The Effects of Prenatal Exposure to the Great Chinese Famine on Mortality and Education: A Sibling Fixed Effects Approach

Weina Zhou*

Hiroyuki Kasahara[†]

June 15, 2023

Abstract

This study investigates the long-term impacts of prenatal exposure to the Great Chinese Famine (1959-1961) on mortality rates and educational attainment. To mitigate sample selection biases associated with selective mortality during the famine, we compare exposed individuals with their unexposed same-sex siblings. Utilizing a measure of famine intensities based on excess death rates at the county-year level, we find that males exposed to the famine in utero exhibit higher mortality rates in their 40s compared to their unexposed male siblings, a trend that intensifies over time. This elevated mortality is associated with worsened health conditions in adulthood. Conversely, we find no evidence of a similar effect on female mortality. Findings from individual-level survey data are supported by aggregate-level analysis. The famine also reduces educational attainment for both exposed males and females.s

JEL Classification: I1, I2, J13, J16, N35, O15

^{*}Department of Economics, Dalhousie University. (Email: weina.zhou@dal.ca)

[†]Vancouver School of Economics, University of British Columbia. (E-mail: hkasahar@mail.ubc.ca)

1 Introduction

The fetal origins hypothesis states that a lack of nutrition during fetal development significantly impacts health outcomes in later life. Over the past decade, a growing body of empirical evidence has demonstrated that severe fetal malnutrition shocks are associated with various adverse health outcomes (Currie and Almond, 2011; Currie and Vogl, 2013; Almond et al., 2018). However, the potential influence of such shocks on exposed individuals' mortality in adulthood, and the age at which this effect becomes evident, remains ambiguous.

Any investigation into the long-term impact of a severe fetal malnutrition shock, such as famine, encounters identification challenges due to selection biases. Families of lower socioeconomic status (SES) may opt to postpone or even abstain from conception, leading to selective fertility. Moreover, only children with healthier genes or higher family SES are likely to be born and survive early childhood during famine, resulting in selective mortality. Consequently, individuals who were born and were observed to have been exposed to adversity in utero may exhibit different outcomes compared with unexposed individuals, due not only to the impact of the in utero shock but also to differences in unobserved family background characteristics and genes. These selection issues are particularly relevant in the context of famines, where malnutrition is widespread, and the biases associated with these selections are likely to be significant (Almond, Currie, and Duque, 2018). While most existing studies in the famine literature acknowledge these issues, they largely remain unaddressed, potentially obscuring the true consequences of fetal malnutrition in earlier studies.

This paper examines the long-term effect of prenatal exposure to the Great Chinese Famine (1959-1961), an event leading to 16.5-30 million fatalities and over 30 million delayed or lost births (Lin and Yang, 2000; Kasahara and Li, 2020). We aim to address gaps in the literature by employing a sibling fixed-effects model to tackle selection issues and estimate the impact on education and mortality as individuals exposed to the famine in utero reach mid to late adulthood. Historical events such as famines yield rare data that enable researchers to observe the long-term effects of a fetal malnutrition shock over half a century post-exposure. By comparing individuals prenatally exposed to the famine with their unexposed siblings, we mitigate the selection biases arising from differences in unobserved family genes and family SES.

Our findings reveal that prenatal famine exposure increases the mortality rate of males in their early 40s by 0.03 on average, approximately 14% of a standard deviation, compared to their unexposed male siblings. Furthermore, the impact on mortality only becomes evident after exposed males reach their 40s and gradually intensifies throughout their late 40s and early 50s. Conversely, we observe no effect on the mortality rates of exposed females across different age groups. Regarding education, we find that the famine equally impacted both exposed males and females, resulting in an average reduction of 0.3 years of education, approximately 7% of a standard deviation. This estimated effect on education is larger compared to findings in previous studies where selection issues are not fully addressed (Meng and Qian, 2009; Fan and Qian, 2015).

To explore the underlying mechanism, we find suggestive evidence that the increased mortality among males is linked to deteriorated health conditions in adulthood: exposed males are more likely to have been bedridden or hospitalized for over a month in adulthood. This finding aligns with the existing literature which suggests that prenatal exposure to severe nutritional shocks correlates with poor health outcomes in adulthood, including high blood glucose, high blood pressure, and increased risk of stroke, heart disease, diabetes, schizophrenia, and disability.¹ On the other hand, we find no evidence to suggest that the higher mortality rate among males is due to lack of healthcare access or a higher likelihood of physical injury. The fact that only males experience early mortality—while both genders suffer in terms of educational attainment—suggests that the primary cause of early death is unlikely to operate through the education channel. Similarly, given the well-documented son preference in rural China, it is also unlikely that unequal distribution of parental resources across siblings is the main cause of early male mortality, as opposed to females.

Our findings regarding midlife male mortality are corroborated by evidence from an alternative data source: Using aggregate-level mortality data from 2005, 2010, and 2015, we find that famine exposure in the birth year correlates with a 6.7% standard deviation increase in mortality rates in individuals' mid-40s and 50s. This effect is once again observed only in males; no significant impact on female mortality rates is detected. Our aggregate-level analysis employs a generalized Difference-in-Difference-in-Differences (DDD) strategy, leveraging variations in famine intensity across birth cohorts, ages, and provinces. However, as sibling information at the aggregate level is less meaningful, selection issues persist; the effect size is smaller compared to the result from the sibling fixed-effects model, which utilizes individual-level data.

This paper uses a more detailed famine intensity measure than most existing studies: the excess death rate at the county-year level. This level of data aggregation is more precise than the province-year or prefecture-year level data typically used in previous Chinese famine studies. China's administrative divisions comprise five levels: province (1st level), prefecture (2nd level), county (3rd level), and two smaller divisions below the county level. The county-year level death rate data, sourced from Kasahara and Li (2020), suggest that over 80% of the death rate variation during the famine occurs within provinces. This detailed and accurate measure of famine intensity enables more precise impact estimation in our study.

Our study contributes to the literature by investigating the long-term impact of prenatal shocks on adult mortality rates, which remains a relatively understudied area. Animal research has long established that prenatal nutrition shocks not only increase disease risk but also lead to higher mortality rates later in life.² However, there is a lack of consistent and robust evidence

¹See reviews in Almond and Currie (2011); Currie and Almond (2011)' Currie and Vogl (2013); Almond et al. (2018) and Chen (2022).

 $^{^{2}}$ See Sayer et al. (2001); Ozanne and Hales (2004); and Langley-Evans and Sculley (2006).

regarding whether humans also suffer the similar consequences. Lindeboom et al. (2010) find that the Dutch potato famine of 1846-1847 associated with higher mortality rates for males aged 50 and older, with a small effect observed for lower-SES females. Conversely, van Abeelen et al. (2012) find that prenatal exposure to the Dutch winter famine of 1944-1945 associated with higher adult mortality rates for females but not males. Like many other studies in the field, both rely on cross-cohort comparison, leaving selection issues unresolved. We are not aware of any examination of the effect of prenatal famine exposure on adult mortality rates in other countries. Beyond famine studies, a small body of research explores the relation between economic conditions at birth and mortality in adulthood (Van den Berg et al., 2006, 2011; Yeung et al., 2014). These studies use business-cycle fluctuations at the aggregate level as exogenous shocks to individuals and find that being born during an economic downturn is associated with higher mortality rates in later life. Our study contributes to the literature by being the first to examine the long-term impact on adult mortality in the context of the Chinese famine. Additionally, it is also the first to employ a sibling fixed-effects model to address the selection issue in examining the impact on the mortality rate in adulthood.

This paper further contributes to the literature by suggesting that the true effect of prenatal exposure to the Chinese famine on educational attainment appears to be much larger after correcting for selection bias. Although the impact on education has been relatively well-examined, the estimated effects found in prior studies are relatively small. For example, the size of the estimated effect on years of education using sibling comparisons in this paper are at least twice as large as the size reported in early studies (Meng and Qian, 2009; Fan and Qian, 2015), in which cross-cohort comparison is the main estimation strategy.

The remainder of the paper is structured as follows. Section 2 provides a brief background of the Chinese Famine and a literature review on the impact of in utero exposure to the Chinese Famine on later life outcomes. Section 3 describes our empirical methodology. Section 4 reports our data sources and sample restrictions. Section 5 presents our results. Section 6 provides additional analysis, which include using alternative data sources and a birth-month-weighted famine intensity measure. Section 7 explores and discusses the potential channels through which the effects may operate. Section 8 concludes the paper.

2 Background and literature review

The Great Chinese Famine (1959-1961) resulted in an estimated loss of 16.5 to 30 million lives, predominantly in rural areas (Lin and Yang, 2000). One contributing factor was the central government's policy of diverting substantial grain resources from rural to urban areas (Meng et al., 2015). Additionally, strict internal migration controls in place before the famine prevented individuals from escaping regions severely affected by hunger. Kasahara and Li (2020) further suggest that an increase in grain exports during the famine likely worsened the situation.

A growing number of studies have investigated the effect of prenatal exposure to famine on later-life health outcomes, primarily using two individual-level survey data sets: the China Health and Nutrition Survey (CHNS) and the China Health and Retirement Longitudinal Study (CHARLS). Previous research indicates that early-life famine exposure is associated with lower height (Chen and Zhou, 2007; Meng and Qian, 2009; Fung and Ha, 2010; Gørgens et al., 2012); anemia (Shi et al., 2013), the prevalence of hyperglycemia (Li et al., 2010), high blood pressure (Wang et al., 2012), overweight or obesity (Luo et al., 2006; Meng and Qian, 2009), and risk of developing schizophrenia (St Clair et al., 2005; Xu et al., 2009). Also, Almond et al. (2007) use 2000 Chinese Census data and find famine has a negative effect on the exposed cohort's literacy, labor market status, wealth, and marriage market outcomes. Chen (2022) provides a comprehensive summary of the effects studied in the literature to date. While most studies approximate famine intensity using rough measures of province-level death rates or county-level cohort sizes decades after the famine, two recent studies use hunger recall to minimize potential measurement error. Cui et al. (2020) find that exposure to famine in childhood increases the probability of being overweight, having difficulty with daily living activities, and depression in old age, and Deng and Lindeboom (2022) find that hunger exposure increases the risk of metabolic syndrome in later life.

3 **Estimation Strategy**

We use the excess death rate (EDR) to measure famine intensity in year y and county c. EDR represents the difference between the death rate in a famine year and the average death rate before the famine period:

$$EDR_{yc} = Death \, Rate_{yc} - Base \, Death \, Rate_c, \tag{1}$$

where $Death Rate_{yc}$ is the number of deaths per 100,000, and $Base Death Rate_c$ is the average number of deaths per 100,000 over the 5 years before the famine, 1954-1958.³ Note that we assume there was no excess death rate in the non-famine period.⁴

The following regression model estimates the impact of prenatal exposure to the famine on later life outcomes.

$$Y_{ijyc} = \alpha E D R_{yc} + \beta X_{ijyc} + \theta_j + \delta_y + \epsilon_{ijyc}, \tag{2}$$

where Y_{ijyc} represents the outcome of individual *i*, born to family *j* in year *y* and country c. X_{ijyc} indicates individual i's characteristics. In the baseline model, X_{ijyc} only includes a

³Defined as Base Death Rate_c= $\frac{1}{5}\sum_{j=1954}^{1958}$ Date Rate_{jc}. ⁴In other words, $EDR_{yc} = 0$ if y < 1959 or y > 1961.

female dummy. θ_j is the sibling fixed effects of family j while δ_y is the birth year fixed effects, controlling for year-specific unobserved factors. ϵ_{ijyc} is the error term clustered at the county level to allow within-county correlations. County fixed effects are included in sibling fixed effects, as we assume that siblings were born in the same county.

As discussed earlier, selective fertility and selective mortality pose significant challenges in identifying the long-term impacts of severe in-utero stress factors such as a famine. Because of these two selection processes, the famine cohort—that is, individuals who were born and survived during the famine—is likely to have higher parental SES and/ or better innate biological endowment compared with the non-famine cohort, thereby underestimating the adverse effects of the famine.

The sibling fixed-effects model mitigates this issue by comparing the outcomes of a child born during the famine with those of a sibling from the same family who was not born during the famine. This identification strategy effectively eliminates all bias caused by unobserved family background characteristics, such as family SES and genetic quality.

In addition to within-family variation, the identification strategy also leverages cross countyyear level variation in famine intensity to estimate a differential effect. Essentially, α is identified by comparing the within-family difference in a family with a child exposed to high famine intensity, with the within-family difference in a family with a child who was exposed to low famine intensity. County-year level variation in EDR facilitates such a comparison.

The estimated prenatal effect may also include a postnatal malnutrition effect, as individuals born between 1959 and 1960 were not only affected in utero but may have also experienced malnutrition in the first one or two years after birth. We address this issue in Section 6.2 by excluding all individuals born in the first two years of the famine and further use a birth-month weighted measure of famine intensity.

A potential concern is that regional factors associated with famine intensity might also affect individual outcomes in the long run. Famine intensity has been linked not only to weather conditions but also to various regional factors, such as labor productivity, land quality, and propensity to export during a famine (Li and Yang, 2005; Meng et al., 2015; Kasahara and Li, 2020). These factors, although challenging to measure, are likely to have long-term impacts on individuals' well-being. However, these factors can be controlled for using the sibling fixedeffects model. Sibling fixed-effects ensure that our famine intensity measure, EDR_{yc} , is not correlated with such historical regional factors, as siblings within a family share the same regional factors.

A concern related to using the fixed-effects model is that siblings may have different experiences within the same family, leading to different outcomes. Birth order has been identified as a significant factor driving within-family differences (Black et al., 2021). To address this concern, we control for birth order fixed effects in our robustness test.

Similar to many sibling studies that examine prenatal shocks (Currie and Walker, 2011;

Black et al., 2021; Daysal et al., 2022), this study is based on the assumption that genetic variation within families is not strong enough to cause differences in outcomes. Half of the genome that varies between humans is identical in siblings, while the other half shows differences but still exhibits some degree of similarity. (Gagnon et al., 2005; Lin et al., 2007). If within family variation is strong enough to affect a sibling's mortality in early childhood, our parameter of interest, α , represents a lower bound of the true effect of prenatal famine exposure. However, the downward bias caused by selective mortality driven by within-family difference is likely to be much smaller compared with the bias in cohort comparison studies in the existing literature.

Another common issue in estimating a sibling fixed-effects model is potential family relocation in response to environmental factors (Currie and Vogl, 2013). However, in the context of rural China during the 1950s–1970s, internal migration was rare because of the household registration system (Chen and Zhou, 2007; Wang and Zhou, 2017).

4 Data

4.1 Famine-era Death Rate Data

Our death rate data are from Kasahara and Li (2020), who document county-level death rates between 1954 and 1965 in 1,803 rural counties. Kasahara and Li (2020) collected data from population statistics yearbooks published by regional statistics bureaus in the 1980s, which were cross-checked with the province-level data used by Lin and Yang (2000) and Meng et al. (2015). The county-year level EDR data between 1959 and 1961 (computed using Equation 1) in Kasahara and Li (2020)'s whole sample has a mean of 0.081 with a standard deviation of 0.178. The standard deviation of EDR after factoring out province fixed effects is 0.15, suggesting that about 84% (=0.15/0.178) of the variation in death rates occurs within provinces.

4.2 Individual-level Survey Data

The China Family Panel Study (CFPS) is a biannual longitudinal survey conducted by Peking University. This paper uses the CFPS's first wave, 2010, to estimate the long-term effect of in utero exposure to the famine. An important feature of CFPS 2010 is that it contains detailed information on all siblings that respondents have ever had, including deceased siblings. CFPS 2014 further asks respondents to confirm whether the information reported in 2010 regarding deceased siblings is correct to improve data accuracy. Such a survey feature is rare, yet it is essential for the comparison of mortality across siblings. The CFPS 2010 covers 163 counties in 25 provinces and is considered to be a nationally representative survey.

4.3 Regression Sample

We match the data on 1,803 county-year-level famine-era death rates from Kasahara and Li (2020) with the 164 counties from the CFPS data. Fifty-four counties across 18 provinces are matched based on the two data sources. Figure A.1 in Appendix reports the matched counties on a map. Note that Kasahara and Li (2020) only document death rates in rural counties, because famines mostly occurred in rural areas (Meng et al., 2015). For this reason, urban areas in the CFPS, such as Shanghai and Guangzhou, in which the CFPS is over sampled, are not included in our analysis. The average EDR in the matched counties is similar to that in Kasahara and Li (2020)'s full sample, which is about 0.08.

In the 54 matched rural counties, we restrict our final sample based on the following criteria:

- 1. Individuals who have at least one sibling.
- 2. Information related to a sibling's death is consistently reported across the CFPS 2010 and CFPS 2014.
- 3. Individuals born after introduction of the one-child policy (1978) are excluded, because the number of siblings and siblings' gender composition are likely to be distorted after the policy (Chen, Li, and Meng, 2013).
- 4. Individuals whose birth province is the same as their current province, which minimizes the impact of migration after the 1980s.⁵

After further dropping missing values, our final sample consists of 7392 individuals from 2418 families across 54 counties.

4.4 Summary statistics

Table 1 reports summary statistic. "Famine cohort" is defined as individuals born during the famine (1959-1961) while "Non-famine cohort" represents individuals who were born before or after the famine. The EDR during the famine has a mean of 0.083 with a standard deviation of 0.159. By definition, the EDR for the non-famine cohort is zero.

We observe that the famine cohort has a higher education level and a lower proportion of deceased individuals compared with the non-famine cohort, likely due to selection effects. The famine cohort on average has 6.9 years of education, which is about 1 year more than the non-famine cohort. The difference is statistically significant at the 1% level. The variable "Deceased" equals one if an individual had died by the survey year 2010 and zero otherwise. The famine cohort has a slightly smaller percentage of people who had died by the survey year, compared to the non-famine cohort: 4.1% vs 4.8%. However, the difference is not statistically

⁵Migration within a province could occur. Using China Health and Retirement Longitudinal Study (CHARLS) data, we find that 6% of rural individuals migrated to another county in the same province.

significant. The average family size in the sample is about 4. The larger sibling size is likely due to the population expansion policies implemented during the 1950s and 1960s (Zhou, 2014).

5 Estimation Results

Table 2 presents the results of the estimation of equation (2). Panel A uses "Years of Education" as an outcome, while Panel B uses the binary variable "Deceased" as an outcome. Column 1 controls for county fixed effects, while the remaining columns control for sibling fixed effects.

In Panel A, Column 1, where county fixed effects are controlled for instead of sibling fixed effects, the coefficient on EDR is positive. However, when we control for sibling fixed effects in Column 2, the coefficient turns negative and the standard error also decreases, although the coefficient is not statistically significant at conventional levels. These results align with our discussion suggesting that the sibling fixed-effects model corrects biases that underestimate the famine effect. In Columns 3 and 4, where the sample is divided by gender, we observe that the coefficient on EDR becomes negative and statistically significant. Applying the average EDR during the famine, the estimation results suggest that the famine has, on average, reduced males' and females' years of education by $0.33 (=0.083 \times 3.940)$ and $0.30(=0.083 \times 3.63)$ respectively, which is about 7% ($\approx 0.30/4.392$) of the standard deviation for years of education in the sample.

In Panel B, using "Deceased" as an outcome variable, we observe a similar tendency where the effect of the famine is likely to be underestimated without sibling fixed effects. We also find a significant gender difference. While the famine increases the probability of death for males, it does not affect females. Column 3 of Panel B suggests that the famine has on average increased males' probability of death by 4 percentage points (= 0.083×0.49), which is about 19% (=0.04/0.21) of the standard deviation in the whole sample. Conversely, the coefficient on EDR in the female sample (Column 4) is negative and not statistically significant.

To verify the robustness of the results, Panel A of Table 3 incorporates birth order fixed effects into our baseline estimation. The results remain consistent. The impact of the famine on males' education is not statistically significant at a conventional level. In Panel B and C, we further falsely assign famine-era EDR to individuals born 10 years before and 10 years after the famine. We do not observe any effect from the falsely assigned EDRs on these cohorts. Additional robustness checks are provided in the subsequent section.

To our knowledge, no studies have investigated the effect of prenatal exposure to the Chinese famine on mortality during adulthood. However, studies that utilize cross-section variation in famine intensity have found the effect of the famine on years of education to be smaller than the effect observed in our study. For example, Meng and Qian (2009) find that prenatal exposure to the famine only reduces years of education for individuals in the 90th percentile of the education distribution by 0.012 to 0.016 years, with no average effect observed.⁶ Fan and Qian (2015) find no effect on years of education. In contrast, Almond et al. (2007) find that famine-exposed cohorts were 7.5% more likely to be illiterate for women, and 9% more likely to be illiterate for men. Both Mu and Zhang (2011) and Shi (2011) find that the effect on education only affected females and not males. Our results, which correct for selection bias, suggest much larger effects on education.

6 Additional Analysis

6.1 The effect of famine on death by age

Panel B of Table 2 estimates the effect of famine on the likelihood of being deceased by 2010, when the famine cohort is approximately 50 years old. To further investigate the age at which the famine impacts an individual's likelihood of being deceased, we estimate the following:

$$Deceased^a_{ijuc} = \lambda EDR_{yc} + \theta_j + \delta_y + e_{ijyc}, \tag{3}$$

where $Deceased_{ijyc}^{a}$ indicates whether an individual died before age a. We use 5-year increments in age a, where a is 5, 10, 15, and so on. Note that the variable is conditional on an individual having been born at least a-5 years ago; individuals born less than a-5 years ago are treated as missing values. For example, individuals currently aged under 40 in the sample are treated as missing values in variable $Deceased_{ijyc}^{45}$, because the mortality of such an individual between the ages of 40 and 45 is unobserved at this point. The parameter λ estimates the effect of exposure to the famine in a birth year on an individual's likelihood of being deceased by age a. We report the estimation results in Table A.1 in Appendix and plot the coefficient on EDR along with its 95% confidence interval in Figure 1.

In Panel A of Figure 1, which presents the brothers' sample, we observe that the impact on male mortality at each age is either very small or statistically insignificant until the age of 40. Beyond 40, the magnitude of the effect gradually increases, and the effects become statistically significant. The estimation results suggest that in utero exposure to famine increases the probability of being deceased by age 45 by 3 percentage points, accounting for approximately 14% of a standard deviation. We further conduct a placebo test to examine the reliability of the results. Figures b and c of Panel A falsely assign the famine-era EDR to individuals born 10 years before and 10 years after the famine, respectively. As expected, we do not observe a similar effect on their mortality.

Panel B of Figure 1 repeats the same exercises using the sisters' sample. Consistent with

 $^{^{6}}$ For individuals in the 90th percentile of the distribution of the outcome, Meng and Qian (2009) report that in-utero exposure to famine reduces those individuals' years of education by 0.174% in their OLS estimation and 0.238% in their 2SLS estimation.

our main results in Table 2, we do not observe any impact of the famine on female mortality across any age group.

6.2 Using birth-month-weighted EDR

To measure an individual's *in utero* exposure to the famine's intensity, we weight EDR by months *in utero* during birth year y - 1 and year y. For example, an individual born in January 1961 in a county is assigned 1/9 of the county's excess death rate in 1961 and 8/9 of the county's EDR in 1960; an individual born during September to December of a year is assigned that year's excess death rate. We define the weighted excess death rate (WEDR), denoted as $WEDR_{umc}$, as the following

$$WEDR_{ymc} = \begin{cases} \frac{m}{9} \times EDR_{yc} + \frac{9-m}{9} ERD_{y-1,c} & if \ m \le 8\\ EDR_{yc} & if \ 9 \le m \le 12, \end{cases}$$
(4)

where m denotes the individual's birth month. $WEDR_{ymc}$ equals zero if the individual was born before January 1959 or after August 2022, since they were not exposed to the famine in utero.

We re-estimate equation (2) using $WEDR_{ymc}$ instead of EDR_{yc} . Birth month fixed effects are also added to the equation to control for month-specific unobserved factors. Panel D of Table 3 suggests that the estimation results are essentially unchanged.

Panel E of Table 3 further excludes the famine cohort born in 1959 and 1960, including only individuals born in the last year of the famine, 1961, as the treatment group. This approach minimizes the impact of post-birth exposure to famine experienced by individuals born in the first 2 years of the famine. However, as a trade-off, we lose two-thirds of the observations in the treatment group, leading to imprecise estimation. Our findings show that the estimation results for mortality and female education remain unchanged, although the effect on male education becomes statistically insignificant, potentially reflecting a loss of power associated with excluding treated observations in 1959 and 1960.

6.3 Estimating the effect on mortality using aggregate level data

This subsection explores the impact of the famine on mortality using aggregate-level data. A detailed analysis is provided in Appendix A, with a summary of the results presented in the remainder of this subsection.

We procured province-by-age-group-level mortality rate data in three census years (2005, 2010, and 2015) from the National Bureau of Statistics of China. These data were matched with the Excess Death Rate (EDR) data gathered by Kasahara and Li (2020). In our estimation, we controlled for age group, survey year, and province fixed effects. Our identification strategy

ensures that the intensity of the famine is exogenously given; once these three types of fixed effects are accounted for, the intensity of the famine is not systematically correlated with other unobserved factors that could affect individuals' mortality later in life. However, our identification strategy does not eliminate bias from selective mortality or selective fertility, potentially leading to an underestimation of the true effect of the famine.

In line with our main findings using the China Family Panel Studies (CFPS) data, the estimation results based on the aggregate-level data also indicate that the famine increases male mortality when the famine cohort reaches their 40s and 50s. However, no significant effect is observed for females. The results suggest that exposure to famine during the birth year increases the male mortality rate in adulthood by 6.7% of the standard deviation of the mortality rate in the aggregate sample. This effect's magnitude is smaller compared to the results obtained from the CFPS data (approximately 19% of the standard deviation). A potential explanation for this discrepancy is that the use of cross-sectional variation in the data introduces selection issues, leading to an underestimation of the results.

7 Potential mechanisms

This section investigates several potential mechanisms that could lead to premature death for exposed males. The first plausible mechanism is that the adverse in utero environment created by the famine could have *programmed* individuals to have deteriorated health conditions in adulthood, as suggested by the fetal origins hypothesis. This hypothesis is supported by substantial empirical evidence indicating that adult disease risks are associated with adverse environmental conditions during fetal development. Researchers in epidemiology have also found that individuals negatively affected in utero exhibit epigenetic marks related to chronic diseases and accelerated aging in DNA markers (Heijmans et al., 2008; Kreier, 2022). Thus, poorer health condition in adulthood could contribute to an individual's premature death. We further examine this channel and add evidence to this extensively studied area. Secondly, we examine whether an individual could also die earlier due to physical injury. Given that the famine causes individuals to have worse educational outcomes, the famine cohort could be more likely to experience physical injury or accidents that could be due to lower cognitive skills. Thirdly, we further examine whether premature death is attributable to a lack of accessible healthcare services, as this cause of death in rural China cannot be overlooked (Gong et al., 2012).

We use the Life History data in the China Health and Retirement Longitudinal Study (CHARLS) 2014 to examine an individual's health history in adulthood and childhood. The advantage of using these data is that the health related questions span across an individual's entire lifespan, rather than being limited to a specific point in time, which is common in standard surveys. Estimation results are presented in Table 4. Summary statistics of the

outcomes variables are reported in Table A.2. All estimations control for birth year fixed effects and county fixed effects. Unfortunately, we are unable to control for sibling fixed effects because of a lack of sibling data, and the cross-sectional comparison could thus underestimate the true effect of the famine.

We find that males born during the famine are more likely to report having poor health conditions in adulthood than males not born during the famine. Specifically, they are more likely to report being confined to bed for health reasons for one month or more, being hospitalized for one month or more, or taking job leave due to health reasons for one month or more, though the difference in the last question is only statistically significant at the 15% level. The estimation results in column 5 further reveal that the poor health outcomes in adulthood are unlikely related to physical injury. We also do not find significant differences between these two groups regarding health conditions before age 15 (column 4) or the availability of health care services to individuals over different ages (columns 6-10). Overall, the results suggest that the premature death of males born during the famine is likely caused by deteriorated health conditions in adulthood, and is unlikely to be caused by accidents or a lack of access to health care services. By contrast, we do not find evidence that females born during the famine are more likely to experience poor health outcomes compared with their counterpart.

Another potential explanation for the observed impact on mortality could be the unequal distribution of resources among siblings, as parents may allocate resources according to their preferences. However, in the context of rural China, where a preference for sons is often observed, female siblings are generally less likely to receive resources compared to their male counterparts. Despite this, we only observe an impact on the mortality of males and not females. This suggests that differences in resource allocation from parents among siblings are unlikely to be the primary cause of the effect of famine on mortality.⁷

Given our findings that being born during a famine results in lower educational outcomes, the higher mortality rate in adulthood could also be a consequence of a lower education level. However, while we find that the impact on females' education is stronger or at least equal to the impact on males' education, we do not observe increased mortality in females. This discrepancy may suggest that education is unlikely to be the primary factor contributing to the high mortality of males. One concern is that a female's health outcomes in adulthood are not only related to her own education, but also to her husband's. If a female born during the famine marries a well-educated husband, the absence of the negative impact on mortality could be explained by the higher education or higher income of her husband. However, we find that conditional on females' own education, being born during a famine is not related to having a relatively well-educated husband.⁸ Using Census data, Almond et al. (2007)find that females born during the famine are more likely to have a husband with lower education. We

⁷Furthermore, we do not observe that the effect of famines differs between first-born son(/daughter) and non-first-born son(/daughter). Estimation results are available upon request.

⁸Estimation results are available upon request.

thus conclude that such an impact is likely to be minimal.

8 Conclusion

This paper investigates the long-term impact of prenatal exposure to the Great Chinese Famine. Our findings indicate that males who were exposed to the famine in utero exhibit a higher mortality rate in their early 40s, which gradually increases through their later 40s and early 50s. However, we do not observe a similar effect on the middle-age mortality of females. In addition, prenatal exposure to the famine also reduces individuals' years of education, with this negative effect affecting both genders equally. The estimation results are obtained by comparing exposed individuals with their same-sex unexposed siblings while controlling for birth year fixed effects in the estimations. Furthermore, our findings provide suggestive evidence that the famine likely contributes to worse health outcomes in exposed males during adulthood, leading to premature death.

The gender differences observed in this study are striking. It is well-known that males tend to experience higher mortality at birth compared with females when exposed to severe malnutrition in utero. Our study's results further suggest that males exposed to severe malnutrition in utero are more likely to face increased mortality in middle age. Interestingly, this effect remains latent during their early adulthood and only becomes evident later in middle age.

Future research could examine the impact on both females' and males' mortality rates over a longer time span throughout their later middle ages and senior life, once the data become available. Given the empirical evidence in the literature that prenatal exposure to malnutrition adversely affects females' later-life health outcomes, it is possible that the mortality rate of females is also affected but only at a later age.

References

- Almond, D. and J. Currie (2011, aug). Killing Me Softly: The Fetal Origins Hypothesis. Journal of Economic Perspectives 25(3), 153–172.
- Almond, D., J. Currie, and V. Duque (2018, December). Childhood Circumstances and Adult Outcomes: Act II. Journal of Economic Literature 56(4), 1360–1446.
- Almond, D., L. Edlund, H. Li, and J. Zhang (2007). Long-term effects of the 1959-1961 china famine: Mainland china and hong kong.
- Black, S. E., S. Breining, D. N. Figlio, J. Guryan, K. Karbownik, H. S. Nielsen, J. Roth, and M. Simonsen (2021, aug). Sibling Spillovers. *The Economic Journal* 131(633), 101–128.
- Chen, X. (2022). Early Life Circumstances and the Health of Older Adults: A Research Note. SSRN Electronic Journal.
- Chen, Y., H. Li, and L. Meng (2013). Prenatal sex selection and missing girls in china: Evidence from the diffusion of diagnostic ultrasound. *Journal of Human Resources* 48(1), 36–70.
- Chen, Y. and L.-A. Zhou (2007, jul). The long-term health and economic consequences of the 1959–1961 famine in China. *Journal of Health Economics* 26(4), 659–681.
- Cui, H., J. P. Smith, and Y. Zhao (2020, mar). Early-life deprivation and health outcomes in adulthood: Evidence from childhood hunger episodes of middle-aged and elderly Chinese. *Journal of Development Economics* 143, 102417.
- Currie, J. and D. Almond (2011). Human capital development before age five. pp. 1315–1486.
- Currie, J. and T. Vogl (2013, aug). Early-Life Health and Adult Circumstance in Developing Countries. Annual Review of Economics 5(1), 1–36.
- Currie, J. and R. Walker (2011, jan). Traffic Congestion and Infant Health: Evidence from E-ZPass. American Economic Journal: Applied Economics 3(1), 65–90.
- Daysal, N. M., M. Simonsen, M. Trandafir, and S. Breining (2022). Spillover effects of early-life medical interventions. *The Review of Economics and Statistics* 104(1), 1–16.
- Deng, Z. and M. Lindeboom (2022, mar). Early-life famine exposure, hunger recall, and laterlife health. Journal of Applied Econometrics 37(4), 771–787.
- Fan, W. and Y. Qian (2015, jan). Long-term health and socioeconomic consequences of early-life exposure to the 1959–1961 Chinese Famine. Social Science Research 49, 53–69.

- Fung, W. and W. Ha (2010). Intergenerational Effects of the 1959–61 China Famine. pp. 222–254.
- Gagnon, A., J. Beise, and J. W. Vaupel (2005). Genome-wide identity-by-descent sharing among CEPH siblings. *Genetic Epidemiology* 29(3), 215–224.
- Gong, P., S. Liang, E. J. Carlton, Q. Jiang, J. Wu, L. Wang, and J. V. Remais (2012). Urbanisation and health in china. *The Lancet* 379(9818), 843–852.
- Gørgens, T., X. Meng, and R. Vaithianathan (2012, jan). Stunting and selection effects of famine: A case study of the Great Chinese Famine. *Journal of Development Economics* 97(1), 99–111.
- Heijmans, B. T., E. W. Tobi, A. D. Stein, H. Putter, G. J. Blauw, E. S. Susser, P. E. Slagboom, and L. Lumey (2008). Persistent epigenetic differences associated with prenatal exposure to famine in humans. *Proceedings of the National Academy of Sciences* 105(44), 17046–17049.
- Kasahara, H. and B. Li (2020, sep). Grain exports and the causes of China's Great Famine, 1959–1961: County-level evidence. Journal of Development Economics 146, 102513.
- Kreier, F. (2022). How the great depression shaped people's dna. Nature.
- Langley-Evans, S. C. and D. V. Sculley (2006, jun). The association between birthweight and longevity in the rat is complex and modulated by maternal protein intake during fetal life. *FEBS Letters* 580(17), 4150–4153.
- Li, W. and D. T. Yang (2005, aug). The Great Leap Forward: Anatomy of a Central Planning Disaster. Journal of Political Economy 113(4), 840–877.
- Li, Y., Y. He, L. Qi, V. W. Jaddoe, E. J. Feskens, X. Yang, G. Ma, and F. B. Hu (2010, jul). Exposure to the Chinese Famine in Early Life and the Risk of Hyperglycemia and Type 2 Diabetes in Adulthood. *Diabetes* 59(10), 2400–2406.
- Lin, C. C. H., C.-H. Su, P.-H. Kuo, C. K. Hsiao, W.-T. Soong, and W. J. Chen (2007, sep). Genetic and Environmental Influences on Schizotypy among Adolescents in Taiwan: A Multivariate Twin/sibling Analysis. *Behavior Genetics* 37(2), 334–344.
- Lin, J. Y. and D. T. Yang (2000, jan). Food Availability, Entitlements and the Chinese Famine of 1959–61. The Economic Journal 110(460), 136–158.
- Lindeboom, M., F. Portrait, and G. J. van den Berg (2010, sep). Long-run effects on longevity of a nutritional shock early in life: The Dutch Potato famine of 1846–1847. *Journal of Health Economics* 29(5), 617–629.

- Luo, Z., R. Mu, and X. Zhang (2006). The Long Term Health Impact of the Chinese Great Famine (Preliminary).
- Meng, X. and N. Qian (2009, apr). The Long Term Consequences of Famine on Survivors: Evidence from a Unique Natural Experiment using China's Great Famine. Technical report.
- Meng, X., N. Qian, and P. Yared (2015, apr). The Institutional Causes of China's Great Famine, 1959–1961. The Review of Economic Studies 82(4), 1568–1611.
- Mu, R. and X. Zhang (2011, jan). Why does the Great Chinese Famine affect the male and female survivors differently? Mortality selection versus son preference. *Economics & amp Human Biology* 9(1), 92–105.
- Ozanne, S. E. and C. N. Hales (2004, jan). Catch-up growth and obesity in male mice. Nature 427(6973), 411–412.
- Sayer, A. A., R. Dunn, S. Langley-Evans, and C. Cooper (2001). Prenatal Exposure to a Maternal Low Protein Diet Shortens Life Span in Rats. *Gerontology* 47(1), 9–14.
- Shi, X. (2011). Famine, fertility, and fortune in china. China Economic Review 22(2), 244–259.
- Shi, Z., C. Zhang, M. Zhou, S. Zhen, and A. W. Taylor (2013, oct). Exposure to the Chinese famine in early life and the risk of anaemia in adulthood. *BMC Public Health* 13(1).
- St Clair, D., M. Xu, P. Wang, Y. Yu, Y. Fang, F. Zhang, X. Zheng, N. Gu, G. Feng, P. Sham, and L. He (2005, 08). Rates of Adult Schizophrenia Following Prenatal Exposure to the Chinese Famine of 1959-1961. JAMA 294(5), 557–562.
- van Abeelen, A. F., M. V. Veenendaal, R. C. Painter, S. R. de Rooij, M. G. Dijkgraaf, P. M. Bossuyt, S. G. Elias, D. E. Grobbee, C. S. Uiterwaal, and T. J. Roseboom (2012, dec). Survival effects of prenatal famine exposure. *The American Journal of Clinical Nutrition* 95(1), 179–183.
- Van den Berg, G. J., G. Doblhammer-Reiter, and K. Christensen (2011). Being born under adverse economic conditions leads to a higher cardiovascular mortality rate later in life: Evidence based on individuals born at different stages of the business cycle. *Demography* 48(2), 507–530.
- Van den Berg, G. J., M. Lindeboom, and F. Portrait (2006). Economic conditions early in life and individual mortality. *American Economic Review 96*(1), 290–302.
- Wang, P.-X., J.-J. Wang, Y.-X. Lei, L. Xiao, and Z.-C. Luo (2012, nov). Impact of Fetal and Infant Exposure to the Chinese Great Famine on the Risk of Hypertension in Adulthood. *PLoS ONE* 7(11), e49720.

- Wang, S. and W. Zhou (2017, feb). The Unintended Long-Term Consequences of Mao's Mass Send-Down Movement: Marriage, Social Network, and Happiness. World Development 90, 344–359.
- Xu, M.-Q., W.-S. Sun, B.-X. Liu, G.-Y. Feng, L. Yu, L. Yang, G. He, P. Sham, E. Susser,
 D. S. Clair, and L. He (2009, jan). Prenatal Malnutrition and Adult Schizophrenia: Further
 Evidence From the 1959-1961 Chinese Famine. Schizophrenia Bulletin 35(3), 568–576.
- Yeung, G. Y., G. J. Van den Berg, M. Lindeboom, and F. R. Portrait (2014). The impact of early-life economic conditionson cause-specific mortality during adulthood. *Journal of Population Economics* 27, 895–919.
- Zhou, W. (2014, nov). Brothers, household financial markets and savings rate in China. *Journal* of Development Economics 111, 34–47.

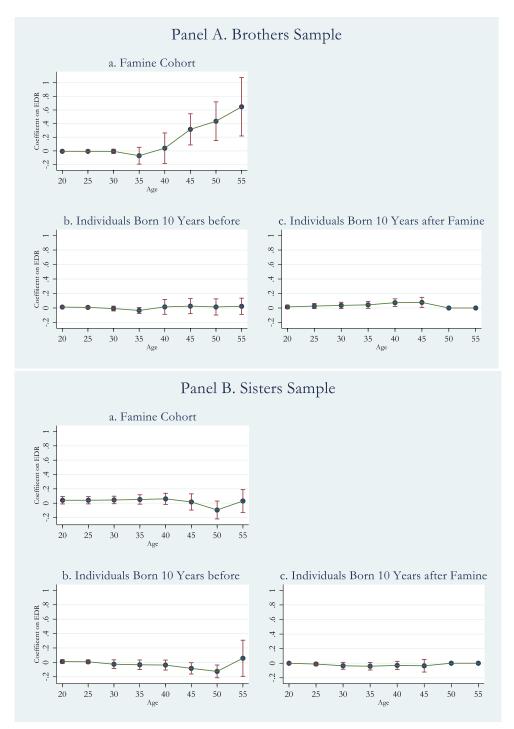


Figure 1: Effects of prenatal exposure to the Chinese famine on mortality by age

Notes: Each point and the corresponding 95% confidence interval are based on a separate regression of the following equation: $Deceased^a_{ijyc} = \lambda EDR_{yc} + \theta_j + \delta_y + e_{ijyc}$, where $Deceased^a_{ijyc}$ equals 1 if an individual is dead by age a, and 0 otherwise. The variable is conditional on the individual was born at least a-5 years ago. Each regression estimates the effect of famine on mortality at age a. Panel (a) use the excess death rate (EDR_{yc}) in the year an individual was born; figures (b) and (c) are falsification tests that apply the famine-era EDR to individuals born 10 years before the famine and individuals born 10 years after the famine, respectively. Since individuals born 10 years after the famine have not yet reached 50 years at the time of the survey, the estimate for these individuals uses the null hypothesis value of zero.

		Famine	Non-famine	
	All	cohort	cohort	Difference
	(1)	(2)	(3)	(4)
EDR (in birth year)	0.004	0.083		
	[0.039]	[0.159]		
Years of education	5.958	6.861	5.914	0.947***
	[4.392]	[4.523]	[4.381]	(0.242)
Deceased	0.047	0.041	0.048	-0.007
(=1 if died by 2010)	[0.213]	[0.198]	[0.213]	(0.012)
Birth year	1961.85	1959.97	1961.94	-1.97***
·	[11.109]	[0.783]	[11.368]	(0.612)
Family size	3.945	4.270	3.929	0.340***
v	[1.627]	[1.693]	[1.622]	(0.09)
Observations	7392	345	7047	7392

Table 1: Summary Statistics

Notes: EDR (excess death rate) is at county and birth year level. It is defined as subtracting the average death rate 5 years before the famine from the death rate in a famine year. The EDR in a non famine year is zero by definition. Deceased equals one if an individual died by 2010, and zero otherwise. The first three columns report the mean and standard deviation. The last column reports the differences in mean between the famine cohort and the non famine cohort. Standard deviations are in square brackets. Standard errors are in parentheses.

	All	All	Male	Female					
	(1)	(2)	(3)	(4)					
Panel A. Dependent variable: Years of education									
EDR	0.208	-1.958	-3.940*	-3.630**					
	(1.322)	(1.241)	(2.324)	(1.562)					
Observations	7,390	7,390	3,817	$3,\!573$					
R-squared	0.154	0.138	0.091	0.161					
Panel B. Dependent variable: Deceased									
EDR	0.003	0.109	0.490^{***}	-0.079					
	(0.068)	(0.079)	(0.153)	(0.070)					
Observations	7,390	7,390	3,817	$3,\!573$					
R-squared	0.094	0.068	0.124	0.124					
Control for both pane	els								
County FE	Yes								
Sibling FE		Yes	Yes	Yes					
Birth year FE	Yes	Yes	Yes	Yes					

Table 2: The long-term effects of prenatal exposure to the Chinese famine

Notes: EDR (excess death rate) is at county and birth year level. It is defined as subtracting the average death rate 5 years before the famine from the death rate in a famine year. The EDR in a non famine year is zero by definition. Deceased equals one if an individual died by 2010, and zero otherwise. Robust standard errors clustered at the county level are in parentheses.*** p < 0.01, ** p < 0.05, * p < 0.1

	Years of	f education	Deceased		
	Male	Female	Male	Female	
	(1)	(2)	(3)	(4)	
	Panel A.	Control for birth	order FE		
EDR	-3.748	-3.390**	0.498^{***}	-0.079	
	(2.412)	(1.625)	(0.153)	(0.073)	
Observations	$3,\!817$	$3,\!573$	$3,\!817$	$3,\!573$	
R-squared	0.095	0.170	0.131	0.131	
	Panel B. Pla	cebo test: Assign	famine-era		
E	EDR to individua	als born 10 years	after the famine		
EDR	-0.100	0.261	0.077	-0.011	
	(0.744)	(0.929)	(0.047)	(0.042)	
Observations	6,968	$6,\!802$	6,969	6,804	
R-squared	0.090	0.163	0.121	0.125	
	Panel C. Pla	cebo test: Assign	famine-era		
E	DR to individua	ls born 10 years l	before the famine		
EDR	-0.891	-1.344	-0.047	0.128	
	(1.441)	(0.989)	(0.087)	(0.138)	
Observations	7,010	6,848	7,011	$6,\!850$	
R-squared	0.092	0.161	0.123	0.128	
	Panel D. Us	e birth-month-we	ighted EDR		
	$and \ con$	trol for birth mo	nth FE		
Weighted EDR	-4.464*	-5.797***	0.489^{***}	-0.073	
	(2.359)	(1.772)	(0.153)	(0.078)	
Observations	3,808	3,564	3,808	3,564	
R-squared	0.098	0.165	0.137	0.134	
		Only include fami			
	born in tl	he last year of the	•		
Weighted EDR	0.420	-5.031*	0.501^{*}	-0.101	
	(4.442)	(2.857)	(0.261)	(0.148)	
Observations	$3,\!693$	$3,\!434$	$3,\!693$	$3,\!434$	
R-squared	0.099	0.167	0.141	0.136	

 Table 3: Robustness Checks

Notes: All panels control for sibling fixed effects and birth year fixed effects. EDR (excess death rate) is at county and birth year level. It is defined as subtracting the average death rate 5 years before the famine from the death rate in a famine year. The EDR in a non famine year is zero by definition. Deceased equals one if an individual died by 2010, and zero otherwise. Robust standard errors clustered at the county level are in parentheses.*** p < 0.01, ** p < 0.05, * p < 0.1

	Health conditions in adulthood			Health	Physical	Availability of health care services				
	Confined Hospitali- Job		conditions in	\mathbf{injury}^{e}	Had a source of care/doctor/clinic at age^{f}			Vaccination		
	to bed^a	zed^b	$leave^{c}$	${f childhood}^d$		0-15	16-25	26-40	41-55	before age 15
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Male										
EDR	0.271^{**}	0.260^{*}	0.187	0.375	0.145	-0.040	-0.030	-0.026	0.005	-0.017
	(0.132)	(0.141)	(0.125)	(0.323)	(0.194)	(0.069)	(0.070)	(0.052)	(0.038)	(0.076)
Obs.	7,466	$7,\!470$	7,466	$7,\!428$	7,467	$7,\!446$	$7,\!446$	$7,\!446$	$7,\!446$	$7,\!379$
R-sq.	0.014	0.017	0.009	0.010	0.007	0.030	0.032	0.024	0.018	0.063
Fema	ele									
EDR	0.057	-0.039	-0.057	-0.170	-0.053*	-0.056	-0.047	-0.019	-0.016	-0.167
	(0.112)	(0.066)	(0.125)	(0.207)	(0.030)	(0.038)	(0.036)	(0.028)	(0.023)	(0.131)
Obs.	8,335	8,339	8,333	8,274	8,335	8,246	8,243	8,243	8,243	8,210
R-sq.	0.022	0.015	0.010	0.007	0.014	0.036	0.033	0.023	0.021	0.056

Table 4: Mechanism

Notes: CHARLS 2014 Life History Data are used. All outcome variables are dummy variables, equal to 1 if the respondent answered yes and 0 otherwise, except for the outcome variable used in column 4, in which the answer is expressed on a 5-point scale ranging from 1 (healthy) to 5 (unhealthy). Summary statistics of all outcomes variables are reported in Appendix Table 2. County fixed effects and birth year fixed effects are controlled for in all estimations. Robust standard errors clustered at the county level are in parentheses. (a) Confined to bed due to health reasons for 1 month or more (yes=1, no=0). (b) Hospitalized for 1 month or more (yes=1, no=0). (c) Job leave due to health reasons for 1 month or more (yes=1, no=0). (d) Health status before age 15: healthy=1, unhealthy=5. (e) Had physical injury that led to disability (yes=1, no=0). (f) Had a usual source of care/doctor/clinic at the following ages (yes=1, no=0). p < 0.01, ** p < 0.05, * p < 0.1