Windows of Vulnerability: Heterogeneous late-life consequences contingent on timing of Dutch Hunger Winter exposure[1]

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Abstract

Prior research on early-life exposures to famine and its consequences on late-life health and socioeconomic status has established in-utero development as a critical period of vulnerability to malnutrition. Yet, previous research tends to focus narrowly on this stage, at the expense of a more comprehensive examination of childhood. As a result, the literature has yet to compare the severity of the consequences of exposure to malnutrition across developmentally salient periods. Such comparison is crucial not only in magnitude of effects, but also in the nature of outcomes. Using a unique combination of population registries and detailed health surveys to study the Dutch Hunger Winter, this study provides a comprehensive examination of the long-term consequences for in-utero, infant, childhood, and adolescent exposure to famine. The results show malnutrition leads to heterogeneous effects depending on when the exposure occurs. Exposure to malnutrition during in-utero stages leads to deleterious conditions in physical health and lower position in socioeconomic hirearchies. For older cohorts, results suggest a resilience to the effects of malnutrition on physical health in late life, but a higher vulnerability to cognitive abilities and socioeconomic indicators. Furthermore, the results suggest important gender differences in the long-term impact of malnutrition. Male babies exposed while in-utero show stronger negative consequences and a wider array of conditions. Ultimately, this study contributes to the notion that there are multiple critical periods of exposure to malnutrition and these vary in vulnerability and nature of outcomes.

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Abstract

Prior research on early-life exposures to famine and its consequences on late-life health and socioeconomic status has established in-utero development as a critical period of vulnerability to malnutrition. Yet, previous research tends to focus narrowly on this stage, at the expense of a more comprehensive examination of childhood. As a result, the literature has yet to compare the severity of the consequences of exposure to malnutrition across developmentally salient periods. Such comparison is crucial not only in magnitude of effects, but also in the nature of outcomes. Using a unique combination of population registries and detailed health surveys to study the Dutch Hunger Winter, this study provides a comprehensive examination of the long-term consequences for in-utero, infant, childhood, and adolescent exposure to famine. The results show malnutrition leads to heterogeneous effects depending on when the exposure occurs. Exposure to malnutrition during in-utero stages leads to deleterious conditions in physical health and lower position in socioeconomic hirearchies. For older cohorts, results suggest a resilience to the effects of malnutrition on physical health in late life, but a higher vulnerability to cognitive abilities and socioeconomic indicators. Furthermore, the results suggest important gender differences in the long-term impact of malnutrition. Male babies exposed while in-utero show stronger negative consequences and a wider array of conditions. Ultimately, this study contributes to the notion that there are multiple critical periods of exposure to malnutrition and these vary in vulnerability and nature of outcomes.

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Introduction

Human development can be conceived as a continuous interaction between the inherited genetic makeup of the individual and the external stimuli received throughout the life course (Berens, Jensen, and Nelson 2017; Hertzman 1999). Accordingly, the body of research known as the Developmental Origins of Health and Disease (DOHaD) highlights the important role that material conditions in early life play in the development of later life chronic disease and mortality risk. In particular it emphasizes gestation as a life stage highly vulnerable to insults such as malnutrition (Barker 1990, 2004; Kuh and Shlomo 2004; Palloni et al. 2009). This is reflected in research that has shown that in-utero malnutrition can result in higher propensity to develop an array of conditions associated with increased mortality risk, including cardiovascular disease, cerebrovascular disease, and diabetes, as well as reduced cognitive ability and stunted socioeconomic attainment (Almond and Currie 2011; Basso 2008; Conley and Bennett 2000; Painter, Roseboom, and Bleker 2005; Roseboom et al. 2011, 2011).

Though implicitly based in a life course developmental perspective, prior DOHaD research has tended to focus narrowly on in-utero exposures and cardiometabolic outcomes, at the expense of a more comprehensive life course approach. Like gestation, infancy, childhood, and adolescence are life stages characterized by key developmental changes (Nelson 2013, 2017). Focusing exclusively on the gestational period has left crucial questions underexplored. For example, to what extent does vulnerability due to exposures like malnutrition extend beyond gestation? How does the impact of early life malnutrition vary across developmental domains, either in their magnitude or in terms of heterogenous timing effects?

Adopting a natural experiment framework and using a unique dataset, which combines the population registry of the Netherlands with a large nationally representative health survey, this study re-examines the case of the Dutch Hunger Winter to investigate the heterogeneous effects of malnutrition by examining the timing of exposure beyond gestation. In addition, this study explores a wide array of health and socioeconomic outcomes in late life. Furthermore, this data allows us to overcome important limitations of most prior research, which has been constrained by small samples and a limited array of outcomes. This includes examining gender differences in the impact of famine exposure on health and socioeconomic outcomes over the long-term. This study expands the knowledge on the long-term effects of malnutrition on life course health and socioeconomic outcomes. The findings contribute to the understanding of what conditions arise as a consequence of malnutrition experienced at different stages in early life. While famine is a rare event in high income contexts, hundreds of millions of people are still subject to hunger, severe levels of food insecurity, and malnutrition around the world. Globally, nearly nearly a quarter of children under the age of 5 were affected by stunting or wasting in 2020 (UNICEF-WHO-WB 2021). How such exposures will effect their lives over the long-term remains a very important policy question. Knowledge of how famine affects people in the long run is crucial for potential policy interventions designed to improve human development.

Background

The Dutch famine context

Much of what is known about the long-term consequences of early life famine exposure come from examining the Dutch Hunger Winter. At the outbreak of the Second World War, the Dutch government began rationing food (Banning 1946; Dols and Arcken 1946). In September 1944, in response to a railroad strike called by the government in exile, Germany imposed a food

embargo on the Western provinces (i.e., Noord-Holland, Zuid-Holland, and Utrecht). In addition, an early extreme winter resulting in frozen waterways, precluded supplying the Western provinces with resources from other areas. As a result, approximately 4.5 million people were exposed to a context of severe famine. Average caloric intake in these provinces declined from approximately 1500 calories/day in September of 1944 to as low as 700 calories for children and just over 500 calories for adolescents and adults during January-March 1945 (Dols and Arcken 1946). Pregnant and lactating women were entitled to extra rations, however, at the peak of the famine these extra rations could not be sustained. Nutrient composition was also affected, as rations shifted heavily towards cereals and potatoes, in substitution of meat and dairy. This increased the proportionate intake of carbohydrates and decreased consumption of proteins and fats as well as key vitamins and minerals (particularly calcium) (Dols and Arcken 1946). Caloric intake would not recover until June 1945. Between January-July 1945 there were 45,000 excess deaths in the Netherlands, 68% of which occurred in the famine-affected Western regions (Ekamper et al. 2017). Among war-related or otherwise unspecified mortality, hunger/thirst was responsible for less than 1% of deaths nationwide in 1944 but rose to 23% in 1945 (Ekamper et al. 2017).

[Figure 1 here]

Windows of vulnerability in early life.

A core principal of life course theory asserts that the impact that events, exposures, or transitions have on life trajectories depends heavily on the timing at which they occur (Elder 1998). As developmental stages of the life course unfold from conception to adulthood, individuals traverse multiple critical/sensitive periods that shape their subsequent social and biological development. During these critical/sensitive periods, windows of vulnerability open to adversity. While

individuals may be vulnerable to adverse experiences during a given developmental interval, they may be resilient to the same experiences outside of it (Kuh et al. 2003; Kuh and Shlomo 2004).

In-utero windows of vulnerability

According to DOHaD research, in-utero nutritional deprivation triggers a set of fetal adaptations to critical structures and tissues. This process—known as predictive adaptive responses (PAR)—consists of phenotypic adjustments to experienced or predicted environmental challenges (Bateson and Gluckman 2012a; Gluckman et al. 2008; Gluckman, Hanson, and Spencer 2005). These adaptations range from disruptions of organ formation (cell division, growth, and functional specialization) to alterations to basal metabolic function including blood pressure, heartrate, as well as to glucose and lipid metabolism (Bateson and Gluckman 2012a). PAR adaptations increase fetal survival in the short-term (Kahn, Narayan, and Valdez 1998). However, they are thought to be maladaptive in the long-term, resulting in higher propensities of developing cardiovascular and cerebrovascular disease, diabetes, and other chronic diseases (Almond and Currie 2011; Basso 2008; Black, Devereux, and Salvanes 2005; Conley and Bennett 2000; Palloni et al. 2020; Roseboom et al. 2001, 2011a). For example, in animal models, induced protein deficiency has been shown to result in permanently elevated blood pressure, impairment in glucose tolerance, and increases in the likelihood of obesity (Aiken and Ozanne 2013; Heijmans et al. 2008; Lorente-Pozo et al. 2018). Results from observational studies and natural experiments on human populations have similarly demonstrated adverse effects of in-utero nutritional deprivation on later life health across a wide variety of case studies, (e.g., the Leningrad siege, the Rwandan civil war, the Dutch hunger winter, and the Chinese revolution)

(Akresh, Verwimp, and Bundervoet 2011; Schulz 2010; Stanner and Yudkin 2001; Zhang, Gu, and Hayward 2010).

Beyond the womb and cardiometabolic disease

The focus in the literature on the gestational period and on late life cardiometabolic outcomes tends to obviate other critical periods within childhood and adolescence and other important domains of life course outcomes. Malnutrition during subsequent stages of development (e.g., in early childhood) may also adversely impact adult health. For example, post-natal nutritional deprivation can also permanently alter the microbiome—crucial for subsequent nutrient acquisition and energy harvesting throughout the life course (Devaraj, Hemarajata, and Versalovic 2013). Recent evidence suggests that the gut microbiome plays a critical role in physiological functions throughout the life course and influences of a wide array of chronic diseases in late life, including obesity, diabetes, and cardiovascular disease (Devaraj et al. 2013; Alur 2019). There is evidence supporting the notion that nutrient acquisition and energy harvesting that are intimately related to physical growth and weight regulation rely on processes embedded within the microbiome (Herd et al. 2018; Ng et al. 2014). As humans are born microbially sterile, infant and childhood nutrition is central to building the microbiome, thus implicating the post-natal developmental environment (Burmeister et al. 2020; Devaraj et al. 2013; Herd et al. 2018). Similarly, there is a growing body of research that shows greater loss of muscle strength and bone mass in late life among those exposed to poor nutrition in early life (Bartz et al. 2014; Huang, Soldo, and Elo 2011). Low calcium and protein intake during infancy, childhood, and adolescence are key determinants of bone density and muscle development (McFie and Welbourn 1962; Sayer and Cooper 2002). Bone stability and height have also been directly linked to the quantity and composition of childhood nutrition (Akachi and Canning

2007; Deaton 2007). It's important therefore to expand the investigation of the long-term impacts of famine beyond gestational exposures.

Extending the developmental critical periods under consideration beyond the womb further expands the array of outcomes subject to influence of malnutrition. Beyond cardiometabolic outcomes, there is evidence that suggests exposure to malnutrition may increase risk of mental health problems and addiction, lower cognitive ability, and adversely impact socioeconomic outcomes (i.e, labor market earnings and employment) (Franzek et al. 2008; Huang et al. 2013; Lumey, Stein, and Susser 2011; Lumey and Van Poppel 1994; Roseboom et al. 2011; Susser and Lin 1992). The concept of *biological embedding* asserts that the material and psychosocial conditions under which physical, neurocognitive, and psychosocial development occurs shapes essential bodily systems, such as the central nervous system, in fundamental ways that persist over the life course (Hertzman 1999a–b). If the childhood environment is not conducive to healthy development this may lead to adverse physiological and neurocognitive outcomes, as well as poor emotional and psychosocial coping mechanisms, and thus higher lifetime levels of stress and subsequently poor health (Cynader and Frost 1999; Hertzman 1999a–b). Executive function, which regulates how individuals respond to social and emotional stimuli, develops between approximately ages 3-9 (Vineis et al. 2016). Cognitive capacities related to attention, memory, sensory function, coordination, and broad motor skills—key variables for school and occupational success—are developed during mid-childhood as well. (Demetriou et al. 2015; Hertzman 1999; Hertzman and Keating 1999). Research in the last decade has extended critical periods up through adolescence as well, as brain plasticity and rapid pubertal maturation of all organ systems occur during this period (Barouki et al. 2012; Bateson and Gluckman 2012b; Belsky and Pluess 2009). In parallel with such biological developments

during adolescence, there are marked psychological developmental changes such as the development of peer networks, friendships, and intimate partners, as well as personality formation, which all have a strong influence on mental health vulnerabilities in later life (Viner et al. 2015).

Gender differences

There is evidence within both clinical and demographic research of striking gender differences in resilience to malnutrition while in-utero and in early infancy. This difference in resilience is referred to as the "frail male" hypothesis in demographic literature, and often relies on imbalanced sex ratios at birth and disproportionate infant mortality among male babies during times of hardship as evidence (Bisioli 2004; James 2009; Schacht, Tharp, and Smith 2019). According to the developmental biology literature, males tend to show a higher resilience during the 1st trimester of gestation as compared to their female counterparts. However, this is only the case for the 1st trimester of gestation, after this stage, females exhibit a stronger resilience during the fetal-to-neonatal transition, in the newborn period, as well as in the first years of life (Alves et al. 2019; Rosenfeld 2015). Speed in maturation may be key to understanding how windows of vulnerability operate. If maturation is slower, this translates to a wider window of time during which insults can have adverse effects. For instance, functional and structural development of both the cardiovascular and respiratory systems mature faster in female fetuses, shortening the window of vulnerability to insults during critical developmental periods related to cardiometabolic maturation (Franzek 2019; Lorente-Pozo et al. 2018).

Similar patterns have been observed in animal models. Functional and structural development of both the cardiovascular and respiratory systems mature faster in female fetuses, decreasing vulnerability to insults during critical periods in early life (Franzek 2019; Lorente-

Pozo et al. 2018). For instance, mouse models of in-utero malnutrition has shown that the placenta of the female fetus may be more efficient in terms of extracting and transporting nutrients from the maternal circulation (O'Connell et al. 2011). Females further exhibit a greater degree of maturation in early stages of development, which translates into a lower incidence of prematurity and prematurity-associated morbidities, as well as a higher capacity for catch-up growth (Heijmans et al. 2008; Lorente-Pozo et al. 2018).

Therefore, one possible explanation behind the "frail male" hypothesis is that males have larger windows of vulnerability because of longer developmental periods of cardiovascular and respiratory systems. However, the fact that males have a longer maturation period, may also mean that males' possibilities of recuperation from insults may be higher, which would translate to an observable resilience or "dampened" effects of the famine as compared to females (Gilbert and Gilbert 2000; Palloni et al. 2020). An important limitation of this literature is that it has focused almost exclusively on gender differences in survival in-utero and in the neonatal period with little attention given to health and mortality differences later in life. This limitation exists in large part due to the lack of adequate data on exposure to early life nutritional deprivation among cohorts of older adults. As such, it is unclear how gender may shape the effects of early life famine exposure over the long-term among those who survive into adulthood. Do gender differences in the impact of famine exposure follow the same pattern in later life as they do in early childhood? The present study leverages a large sample derived from population registry linked survey data which allows for the examination of gender differences in the effects of early life malnutrition on life course outcomes in ways that were not possible in prior research.

The present study

Following both the developmental biology and social science literatures, we propose that the timing of exposure to malnutrition yields differential consequences in terms of health and socioeconomic development. As expressed in figure 2, and consistent with the prior literature, we hypothesize that in-utero exposure to malnutrition results in higher vulnerability to biological adaptations, increasing risk of cardiometabolic and muscular-skeletal physical problems in later-life. However, as the timing of exposure shifts to the post-natal and later childhood periods, different developmental processes come in to play and vulnerabilities shift from being primarily biological in nature to increasingly impacting processes implicated in social development and outcomes.

[Figure 2 here]

Accordingly, we hypothesize that exposure to famine during childhood and adolescence is less likely to increase cardiometabolic or other physical health risks than in-utero exposure. However, we propose that vulnerabilities during these stages are still important, with socioeconomic and socioemotional development becoming central. Finally, we examine gender differences in these patterns. Hence, we examine the following research questions:

R1: To what extent is there heterogeneous vulnerability to famine contingent on the timing of exposure throughout in-utero, infancy, childhood, and adolescence?

R2: Are there specific patterns in health conditions and socioeconomic attainment contingent on the timing of exposure throughout in-utero, infancy, childhood, and adolescence?

R3: In what ways does vulnerability to famine vary by gender?

Data

This study relies on the unique linkage of two major sources of data: The Adult and Elderly Health Monitor (GEMON) and the Dutch Census. GEMON is a nationally representative survey of the Dutch population aged 18 years and older. We utilize the latest available waves from 2012 and 2016, which provide information on approximately 400,000 individuals. The second data source is the restricted Dutch Census data provided by Microdata CBS (Statistics Netherlands)[3]. When linked to GEMON this provides date and place of birth of respondents, allowing the identification the location and age of the respondent—including those in-utero—during the Dutch Hunger Winter, and thus exposure to famine. Because we are interested in studying those cohorts who had the potential to be exposed to the Dutch Hunger Winter or those cohorts born immediately after, we exclude any respondents born after 1949. Overall missing data accounts for approximately 15%[4]. After excluding individuals born after 1949 and accounting for 15% of missing data, we obtain a total sample of 123,789 individuals[5].

Exposure to famine: cohort and gestation groups

The study focuses on all individuals at risk of exposure to the Dutch Hunger Winter, hence; with the exception of a control group comprised of cohorts born 1945–1949, we exclude individuals born after 1950. Following previous research, this study distinguishes between three groups 1) cohorts in-utero during famine, 2) cohorts born before the famine period, and 3) cohorts born between 1945 and 1949. We further partition in-utero cohorts by trimester of first exposure. Figure 3 presents a diagram pertaining to differential groups based on the timing of exposure.

[3] Results based on calculations by Daniel Ramirez and Steven Haas, using non-public microdata from Statistics Netherlands. Under certain conditions, these microdata are accessible for statistical and scientific research. For further information: microdata@cbs.nl.

[4] Previous research has asserted percentages of missing data between 10%-15% are inconsequential for the results of analyses (Dong and Peng 2013; Enders 2010; Johnson and Young 2011). For this reason, we opted to utilize listwise deletion approaches in our analysis.

[5] Sample size by cohort is available in Table 1. descriptive statistics.

The shaded area in figure 3 relates to the famine period for which average daily rations fell below 1,000 calories (i.e., November 1944 to June 1945) (Dols et al. 1946). Assuming a 9-month gestation interval, the study identifies differential timing of exposure based on the trimester in which famine began. For instance, respondents are considered to be at risk of exposure during the 3rd trimester if they were born 1-3 months after October 1944. All others are grouped into 5-year age cohorts corresponding to different stages of childhood including infancy and early childhood (ages 1-4), the early school years (ages 5-9), and early adolescence (ages 10–14). In terms of current age, this study examines individuals aged 72-87 in 2016.

[Figure 3 here]

Dependent variables

Self-rated health (SRH). A self-assessed summary statement of the respondent's overall health status that was dichotomized to express positive health (excellent/very good = 1; otherwise=0). SRH is a widely utilized indicator of health that has been deemed a valid approximation of overall health that successfully predicts health conditions as well as mortality (Idler and Benyamini 1997; Jylhä 2009). Self-rated health captures a wide range of both health conditions (physical, functional, and psychological), and underlying mechanisms (social and biological). Utilizing an ordinal form of SRH yielded similar results.

Cardiometabolic and muscular-skeletal physical health conditions

GEMON respondents were asked if they had ever been diagnosed by a doctor with a series of health conditions and diseases. Indicator variables identify those affirming diagnosis for *Cardiovascular disease* or *Diabetes* (yes=1; no=0). A third indicator identifies those with a body-mass index (BMI) equal or above 30.0 as *Obese*. As a measure of muscular-skeletal health, we use *Number of functional limitations* (0-3). The measure is based on 3 items that assess

whether the individual has difficulty walking 400 meters without stopping, carrying an object of 5kg a distance of 10 meters, and bending to pick up something from the ground.

Psychological and sensory health

GEMON includes the Hospital Anxiety and Depression (HAD) screening scale. The HAD is an extensively validated 7-item self-report screening scale initially developed to screen for the presence of anxiety and depressive states in the setting of a medical out-patient clinic (Mykletun, Stordal, and Dahl 2001; Snaith 2003). The operationalization results in a dichotomous indicator designating those individuals who screened positively for being at risk of *depression* or *anxiety*. *Auditory impairment* is a dichotomous indicator designating whether the respondent has trouble following a conversation with three or more people. *Visual impairment* is a dichotomous measure of whether individuals have issues reading small letters or whether they have trouble recognizing people at a distance of four meters.

Socioeconomic outcomes

Educational attainment is an ordinal measure capturing respondents' highest educational degree achieved, where 1=secondary or less, 2=professional degree (equivalent to an associate degree), 3=bachelor's degree, and 4=postgraduate degree (MA/PhD)[6]. *Income quintile* indicates the quintile the respondent occupies in the Dutch income distribution.

Analytic approach

We utilize a difference-in-difference (DID) approach to estimate the treatment effect of being exposed to a context of famine, using the following equation:

$$Y_{ji} = \alpha + \beta_1 \mathbf{T}_i + \beta_2 \mathbf{R}_i + \beta_3 \mathbf{T}_i x \mathbf{R}_i + \beta_4 \mathbf{Z}_i + \varepsilon_i$$

[6] This educational scale is taken as provided by the GEMON dataset and is particular to the Dutch society.

Where Y_{ji} represents the outcome of interest *j* for individual *i.* $\mathbf{T}_i$ represents a vector of dummy variables that stem from the categorical variable that delimits the timing of exposure to famine for individual *i,* and $\mathbf{R}_i$ represents a vector of dummy variables that stem from the categorical variable for region of birth for individual *i*. These two sources of variation—time and place of birth—are random in nature, allowing a DID estimator. The parameters of interest are the set of interaction terms $\mathbf{T}_i x \mathbf{R}_i$ (i.e., β_3) representing the treatment effect of being a particular age in the Western provinces of the Netherlands during the winter of 1944-45. They represent the effect of famine exposure over and above the effect of birth in a region in the West, and the period effect effect of being born at a particular moment in time. Finally, $\mathbf{Z}_i$ represents survey fixed effects variables which capture the variation between the two waves of GEMON.

Results

Table 1 provides descriptive statistics for all outcomes by age of risk of exposure and by treatment group. For simplicity, we summarize descriptive statistics for childhood as one group. In the analysis we disaggregate the groups. Overall, children living in famine regions show higher prevalence of negative health outcomes, including poor SRH, cardiovascular disease, diabetes, depression and anxiety, and auditory and visual problems. However, there are no differences in socioeconomic standing. Those exposed in-utero show stark differences in self-rated health, cardiovascular disease, diabetes, obesity, and auditory problems. Conversely, the differences between famine areas and non-famine areas for those born post-famine show the reverse tendency, with the areas previously effected by family demonstrating better health across nearly every outcome. Important differences in SRH are observed for those who were children during the famine. 62% of those living in the famine region reported positive SRH, compared to 72% among those living in non-famine provinces. Similarly, those who were in-utero in the

famine region also had lower SRH than those in non-famine regions, though the gap was smaller. Conversely, for the post-famine control group those residing in the region previously affected by famine show better SRH than in the non-famine region. Similar patterns between famine and non-famine regions at distinct age groups can be found across multiple outcomes. For instance, cardiovascular disease prevalence is 11.4% for those who lived in a famine region during childhood, whereas their unexposed counterparts had a 7.8% prevalence..

<Insert table 1 here>

Table 2 presents DID estimates for positive self-rated health. The treatment effect of being exposed to famine for each age group is reflected in the interaction terms. Each of these estimates are calculated separately by gender. As can be seen in the table, there are no significant effects of famine exposure at any stage for females. However, for males there are effects for those exposed in the 1st and the 2nd trimesters. Those exposed in the first trimester had a 27% ($e^{-0.32}$ = (1-0.73 * 100)) lower odds of reporting a positive self-rated health above and beyond regional and cohort differences. Similarly, those exposed during the second trimester had 24% ($e^{-0.27}$ = (1-0.76 *100)) lower odds of a positive self-rated health above and beyond regional and cohort differences.

<Insert table 2 here>

Table 3 presents DID estimates of effects of famine exposure on physical health for each cohort and by gender. The results show a pattern where the earlier the timing of exposure, the higher the risk of developing diseases due to famine exposure. This is true not only because most effects are found among respondents who lived through the famine while in-utero, but

additionally because the results show the strongest effects concentrate in the 1st and 2nd trimester of gestation. Males exposed in the first trimester had a 33.6% ($e^{0.29}$ = (1-1.336 * 100)) higher odds of reporting a cardiovascular disease above and beyond regional and cohort differences. There is a similar effect for females as well, as they show 35% higher odds of reporting cardiovascular disease if they were exposed in their first trimester. Additionally, males exposed in their second trimester had a 55.3% ($e^{0.44}$ = (1-1.553 * 100)) higher odds of reporting a diabetes above and beyond regional and cohort differences.

Beyond an enhanced vulnerability as a function of how early on the exposure to famine occurs, there are stark gender differences. Males are considerably more vulnerable to famine effects while in-utero overall. Results suggest males experience increased probabilities of developing diabetes, cardiovascular disease, obesity, as well as higher number of functional limitations as a cause of famine exposure. For females, there seems to be greater resilience in terms of developing most physical conditions. Females who were in-utero during the famine show increased probabilities of developing cardiovascular disease and having higher number of functional limitations.

<Insert table 3 here>

Figure 4 presents average marginal effects for the difference in difference regression estimates of famine exposure on various later life physical health outcomes for each exposure and gender group. Overall, effects of famine above and beyond cohort and regional differences are concentrated within in-utero periods. The only condition that registers statistically significant effects outside of the in-utero stage is obesity, where women exposed at ages 1-4 experience higher risk. The results show there is an increase in risk of developing cardiovascular disease due

to exposure to famine for cohorts in the 1st trimester of approximately 7% for males and females. For diabetes, there is an increase in risk due to exposure to famine of approximately 6% for males exposed during the 1st trimester of gestation. Similar effects in terms of timing and magnitude can be found for obesity and functional limitations.

[Figure 4 here]

Table 4 presents DID estimates of mental health risk, auditory and visual impairments. The first column shows estimates for risk of depression and anxiety. No treatment effects are statistically significant, despite coefficients showing a positive sign—indicating exposure to famine increases risk of depression and anxiety. For visual impairment, there are no treatment effects that are statistically significant either. Yet, famine exposure does increase the risk of developing auditory impairment for males. Males exposed during the second trimester show 71% ($e^{0.535} = (1.71 - 1 * 100)$) higher odds of developing an auditory impairment.

<Insert table 4 here>

Table 5 presents estimated famine effects on socioeconomic outcomes. As can be seen in the first set of columns, exposure to famine decreases the odds of attaining a higher educational degree for all individuals (with the exception of females exposed in the first trimester of gestation or at ages 10-14). Severity of the impact is larger for males than they are for females, in line with the previous results related to health. Importantly, as predicted the effect of famine exposure on educational attainment is more severe if it occurred post-natal. For instance, the largest treatment effect for males is concentrated among those exposed at ages 1-4. Those exposed to famine at ages 1-4 show increased odds by 47% ($e^{-0.64} = 1\text{-}0.53 *100$)) to be in a

lower educational category compared to those unexposed. Those exposed in the third trimester as well as in ages 5-14 show comparable effects.

<Insert table 5 here>

The results for income there share notable similarities to those for education. Effects are concentrated among males with females showing no statistically significant effects (though coefficients were in the expected direction). The largest treatment effect is found for those who were exposed in the third trimester of gestation. Those exposed at this stage of gestation show a 24% ($e^{-0.27}$ = (1-0.76 *100)) increase in the odds of being in a lower income. To a lesser extent, there are famine effects concentrated amongst individuals who lived through the famine at ages 1-4 and 5-9 as well. For an easier appreciation of these differences, we plot the treatment effects in figure 5 for each cohort and gender. It is important to note in this figure, we only show treatment effects, this is the coefficient of the interaction effect in the difference in difference models. The results show there is decrease in educational attainment for most cohort groups due to exposure to famine[7]. The largest effect is located in males exposed at ages 1-4, where famine exposure decreased the likelihood of having a higher educational attainment category by approximately 22%

<Insert figure 5 here>

Sensitivity analysis

[7] It is important to note we do not explore indirect effects acting via educational attainment. It is possible reductions in income (and other outcomes related health) may operate via indirect effects. Because this expands beyond the scope of this particular study we opted to exclude such analysis.

The DID approach used here rests on a set of assumptions. Most crucial is the parallel trend assumption (Branas et al. 2011; Gangl 2010; King et al. 2013). That is, in the absence of the treatment, the trends in the outcome between the control group and treatment group are the same. In the current case, suggestive evidence that this assumption is met can be seen in overall differences in health in between famine regions and non-famine regions. They are either non-existent or are positively higher in famine regions for those cohorts who were born after the famine period.

In addition, we perform two placebo tests based on variation in time and in place to provide further evidence. The first test restricts the sample to those individuals outside of the affected areas. Subsequently, we randomly dichotomize regions into "affected" and "non-affected" regions within the non-affected areas, this is all regions besides the Western provinces (i.e. Noord-Holland, Zuid-Holland, and Utrecht). These estimates provide a "placebo" test for the source of variation that stems from region of birth. The second robustness test consists of restricting the analysis to the post-WWII cohorts and randomly dichotomizing individuals into treated and control groups. These estimates provide a "placebo" test for the source of variation that comes from the timing of birth for different cohorts. The results of these tests show the "placebos" had no effect on either of the samples, indicating differences are attributable to famine exposure. These results are available in appendix.

We conducted additional analyses on the ancillary measures number of chronic conditions, stroke, and risky behaviors (i.e., excessive drinking and daily smoking). In regard to number of chronic conditions we find a similar pattern to the results obtained in studying physical health. Males exposed to famine during the second and third trimester of gestation show a higher number of chronic conditions. We did not find any effects on stroke for any cohort

group or gender. In relation to risky behaviors, despite coefficients signal in the expected direction—where famine exposure increases the likelihood of engaging in these behaviors—there are none that are statistically significant. Despite the non-significance from a statistical perspective, there are important differences in daily smoking behaviors in regard to gender, where only males exposed to famine show increases in likelihood of being a daily smoker.

Selection effects

Another potential threat to the validity of the results are selection effects due to differential mortality, migration, and fertility. Selection effects related to mortality can pertain to immediate impacts on mortality or to delayed impacts on mortality (i.e., longevity). Previous research has found immediate excess mortality across all age groups in response to the Dutch Famine, yet; there was a heightened excess mortality for those who were exposed during their first year of life and for those who were 70 years of age and above (Ekamper et al. 2017; Lumey and Van Poppel 1994). Furthermore, there are important gender differentials to consider.

In terms of longevity, those exposed during adolescence and early childhood show small effects of famine exposure on mortality, and such effects become more pronounced for cohorts exposed during infancy and in-utero. Shortened longevity essentially disappears for cohorts born immediately after the famine. Considering the results show heightened vulnerabilities for cohorts exposed in-utero and infancy, and a resilience for cohorts exposed at older ages, it is likely the effects shown in this study's estimations are understated. Appendix B presents survival analysis estimates of differential hazard rates by gender and by timing of exposure. We find males experience a reduction in longevity as a consequence of the exposure to famine, with heightened effects if exposure occurred during the gestational periods. This implies our findings in this study

are like to be underestimates, as those males that were most affected by the famine were less likely to survive to participate in 2016.

In terms of fertility, previous research has shown famine exposures can have strong effects on both fertility and fecundity. Stein and Suzzer (1975) conducted a study focusing on cohorts exposed to famine during the Dutch Hunger Winter. Their findings show steep drops in births during the famine period followed by a pronounced catch-up trend in births once the famine ended. Within these fluctuations, Stein and Suzzer (1975) find there is a social class gradient, where parents holding a manual occupation experience the most pronounced drops in fertility during the famine. Similarly to the pronounced drops, catch-up fertility following the famine was concentrated amongst those parents holding a manual occupation (Stein and Susser 1975). In short, Stein and Suzzer show higher social classes display a certain robustness to famine effects. Razzaque (2008) found similar patterns in studying the effects of the 1974 on differential fertility in a rural population of Bangladesh. The results suggested overall fertility declined by 34% percent as a consequence of the famine, furthermore, there appeared to be a strong socioeconomic gradient where women from low SES groups experienced more pronounced effects. Hence, it is possible there are strong selection effects regarding SES. Potentially, a larger number of babies selected into the treatment groups come from a higher SES background as families from this gradient were resilient to famine effects. Similarly, the control group is potentially comprised in greater proportion of babies born to lower SES backgrounds. If this were the case, it is likely the famine effect is understated.

Our estimates are also potentially subject to selection effects due to migration. Previous research has discussed similar concerns regarding migrations prior to WWII and in response to it (Castles, Haas, and Miller 2013; Crafts and Toniolo 1996; Kesternich et al. 2014; Van Mol and

de Valk 2016). Previous research has shown that, in light of conflict, socioeconomic status at both the individual level and the regional level moderate the likelihood of migration (Williams 2013; Williams et al. 2010). Considering migration tends to occur amongst sectors with a higher socioeconomic background, it is possible individuals in this study's sample is composed of individuals with a comparatively lower SES. If this were the case, effects would be overstated. In order to assess the potential selection effects, we examined patterns of internal migration of the Dutch population for each cohort using Survey of Health and Retirement in Europe Life History data. Approximately, 4-6% of the Dutch population experienced migration prior to the beginning of WWII. During WWII this percentage rises to approximately 10%. External migration was unlikely at that time, as only 2% of the Dutch population left the country. These results are available in the appendix.

Discussion

While the literature has established the in-utero period as one highly sensitive to malnutrition, prior research tends to focus narrowly on that window of time, eschewing a more comprehensive life course approach. Furthermore, most research to date has only examined one or two specific health conditions at a time in isolation, with few comparisons across a wider array of exposure timings and life course outcomes. As a result, questions about how various early life sensitive periods across developmental domains differ in terms of vulnerability to the effects of malnutrition remain largely underexplored. In addition, scant research has examined the extent to which there are gender differentials in the long-term effects of early life malnutrition.

Using a unique dataset that combines large nationally representative health surveys and a population registry, this attempted to fill these gaps by re-examining the Dutch Hunger Winter. At its core this study highlights the notion that windows of vulnerability to malnutrition open and

close contingent on the stage of development, not only in terms of severity but also in regard to the nature of consequences. We hypothesized that in-utero exposure to malnutrition results in higher vulnerability to biological adaptations, increasing risks of cardiometabolic, muscular-skeletal physical problems, and sensorial impairment in later-life. However, as the timing of exposure shifts to the post-natal and later childhood periods, different developmental processes come into play. As such, we hypothesized vulnerabilities to famine exposure shift from being primarily biological in nature to increasingly impacting processes implicated in social development and outcomes.

Our results show the in-utero window is vulnerable to malnutrition effects that lead to physiological consequences consistent with the metabolic syndrome in later life (e.g., diabetes, cardiovascular disease, obesity) as well as muscular-skeletal deficiencies and auditive impairment. Conversely, for cohorts exposed at later developmental stages (childhood and adolescence), results suggest a resilience to the effects of malnutrition on physical health in late life, but a higher vulnerability with regard to socioeconomic indicators.

Finally, research from developmental biology has shown that some structures and systems develop faster in female fetuses, shortening the window of vulnerability to insults during critical developmental periods for males. Therefore, we hypothesized important gender differentials in which famine exposure would result in more severe and frequent deleterious physical health conditions in later life for males relative to females. The results support this hypothesis. Male babies exposed in-utero show stronger negative consequences across a wider array of conditions (self-rated health, cardiovascular disease, obesity, functional limitations, and auditory impairment).

We also found important gender differences in the impact of on socioeconomic outcomes. Compared to their similarly exposed female peers males show substantially larger reductions in educational attainment rsulting from famine exposure. Interestingly, the differences are especially pronounced if the exposure occurred during childhood and adolescence. Furthermore, in terms of reductions in income, we only find effects among males. We believe part of the gap is likely due to differential gender and biological processes contingent on when the exposure occurred in the life course. Famine exposure occurring in-utero or early childhood is likely to impact brain development and cognitive abilities, which would in turn affect educational attainment. However, for exposures occurring during later childhood and adolescence, processes related to social and gender role expectations are likely to be more important. The cohorts examined here were brought up prior to the gender revolution of the 1960s and 1970s (Goldin 2006). Hence, it is likely that highly gendered social and educational expectations played an important role in the extent to which early life exposure to famine impacted socioeconomic attainment differentially among boys and girls. If educational expectations and investments were substantially curtailed for girls relative to boys, then early life exposures that adversely stunted the top of cognitive and achievement distributions would appear to be less consequential for the socioeconomic outcomes of girls.

The Dutch Hunger Winter literature often overlooks the fact that war and famine are contextual phenomena that exceed individuals' exposure to physical violence or food shortages. Although the primary hazard was severe food shortage, the period of the Dutch Hunger Winter was further characterized by high levels of violence, infrastructure and institutional breakdown, extreme cold, overcrowding, military executions, and breakdown of sanitation systems (Ekamper et al. 2017; Lumey et al. 2011). Such contextual exposures are likely to have important implications for cognitive and socio-emotional development, as well as educational and

occupational outcomes. Furthermore, exposure to the trauma of war and hunger can induce psychopathology and mental illnesses in adult life (Anda et al. 2006; Chapman et al. 2004; O'Rourke, Carmel, and Bachner 2018).

As such war-related exposures can induce high levels of stress, which may have a long-lasting physiological impacts that opperate independently from malnutrition. A wide array of laboratory studies conducted on animals show that young adults exposed in-utero to maternal psychosocial stress—and postnatal stress—lead to a dysregulation in key physiological systems, increasing the risk for developing higher BMI and percent body fat, primary insulin resistance, and a lipid profile consistent with the metabolic syndrome (Entringer and Wadhwa 2013; Kuzawa and Quinn 2009; Paternain et al. 2013; Thayer and Kuzawa 2015). While we are not able to test differential effects via malnutrition or via psychological stress, it is important to recognize that stress processes induced by the famine and the broader context of war it accompanied likely contributed to adverse health in later life, independently of those related to nutritional deprivation presented here.

It is worth noting the Dutch Hunger Winter lasted approximately 6-7 months. While the effects found in this study might seem small at a first glance, if one takes into consideration the duration of the famine, it is clear the effect sizes are quite substantial. One can only imagine the long-term effects of famines that have been known to last for years (e.g., the Spanish post-Civil War famine lasted from 1939 to 1950) or persistent and prolonged malnutrition experienced in many contemporary low-income contexts. As such the findings have important policy implications. While substantial progress has occured over the past 2 decades in reducing malnutrion, particularly in Asia, Latin America, and the Carribean, globally, stunting and wasting continue to be a major concern. Around the world approximately 150 million children

under the age of 5 suffer from stunting[8] another 45 million suffer from wasting[9] (UNICEF-WHO-WB 2021). UNICEF, WHO and other organizations specialized in famines, wars, and natural disasters have placed their focus on what is known as the essential nutrition actions (ENA). These policy actions provide nutrition interventions targeting the first 1000 days of life with the aim of reducing infant and child mortality, improve physical and mental growth and development, and improve productivity. While the first 1000 days of life are crucial, the present study shows interventions should also focus on expectant mothers and women in childbearing ages. Additionally, children outside the 1000 first days of life window are also vulnerable, and at least for some outcomes, maybe even more so than those in-utero and infantcy. Our study shows compelling evidence supporting the notion that exposure during childhood and adolescence has the greatest effects on reductions in educational attainment. These education effects are likely to have lasting impact on a wide variety of health and social outcomes across the life course. Overall the results highlight that policies to improve early life nutrition and eliminate malnutirion are likely to yield large long-term population health benefits beyond specific improvements to childhood health and survival.

Ultimately, with regard to the long-term impact of childhood malnutrition or similar early life exposures, the present study highlights the importance of considering multiple windows of vulnerability, spanning various developmental domains. As individuals move through different developmental stages, a multiplicity of critical periods can lead to heterogeneous outcomes

[8] Stunting is defined as an impaired growth and development that children experience as a consequence of poor nutrition and infections and can have irreversibly effects on cognitive and physical development, as well as an increased risk of chronic and degenerative diseases.
[9] Low weight-for-height is known as wasting. It usually indicates recent and severe weight loss, because a person has not had enough food to eat and/or they have had an infectious disease (e.g., diarrhea).

contingent on when the exposure to malnutrition occurrs. It also emphasizes the important role that gender plays in modulating malnutrition-induced alterations to developmental trajectories.

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Figure 1: Famine-affected provinces of Netherlands during the 1944-1945 Winter.

Figure 2. Hypothesized vulnerability by timing of exposure to famine.

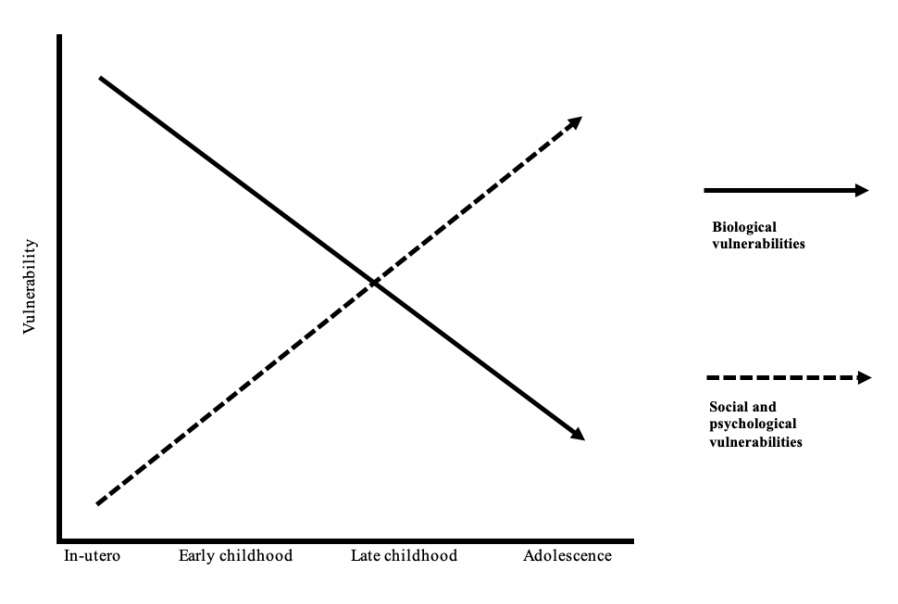


Figure 3. Cohort delimitation chart based on timing of exposure to famine.

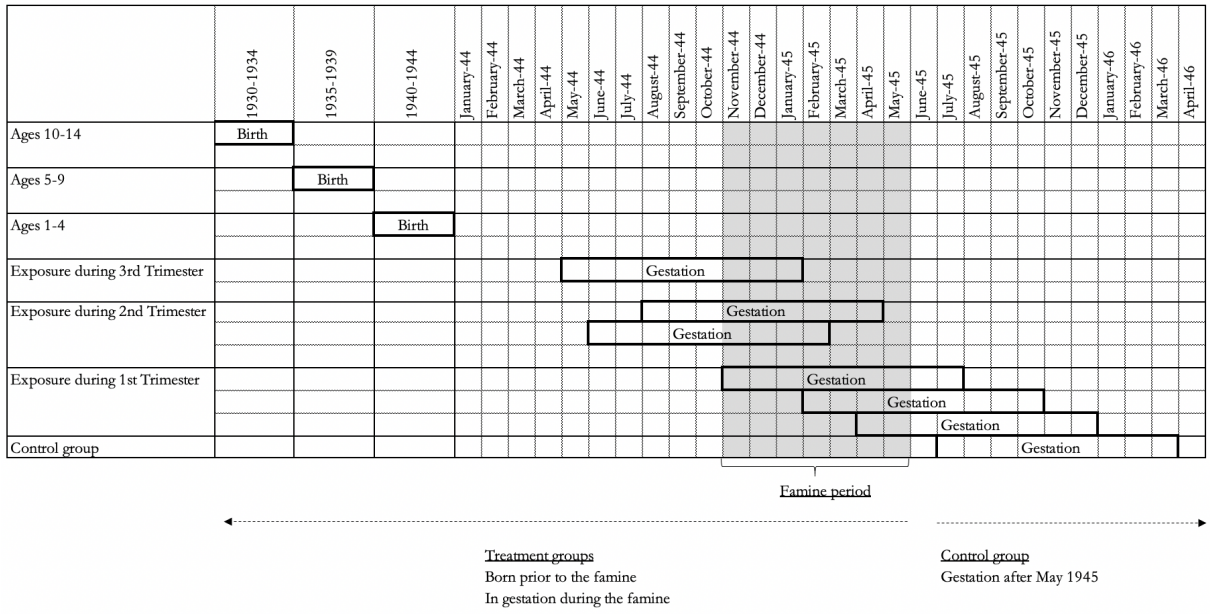


Figure 4. Average marginal effects of famine exposure on physical health by timing of exposure and gender.

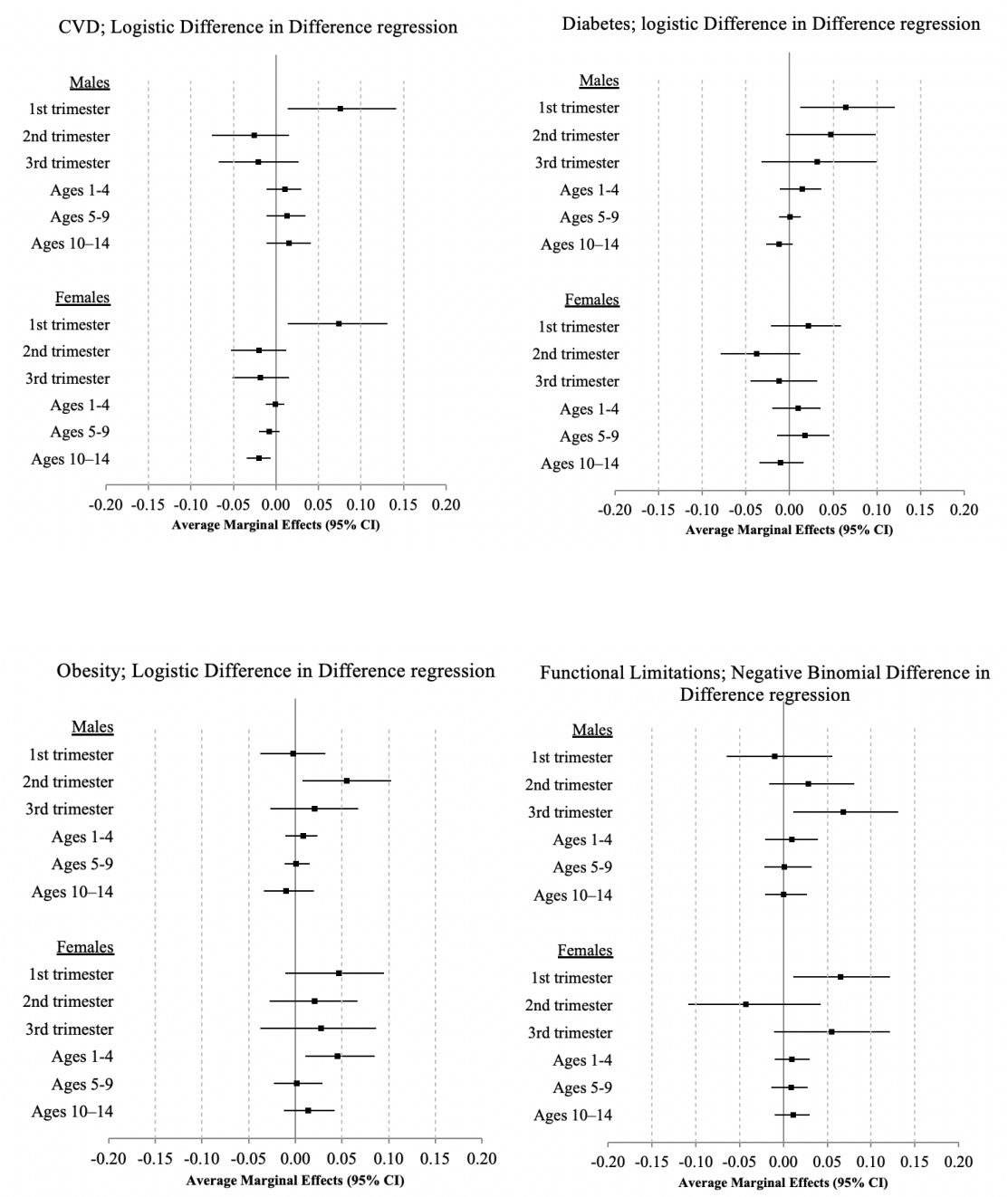


Figure 5. Average marginal effects of famine exposure on socioeconomic indicators by timing of exposure and gender.

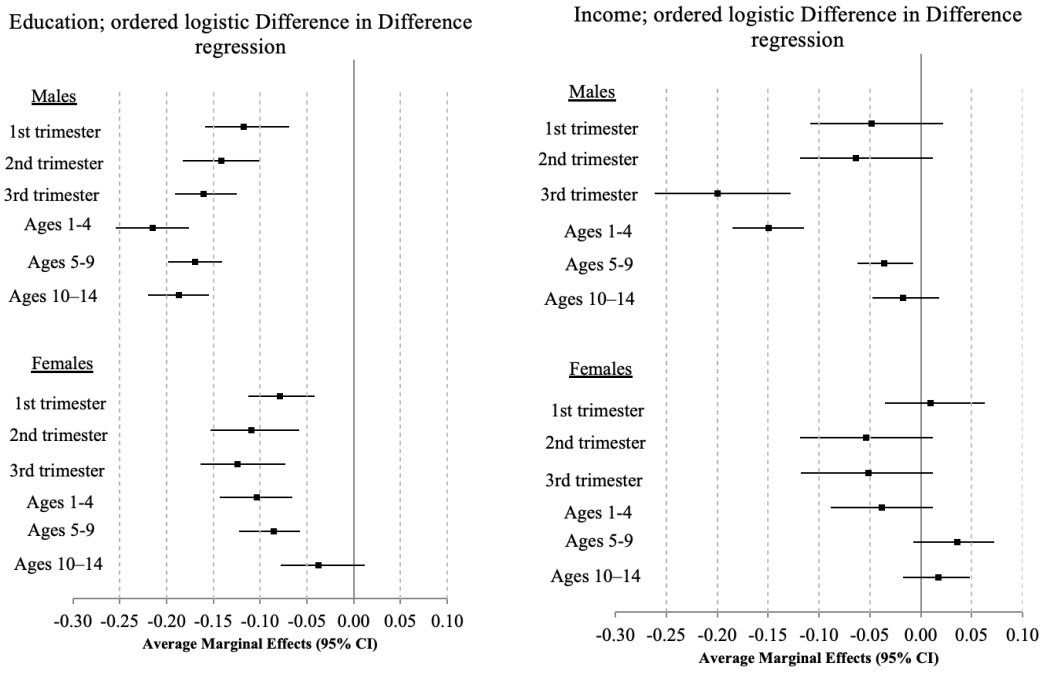


Table 1. Descriptive statistics

Variable	Childhood (birth-Age 14)						In-Utero						Post-famine					
	Famine region (67,568)			Non-famine region (32,953)			Famine region (3,041)			Non-famine region (7,017)			Famine region (4,672)			Non-famine region (8,903)		
	%	Mean	SD	%	Mean	SD	%	Mean	SD	%	Mean	SD	%	Mean	SD	%	Mean	SD
Physical Health																		
Positive self-rated health	62.10%			71.80%			70.60%			72.50%			65.30%			61.90%		
Cardiovascular disease	11.40%			7.80%			7.60%			6.80%			9.90%			10.40%		
Diabetes	16.70%			14.60%			13.30%			12.80%			15.70%			16.40%		
Obesity	13.50%			16.20%			17.50%			15.70%			14.70%			15.20%		
Functional limitations		1.4	0.02		1.2	0.01		1.3	0.01		0.6	0.02		0.7	0.02		0.7	0.03
Mental and Sensory Health																		
Depression & Anxiety	40.60%			32.00%			31.50%			31.70%			37.70%			40.00%		
Auditory impairment	7.40%			4.20%			5.90%			4.40%			6.60%			8.20%		
Visual impairment	6.30%			4.00%			4.10%			4.10%			5.90%			6.80%		
Socioeconomic status																		
Education		2.5	0.01		2.7	0.01		2.6	0.01		2.7	0.02		2.6	0.01		2.4	0
Income quintile		3.3	0.01		3.5	0.01		3.5	0.02		3.7	0.03		3.4	0.01		3.2	0.01

Notes: Total N = 123,794.

Table 2. Difference In Difference Logistic regression estimates for positive self-rated health.

	Females		Males	
Interactions	β	SE	β	SE
Famine reg. x 1st trimester	-0.02	(0.14)	-0.32**	(0.14)
Famine reg. x 2nd trimester	0.07	(0.13)	-0.27*	(0.13)
Famine reg. x 3rd trimester	-0.09	(0.13)	-0.07	(0.13)
Famine reg. x Ages 1-4	0.1	(0.06)	-0.04	(0.06)
Famine reg. x Ages 5-9	0.11	(0.07)	0.01	(0.07)
Famine reg. x Ages 10 – 14	0.07	(0.07)	0.01	(0.07)
Famine region	0.11+	(0.06)	0.04	(0.06)
Birth groups				
1st trimester	-0.25**	(0.09)	-0.139	(0.10)
2nd trimester	-0.25**	(0.76)	-0.023	(0.08)
3rd trimester	-0.02	(0.07)	-0.01	(0.07)
Ages 1-4	-0.24***	(0.04)	-0.247***	(0.04)
Ages 5-9	-0.51***	(0.04)	-0.642***	(0.05)
Ages 10–14	-0.88***	(0.05)	-1.045***	(0.05)

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001 ;. Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Table 3. Difference in difference regression estimates for physical health

	Cardiovascular disease (logistic regression)				Diabetes (logistic regression)				Obesity (logistic regression)				Functional lim. (Neg. Binomial)			
	Females		Males		Females		Males		Females		Males		Females		Males	
Interactions	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE
Famine reg. x 1st trim.	0.30*	(0.14)	0.29*	(0.14)	0.17	(0.21)	0.44**	(0.14)	0.29†	(0.17)	-0.06	-0.15	0.27*	-0.12	-0.05	-0.1
Famine reg. x 2nd trim.	-0.14	(0.21)	-0.1	(0.17)	-0.21	(0.21)	0.29	(0.20)	0.08	(0.18)	0.42**	(0.15)	-0.17	-0.16	0.12	-0.1
Famine reg. x 3rd trim.	-0.09	(0.25)	-0.06	(0.22)	-0.13	(0.23)	0.15	(0.20)	0.1	(0.20)	0.25	(0.17)	0.22	-0.17	0.30**	-0.11
Famine reg. x Ages 1-4	-0.03	(0.13)	0.18	(0.14)	0.03	(0.15)	0.16	(0.12)	0.28**	(0.11)	0.07	(0.08)	0.06	-0.1	0.09	-0.06
Famine reg. x Ages 5-9	-0.05	(0.13)	0.13	(0.14)	0.03	(0.15)	0.06	(0.12)	0.06	(0.12)	0	(0.08)	0.05	-0.1	0.02	-0.06
Famine reg. x Ages 10 – 14	-0.08	(0.13)	0.12	(0.14)	-0.07	(0.15)	-0.05	(0.12)	0.16	(0.12)	-0.09	(0.09)	0.06	-0.1	0.02	-0.06
Famine region	-0.04	(0.12)	-0.14	(0.12)	0.02	(0.14)	-0.15	(0.11)	-0.18	(0.10)	-0.05	(0.07)	-0.1	-0.09	-0.04	-0.05
Birth groups																
1st trimester	0.01***	(0.09)	0.47***	(0.08)	-0.18	(0.12)	-0.56***	(0.10)	0.21***	(0.07)	0.05	(0.08)	0.33***	-0.07	0.61***	-0.03
2nd trimester	0.09***	(0.08)	0.81***	(0.08)	0.08	(0.12)	-0.51***	(0.12)	0.32***	(0.07)	0.02	(0.09)	0.74***	-0.06	1.07***	-0.03
3rd trimester	0.33***	(0.08)	1.08***	(0.29)	0.01	(0.14)	-0.63***	(0.14)	0.46***	(0.06)	0.11	(0.11)	1.22***	-0.06	1.54***	-0.03
Ages 1-4	0.65***	(0.08)	1.37***	(0.08)	0.07	(0.09)	-0.33***	(0.07)	0.61***	(0.12)	0.11*	(0.05)	1.65***	-0.06	1.77***	-0.07
Ages 5-9	0.65***	(0.12)	1.52***	(0.19)	0.15	(0.09)	-0.09	(0.07)	0.65***	(0.10)	0.11*	(0.05)	1.82***	-0.1	1.68***	-0.06
Ages 10–14	0.99***	(0.14)	1.76***	(0.18)	0.31	(0.09)	0.14	(0.09)	0.68***	(0.09)	0.19***	(0.05)	1.63***	-0.09	1.74***	-0.05

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001; Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Table 4. Difference in difference logistic regression estimates mental health risk, auditive impairment, and visual impairment.

	Depression & Anxiety		Auditive impairment		Visual impairment	
	Females	Males	Females	Males	Females	Males
Interactions						
Famine reg. x 1st trim.	0.04	0.14	0.22	0.32	0.31	-0.04
	(0.15)	(0.14)	(0.29)	(0.31)	(0.31)	(0.28)
Famine reg. x 2nd trim.	0.15	0.14	-0.03	0.535**	-0.3	0.02
	(0.14)	(0.12)	(0.24)	(0.23)	(0.30)	(0.22)
Famine reg. x 3rd trim.	0.1	0.01	-0.04	-0.29	-0.16	0.1
	(0.14)	(0.11)	(0.26)	(0.32)	(0.31)	(0.23)
Famine reg. x Ages 1-4	0.06	-0.02	-0.01	0.13	-0.06	0.06
	(0.07)	(0.06)	(0.10)	(0.11)	(0.12)	(0.09)
Famine reg. x Ages 5-9	0.07	-0.03	0	-0.04	0.02	-0.12
	(0.07)	(0.06)	(0.10)	(0.10)	(0.12)	(0.09)
Famine reg. x Ages 10 – 14	0.04	-0.06	0.01	0.14	-0.06	-0.08
	(0.07)	(0.06)	(0.10)	(0.09)	(0.12)	(0.09)
Famine region	0.01	0.025	-0.249**	-0.21**	-0.14	-0.09
	(0.06)	(0.05)	(0.08)	(0.07)	(0.09)	(0.06)
Birth groups						
1st trimester	0.761***	0.666***	-1.191***	-1.702***	1.114***	-1.227***
	(0.04)	(0.03)	(0.05)	(0.19)	(0.07)	(0.05)
2nd trimester	0.488***	0.4***	-0.871***	-1.203***	0.918***	-0.819***
	(0.04)	(0.03)	(0.05)	(0.17)	(0.07)	(0.05)
3rd trimester	0.193***	0.163***	-0.39449	-0.680***	0.421***	-0.371***
	(0.04)	(0.04)	(0.05)	(0.14)	(0.07)	(0.05)
Ages 1-4	0.786***	0.767***	-1.508***	-1.6***	1.293***	-1.25***
	(0.09)	(0.08)	(0.17)	(0.06)	(0.20)	(0.16)
Ages 5-9	0.720***	0.784***	-1.224***	-1.755***	1.049***	-1.09***
	(0.08)	(0.07)	(0.12)	(0.05)	(0.15)	(0.12)
Ages 10–14	0.801***	0.864***	-1.446***	-1.633***	1.261***	-1.423***
	(0.07)	(0.06)	(0.12)	(0.05)	(0.14)	(0.12)

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001; Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Table 5. Difference in difference ordered logistic regression estimates for education and income quintile.

	Education				Income quintile			
	Females		Males		Females		Males	
Interactions	*β*	SE	*β*	SE	*β*	SE	*β*	SE
Famine reg. x 1st trim.	-0.39**	(0.13)	-0.27***	(0.08)	0.02	(0.10)	-0.08	(0.12)
Famine reg. x 2nd trim.	-0.29*	(0.13)	-0.46***	(0.08)	-0.12	(0.10)	-0.12	(0.11)
Famine reg. x 3rd trim.	-0.4**	(0.14)	-0.59***	(0.08)	-0.12	(0.12)	-0.27**	(0.10)
Famine reg. x Ages 1-4	-0.26**	(0.09)	-0.64***	(0.14)	-0.08	(0.05)	-0.17**	(0.06)
Famine reg. x Ages 5-9	-0.19*	(0.10)	-0.51***	(0.12)	0.02	(0.05)	-0.09†	(0.06)
Famine reg. x Ages 10 – 14	-0.11	(0.10)	-0.57***	(0.12)	0.09†	(0.05)	-0.04	(0.06)
Famine region	0.55***	(0.09)	0.86***	(0.08)	0.25	(0.04)	0.43	(0.05)
Birth groups								
1st trimester	0.73***	(0.07)	1.63***	(0.07)	1.0***	(0.05)	1.01***	(0.05)
2nd trimester	0.78***	(0.08)	1.63***	(0.07)	1.08***	(0.06)	1.03***	(0.06)
3rd trimester	0.83***	(0.09)	1.65***	(0.08)	1.12***	(0.07)	0.98***	(0.07)
Ages 1-4	0.66***	(0.06)	1.49***	(0.05)	0.81***	(0.03)	0.77***	(0.03)
Ages 5-9	0.36***	(0.06)	1.01***	(0.05)	0.34***	(0.03)	0.29***	(0.03)
Ages 10–14	0.19**	(0.06)	0.62***	(0.05)	0.1***	(0.03)	0.04	(0.04)

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001; Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Appendix

Table A1. Difference in difference logistic regression estimates for stroke.

	Females		Males	
Interactions	*β*	SE	*β*	SE
Famine reg. x 1st trim.	0.14	(0.24)	-0.24	(0.32)
Famine reg. x 2nd trim.	-0.40	(0.25)	0.02	(0.25)
Famine reg. x 3rd trim.	-0.30	(0.29)	0.03	(0.32)
Famine reg. x Ages 1-4	-0.12	(0.14)	-0.24	(0.33)
Famine reg. x Ages 5-9	-0.21	(0.14)	-0.14	(0.13)
Famine reg. x Ages 10 – 14	-0.10	(0.14)	-0.02	(0.13)
Famine region	0.06	(0.13)	0.05	(0.11)
Birth groups				
1st trimester	-1.13***	(0.13)	-1.49***	(0.16)
2nd trimester	-0.79***	(0.13)	-0.99***	(0.15)
3rd trimester	-1.02***	(0.17)	-1.25***	(0.20)
Ages 1-4	-0.86***	(0.09)	-1.06***	(0.08)
Ages 5-9	-0.55***	(0.09)	-0.71***	(0.08)
Ages 10–14	-0.32***	(0.09)	-0.37***	(0.08)

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001 ;. Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Table A2. Difference in difference negative binomial regression estimates for number of chronic conditions.

	Females		Males	
Interactions	*β*	SE	*β*	SE
Famine reg. x 1st trim.	0.2	-0.13	0.08	-0.12
Famine reg. x 2nd trim.	0.08	-0.14	0.21 †	-0.12
Famine reg. x 3rd trim.	0.17	-0.15	0.45**	-0.14
Famine reg. x Ages 1-4	0.16	-0.1	0.07	-0.08
Famine reg. x Ages 5-9	0.15	-0.1	0.02	-0.08
Famine reg. x Ages 10 – 14	0.17	-0.11	0.04	-0.08
Famine region	-0.22	-0.09	-0.05	-0.07
Birth groups				
1st trimester	0.28***	-0.06	0.34**	-0.05
2nd trimester	0.49***	-0.06	0.65***	-0.05
3rd trimester	0.78***	-0.06	0.98***	-0.05
Ages 1-4	0.89***	-0.09	1.2***	-0.08
Ages 5-9	0.69***	-0.08	1.16***	-0.07
Ages 10–14	0.95***	-0.08	1.23***	-0.07

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001; IRR = Incidence Risk Ratios; Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Table A3. Difference in difference logistic regression estimates for excessive drinking.

	Females		Males	
Interactions	β	SE	β	SE
Famine reg. x 1st trim.	-0.08	-0.21	0.12	-0.22
Famine reg. x 2nd trim.	0.22	-0.21	0.01	-0.23
Famine reg. x 3rd trim.	0.11	-0.23	0.06	-0.25
Famine reg. x Ages 1-4	0.17	-0.16	-0.04	-0.16
Famine reg. x Ages 5-9	0.16	-0.16	0.25	-0.17
Famine reg. x Ages 10 – 14	0.09	-0.17	0.19	-0.18
Famine region	0.17	-0.15	0.27†	-0.16
Birth groups				
1st trimester	0.39***	-0.11	0.33***	-0.12
2nd trimester	0.65***	-0.1	0.66***	-0.11
3rd trimester	1.01***	-0.1	1.27***	-0.1
Ages 1-4	1.23***	-0.15	1.37***	-0.16
Ages 5-9	1.16***	-0.13	1.47***	-0.14
Ages 10–14	1.37***	-0.12	1.43***	-0.13

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001; Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Table A4. Difference in difference logistic regression estimates for daily smoking.

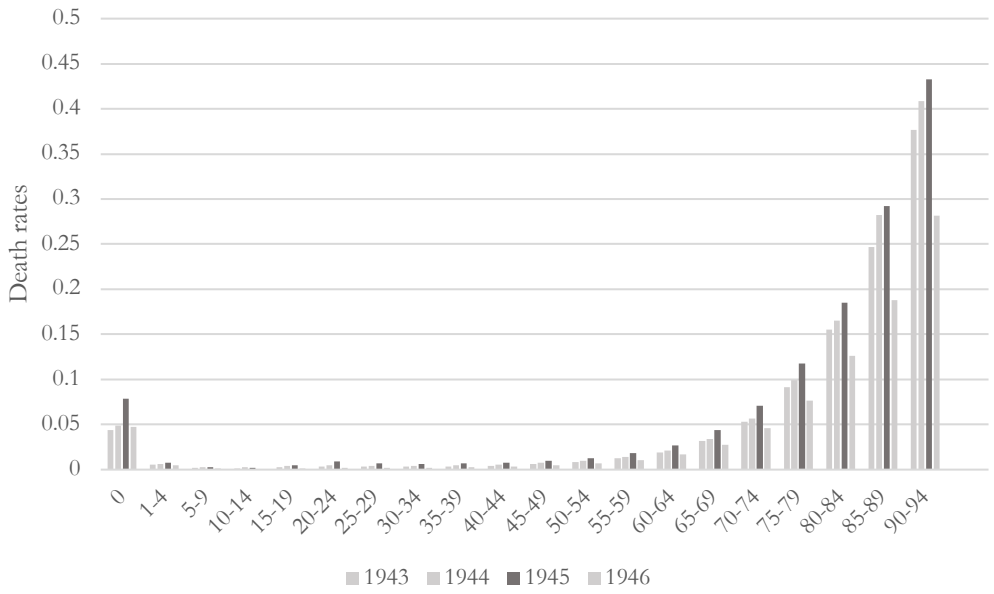
	Females		Males	
Interactions	β	SE	β	SE
Famine reg. x 1st trim.	-0.35	-0.47	0.6	-0.39
Famine reg. x 2nd trim.	-0.44	-0.45	0.29	-0.39
Famine reg. x 3rd trim.	-0.53	-0.5	0.37	-0.46
Famine reg. x Ages 1-4	-0.3	-0.34	0.33	-0.32
Famine reg. x Ages 5-9	-0.28	-0.35	0.24	-0.33
Famine reg. x Ages 10 – 14	-0.03	-0.38	0.52	-0.35
Famine region	0.46	-0.33	-0.18	-0.3
Birth groups				
1st trimester	0.52***	-0.25	0.11***	-0.2
2nd trimester	1.11***	-0.23	0.60***	-0.18
3rd trimester	1.50***	-0.23	1.05***	-0.18
Ages 1-4	1.95***	-0.31	1.03***	-0.28
Ages 5-9	1.98***	-0.28	1.55***	-0.22
Ages 10–14	1.63***	-0.28	1.21***	-0.22

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001 ; Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Mortality

In this section we discuss selection effects on mortality in further detail. Figure A1 shows death rates by age groups in Netherlands between 1943 and 1946 sourced from the Human Mortality Database [10]. As can be seen in both extremes of the figure, there is a substantial increase in death rates for ages 0 and 60+ in 1944. This clearly indicates individuals affected during their postnatal stage and the elderly were subject to heightened mortality during 1944. As such, this evidence indicates there are mortality selection effects on account of the immediate death tolls stemming from famine exposure.

Figure A1. Death rates by age groups in Netherlands 1943-1946



Notes: Death rates are expressed as a ratio of the death count for a given age-time interval divided by an estimate of the exposure-to-risk in the same interval. Source: Human Mortality Database.

However, there is another source of mortality selection beyond immediate or short term deaths - i.e. survivors. Individuals who did not experience death between 1944 and 1946, and survived, could have experienced a decrease in longevity strong enough to condition individuals' ability to reach the GEMON survey in 2016. To model such selection effects we study the timing of all-cause mortality, using the *Doodorztab* dataset provided by Microdata CBS. This dataset contains the date of death of all Dutch people who have died since January 1st, 1995 and were registered in the Personal Records Database (BRP) on the date of death. This dataset is subsequently linked to information on place of birth and date of birth relying on census data from population

[10] It is worth noting these death rates are national averages. Hence, given there were only three famine-affected provinces, the concentrated death rates related to the Dutch Famine are understated.

registries provided by Microdata CBS as well. The linkage of these sources of information relies on Netherlands' unique identification number (i.e., RIN).

The first stage of this study's analytical approach consists of a series of Cox proportional hazards models applied to each age group of interest with an age-time scale. Because of data availability within the *Doodorstab* dataset, the earliest recorded death that is linkable to individual identifiers is in 1995 -i.e., the entry date to the study in this survival analysis. Additionally, the data is right-censored to 2019 on account of data availability as well. The Cox proportional hazards model utilized in the study is expressed in the following functional form:

$$h_{ij}(t) = h_0 * \exp\left(\beta_1 \mathbf{R}_i + \beta_2 \mathbf{X}_i\right)$$

Where $h_{ij}(t)$ is the hazard function determined by the covariates for subject *i* in the age group *j*, h_0 is the baseline hazard, **R** is a dichotomous variable that captures whether individuals were born in a famine region, and ***X*** is a covariate indicating gender. In subsequent models I estimate the same equation separately for females and males to gauge gender differences in vulnerability of exposure to famine.

In order to estimate the causal effect of being exposed to a context of famine, I take on a Difference in Difference approach in estimating the following Cox regression equation:

$$h_i(t) = h_0 * \exp\left(\beta_1 \mathbf{T}_i + \beta_2 \mathbf{R}_i + \beta_3 \mathbf{T}_i x \mathbf{R}_i + \beta_4 \mathbf{x}_i\right)$$

Where $h_i(t)$ represents the hazard function determined by the covariates for subject *i.* $\mathbf{T}_i$ represents a set of dummy variables that stem from the categorical variable that delimits the timing of exposure to famine for individual *i,* and $\mathbf{R}_i$ represents a set of dummy variables that stem from the categorical variable for region of birth for individual *i*. These two sources of variation—time and place of birth—are random in nature, allowing to set a difference-in-difference estimation method (Branas et al. 2011). Hence, the parameters of interest is β_3 as it stems from the estimated coefficients of the set of interaction terms $\mathbf{T}_i x \mathbf{R}_i$ .These interaction terms represent the treatment effect of being born in the Western area of the Netherlands during the Dutch Hunger winter. They represent the effect of exposure over and above the effect of birth in a region in the West, and over and above the effect of being born at a particular moment in time. Finally, $\mathbf{x}_i$ is a gender covariate. we estimate subsequent models by gender to gauge whether treatment effects of the famine differ by gender.

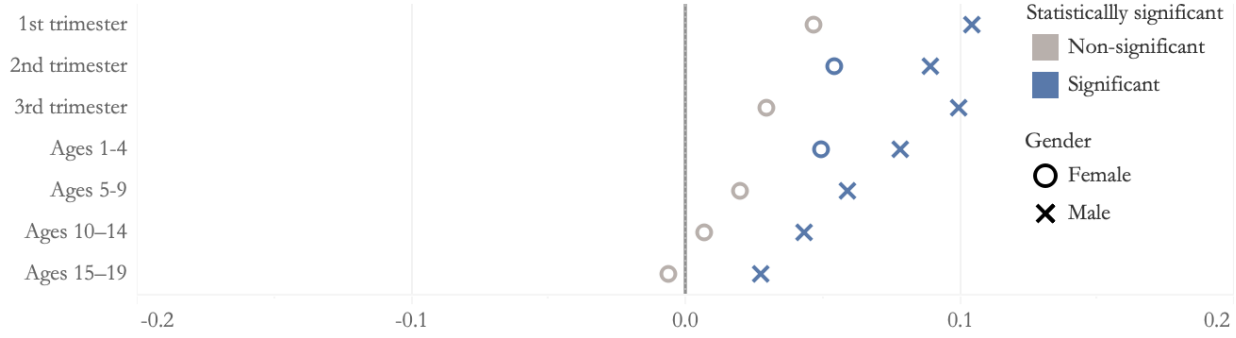
Table A5 shows risk of death for famine exposure estimates utilizing difference-in-difference Cox regression estimates by gender. The first set of columns shows the famine effects for males, whereas the second column shows the effects for females. The key coefficients are the ones belonging to the interaction terms, as they represent the famine effects on the risk of death. As can be seen in the table, males exposed while in the 1st trimester of gestation show a hazard rate of 1.12 ($e^{0.105}$) above and beyond differences brought about region and cohort, and this coefficient is statistically significant at a P<0.001. Males in the remaining in-utero stages show similar coefficient sizes, yet; those cohorts exposed in early infancy show a drop in coefficient size of approximately 0.02. This drop is progressive throughout the age gradient, indicating that vulnerability to famine is higher in-womb (the 1st trimester of gestation in particular). Females, on the other hand, show conflicting evidence. There is somewhat of a concentrated set of effects in the in-womb and early infancy stages -the 2nd trimester and ages 1-4 in particular. Females exposed while in the 2nd trimester of gestation show a hazard rate of 1.055 ($e^{0.054}$) above and beyond differences brought about region and cohort, and this coefficient is statistically significant at a P<0.001.

Table A5. Risk of death for famine exposure; Difference-in-difference Cox regression estimates by gender.

	Males			Females		
	ℓ		SE	*ℓ*		SE
Interactions						
Famine reg. x 1st trim.	0.105	***	0.014	0.046	†	0.025
Famine reg. x 2nd trim.	0.089	***	0.018	0.054	*	0.025
Famine reg. x 3rd trim.	0.099	***	0.030	0.029		0.029
Famine reg. x Ages 1-4	0.078	***	0.012	0.049	***	0.011
Famine reg. x Ages 5-9	0.059	***	0.011	0.020	†	0.011
Famine reg. x Ages 10 – 14	0.043	***	0.010	0.007		0.011
Famine reg. x 1st trim.	0.028	**	0.010	-0.006		0.011
Famine region	0.001		0.009	-0.022	*	0.010
Birth groups						
1st trimester	-0.090	***	0.003	-0.386	***	0.013
2nd trimester	-0.030	***	0.003	-0.331	***	0.014
3rd trimester	-0.062	***	0.003	-0.361	***	0.016
Ages 1-4	-0.107	***	0.003	-0.380	***	0.006
Ages 5-9	-0.086	***	0.008	-0.313	***	0.006
Ages 10–14	0.035	***	0.007	-0.164	***	0.006
Ages 15–19	0.167	***	0.006	-0.051	***	0.006

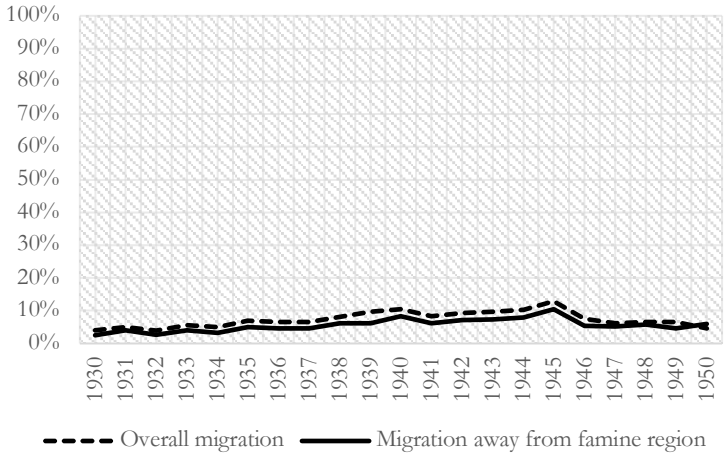
Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001 ;. Ref. group for cohort variables: Post-famine (born 1-4 years after famine). N (4,337,779) ; N Males (2,152,990) ; N Females (2,184,789).

Figure A2. DID treatment effects of famine exposure by age group and by gender.



As can be seen in the figure A2, both genders show a clear gradient of vulnerability by age, where the earlier in the life course (including in-womb stages) the stronger the famine effects on mortality. Additionally, there is a clear difference in vulnerability by gender. Males at all ages (including in-womb stages) show larger treatment effects of famine on risk of death, as compared to females. Furthermore, it seems as there is a slight increase in the gap between the genders during in-womb stages and early infancy. Such gap appears to converge the older the cohort are at the time of exposure. We conclude there is a selection effect on mortality, especially concentrated on males. However, as discussed in the main text, we firmly believe the models show above reveal our main results are bias downward, as our current sample if comprised of individuals who have survived long enough to make the survey in 2016.

Figure A3. Internal migration patterns in Netherlands (1930-1950) obtained from SHARELIFE data.



Placebo models

Table A5. DID Logistic regression estimates for self-rated health (placebo test)

	Females		Males	
Interactions	*β*	SE	*β*	SE
FR x 1st trimester	-0.06	(0.12)	0.03	(0.09)
FR x 2nd trimester	0.02	(0.12)	0.10	(0.08)
FR x 3rd trimester	-0.06	(0.12)	0.12	(0.08)
FR x Ages 1-4	0.31	(0.31)	0.02	(0.17)
FR x Ages 5-9	-0.30	(0.30)	0.01	(0.15)
FR x Ages 10 – 14	-0.16	(0.31)	-0.03	(0.15)
Famine region	-0.13***	(0.09)	0.27†	(0.16)
Birth groups				
1st trimester	-0.42***	(0.07)	0.027***	(0.09)
2nd trimester	-0.91***	(0.07)	0.1***	(0.08)
3rd trimester	-1.11***	(0.07)	0.12***	(0.08)
Ages 1-4	-1.29***	(0.20)	0.016***	(0.17)
Ages 5-9	-1.05***	(0.15)	0.01***	(0.15)
Ages 10–14	-1.261***	(0.14)	-0.03***	(0.15)

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001 ; Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Table A6. DID Logistic regression estimates for diabetes (placebo test)

	Females		Males	
Interactions	*β*	SE	*β*	SE
FR x 1st trimester	0.70	(0.27)	0.13	(0.23)
FR x 2nd trimester	-0.11	(0.30)	0.29	(0.22)
FR x 3rd trimester	-0.12	(0.23)	0.15	(0.23)
FR x Ages 1-4	0.13	(0.15)	0.16	(0.12)
FR x Ages 5-9	0.23	(0.16)	0.06	(0.13)
FR x Ages 10 – 14	1.07	(0.16)	0.02	(0.14)
Famine region	0.04	(0.16)	-0.14	(0.11)
Birth groups				
1st trimester	-0.11	(0.12)	-0.56***	(0.10)
2nd trimester	0.08	(0.12)	-0.51***	(0.12)
3rd trimester	0.2	(0.14)	-0.63***	(0.14)
Ages 1-4	0.07	(0.09)	-0.32***	(0.07)
Ages 5-9	0.15	(0.09)	-0.08	(0.07)
Ages 10–14	0.31	(0.09)	0.12	(0.09)

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001 ; FR = Famine Region; Ref. group for cohort variables: born 1-2 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Table A7. DID Logistic regression estimates for heart attack (placebo test)

	Females		Males	
	β	SE	*β*	SE
FR x 1st trimester	-0.06	(0.12)	0.05	(0.10)
FR x 2nd trimester	0.02	(0.12)	0.13	(0.23)
FR x 3rd trimester	-0.06	(0.12)	0.02	(0.08)
FR x Ages 1-4	0.31	(0.31)	0.02	(0.17)
FR x Ages 5-9	-0.30	(0.30)	0.01	(0.15)
FR x Ages 10 – 14	-0.16	(0.31)	-0.01	(0.12)
Famine region	-0.10	(0.19)	0.07	(0.16)
1st trimester	0.01***	(0.09)	0.47***	(0.08)
2nd trimester	0.09***	(0.08)	0.81***	(0.08)
3rd trimester	0.32***	(0.08)	1.08***	(0.29)
Ages 1-4	0.65***	(0.08)	1.34***	(0.08)
Ages 5-9	0.64***	(0.12)	1.55***	(0.19)
Ages 10–14	0.98***	(0.14)	1.06***	(0.18)

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001 ;. Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Table A8. DID Logistic regression estimates for obesity (placebo test)

	Females		Males	
Interactions	*β*	SE	*β*	SE
FR x 1st trimester	-0.05	(0.12)	0.03	(0.09)
FR x 2nd trimester	0.03	(0.12)	0.10	(0.08)
FR x 3rd trimester	-0.03	(0.12)	0.12	(0.08)
FR x Ages 1-4	0.31	(0.31)	0.02	(0.17)
FR x Ages 5-9	0.24	(0.30)	0.01	(0.15)
FR x Ages 10 – 14	-0.10	(0.31)	-0.03	(0.15)
Famine region	-0.14	(0.10)	-0.05	(0.07)
Birth groups				
1st trimester	0.19***	(0.07)	0.05	(0.08)
2nd trimester	0.31***	(0.07)	0.02	(0.09)
3rd trimester	0.43***	(0.06)	0.11*	(0.06)
Ages 1-4	0.6***	(0.09)	0.12**	(0.05)
Ages 5-9	0.62***	(0.09)	0.11*	(0.05)
Ages 10–14	0.69***	(0.07)	0.19***	(0.05)

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001; Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Table A9. DID Logistic regression estimates for auditive impairment (placebo test)

	Females		Males	
Interactions	*β*	SE	*β*	SE
FR x 1st trimester	0.03	(0.12)	0.03	(0.09)
FR x 2nd trimester	0.04	(0.12)	0.10	(0.08)
FR x 3rd trimester	0.01	(0.11)	0.12	(0.08)
FR x Ages 1-4	0.13	(0.31)	0.02	(0.17)
FR x Ages 5-9	0.23	(0.20)	0.01	(0.15)
FR x Ages 10 – 14	-0.10	(0.21)	-0.03	(0.15)
Famine region	-0.14	(0.10)	-0.05	(0.07)
Birth groups				
1st trimester	0.19***	(0.07)	0.05	(0.08)
2nd trimester	0.31***	(0.07)	0.02	(0.09)
3rd trimester	0.43***	(0.06)	0.11*	(0.06)
Ages 1-4	0.6***	(0.09)	0.12**	(0.05)
Ages 5-9	0.62***	(0.09)	0.11*	(0.05)
Ages 10–14	0.69***	(0.07)	0.19***	(0.05)

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001; Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Table A10. DID Ordered logistic regression estimates for education (placebo test).

	Females		Males	
Interactions	*β*	SE	*β*	SE
FR x 1st trimester	0.05	(0.10)	-0.06	(0.12)
FR x 2nd trimester	0.13	(0.23)	0.02	(0.12)
FR x 3rd trimester	-0.04	(0.14)	0.31	(0.31)
FR x Ages 1-4	0.03	(0.12)	-0.06	(0.12)
FR x Ages 5-9	0.05	(0.12)	0.30	(0.30)
FR x Ages 10 – 14	0.06	(0.10)	-0.13	(0.21)
Famine region	0.05	(0.09)	0.06	(0.15)
Birth groups				
1st trimester	0.73***	(0.07)	1.63***	(0.07)
2nd trimester	0.76***	(0.08)	1.62***	(0.07)
3rd trimester	0.82***	(0.09)	1.64***	(0.08)
Ages 1-4	0.61***	(0.06)	1.49***	(0.05)
Ages 5-9	0.31***	(0.05)	1.01***	(0.05)
Ages 10–14	0.19**	(0.06)	0.62***	(0.05)

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001 ; Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).

Table A11. Ordered logistic regression estimates for income quintile (placebo test).

	Females		Males	
Interactions	*β*	SE	*β*	SE
FR x 1st trimester	0.02	(0.10)	-0.08	(0.12)
FR x 2nd trimester	-0.03	(0.12)	0.12	(0.08)
FR x 3rd trimester	-0.12	(0.23)	0.15	(0.23)
FR x Ages 1-4	0.13	(0.15)	0.16	(0.12)
FR x Ages 5-9	0.02	(0.05)	-0.1†	(0.06)
FR x Ages 10 – 14	-0.03	(0.38)	0.52	(0.35)
Famine region	0.06	(0.15)	0.41	(0.35)
Birth groups				
1st trimester	1.0***	(0.05)	1.02***	(0.05)
2nd trimester	1.02***	(0.06)	1.13***	(0.06)
3rd trimester	1.02***	(0.07)	0.94***	(0.07)
Ages 1-4	0.89***	(0.03)	0.78***	(0.03)
Ages 5-9	0.32***	(0.03)	0.29***	(0.03)
Ages 10–14	0.1***	(0.03)	0.04	(0.04)

Notes: Standard errors in parentheses; † p<0.1, * p<0.05, ** p<0.01, *** p<0.001 ; Ref. group for cohort variables: born 1-4 years after famine. Survey fixed effects to capture potential differences through GEMON waves are included (not shown).