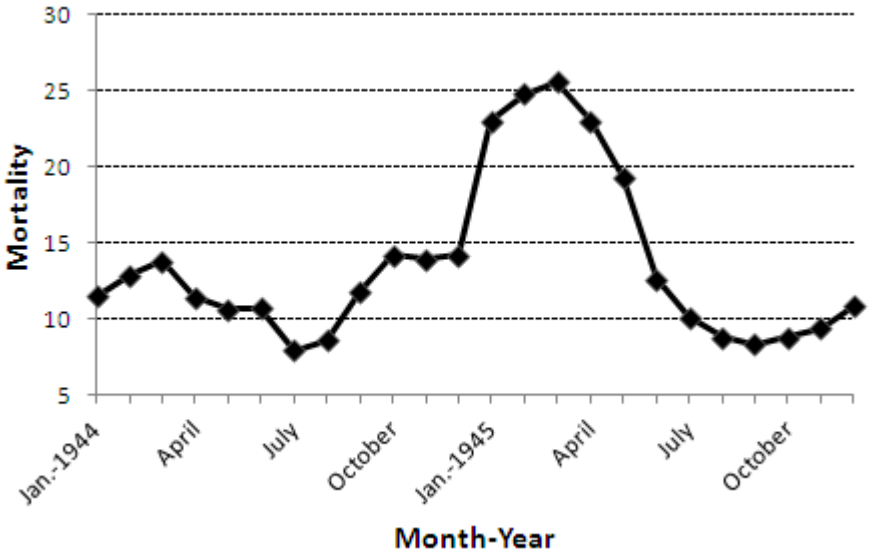

Figure 4: Yearly mortality per 1,000 inhabitants, the Netherlands, 1935-1949 (source: Statistics Netherlands, statline.cbs.nl)

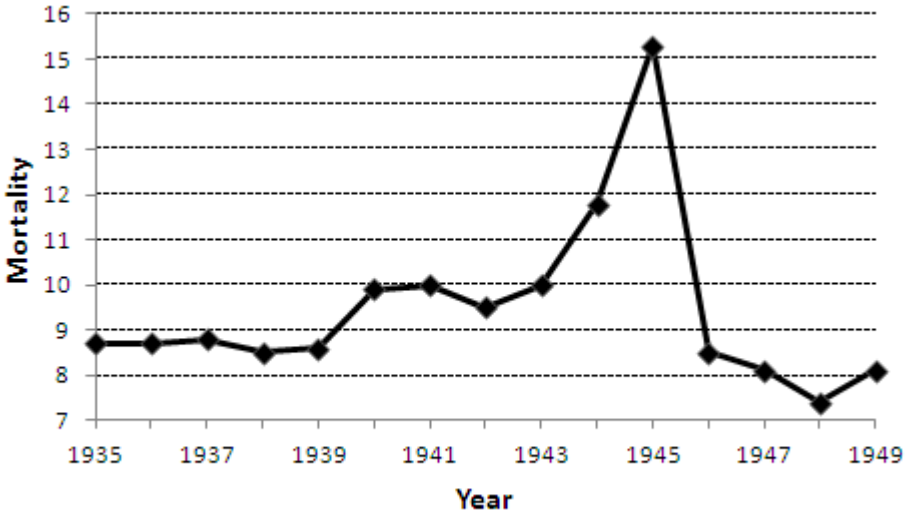


Figure 5: Deaths within the first year of life per 1,000 live births, the Netherlands, 1940-1949 (source: Statistics Netherlands, statline.cbs.nl)

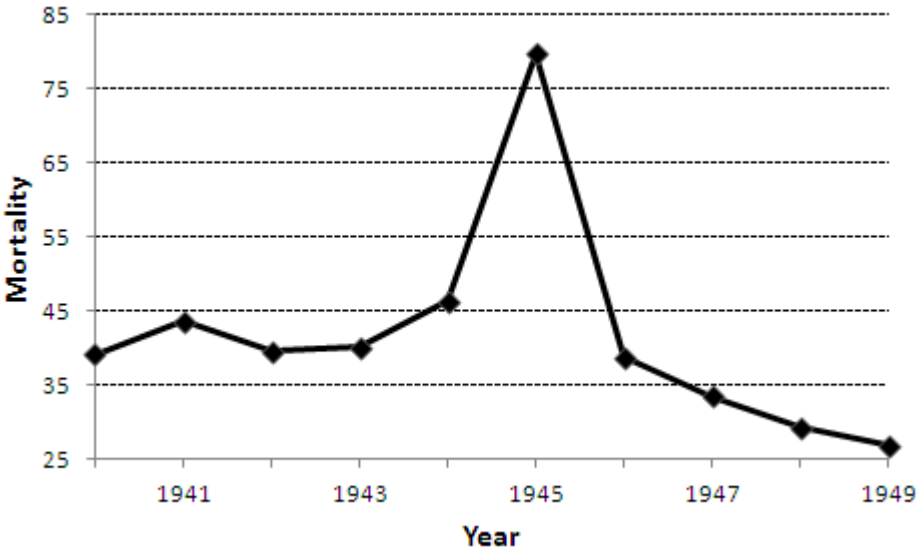

Figure 6: Composition of mortality by age and birth year, the Netherlands, 1943-1947 (source: Statistics Netherlands Archive)

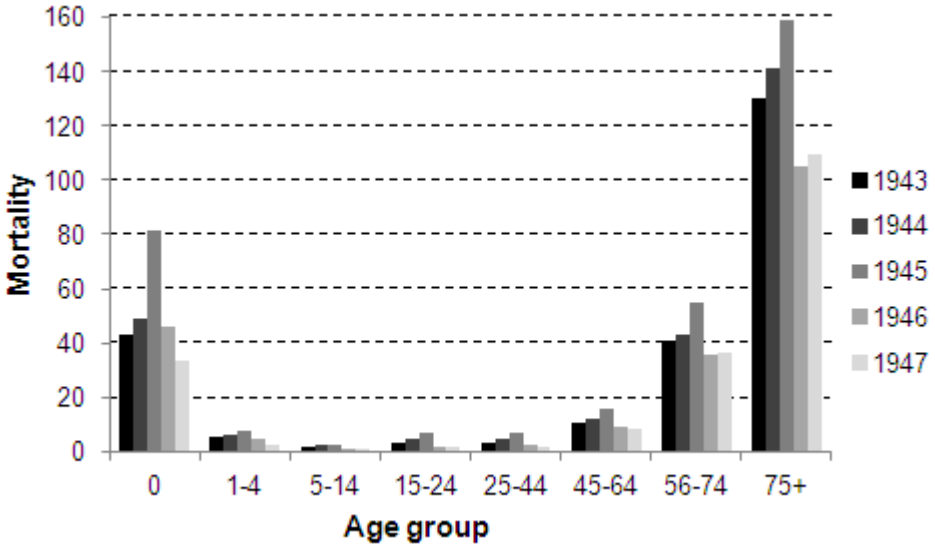


Figure 7: Yearly mortality per 1,000 inhabitants by region, 1941-1949 (source: Statistics Netherlands Archive)

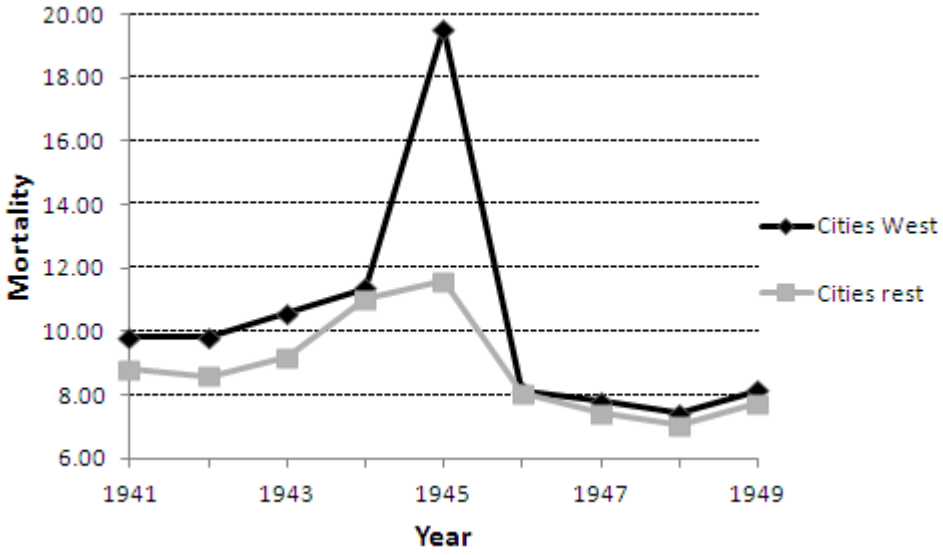

Figure 8: Live births per 1, 000 inhabitants, the Netherlands, 1940-1949 (source: Statistics Netherlands, statline.cbs.nl)

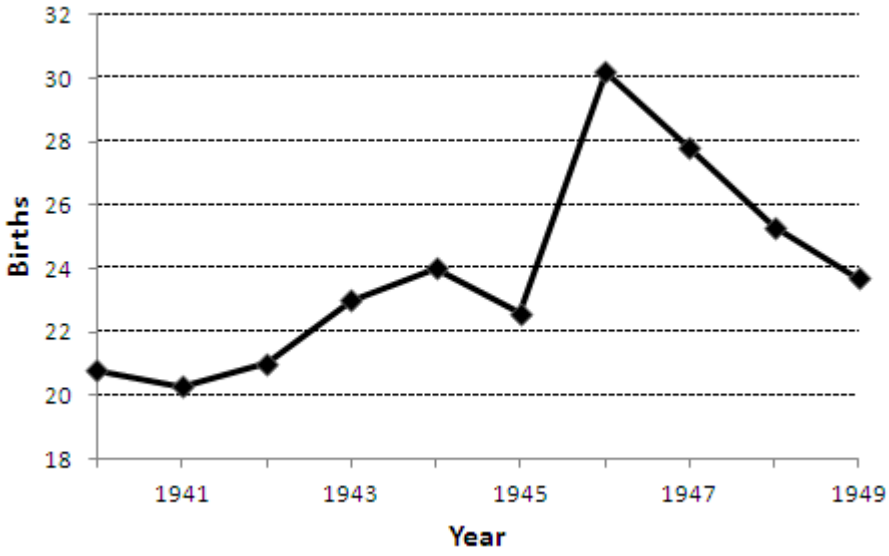

Table 1: List of outcome variables

| **Outcome** | **Definition** |
|---|---|
| *Labor market* | |
| Employment | Binary indicator for whether the individual is in paid employment in 1999. |
| Labor income | The logarithm of the fiscal labor income in 1999. |
| *Hospitalization* | |
| Hospital | Binary indicator for at least one hospital visit (all causes) in 1999-2005. |
| CVD | Binary indicator for at least one cardiovascular-related hospital visit in 1999-2005, i.e. ICD-9 CM codes 390-459. |
| Cancer | Binary indicator for at least one cancer- related hospital visit in 1999-2005, i.e. ICD-9 CM codes 140-208. |

Table 2: Descriptive statistics

| | **Cities West** | **Cities rest** |
|---|---|---|
| # individuals | 31039 | 16588 |
| | | |
| *Birth group* | | |
| Pre-famine | 9302 | 4046 |
| Trimester 3 | 6174 | 3199 |
| Trimester 2 | 3788 | 2311 |
| Trimester 1 | 3131 | 3041 |
| Post famine | 8644 | 3991 |
| | | |
| *Labor market outcomes* | | |
| Employment | .754 | .760 |
| | (.431) | (.427) |
| Labor income | 10.403 | 10.309 |
| | (.737) | (.757) |
| | | |
| *Hospitalization outcomes* | | |
| Hospital | .410 | .417 |
| | (.492) | (.493) |
| CVD | .124 | .131 |
| | (.330) | (.338) |
| Cancer | .033 | .031 |
| | (.179) | (.173) |

Note: standard deviations within brackets

Table 3: Cohort means of outcome variables - cities West

| | **Labor market outcomes** | | **Hospitalization outcomes** | | |
|---|---|---|---|---|---|
| | Employment | Labor income | Hospital | CVD | Cancer |
| Pre-famine | .757 | 10.403 | .406 | .116 | .036 |
| Trimester 3 | .759 | 10.401 | .425 * ** | .132 * ** | .034 |
| Trimester 2 | .739 * ** | 10.396 | .422 * ** | .135 * ** | .031 |
| Trimester 1 | .729 * ** | 10.401 | .423 * ** | .130 * * | .032 |
| Post-famine | .761 | 10.406 | .394 | .119 | .031 |

Notes: * , ** and *** denote the significance level of the t-statistic of the one sided t-test comparing the exposed group vs. the pre- and post-famine comparison groups, allowing for unequal variances.
* p<0.1, ** p<0.05, *** p<0.01.

Table 4: Cohort means of outcome variables - cities rest

| | **Labor market outcomes** | | **Hospitalization outcomes** | | |
|---|---|---|---|---|---|
| | Employment | Labor income | Hospital | CVD | Cancer |
| Pre-famine | .760 | 10.314 | .440 | .136 | .040 |
| Trimester 3 | .760 | 10.280 ∗ ∗ | .416 | .132 | .031 |
| Trimester 2 | .747∗ | 10.314 | .411 | .129 | .028 |
| Trimester 1 | .762 | 10.325 | .418 | .137 | .027 |
| Post-famine | .768 | 10.312 | .396 | .123 | .027 |

Notes: * , ** and *** denote the significance level of the t-statistic of the one sided t-test comparing the exposed group vs. the pre- and post-famine comparison groups, allowing for unequal variances.
* p<0.1, ** p<0.05, *** p<0.01.

Table 5: Estimates of famine exposure effects, based on between-cohort variation in the famine-stricken cities

| | **Labor market outcomes** | | **Hospitalization outcomes** | | |
|---|---|---|---|---|---|
| | Employment | Labor income | Hospital | CVD | Cancer |
| Trimester 3 | −.005 | −.005 | .078 ∗ ∗∗ | .065 ∗ ∗ | .046 |
| | (.023) | (.014) | (.021) | (.027) | (.041) |
| Trimester 2 | −.068 ∗ ∗ | −.010 | .070 ∗ ∗∗ | .077 ∗ ∗ | .011 |
| | (.026) | (.016) | (.025) | (.031) | (.048) |
| Trimester 1 | −.100 ∗ ∗∗ | −.005 | .074 ∗ ∗∗ | .054 | .023 |
| | (.028) | (.019) | (.026) | (.033) | (.051) |
| Pre-famine | −.013 | −.003 | .031 | −.015 | .071∗ |
| | (.021) | (.013) | (.019) | (.024) | (.036) |
| # individuals | 31039 | 23391 | 31039 | 31039 | 31039 |

Notes: * p<0.1, ** p<0.05, *** p<0.01. Employment, hospital, CVD and cancer estimated by binary probit; labor income by OLS. Robust s.e.'s within brackets.
Notice that the chronological order goes from "pre- famine" to trimesters 3, 2 and 1.

Table 6: Estimates of cohort variation in outcomes in cities in the rest of the country

| | **Labor market outcomes** | | **Hospitalization outcomes** | | |
|---|---|---|---|---|---|
| | Employment | Labor income | Hospital | CVD | Cancer |
| Trimester 3 | −.026 | −.032 | .050∗ | .044 | .063 |
| | (.033) | (.021) | (.030) | (.038) | (.060) |
| Trimester 2 | −.066∗ | .002 | .037 | .029 | .014 |
| | (.036) | (.023) | (.033) | (.042) | (.068) |
| Trimester 1 | −.019 | .013 | .055∗ | .065∗ | .008 |
| | (.033) | (.020) | (.030) | (.038) | (.063) |
| Pre-famine period | −.027 | .002 | .112 ∗ ∗ | .062∗ | .177 ∗ ∗ |
| | (.031) | (.019) | (.028) | (.036) | (.055) |
| # individuals | 16588 | 12615 | 16588 | 16588 | 16588 |

Notes: * p<0.1, ** p<0.05, *** p<0.01. Employment, hospital, CVD and cancer estimated by binary probit; income by OLS. Robust s.e.'s within brackets.
Notice that the chronological order goes from "pre- famine period" to trimesters 3, 2 and 1, where each of these names refers to the contemporaneous interval in cities in the West.

Table 7: Estimates of famine exposure effects, based on within- and between- cohort variation

| | **Labor market outcomes** | | **Hospitalization outcomes** | | |
|---|---|---|---|---|---|
| | Employment | Labor income | Hospital | CVD | Cancer |
| Interaction cities West X trimester 3 | .015 | .031 | .070 ∗ ∗ | .063 | .044 |
| | (.035) | (.022) | (.032) | (.041) | (.062) |
| Interaction cities West X trimester 2 | −.003 | −.011 | .077 ∗ ∗ | .093 ∗ ∗ | .053 |
| | (.040) | (.026) | (.038) | (.047) | (.075) |
| Interaction cities West X trimester 1 | −.087 ∗ ∗ | −.014 | .062∗ | .031 | .069 |
| | (.039) | (.026) | (.036) | (.046) | (.072) |
| Trimester 3 | −.009 | −.120 ∗ ∗∗ | −.040 | −.014 | −.003 |
| | (.054) | (.033) | (.050) | (.063) | (.094) |
| Trimester 2 | .020 | .069∗ | .089 | .079 | −.059 |
| | (.064) | (.041) | (.059) | (.075) | (.113) |
| Trimester 1 | −.028 | .100 ∗ ∗∗ | .017 | .061 | −.131 |
| | (.052) | (.032) | (.048) | (.061) | (.093) |
| Cities West | −.017 | .090 ∗ ∗∗ | −.047 ∗ ∗∗ | −.061 ∗ ∗∗ | .003 |
| | (.019) | (.011) | (.017) | (.022) | (.033) |
| # months born before May 1946 | −.001 | .007 ∗ ∗∗ | .005 | .003 | .001 |
| | (.004) | (.002) | (.003) | (.004) | (.006) |
| # individuals | 47627 | 36006 | 47627 | 47627 | 47627 |

Notes: * p<0.1, ** p<0.05, *** p<0.01. Robust s.e.'s included within brackets. Employment, hospital, CVD and cancer estimated by binary probit; labor income by OLS. Binary indicators for month of birth are included in the model but are omitted from the table.

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