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Long-Term Effects of Famine on Chronic Diseases: Evidence from China's Great Leap Forward Famine

Running Title: Famine and Chronic Diseases

Xuefeng Hu[a], Gordon G. Liu[b], and Maoyong Fan[c]*

[a] Xuefeng Hu, Department of Biology, University of Ottawa.
[b] Gordon Liu, National School of Development, Beijing University.
[c] Maoyong Fan, Department of Economics, Ball State University.

ABSTRACT

We evaluate the long-term effects of famine on chronic diseases using China's Great Leap Forward Famine as a natural experiment. Using a unique health survey, we explore the heterogeneity of famine intensity across regions and find strong evidence supporting both the adverse effect and the selection effect. The two offsetting effects co-exist and their magnitudes vary in different age cohorts at the onset of famine. The selection effect is dominant among the prenatal/infant famine-exposed cohort, while the adverse effect appears dominant among the childhood/puberty famine-exposed cohort. The net famine effects are more salient in rural residents and non-migrants subsamples. Gender differences are also found, and are sensitive to smoking and drinking behaviors. Our conclusion is robust to various specifications.

Keywords: Great Leap Forward Famine, Chronic Disease, Adverse Effect, Selection Effect, Gender Difference

JEL Classifications: C21; I18; J13

*** Corresponding to:**
Maoyong Fan
Department of Economics
Miller College of Business
Whitinger Business Building, room 201
Ball State University
Muncie, Indiana 47306

Email: mfan@bsu.edu
Phone: 765-285-5741
Fax: 765-285-4343

INTRODUCTION

A fetus's early adaptation in response to maternal malnutrition may have long-term health impacts (Barker 1997). Identifying the links between malnutrition and health is important for designing nutrition intervention programs due to the prevalence of malnutrition among pregnant women and children in developing countries (UNICEF et al. 2014). The Great Leap Forward Famine (GLFF) in China (1959 to 1961) provides an opportunity for researchers to study the long-term health effects of famine. The GLFF had severe consequences on mortality, with an estimated 15 to 30 million premature deaths being attributed to it (Ashton et al. 1984; Peng 1987; Song 2010). The size of the population affected and its duration make the GLFF the most severe famine ever recorded in human history. Birth rates also dropped dramatically with an approximate one-third reduction in the cohort size of children born during the famine (An et al. 2001; Coale 1981; Yao 1999).

For the survivors of the GLFF, researchers have highlighted two major health effects, namely the adverse effect and the selection effect (Gørgens et al. 2012; Meng & Qian 2009). The adverse effect, also known as Barker's hypothesis or thrifty phenotype hypothesis (Barker 1998; Wells 2007), refers to a higher probability of developing chronic diseases among famine survivors. The hypothesis proposes that the development process of the fetus can be modified during insufficient nutrition conditions (e.g., a pregnant woman with constrained energy and nutrient intake) to better prepare for survival in the face of limited resources. However, this modification may cause a failure to adapt to normal or prosperous nutrition conditions, thereby resulting in increased risks of chronic diseases. The chronic diseases include but are not limited to type 2 diabetes, obesity, hypertension, and coronary heart disease (Barker 1995; Hales & Barker 1992; Hales & Barker 2001; Ravelli et al. 1999).

The selection effect proposes that famine survivors, on average, may actually be

healthier than those who are not affected by the famine. There are two possible mechanisms for the selection effect. First, famine survivors are supposed to be genetically healthier because only parents who are strong enough are able to give birth during difficult years, and thus, the newborns inherit their parents' strong genes. Furthermore, among the newborns, the unhealthy ones are less likely to survive a famine (Akachi & Canning 2010; Alderman et al. 2011; Fawzi et al. 1997). Second, the quantity-quality hypothesis proposes that the reduced competition for family and social resources due to the reduction of the birth cohort size may produce better health among famine survivors (Easterlin 1980; Schultz 2005). Consequently, the observed famine effect is the net effect after the adverse effect and the selection effect have offset each other. Figure 1 shows part of the population pyramid of China based on China's Census 1982. As population growth was severely disrupted during the GLFF period, the cohort size decreased dramatically during the 1959 to 1961 period and did not return to a normal level until 1963, thus indicating mortality selection during the GLFF.

This study aims to demonstrate the co-existence of the adverse and selection effects by comparing the net famine effects on two different age cohorts at the onset of famine, specifically, the prenatal/infant famine-exposed cohort and the childhood/puberty famine-exposed cohort. As both cohorts are at crucial stages of body development, having experienced restricted nutrition intake may permanently affect their health. The major difference between the two cohorts is that the prenatal/infant cohort experienced a more severe mortality selection. Thus, our hypothesis is that if the selection effect exists, it should be strongest in the prenatal/infant famine-exposed cohort and the net famine effect in this cohort should differ from other famine-exposed cohorts. We used a large-scale health survey conducted in 2008 for our empirical analysis. The famine effect throughout the paper refers to the net effect without further

specification.

This study makes three contributions to the literature. First, this study shows the co-existence of the adverse and selection effects of the GLFF. The majority of previous GLFF studies report the net effect of famine as the sum of the adverse effect and selection effect. However, the estimation and discussion about the relative sizes of the adverse effect and the selection effect are rare. Moreover, the discussion about selection is limited to mortality and height. This study fills the gap in the literature by providing empirical evidence of both the adverse effect and the selection effect of the GLFF. We find that the adverse effect dominates the selection effect in the childhood/puberty famine-exposed cohort, which is consistent with our hypothesis, and the selection effect outweighs the adverse effect in the prenatal/infancy famine-exposed cohort. Second, we eliminate the bias caused by migration using a non-migrant subsample. To our best knowledge, this is the first GLFF study to rule out migration as a confounding factor. We find that the estimates in the non-migrant subsamples are greater in magnitude and more significant for both the prenatal/infant famine-exposed and the childhood/puberty famine-exposed cohorts. The difference is particularly large when the selection effect dominates in the prenatal/infant famine-exposed cohort. Third, as rural and urban residents experience different levels of famine exposure, we compare regions with the highest EDR to regions with the lowest EDR to estimate the famine effect separately for rural and urban residents. This approach complements two commonly used empirical strategies, specifically, the comparison of rural residents with urban residents and the comparison of residents in different regions. Adverse effects are found among both rural and urban residents. However, a net selection effect, which is the result of the selection effect and the adverse effect offsetting each other, is only found among rural residents who suffered more during the GLFF than urban residents. We also find

a net selection effect among the prenatal/infant famine-exposed cohort. In contrast, however, there are many GLFF studies that fail to find a net selection effect. We conclude that the net selection effect exists and could be identified if researchers compare two cohorts with large differences in the level of famine intensity (e.g., Sichuan vs. Shaanxi). Comparing areas with similar levels of famine intensity may be why the selection effect has not been found in many previous GLFF studies.

This paper is arranged as follows. Section 2 describes the GLFF and its geographic heterogeneities of famine intensity and also discusses the links between famine exposure and chronic diseases in later lives. Section 3 describes the data and empirical strategy used in this paper. Section 4 presents the results and examines the heterogeneous famine effects, and section 5 discusses the findings and concludes the paper.

1. BACKGROUND AND LITERATURE REVIEW

2.1 The Great Leap Forward Famine

The Great Leap Forward Campaign, which began in 1958, attempted to exploit China's vast population to transform the country from an agrarian economy to a modern communist society through rapid industrialization and collectivization (Bachman 1991). During the campaign, agricultural production dropped sharply[1] as labor was diverted from agriculture to industry and people's communes replaced private farming (Li 2005; Lin 1990). At the same time, grain procurement from rural areas increased because the political climate encouraged provincial leaders to overstate grain production.[2] Over the

[1] China's grain output was increasing steadily before 1958 and reached a peak of 200 million tons in 1958. However, grain output dropped sharply by 15 percent in 1959, and during the following two years, the output continued to decline to approximately 70 percent of the 1958 record level (State Statistical Bureau 1990).

[2] Despite widespread starvation, China was a net exporter of grain throughout 1960 (Lin & Yang 1998; Yao 1999) .

next three years (1959 to 1961), famine affected all regions of China. While weather conditions may have contributed to the GLFF, the radical economic policies of the Great Leap Forward Campaign (1958) were primarily to blame (Chang & Wen 1998). Though the GLFF ended in 1962, the cause of its conclusion is still under debate. Evidence indicates that the abolition of communal kitchens, the importation of grain, the reduction in the urban appropriation of grain, and the retreat from collectivization, i.e., land was returned to farmers, all contributed to mitigating the famine (Johnson 1998; Yang & Su 1998).

Even though the GLFF was widespread in China, famine intensity varied significantly across provinces[3] (Chang & Wen 1997; Peng 1987). The major reason for these differences in intensity was the disparities in the local enforcement of the grain procurement quota established by the central government. Provinces such as Sichuan that fully complied with quota orders from the central government were the most severely affected, while provinces such as Guangdong and Jilin were relatively spared because their local governments successfully reduced the quota (Lin 2000). Table I presents the death rates from 1954 to 1962 as well as the average excess death rates (EDR) during the famine period for each province and for the nation as a whole (State Statistical Bureau 1990). The EDR was defined as the difference between the average death rate during the famine years (1959 to 1961) and the EDR prior to the famine (1954 to 1958). It was clear that the EDR varied dramatically across provinces with Sichuan being one of the most severely affected provinces and the other three provinces

[3] Besides the inter-province heterogeneity of famine intensity, there is also a noticeable difference in famine intensity between urban and rural areas. In 1958, the household register system, the Hukou, was officially instated by the Chinese government to prohibit free migration between different regions, especially from rural areas to cities (Wang 2005). The Chinese population is broadly divided into rural residents and urban residents. A grain rationing system is used to control unauthorized migration from rural areas to cities during this same period. Rural people have to deliver heavy quotas to procurement agencies and could only keep the residual grain after fulfilling obligations. People not registered as urban residents could not qualify for grain rations (Cheng & Selden 1994).

in our sample being among the least affected areas.

2.2 The long-term health effects of the GLFF

Long-term effects of the GLFF have been discussed in both health economics and public health literatures. Some GLFF studies have discussed famine exposure and its long-term impact on mortality, sex ratio, education, marriage, as well as labor market outcomes (Almondy et al. 2007; Shi 2011; Song 2010; Song 2012). Herein, we focus on the health effects with respect to the GLFF. Although the previous studies used different outcomes and were based on different data sources, there are some notable similarities among them.

For example, difference-in-differences was the most commonly used strategy in the literature. The difference between survivors' age at the onset of the GLFF provided a natural dimension of difference. The comparison between infants born during and after the famine was a prevailing strategy found in many of the studies, whereas several studies also included older children cohorts. Some of the GLFF studies used the CHNS (China Nutrition and Health Study) 2002[4] data and the rural-urban difference to define other dimensions of difference with respect to famine exposure (State Statistical Bureau 1991; Gørgens et al. 2012; Chen & Zhou 2007). Still other studies used the CNHS 2002 and regional differences to define famine intensity (Li et al. 2010; Li et al. 2011a; Li et al. 2011b).

The adverse effect was observed in many of the GLFF studies. For example, famine exposure was associated with shorter height, increased risk of hypertension, metabolic syndrome, and obesity (Chen & Zhou 2007; Li et al. 2010; Li, Jaddoe, Qi,

[4] CNHS 2002 is a stratified, multistage probability cluster-sampling designed nationally representative cross-sectional study on nutrition and chronic diseases conducted by the China CDC. See details of the dataset in the following publication: http://www.ncbi.nlm.nih.gov/pubmed/16334996.

He, Wang, et al. 2011; Li, Jaddoe, Qi, He, Lai, et al. 2011; St Clair et al. 2005; Zheng et al. 2012; Cheng Huang et al. 2010). The selection effect was also proposed and discussed in some of the GLFF studies (Meng & Qian 2008; Gørgens et al. 2012; C Huang et al. 2010; Shi 2011; Meng & Qian 2009). For example, gender difference was observed in some studies where females were found to more likely be obese and have certain chronic conditions (Mu & Zhang 2011; Zheng et al. 2012; Luo et al. 2006; Z. Yang et al. 2008; Chen et al. 2014).

2. DATA AND EMPIRICAL STRATEGY

3.1 The Urban and Rural Health Survey

We use the Urban and Rural Health Survey (URHS) 2008 for our empirical analysis.[5] The URHS 2008 is a large-scale cross-section individual survey administered by the Guanghua School of Management and the Center for Health Economics Research of Peking University. The overall objective of the URHS 2008 is to provide information regarding the current health status of urban and rural residents in China. It also examines the effects of health determinants, such as environmental factors, living conditions, lifestyle, and access to health facilities, on population health outcomes. The URHS 2008 collects rich socioeconomic and health information from a random sample of 12,700 individuals from four provinces, namely Beijing, Shaanxi, Sichuan and Yunnan).[6] Data gathered include demographic background (race, education, occupation, income etc.), health status, perceived stress, two-week morbidity rate, annual hospitalization rate, tobacco and alcohol consumption, health care utilization

[5] We do not use the China Health and Nutrition Survey because it has very limited information about respondents' chronic conditions and it does not include the most severely affected regions.

[6] The selection of the random sample for the URHS is a two-stage stratified cluster approach. First, PSUs are assigned to strata according to region, urban/rural and other socioeconomic variables. Then, systematic sampling is used to select a sample of PSUs independently from each stratum. This was followed by a systematic random sample of households taken within each PSU. The field survey was conducted between 2008.03 and 2008.08, covering approximately 240 primary sampling units and 4,400 households.

and access, knowledge, attitude, and practice of self-medication. Survey participants were specifically asked whether they had doctor-confirmed chronic conditions.[7] We do not use the CHNS or the CNHS 2002 data because neither dataset provides participants' immigration history, whereas the URHS 2008 does.

Our sample includes respondents born during the following periods: 1939 to 1942, 1943 to 1958, 1959 to 1961 and 1963 to 1965. As infancy is a crucial period for body development and long-term health, a fetus/infant exposed to an unfavorable nutritional environment is at increased risk of developing various chronic diseases (Barker 1997; Robinson 2001). The 1959 to 1961 cohort, i.e., those born during the famine, is the prenatal/infant famine-exposed cohort, and the 1963 to 1965 cohort, i.e., those born after the famine, serves as the comparison cohort. We exclude the 1962 cohort because part of this cohort experienced in-womb exposure to the GLFF. Because childhood and puberty are also phases of rapid physical development (Gordon & Laufer 2005; Marshall & Tanner 1968), exposure to restricted nutrition due to famine during these periods also affects health adversely and may lead to chronic conditions (Heald 1975; Meredith & Dwyer 1991). We assume all children under the age of 16 before the famine were affected, and thus, we use the 1939 to 1942 cohort, which had reached adulthood in 1959, as the comparison cohort. In the sensitivity analysis, we separate the children

[7] The following explanations were given to the interviewees when asked about their chronic diseases. "Now I would like to ask about some chronic diseases that you may have. We are interested in long-term diseases that have lasted or are expected to last for at least 6 months. Also, we are only interested in the diseases that have been diagnosed by a health professional (e.g., doctors in a hospital). Please show me the diagnosis." The interviewer then asks, "Have you ever been told by a health professional that you have ***?" The interviewer will read aloud the 14 most prevalent chronic diseases according to the disease spectrum in China, including cardiovascular disease, stroke, any cancer, hypertension, hyperlipidemia, diabetes, COPD, asthma, arthritis, osteoporosis, ulcers, Parkinson's, and hip fracture. Most of the diseases are metabolic-related conditions that have already been linked to famine exposure, with the exception of cancer, arthritis, and ulcers. At the end of the interview, there was an open-ended question that asked the interviewee, "What other chronic diseases do you have?" Our dependent variable of whether or not a respondent had a chronic diseases was based only on the 14 doctor-confirmed chronic diseases.

famine-exposed cohort into 2-year and 1-year age groups to test whether the GLFF had heterogeneous effects on different age groups.

Table II presents the summary statistics. A total of 1,205 participants are available for the prenatal/infant exposure analysis and 3,160 for the childhood/puberty exposure analysis. The prevalence of chronic diseases declines as participants age. Demographic characteristics and social economics status, health behaviors, and environmental variables are comparable across the famine-exposed and control cohorts. Numbers are presented separately for men and women when substantial differences exist. Women had a higher prevalence of chronic diseases, and most women neither smoked nor drank, which is consistent with the social norm. Women were also less likely to be non-migrants, which is in accordance with the Chinese culture as women typically move to live with their husbands after marriage.

3.2 Empirical Strategy

As discussed in section 2, heterogeneous agricultural policies across provinces lead to different levels of famine intensity as measured by the EDR, which enables us to quantify the famine effect. We estimate the following equation:

$$Prob(CD)_i = \alpha + \delta EDR_i * FC_i + \beta_1 EDR_i + \beta_2 FC_i + X_i\gamma + \varepsilon_i \quad (1)$$

where $Prob(CD)_i$ is the probability of having chronic diseases for individual i, EDR_i is the EDR of the province where individual i lived during the famine, and FC_i is a dummy variable taking the value of one if individual i belongs to the famine-exposed cohort (born between 1959 and 1961 for prenatal/infant famine exposure and 1945 and 1947 for childhood/puberty famine exposure) and zero otherwise. The coefficient of the interaction term, δ, represents the impact of the GLFF and measures the changes in the prevalence of chronic diseases as the EDR changes for the famine-exposed cohort

relative to the non-exposed cohort. For presentational simplicity, we use the linear probability model.[8]

The vector, X_i, includes factors that may influence famine intensity (dummies for rural residents and non-migrants); individual characteristics and social economic status (age, ethnic group, marriage status, education, and log income per capita); health behaviors (smoking and drinking status); and self-perceived environmental conditions averaged at the community level. The study also controls for province-fixed effects. We define two dummy variables, non-smoker and non-drinker, to indicate individuals who never smoked and never or only occasionally drank alcoholic beverages. As environmental conditions also play an important role in the development of chronic diseases, self-perceived environmental conditions are a set of scores from all respondents in the survey (total=12,691) averaged at the community (PSU) level. This provides an instrument to measure the environmental conditions associated with health in a relatively exogenous way. Standard errors in the estimation are clustered at the community level to account for within-community serial correlations.

4. RESULTS

4.1 Effects of the GLFF on Chronic Diseases

Table III presents the main results for both the prenatal/infant famine-exposed cohort (panel A) and the childhood/puberty famine-exposed cohort (panel B). Column 1 is based on a specification that adjusts only for gender, while the remaining columns correspond to specifications that gradually add more controls. Column 2 adds factors that may influence famine intensity, i.e., dummies for rural residents and non-migrants.

[8] The corresponding logit estimates are exceptionally close to those of the corresponding linear probability model.

Column 3 adds individual characteristics and social economic status, i.e., age, ethnic background, marriage status, education, and log income per capita. These variables, to some extent, control for the mechanisms through which the GLFF can affect the health of survivors. For example, our results indicate that married people, people with higher levels of education, and people with higher incomes are less likely to have chronic diseases (Appendix Tables A1 and A2). Column 4 adds health behaviors, i.e., smoking and drinking status. Column 5 adds self-perceived environmental conditions averaged at community level, i.e., air, water, noise pollution, and greening rate.

The estimates for prenatal/infant famine exposure (panel A) reveal a negative famine effect on the probability of having chronic diseases in later life for GLFF survivors, and as the estimates are stable across various specifications, it is suggested that the famine effect is less likely to be affected by omitted variables. The estimates of the most restrictive specification (column 5) is -3.98 per mil, indicating that if the EDR increases by one per mil, the probability that people born between 1959 and 1961 will have a chronic disease is 0.4 percentage points lower than it is for those born after the famine. For example, given that the EDR for Sichuan is 29.2 per mil, the probability that people born during the famine in Sichuan will have a chronic disease is approximately 12 percentage points lower relative to people born after the famine. In terms of percentage, this number translates to an approximate 50 percent reduction in the prevalence of chronic diseases for prenatal/infant famine survivors in Sichuan. However, we should not interpret the negative estimates as famine's protective effect on survivors' long-term health because the reduced probability of having a chronic disease is at the cost of the excess death rate for famine survivors' genetically weaker peers. The estimates represent the net effect after the selection effect and adverse effect offset each other. Thus, the negative net effects suggest that the selection effect exists

and dominates the adverse effect in the prenatal/infant famine-exposed cohort.

Panel B presents the estimates for the childhood/puberty famine-exposed cohort. In contrast to panel A, all of the estimates are positive, thereby indicating that exposure to the GLFF during childhood/puberty increases the probability of having a chronic disease later in life. The estimate of the interaction term in the most restrictive specification (column 5) is 6.42 per mil, suggesting that exposure to the GLFF during childhood/puberty increases the probability of having a chronic disease in later life by 0.64 percentage points if the EDR increases by one unit relative to people who had attained adulthood prior to the onset of the GLFF. Using Sichuan as an example, the probability of having a chronic disease for people who experienced the famine during childhood/puberty is approximately 18 percentage points higher relative to people who had reached adulthood before the famine, when considering that the EDR for Sichuan is 29.2 per mil. In relative terms, this represents an approximate 40 percent increase in the probability of having a chronic disease. Therefore, it is further posited that positive net effects suggest that the adverse effect dominates the selection effect in the childhood/puberty exposure cohort.

4.2 Heterogeneous Famine Effects on Chronic Diseases

Whereas China has witnessed the world's largest migration since the 1980s, current local residents may well be migrants, and thus, they may not have experienced the GLFF locally. Therefore, we must consider that migrants may potentially bias our estimation in two ways. First, migration may conceal or exaggerate individual exposure to famine because migrants move between areas with different levels of famine intensity. Second, the health conditions of migrants may be substantially different from the health conditions of local residents due to factors other than the GLFF. Accordingly, the URHS 2008 asks respondents to indicate how many years they have been living in the current

region. Using this information and the ages of the individuals, we are able to separate non-migrants from migrants. With respect to childhood/puberty famine exposure and comparison cohorts, we define non-migrants as individuals who have been living locally since 1958. Regarding the prenatal/infant famine-exposed cohort and comparison cohort, we define non-migrants as individuals who have lived locally since 1978, given that rural-urban and inter-province migrations were extremely rare prior to 1978.[9]

Column 6 of Table III provides the estimates for the famine effect among non-migrants. Panel A indicates that the famine effect for the prenatal/infant famine-exposed cohort increases to -10.04 per mil, which is more than double the estimate when using the full sample (-3.98 per mil in column 5). Furthermore, the corresponding estimate for the childhood/puberty famine-exposed cohort in panel B also increases from 6.42 per mil to 7.48 per mil. Both results suggest that involving migrants in the sample is likely to bias the true effect of the famine, and they also emphasize the importance of identifying a relatively clean sample to obtain proper estimates of the famine effect.

We further estimate the famine effect on urban and rural subsamples separately as they faced drastically different food supply situations during the GLFF. Urban residents, for instance, were subject to certain food quotas throughout the entire GLFF period, while rural residents were not. Thus, rural residents suffered more from the GLFF relative to urban residents in the same region. Column 7 of panel A indicates that rural residents exposed to the GLFF during prenatal/infancy exhibited a lower probability of developing a chronic disease relative to those not exposed to the GLFF (-6.65 per mil); however, we do not find similar statistically significant results for urban residents

[9] We adopt a less strict definition of non-migrants for prenatal/infant exposure and their comparison cohorts to reduce recall bias as we are not able to accurately trace the status of their parents during the GLFF and where they were born.

(column 8 of panel A). Columns 7 and 8 of panel B reveal that both rural and urban residents exposed to the GLFF during childhood/puberty had a higher probability of having a chronic disease relative to those who were not exposed. We observe a rural-urban difference in the prenatal/infancy famine-exposed cohort but not in the childhood/puberty famine-exposed cohort. This is further investigated in a sensitivity analysis.

4.3 Gender Difference

Previous GLFF studies have revealed inconsistent findings with respect to gender difference. Some GLFF studies either do not report gender-specific famine effect estimates or do not find any gender difference (Chen & Zhou 2007; Gørgens et al. 2012; Li et al. 2010; Li et al. 2011a; Li et al. 2011b; Zheng et al. 2012), whereas other studies find that females are more likely to be obese and have metabolic-related chronic conditions (Chen et al. 2014; Luo et al. 2006; Mu & Zhang 2011; Wang et al. 2010; Z Yang et al. 2008; Zheng et al. 2012).

We estimate the gender-specific famine effect and examine the gender difference by re-estimating equation (1) using male/female subsamples. Table IV presents the gender-specific famine effect for the prenatal/infant (panel A) and the childhood/puberty (panel B) famine-exposed cohorts, respectively. Column 1 (panel A) indicates that females exposed to the GLFF during the prenatal/infant period exhibited a lower probability of having a chronic disease (one per mil increase in EDR leads to 0.63 percentage point decrease). In contrast, we do not find an effect for males (column 2 of panel A). Panel B shows that both males and females exposed to the GLFF during childhood/puberty exhibited a higher probability of having chronic diseases, though the estimate for males is one-third less than that for females and significant at the 10 percent level.

Certain important health risk factors that are not well-balanced among males and females may confound the gender-specific estimates. For example, men have much higher smoking and drinking rates than women in China (Yang et al. 1999), and smoking and drinking are among the most important factors known to increase the risk of many metabolic-related chronic conditions (WHO 2010). To rule out the confounding effects of smoking and drinking, we further estimate gender-specific famine effects using non-smoker and non-drinker subsamples. The results are presented in columns 3 to 6 of Table IV. As we expected for both the prenatal/infant and childhood/puberty famine-exposed cohorts, the effects of famine remain the same for females. These results are reasonable given that over 90 percent of the women in China are non-smokers and/or non-drinkers (Yang et al. 1999; Yang et al. 2012). With respect to males, excluding smokers or drinkers had a large impact on estimates as smoking and drinking affect people's health in the same direction as the adverse effect of famine. With respect to the male prenatal/infant famine-exposed cohort, the famine effect becomes statistically significant when we eliminate either smokers or drinkers. However, regarding the male childhood/puberty famine-exposed cohort, the estimates are insignificant when excluding either smokers or drinkers.

To summarize, the results suggest that males and females are affected differently by the GLFF. The difference is more prominent for the childhood/puberty famine-exposed cohort. However, due to the nature of cross-sectional data, we are not able to explore the mechanism of gender difference.

4.4 Falsification tests

We conduct multiple falsification tests using birth cohorts that are not affected by the GLFF. For the infant/prenatal famine-exposed cohort, we re-estimate equation (1)

using the cohort born between 1966 and 1968 as the treatment group. For the childhood/puberty famine-exposed cohort, we use the cohort born between 1935 and 1938 as the treatment group. Table V presents the results of the falsification tests. Panel A presents the results for the prenatal/infant famine-exposed cohort, and panel B displays the results for the childhood/puberty famine-exposed cohort. Columns 1 and 2 report estimates for the full sample and the non-migrant subsample, respectively; columns 3 and 4 present estimates for rural and urban residents, respectively; and columns 5 and 6 present the results for female and male participants, respectively. As none of these estimates are statistically significant, the results from all falsification tests strongly support that the selection and adverse effects found in this study are caused by exposure to the GLFF.

4.5 Robustness checks

We use self-reported doctor-confirmed chronic disease as our main health outcome, as it is a comprehensive health indicator at the individual level and the population level. One concern is that this constructed variable includes both health outcomes linked to famine exposure, such as hypertension and diabetes, and health outcomes lacking direct association with famine, such as cancer and arthritis. We conduct robustness checks for hypertension and diabetes as both are linked to famine exposure. The results for hypertension and diabetes are consistent with our main results (Appendix Tables A5 and A6). Moreover, the estimated effects are similar to the main analysis for the prenatal/infant famine-exposed cohort and greater than those in the main analysis for the childhood/puberty famine-exposed cohort. One plausible explanation is that the association between hypertension, diabetes, and famine exposure are stronger than the combined chronic diseases. It is also possible that the estimates for hypertension and

diabetes are not very stable due to smaller event rates. Similar to the main analysis, famine effects are greater among non-migrants, rural residents, and females.

5. CONCLUSION

The evaluation of the famine effect on health has been an important research topic in economics and public health. However, there are mixed and contradictory findings regarding two offsetting famine effects, namely the adverse effect and the selection effect. In this study, we examine whether famine has long-term health effects and whether its adverse effect and selection effect vary among different famine-exposed cohorts. We use the GLFF as a natural experiment and compare the worst affected region (Sichuan) with three mildly affected regions. We find strong evidence for both the adverse effect and the selection effect. The selection effect is dominant among the prenatal/infant famine-exposed cohort, while the adverse effect appears dominant among the childhood/puberty famine-exposed cohort. Moreover, prenatal/infant exposure to the GLFF leads to a reduced risk of having a chronic disease in later life, whereas childhood/puberty exposure to the GLFF results in an increased risk of having a chronic disease. We also examine the effect of famine on a non-migrant subsample, which thereby removes the confounding effect caused by migration, and find that famine effects are more salient in the non-migrant subsample than they are in the full sample. These findings are robust across different specifications.

This paper adds to the existing literature by discussing the relative magnitude of the adverse effect and the selection effect of the GLFF. The selection effect is not generally observed in previous GLFF studies, and the famine intensity variations in previous studies may not be large enough to detect the net selection effect. We use EDR

as a measurement of famine intensity and investigate the EDRs in previous GLFF studies. The EDR difference in studies exploring the rural-urban difference using the CHNS is approximately 3.4 per mil. The EDR difference in studies exploring regional differences using the CNHS 2002 data is approximately 10 per mil. In our sample, the EDR difference between Sichuan and the other three regions is 27.8 per mil. However, it is possible that the effect of the GLFF on health is non-linear; thus, studies that exploit different levels of famine intensity may reach different conclusions.

There are three caveats to this paper. First, our empirical results are based on cross-sectional data from four provinces. Longitudinal data on health condition changes with a wider geographic coverage may provide better causal estimates of the long-term health effects attributed to the GLFF. Second, undiagnosed chronic diseases, such as hypertension and diabetes, are prevalent in China, especially among rural residents (Yang et al. 2010; Wu et al. 2008), which may bias our estimates. To reduce bias due to undiagnosed diseases, we adopt the derived variable, namely whether the participant has any listed chronic diseases, as our main health outcome. Third, we may underestimate the famine effects, i.e., both the adverse effect and the selection effect, as there was no clean control group that was immune to the GLFF.

In conclusion, this study provides empirical evidence supporting the co-existence of the adverse and selection effects. The current analysis adds to the understanding of GLFF's long-term health impact from several perspectives. First, the actual adverse impact of the GLFF tends to be underestimated if researchers only examine the net effect of the famine. Second, the survivors of the GLFF may have exhibited better long-term health benefits due to the selection effect. Third, in a population with a high prevalence of malnutrition, adolescents should be considered as an intervention target population as they would benefit from nutrition intervention programs in the long run.

Future studies should focus on separating the adverse effect from the selection effect for different birth cohorts exposed to the GLFF. Data with broader famine intensity coverage and more accurate disease diagnoses will also help generate more accurate and reliable famine effect estimates.

References:

Akachi, Y. & Canning, D., 2010. Health trends in Sub-Saharan Africa: Conflicting evidence from infant mortality rates and adult heights. *Economics & Human Biology* **8**: 273–288.

Alderman, H., Lokshin, M. & Radyakin, S., 2011. Tall claims: Mortality selection and the height of children in India. *Economics & Human Biology* **9**: 393–406.

Almondy, D. et al., 2007. Long-Term Effects Of The 1959-1961 China Famine Mainland China and Hong Kong. *NBER Working Paper No. W13384* .

An, Y., Li, W. & Yang, T., 2001. China's Great Leap: Forward or Backward? Anatomy of a Central Planning Disaster. *CEPR Discussion Paper No. 2824*.

Ashton, B. et al., 1984. Famine in China, 1958–61. *Population and Development Review* **10**: 613–645.

Bachman, D., 1991. *Bureaucracy, Economy, and Leadership in China: The Institutional Origins of the Great Leap Forward.* , New York: Cambridge University Press.

Barker, D.J.P., 1995. Fetal origins of coronary heart disease. *BMJ* **311**: 171–174.

Barker, D.J.P., 1997. Maternal Nutrition, Fetal Nutrition, and Disease in Later Life. *Nutrition* **13**: 807–813.

Barker, D.J.P., 1998. *Mothers, Babies, and Disease in Later Life, 2nd ed.*, New York: Churchill Livingstone.

Chang, H. & Wen, G., 1997. Communal dining and the Chinese famine of 1958-1961. *Economic Development and Cultural Change* **46**: 1–34.

Chang, H. & Wen, G., 1998. Food Availability versus Consumption Efficiency: Causes of the Chinese Famine. *China Economic Review* **9**: 157–165.

Chen, H., Nembhard, W.N. & Stockwell, H.G., 2014. Sex-specific effects of fetal exposure to the 1959-1961 Chinese famine on risk of adult hypertension. *Maternal and child health journal* **18**: 527–533.

Chen, Y. & Zhou, L., 2007. The long-term health and economic consequences of the 1959–1961 famine in China. *Journal of Health Economics* **26**: 659–681.

Cheng, T. & Selden, M., 1994. The Origins and Social Consequences of China's Hukou System. *The China Quarterly* 139: 644-668.

Coale, A.J., 1981. Population trends, population policy, and population studies in China. *Population and Development Review* **7**: 85–97.

Easterlin, R., 1980. *Birth and Fortune: The Impact of Numbers on Personal Welfare.*, The University of Chicago Press.

Fawzi, W. et al., 1997. A prospective study of malnutrition in relation to child mortality. *American Journal of Clinical Nutrition* **65**: 1062–1069.

Gordon, C. & Laufer, M., 2005. The physiology of puberty. In M. Laufer, D. Goldstein, & J. Emans, eds. *Pediatric and Adolescent Gynecology: 5th Edition*. Philadelphia: Lippincott, Williams & Wilkins.

Gørgens, T., Meng, X. & Vaithianathan, R., 2012. Stunting and selection effects of famine: A case study of the Great Chinese Famine. *Journal of Development Economics* **97**: 99–111.

Hales, N. & Barker, D., 2001. The thrifty phenotype hypothesis--Type 2 diabetes. *British Medical Bulletin* **60**: 5–20.

Hales, N. & Barker, D., 1992. Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. *Diabetologia* **35**: 595–601.

Heald, F.P., 1975. Adolescent nutrition. *Med Clin North Am.* **59**: 1329–1936.

Huang, C. et al., 2010. Bigger babies born to women survivors of the 1959-1961 Chinese famine: a puzzle due to survival selection? *Journal of developmental origins of health and disease* **1**: 412–418.

Huang, C. et al., 2010. Early life exposure to the 1959-1961 Chinese famine has long-term health consequences. *The Journal of nutrition* **140**: 1874–8.

Johnson, D., 1998. China's Great Famine: Introductory Remarks. *China Economic Review* **9**: 103–109.

Li, W., 2005. The Great Leap Forward: Anatomy of a Central Planning Disaster. *Journal of Political Economics* **113**: 840–877.

Li, Y. et al., 2010. Exposure to the Chinese famine in early life and the risk of hyperglycemia and type 2 diabetes in adulthood. *Diabetes* **59**: 2400–2406.

Li, Y., Jaddoe, V., Qi, L., He, Y., Lai, J., et al., 2011. Exposure to the Chinese famine in early life and the risk of hypertension in adulthood. *Journal of Hypertension* **29**: 1085–1092.

Li, Y., Jaddoe, V., Qi, L., He, Y., Wang, D., et al., 2011. Exposure to the chinese famine in early life and the risk of metabolic syndrome in adulthood. *Diabetes Care* **34**: 1014–1018.

Lin, Y., 1990. Collectivization and China's Agricultural Crisis in 1959–1961. *Journal of Political Economy* **98**: 1228–1252.

Lin, Y., 2000. Food Availability, Entitlements and the Chinese Famine of 1959–61. *The Economic Journal* **110**: 136–158.

Lin, Y. & Yang, T., 1998. On the causes of China's agricultural crisis and the Great Leap famine. *China Economic Review* **9**: 125–140.

Luo, Z., Mu, R. & Zhang, X., 2006. Famine and Overweight in China. *Applied Economic Perspectives and Policy* **28**: 296–304.

Marshall, W. & Tanner, J., 1968. Growth and Physiological Development During Adolescence. *Annual Review of Medicine* **19**: 283–300.

Meng, X. & Qian, N., 2008. The Causes of Cross-Sectional Inequity in Exposure to Chinaís Great Famine and Its Long Run Health and Economic Consequences on Survivors. *Working Paper*.

Meng, X. & Qian, N., 2009. The Long Term Consequences of Famine on Survivors: Evidence from a Unique Natural Experiment using China's Great Famine. *NBER Working Papers 14917*.

Meredith, C. & Dwyer, J., 1991. Nutrition and Exercise: Effects on Adolescent Health. *Annual Review of Public Health* **12**: 309–333.

Mu, R. & Zhang, X., 2011. Why does the Great Chinese Famine affect the male and female survivors differently? Mortality selection versus son preference. *Economics and Human Biology* **9**: 92–105.

Peng, X., 1987. Demographic consequences of the great leap forward in China's provinces. *Population and Development Review* **13**: 639–670.

Ravelli, a C.J. et al., 1999. Obesity at the age of 50y in men and women exposed to famine prenatally. *American Journal of Clinical Nutrition* **70**: 811–816.

Robinson, R., 2001. The fetal origins of adult disease: no longer just a hypothesis and may be critically important in south Asia. *BMJ* **41**: 158–176.

Roseboom, T. et al., 2001. Effects of prenatal exposure to the Dutch famine on adult disease in later life: an overview. *Molecular and Cellular Endocrinology* 185: 93-98.

Schultz, T., 2005. Effects of Fertility Decline on FamilyWell-Being: Evaluationof Population Programs. *MacArthur Foundation ConsultationMeeting*.

Shi, X., 2011. Famine, fertility, and fortune in china. *China Economic Review* **22**: 244–259.

Song, S., 2012. Does famine influence sex ratio at birth? Evidence from the 1959-1961 Great Leap Forward Famine in China. *Proceedings. Biological sciences / The Royal Society* **279**: 2883–2890.

Song, S., 2010. Mortality consequences of the 1959-1961 Great Leap Forward famine in China: Debilitation, selection, and mortality crossovers. *Social Science and Medicine* **71**: 551–558.

St Clair, D. et al., 2005. Rates of Adult Schizophrenia Following Prenatal Exposure to the Chinese Famine of 1959-1961. *JAMA* **294**: 557–562.

Stanner, S. a et al., 1997. Does malnutrition in utero determine diabetes and coronary heart disease in adulthood? Results from the Leningrad siege study, a cross sectional study. *BMJ* **315**: 1342–1348.

State Statistical Bureau, 1990. *Quangguo Gesheng Zizhiqu Zhixiashi Lishi Tongji Ziliao Huibian, 1949–1989 (A Compilation of Historical Statistical Data of Provinces, Autonomous Regions, and Municipalities)*, Beijing: China Statistical Press.

State Statistical Bureau, 1991. *Statistical Yearbook of China 1991*, Beijing.

Wang, F., 2005. *Organizing Through Division and Exclusion: China's Hukou System*, Stanford University Press.

Wang, Y. et al., 2010. The Great Chinese Famine leads to shorter and overweight females in Chongqing Chinese population after 50 years. *Obesity* **18**: 588–592.

Wells, J.C.K., 2007. The thrifty phenotype as an adaptive maternal effect. *Biological Reviews* **82**: 143–172.

WHO, 2010. *Global status report on noncommunicable diseases*, Geneva.

Wu, Y. et al., 2008. Prevalence, Awareness, Treatment, and Control of Hypertension in China: Data from the China National Nutrition and Health Survey 2002. *Circulation* **118**: 2679–2686.

Yang, D. & Su, F., 1998. The Politics of Famine and Reform in Rural China. *China Economic Review* **9**: 141–155.

Yang, G. et al., 1999. Smoking in China--findings of the 1996 national prevalence survey. *JAMA* **282**: 1247–1253.

Yang, L. et al., 2012. Alcohol drinking and overall and cause-specific mortality in China: nationally representative prospective study of 220,000 men with 15 years of follow-up. *International journal of epidemiology* **41**: 1101–1113.

Yang, W. et al., 2010. Prevalence of diabetes among men and women in China. *The New England journal of medicine* **362**: 1090–1101.

Yang, Z. et al., 2008. Impact of famine during pregnancy and infancy on health in adulthood. *Obesity Reviews* **9**: 95–99.

Yao, S., 1999. A note on the causal factors of China's famine in 1959–1961. *Journal of Political Economy* **107**: 1365–1369.

Zheng, X. et al., 2012. Risk of metabolic syndrome in adults exposed to the great Chinese famine during the fetal life and early childhood. *Eur J Clin Nutr* **66**: 231–236.

Table I. Province-Level Death Rate Before, During and After Famine (‰)

Province	1954	1955	1956	1957	1958	1959	1960	1961	1962	EDR [§]
Inner Mongolia	20.9	11.4	7.9	10.5	7.9	11.0	9.4	8.8	9.0	0.3
Shanghai	7.1	8.1	6.8	6.0	5.9	6.9	6.8	7.7	7.3	0.4
Jiangxi	14.2	16.2	12.5	11.5	11.3	13.0	16.1	11.5	11.0	0.7
Zhejiang	13.4	12.6	9.5	9.3	9.2	10.8	11.9	9.8	8.6	0.7
Tianjin	9.3	9.9	8.8	9.4	8.7	9.9	10.3	9.9	7.4	0.8
Shanxi	11.0	10.5	9.9	10.3	11.0	12.7	12.3	8.8	9.4	0.8
Shaanxi	14.7	12.9	11.6	12.7	11.7	12.8	14.2	12.2	11.3	0.8
Heilongjiang	11.1	11.3	10.1	10.5	9.2	12.8	10.6	11.1	8.6	1.2
Beijing	8.6	9.5	7.7	8.2	8.1	9.7	9.1	10.8	8.8	1.5
Xinjiang	16.8	14.4	14.2	14.0	13.0	18.8	15.7	11.7	9.7	1.5
Ningxia	13.1	10.2	10.6	11.1	15.0	15.8	13.9	10.7	8.5	1.7
Yunnan	16.7	13.7	15.2	16.3	21.6	18.0	26.3	11.8	10.9	2.0
Guangdong	11.2	10.6	11.1	8.4	9.2	11.1	15.2	10.8	9.4	2.5
Hebei	12.1	11.6	11.3	11.3	10.9	12.3	15.8	13.6	9.1	2.6
Jilin	10.4	9.9	7.5	9.1	9.1	13.4	10.1	12.0	10.0	2.9
Fujian	10.9	8.9	8.4	7.9	7.5	7.9	15.3	11.9	8.3	3.5
Jiangsu	12.2	11.8	13.0	10.3	9.4	14.6	18.4	13.4	10.4	4.3
Hubei	15.9	11.6	10.8	9.6	9.6	14.5	21.2	9.1	8.8	4.5
Liaoning	8.6	9.4	6.6	9.4	6.6	11.8	11.5	17.5	8.5	5.6
Shandong	11.7	13.7	12.1	12.1	12.8	18.2	23.6	18.4	12.4	7.4
Hunan	17.5	16.4	11.5	10.4	11.7	13.0	29.4	17.5	10.2	7.5
Henan	13.3	11.8	14.0	11.8	12.7	14.1	39.6	10.2	8.0	8.7
Guangxi	15.2	14.6	12.5	12.4	11.7	17.5	29.5	19.5	10.3	9.4
Gansu	11.6	11.9	10.8	11.3	21.1	17.4	41.3	11.5	8.3	9.6
Qinghai	13.3	14.1	9.4	10.4	13.0	16.6	40.7	11.7	5.4	11.3
Guizhou	8.8	8.1	7.5	8.8	13.7	16.2	45.4	17.7	10.4	16.9
Anhui	16.6	11.8	14.3	9.1	12.3	16.7	68.6	8.1	8.2	19.3
Sichuan	8.4	9.2	10.4	12.1	25.2	47.0	54.0	29.4	14.6	29.2
Nation	13.2	12.3	11.4	10.8	12.0	14.6	25.4	14.2	10.0	6.4

Source: A Compilation of Historical Statistical Data of Provinces, Autonomous Regions, and Municipalities (*Quangguo Gesheng Zizhiqu Zhixiashi Lishi Tongji Ziliao Huibian*) 1949–1989 by National Bureau of Statistics of China 1990.

[§] EDR: excess death rate. We calculate the average death rate in normal years (1954-1958) and during the famine (1959-1961) for each province and the national average and then calculate the difference between the two.

Our URHS 2008 data include three mildly affected provinces (Shaanxi, Beijing and Yunnan) and Sichuan, the worst hit province. The worst affected province in CHNS, another popular dataset used for GLFF research, is Guizhou, the EDR of which is only about half that of Sichuan.

Table II. Summary Statistics

	Prenatal/Infant Famine-Exposed and Comparison Cohorts		Childhood/puberty Famine-Exposed and Comparison Cohorts	
	Cohort 59-61	Cohort 63-65	Cohort 39-42	Cohort 43-58
Observations	455	750	359	2801
Independent variables				
Prevalence of chronic disease (% male/female)	19.46/27.35	16.57/17.27	49.39/57.44	33.66/42.27
Prevalence of hypertension (% male/female)	5.88/6.41	2.49/1.80	17.68/23.08	10.33/14.02
Prevalence of diabetes (% male/female)	0.90/1.28	0.55/1.29	6.10/8.21	3.86/4.12
Dependent variables				
Personal characteristics				
Male (%)	48.57	48.27	45.68	48.05
Rural residents (%)	54.07	56.46	50.97	60.51
Non-migrants (% male/female)	80/60	78/57	67/50	75/51
Middle school and above (%)	78.24	78.83	42.34	56.16
Married (%)	98.89	90.28	80.22	89.86
Income per-capita (1000 Yuan) Mean (SD)	12 (21)	14 (25)	11 (20)	12 (32)
Health-related behavior				
Non-smoker (% male/female)	28/97	33/98	40/95	33/97
Non-drinker (% male/female)	40/92	37/91	53/96	39/93
Self-perceived environmental conditions				
Air pollution score Mean (SD)	1.35 (0.35)	1.34 (0.36)	1.32 (0.33)	1.30 (0.35)
Noise pollution score Mean (SD)	1.31 (0.34)	1.35 (0.37)	1.29 (0.31)	1.30 (0.36)
Water pollution score Mean (SD)	1.36 (0.34)	1.36 (0.34)	1.31 (0.32)	1.31 (0.34)
Greening rate score Mean (SD)	2.11 (0.43)	2.18 (0.43)	2.12 (0.43)	2.17 (0.45)
Provinces				
Beijing (%)	27.47	23.17	27.58	24.49
Shaanxi (%)	33.19	27.43	27.58	29.49
Sichuan (%)	16.92	26.36	25.63	27.31
Yunnan (%)	22.42	23.04	19.21	18.71

Notes: Our sample includes people born between 1939 and 1965 excluding 1962.
Prevalence of health outcomes is based on self-reported doctor-confirmed conditions.
Male, rural residents, non-migrants, middle school and above, and married are all dummy variables, with 1 indicating the status defined by variable name, and 0 otherwise.
Non-smoker is a dummy variable indicating that participants never smoked; non-drinker indicates participants who self-reported never or only occasionally drank.
Scores of air pollution, noise pollution, and water pollution range from 1 to 3 (1 - light; 2 - medium and 3 - severe). Greening rate score has the same range (1 - unsatisfied; 2 - neutral; and 3 - satisfied with greening in the community). These scores are averaged at the community (PSU) level from all of the respondents in the survey to provide a relatively objective measure of the environmental conditions.
The province variables indicate the percentage of participants from each province in each exposure/comparison cohort.

Table III. Exposure to Famine and Chronic Disease in Later Life

	Full sample					Non-migrants	Rural	Urban
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: prenatal/infant exposure								
Cohort59-61*EDR	-4.98***	-4.69**	-4.11**	-4.16**	-3.98**	-10.04***	-6.65**	-2.97
	(1.85)	(1.87)	(1.85)	(1.85)	(1.86)	(3.55)	(3.08)	(2.08)
Excess death rate	0.49	0.47	0.27	0.22	-0.13	1.99	0.30	-1.27
	(1.36)	(1.24)	(1.21)	(1.22)	(1.26)	(2.36)	(1.76)	(1.87)
Cohort59-61	0.10***	0.10***	-0.03	-0.03	-0.03	0.05	-0.03	0.02
	(0.03)	(0.03)	(0.06)	(0.06)	(0.06)	(0.12)	(0.08)	(0.08)
Observations	1,205	1,205	1,205	1205	1,205	807	670	535
R-squared	0.01	0.03	0.05	0.05	0.06	0.09	0.06	0.07
Panel B: childhood/puberty exposure								
Cohort43-58*EDR	6.55***	6.29***	6.48***	6.67***	6.42***	7.48***	6.12**	7.29***
	(1.61)	(1.61)	(1.61)	(1.61)	(1.60)	(2.24)	(2.68)	(1.90)
Excess death rate	-10.31***	-10.02***	-10.74***	-10.77***	-11.10***	-12.55***	-12.62***	-10.36***
	(1.96)	(1.92)	(1.94)	(1.93)	(2.04)	(2.52)	(2.76)	(2.60)
Cohort43-58	-0.21***	-0.20***	-0.06	-0.06	-0.06	-0.18***	-0.07	-0.04
	(0.03)	(0.03)	(0.05)	(0.05)	(0.05)	(0.07)	(0.07)	(0.08)
Observations	3,160	3,160	3,160	3,160	3,160	1,956	1,878	1,282
R-squared	0.03	0.03	0.06	0.06	0.06	0.06	0.05	0.11
Control Variables								
Gender	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Rural residents	No	Yes	Yes	Yes	Yes	Yes	No	No
Non-migrants	No	Yes	Yes	Yes	Yes	No	Yes	Yes

Demographic characteristics	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Smoking and alcohol consumption	No	No	No	Yes	Yes	Yes	Yes	Yes
Self-perceived environmental conditions	No	No	No	No	Yes	Yes	Yes	Yes

Notes: For the prenatal/infant famine-exposed cohort, cohort59-61 is the treatment group and cohort63-65 is the comparison group; for the childhood/puberty famine-exposed cohort, cohort43-58 is the treatment group and cohort39-42 is the comparison group. The table presents coefficients of ordinary least square estimation of equation (1) for the prenatal/infant and puberty famine-exposed cohort. Excess death rate is a continuous measurement of famine intensity (definition described in section 2.1). Income per capita is log-transformed, and the self-perceived environmental conditions are averaged within each community. Demographic characteristics include age, marriage status, education, income per capita, and ethnic background. Self-perceived environmental conditions include air, water, noise pollution and community greening status. Robust standard errors clustered by primary sampling unit--community (228 cells for prenatal/infant exposure and 212 cells for childhood/puberty exposure; 240 cells for the entire dataset) are reported in parentheses.

*** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

Table IV Exposure to Famine and Chronic Disease in Later Life — Gender Difference

	Full sample		Non-smoker		Non-drinker	
	Female	Male	Female	Male	Female	Male
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: prenatal/infant exposure						
Cohort59-61*EDR	-6.26**	-1.96	-5.85**	-7.06*	-6.15**	-9.86**
	(2.80)	(2.84)	(2.87)	(4.11)	(3.01)	(4.30)
Excess death rate	0.61	-0.75	0.69	-1.00	1.46	2.84
	(1.55)	(1.70)	(1.58)	(3.27)	(1.64)	(3.43)
Cohort59-61	0.01	-0.09	0.01	-0.12	-0.00	-0.05
	(0.08)	(0.07)	(0.08)	(0.12)	(0.08)	(0.11)
Observations	622	583	606	182	567	218
R-squared	0.09	0.03	0.09	0.09	0.10	0.08
Panel B: childhood/puberty exposure						
Cohort43-58*EDR	6.47**	6.36**	6.23**	1.11	5.99**	4.74
	(2.67)	(2.73)	(2.70)	(4.92)	(2.74)	(4.84)
Excess death rate	-11.20***	-10.86***	-10.96***	-5.29	-10.78***	-9.28**
	(3.00)	(2.64)	(3.02)	(5.01)	(3.08)	(4.56)
Cohort43-58	-0.05	-0.07	-0.07	0.10	-0.05	-0.13
	(0.08)	(0.08)	(0.08)	(0.14)	(0.08)	(0.12)
Observations	1,650	1,510	1,602	508	1,544	606
R-squared	0.10	0.08	0.09	0.10	0.08	0.12
Controls as in column 5 of Table III	Yes	Yes	Yes	Yes	Yes	Yes

Notes: For the prenatal/infant famine-exposed cohort, cohort59-61 is the treatment group and cohort63-65 is the comparison group; for the childhood/puberty famine-exposed cohort, cohort43-58 is the treatment group and cohort39-42 is the comparison group. The table presents coefficients of ordinary least square estimation of equation (1) for prenatal/infant and childhood/puberty exposure. Excess death rate is a continuous measurement of famine intensity (definition described in section 2.1). Income per capita is log-transformed, and the self-perceived environmental conditions are averaged within each community. Robust standard errors clustered by primary sampling unit (community) are reported in parentheses.

*** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

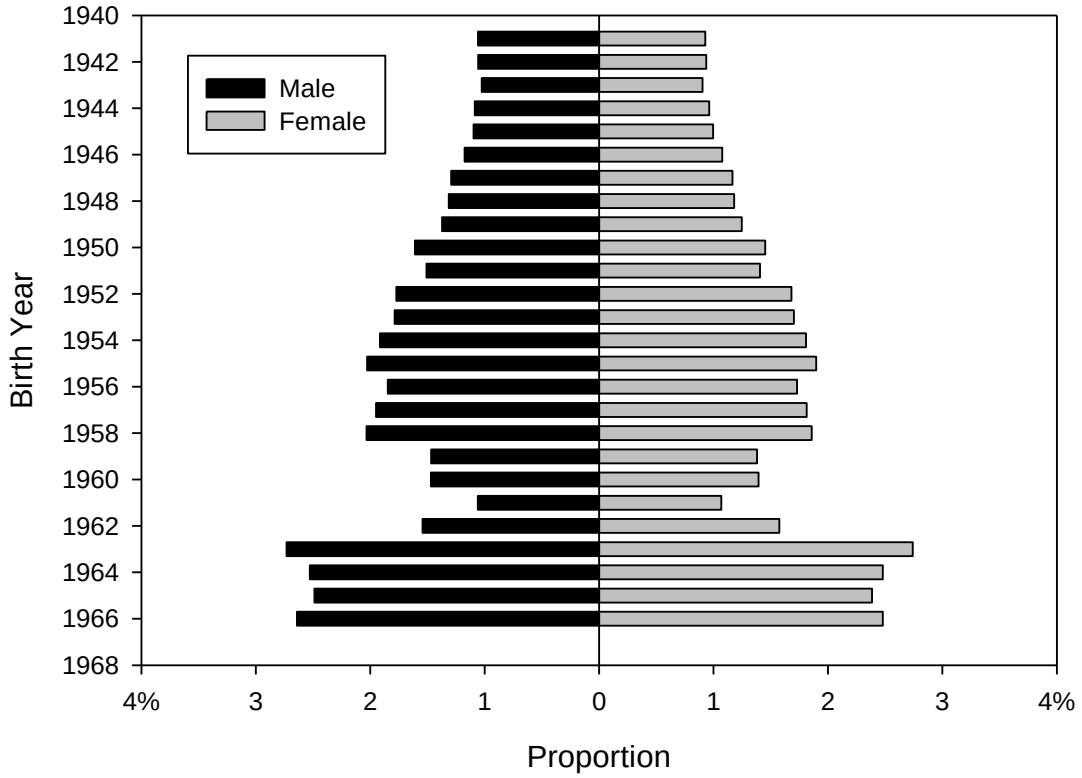
Table V. Falsification tests

	Full sample	Non-migrants	Rural	Urban	Female	Male
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: prenatal/infant exposure						
Cohort66-68*EDR	-2.11	-1.85	-2.54	-1.52	-3.78	0.10
	(1.66)	(1.71)	(2.44)	(1.89)	(3.54)	(2.01)
Excess death rate	-0.10	0.46	0.78	-0.81	3.61	-0.38
	(1.25)	(1.22)	(1.73)	(1.77)	(3.31)	(1.67)
Cohort66-68	-0.04	-0.04	-0.06	0.00	0.01	-0.08
	(0.05)	(0.05)	(0.07)	(0.07)	(0.09)	(0.06)
Observations	1,409	457	827	582	719	690
R-squared	0.04	0.06	0.04	0.05	0.07	0.02
Panel B: childhood/puberty exposure						
Cohort35-38*EDR	0.61	0.23	-1.11	-0.77	0.03	0.95
	(0.91)	(1.57)	(1.26)	(1.35)	(1.34)	(1.03)
Excess death rate	-1.64***	-0.56	-0.60	-0.29	-0.83	-2.31***
	(0.58)	(1.44)	(0.66)	(0.85)	(0.86)	(0.75)
Cohort35-38	-0.03	-0.07	0.05	0.02	0.02	-0.11
	(0.04)	(0.05)	(0.04)	(0.05)	(0.04)	(0.07)
Observations	719	360	347	372	386	333
R-squared	0.09	0.16	0.10	0.13	0.09	0.10
Controls are the same as in column 5 of Table III	Yes	Yes	Yes	Yes	Yes	Yes

Notes: For the prenatal/infant famine-exposed cohort, cohort66-68 is the treatment group and cohort63-65 is the comparison group; for the childhood/puberty famine-exposed cohort, cohort35-38 is the treatment group and cohort39-42 is the comparison group. The table presents coefficients of ordinary least square estimation of falsification tests for prenatal/infant and childhood/puberty exposure. Excess death rate is a continuous measurement of famine intensity (definition described in section 2.1). Income per capita is log-transformed, and the self-perceived environmental conditions are averaged within each community. Robust standard errors clustered by primary sampling unit (community) are reported in parentheses.

*** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

Figure 1. China's Population Pyramid in 1982



Source: National Bureau of Statistics of China. This figure shows the population pyramid of China 1982. From the figure, we observe that the cohort sizes for both males and females drop sharply beginning in 1959, when the GLFF began, and they do not return to normal until 1963. The sizes of the other birth cohorts are normal.

Appendix

Table A1. Prenatal/infant Exposure to Famine and Chronic Disease in Later Life

	Full sample					Non-migrants	Rural	Urban
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Cohort59-61*EDR	-4.98***	-4.69**	-4.11**	-4.16**	-3.98**	-10.04***	-6.65**	-2.97
	(1.85)	(1.87)	(1.85)	(1.85)	(1.86)	(3.55)	(3.08)	(2.08)
Excess death rate	0.49	0.47	0.27	0.22	-0.13	1.99	0.30	-1.27
	(1.36)	(1.24)	(1.21)	(1.22)	(1.26)	(2.36)	(1.76)	(1.87)
Cohort59-61	0.10***	0.10***	-0.03	-0.03	-0.03	0.05	-0.03	0.02
	(0.03)	(0.03)	(0.06)	(0.06)	(0.06)	(0.12)	(0.08)	(0.08)
Female	0.02	0.02	0.02	0.04	0.03	0.04	0.03	0.04
	(0.02)	(0.02)	(0.02)	(0.03)	(0.03)	(0.05)	(0.05)	(0.04)
Rural residents		0.07**	0.07**	0.07**	0.05	0.06		
		(0.03)	(0.03)	(0.03)	(0.03)	(0.07)		
Non-migrants		-0.06**	-0.06**	-0.06**	-0.05**	-0.16	-0.03	-0.06*
		(0.02)	(0.02)	(0.02)	(0.02)	(0.15)	(0.04)	(0.03)
Race			-0.11*	-0.11*	-0.10*	-0.09	-0.13*	-0.10
			(0.06)	(0.06)	(0.06)	(0.08)	(0.07)	(0.10)
Age			-0.35	-0.37	-0.37	-0.66	-0.14	-0.59
			(0.30)	(0.30)	(0.30)	(0.59)	(0.40)	(0.44)
Age^2			0.00	0.00	0.00	0.01	0.00	0.01
			(0.00)	(0.00)	(0.00)	(0.01)	(0.00)	(0.00)
Married			-0.11***	-0.11***	-0.11***	-0.10	-0.21**	-0.09**
			(0.04)	(0.04)	(0.04)	(0.07)	(0.09)	(0.04)
Middle school and above			-0.05	-0.05	-0.05	-0.12**	-0.02	-0.09*
			(0.03)	(0.03)	(0.03)	(0.06)	(0.04)	(0.05)
Income per capita			-0.02	-0.02	-0.02	-0.00	-0.04	-0.00
			(0.03)	(0.03)	(0.03)	(0.05)	(0.05)	(0.04)
Non-smoker				-0.01	-0.01	-0.02	-0.03	0.00
				(0.03)	(0.03)	(0.05)	(0.05)	(0.04)
Non-drinker				0.02	0.01	-0.03	-0.07	0.10**
				(0.03)	(0.03)	(0.05)	(0.04)	(0.04)
Air pollution					0.07	0.28**	0.08	0.14
					(0.09)	(0.13)	(0.11)	(0.19)
Water pollution					0.03	0.10	0.03	-0.04
					(0.07)	(0.11)	(0.09)	(0.15)
Noise pollution					-0.06	-0.34***	-0.02	-0.11
					(0.06)	(0.10)	(0.07)	(0.15)
Greening rate					0.08**	0.04	0.13***	0.01
					(0.04)	(0.07)	(0.05)	(0.08)
Constant	0.16***	0.16***	7.77	8.07	7.94	14.88	2.69	13.31
	(0.02)	(0.03)	(6.62)	(6.71)	(6.72)	(13.07)	(8.94)	(9.75)
Observations	1,205	1,205	1,205	1,205	1,205	807	670	535
R-squared	0.01	0.03	0.05	0.05	0.06	0.09	0.06	0.07

Notes: Cohort59-61 is the treatment group, and cohort63-65 is the comparison group. The table presents coefficients of ordinary least square estimation of equation (1) for prenatal/infant exposure. Excess death rate is a continuous measurement of famine intensity (definition described in section 2.1). Income per capita is log-transformed, and the self-perceived environmental conditions are averaged within each community. Robust standard errors clustered by primary sampling unit (community) are reported in

parentheses.
*** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

Table A2. Childhood/puberty Exposure to Famine and Chronic Disease in Later Life

	Full sample					Non-migrants	Rural	Urban
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Cohort43-58*EDR	6.55***	6.29***	6.48***	6.67***	6.42***	7.48***	6.12**	7.29***
	(1.61)	(1.61)	(1.61)	(1.61)	(1.60)	(2.24)	(2.68)	(1.90)
Excess death rate	-10.31***	-10.02***	-10.74***	-10.77***	-11.10***	-12.55***	-12.62***	-10.36***
	(1.96)	(1.92)	(1.94)	(1.93)	(2.04)	(2.52)	(2.76)	(2.60)
Cohort43-58	-0.21***	-0.20***	-0.06	-0.06	-0.06	-0.18***	-0.07	-0.04
	(0.03)	(0.03)	(0.05)	(0.05)	(0.05)	(0.07)	(0.07)	(0.08)
Female	0.09***	0.07***	0.07***	0.08***	0.08***	0.07**	0.09**	0.06*
	(0.02)	(0.02)	(0.02)	(0.03)	(0.03)	(0.03)	(0.04)	(0.04)
Rural residents		0.02	-0.00	-0.00	-0.03	-0.04		
		(0.03)	(0.03)	(0.03)	(0.04)	(0.04)		
Non-migrants		-0.04*	-0.03	-0.03	-0.03		-0.01	-0.03
		(0.02)	(0.02)	(0.02)	(0.02)		(0.03)	(0.04)
Race			0.06	0.06	0.08	0.08	0.13	0.02
			(0.06)	(0.06)	(0.06)	(0.08)	(0.08)	(0.08)
Age			0.05	0.05	0.06	0.12**	0.02	0.12
			(0.05)	(0.05)	(0.05)	(0.06)	(0.07)	(0.09)
Age^2			-0.00	-0.00	-0.00	-0.00*	-0.00	-0.00
			(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Married			-0.01	-0.00	-0.00	-0.04	-0.01	-0.01
			(0.03)	(0.03)	(0.03)	(0.03)	(0.04)	(0.03)
Middle school and above			-0.06**	-0.06**	-0.05**	-0.05*	-0.05*	-0.08*
			(0.02)	(0.02)	(0.02)	(0.03)	(0.03)	(0.04)
Income per capita			0.02	0.03	0.03	-0.01	-0.01	0.05
			(0.02)	(0.02)	(0.02)	(0.03)	(0.04)	(0.03)
Non-smoker				-0.05**	-0.05**	-0.05	-0.06*	-0.03
				(0.03)	(0.03)	(0.03)	(0.04)	(0.04)
Non-drinker				-0.06***	-0.06***	-0.07**	-0.07**	-0.04

				(0.02)	(0.02)	(0.03)	(0.03)	(0.03)
Air pollution					0.05	-0.07	-0.04	0.49**
					(0.09)	(0.10)	(0.09)	(0.22)
Water pollution					-0.02	-0.01	0.05	-0.15
					(0.08)	(0.08)	(0.07)	(0.21)
Noise pollution					-0.02	0.05	0.05	-0.39**
					(0.07)	(0.07)	(0.07)	(0.17)
Greening rate					0.07*	0.07*	0.15***	-0.06
					(0.04)	(0.04)	(0.04)	(0.07)
Constant	0.58***	0.59***	-1.41	-1.40	-1.68	-3.20*	-0.80	-3.35
	(0.03)	(0.04)	(1.49)	(1.48)	(1.49)	(1.64)	(1.90)	(2.54)
Observations	3,160	3,160	3,160	3,160	3,160	1,956	1,878	1,282
R-squared	0.03	0.03	0.06	0.06	0.06	0.06	0.05	0.11

Notes: Cohort43-58 is the treatment group, and cohort39-42 is the comparison group. The table presents coefficients of ordinary least square estimation of equation (1) for childhood/puberty exposure. Excess death rate is a continuous measurement of famine intensity (definition describes in section 2.1). Income per capita is log-transformed, and the self-perceived environmental conditions are averaged within each community. Robust standard errors clustered by primary sampling unit (community) are reported in parentheses.

*** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level

Table A3. Prenatal/infant Exposure to Famine and Chronic Disease in Later Life—gender difference

	Full sample		Non-smoker		Non-drinker	
	Female	Male	Female	Male	Female	Male
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
Cohort59-61*EDR	-6.26**	-1.96	-5.85**	-7.06*	-6.15**	-9.86**
	(2.80)	(2.84)	(2.87)	(4.11)	(3.01)	(4.30)
Excess death rate	0.61	-0.75	0.69	-1.00	1.46	2.84
	(1.55)	(1.70)	(1.58)	(3.27)	(1.64)	(3.43)
Cohort59-61	0.01	-0.09	0.01	-0.12	-0.00	-0.05
	(0.08)	(0.07)	(0.08)	(0.12)	(0.08)	(0.11)
Rural residents	0.08*	0.01	0.08*	-0.00	0.10*	0.04
	(0.05)	(0.05)	(0.05)	(0.07)	(0.05)	(0.07)
Non-migrants	-0.06*	-0.04	-0.06*	-0.03	-0.07*	-0.06
	(0.03)	(0.04)	(0.03)	(0.07)	(0.04)	(0.07)
Race	-0.17	-0.05	-0.17	-0.11	-0.17	-0.07
	(0.10)	(0.09)	(0.10)	(0.23)	(0.11)	(0.13)
Age	-0.53	-0.17	-0.46	0.02	-0.56	-0.47
	(0.41)	(0.41)	(0.42)	(0.64)	(0.42)	(0.61)
Age^2	0.01	0.00	0.01	0.00	0.01	0.01
	(0.00)	(0.00)	(0.00)	(0.01)	(0.00)	(0.01)
Married	-0.14**	-0.07	-0.14**	-0.12	-0.12**	-0.07
	(0.06)	(0.06)	(0.05)	(0.08)	(0.06)	(0.08)
Middle school and above	-0.03	-0.07	-0.04	-0.08	-0.03	-0.02
	(0.04)	(0.05)	(0.04)	(0.09)	(0.04)	(0.08)
Income per capita	-0.02	-0.02	-0.02	-0.01	-0.04	-0.07
	(0.03)	(0.04)	(0.03)	(0.07)	(0.04)	(0.07)
Non-smoker	-0.01	-0.02			0.10	-0.02
	(0.08)	(0.04)			(0.07)	(0.05)
Non-drinker	-0.00	0.02	-0.03	0.04		
	(0.06)	(0.03)	(0.06)	(0.06)		
Air pollution	0.20	-0.07	0.21*	-0.07	0.22*	-0.27
	(0.12)	(0.12)	(0.12)	(0.26)	(0.13)	(0.25)
Water pollution	0.05	0.03	0.06	0.02	0.02	0.21
	(0.09)	(0.09)	(0.09)	(0.11)	(0.09)	(0.13)
Noise pollution	-0.18*	0.05	-0.19*	-0.08	-0.18	0.02
	(0.11)	(0.10)	(0.10)	(0.27)	(0.11)	(0.25)
Greening rate	0.10*	0.07	0.10*	0.13*	0.11**	0.01
	(0.05)	(0.05)	(0.05)	(0.07)	(0.05)	(0.08)
Constant	11.47	3.22	9.94	-1.11	12.00	10.50
	(9.06)	(9.26)	(9.36)	(14.27)	(9.25)	(13.84)
Observations	622	583	606	182	567	218
R-squared	0.09	0.03	0.09	0.09	0.10	0.08

Notes: Cohort59-61 is the treatment group, and cohort63-65 is the comparison group. The table presents coefficients of ordinary least square estimation of equation (1) for prenatal/infant exposure.

Excess death rate is a continuous measurement of famine intensity (definition described in section 2.1). Income per capita is log-transformed, and the self-perceived environmental conditions are averaged within each community. Robust standard errors clustered by primary sampling unit (community) are reported in parentheses. *** Significant at the 1 percent level. ** Significant at the 5 percent level. *

Significant at the 10 percent level.

Table A4. Childhood/puberty Exposure to Famine and Chronic Disease in Later Life—gender difference

	Full sample		Non-smoker		Non-drinker	
	Female	Male	Female	Male	Female	Male
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
Cohort43-58*EDR	8.87**	6.41*	9.39**	1.71	9.43**	2.96
	(4.27)	(3.74)	(4.25)	(6.78)	(4.31)	(6.30)
Excess death rate	-11.49***	-10.12***	-11.81***	-3.73	-11.39***	-9.27*
	(3.11)	(3.00)	(3.09)	(5.75)	(3.15)	(4.89)
Cohort43-58	-0.05	-0.16	-0.07	-0.14	-0.06	-0.22
	(0.13)	(0.17)	(0.13)	(0.28)	(0.13)	(0.28)
Rural residents	-0.11	0.04	-0.14	0.09	-0.11	-0.03
	(0.08)	(0.08)	(0.09)	(0.17)	(0.09)	(0.13)
Non-migrants	0.08	-0.06	0.07	-0.01	0.08	-0.07
	(0.05)	(0.06)	(0.05)	(0.10)	(0.05)	(0.10)
Race	0.30***	-0.02	0.34***	-0.26	0.30***	-0.07
	(0.11)	(0.13)	(0.11)	(0.16)	(0.11)	(0.28)
Age	0.66	-0.32	0.85	-0.71	0.55	-1.03
	(0.57)	(0.57)	(0.59)	(1.03)	(0.59)	(0.94)
Age^2	-0.00	0.00	-0.01	0.01	-0.00	0.01
	(0.00)	(0.00)	(0.00)	(0.01)	(0.00)	(0.01)
Married	0.00	0.01	0.01	-0.02	0.02	0.04
	(0.07)	(0.08)	(0.07)	(0.16)	(0.07)	(0.14)
Middle school and above	-0.09*	-0.06	-0.10*	-0.02	-0.11*	-0.03
	(0.06)	(0.06)	(0.06)	(0.12)	(0.06)	(0.08)
Income per capita	0.11**	0.10*	0.11*	0.03	0.11**	0.01
	(0.05)	(0.06)	(0.05)	(0.16)	(0.06)	(0.11)
Non-smoker	-0.21	-0.08			-0.09	-0.01
	(0.13)	(0.06)			(0.17)	(0.08)
Non-drinker	0.08	-0.06	-0.02	-0.19*		
	(0.11)	(0.06)	(0.14)	(0.11)		
Air pollution	-0.05	0.32*	-0.03	0.50	-0.02	0.56*
	(0.21)	(0.18)	(0.22)	(0.38)	(0.22)	(0.31)
Water pollution	-0.07	-0.12	-0.08	0.09	-0.06	-0.22
	(0.14)	(0.14)	(0.15)	(0.30)	(0.14)	(0.22)
Noise pollution	0.06	-0.18	0.05	-0.44*	0.04	-0.25
	(0.17)	(0.13)	(0.18)	(0.23)	(0.19)	(0.25)
Greening rate	0.04	0.16**	0.05	0.03	0.03	0.26**
	(0.08)	(0.07)	(0.08)	(0.13)	(0.08)	(0.11)
Constant	-21.05	10.71	-27.62	23.88	-17.81	33.38
	(18.44)	(18.33)	(19.14)	(33.06)	(19.12)	(30.78)
Observations	1,650	1,510	1,602	508	1,544	606
R-squared	0.10	0.08	0.09	0.10	0.08	0.12

Notes: Cohort43-58 is the treatment group, and cohort39-42 is the comparison group. The table presents coefficients of ordinary least square estimation of equation (1) for childhood/puberty exposure.
Excess death rate is a continuous measurement of famine intensity (definition described in section 2.1). Income per capita is log-transformed, and the self-perceived environmental conditions are averaged within each community. Robust standard errors clustered by primary sampling unit (community) are reported in parentheses. *** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

Table A5. Exposure to Famine and Hypertension in Later Life

	Full sample	Non-migrants	Rural	Urban	Female	Male
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: prenatal/infant exposure						
Cohort59-61*EDR	-3.10**	-4.85***	-2.50*	-3.59	-4.96***	-0.78
	(1.44)	(1.86)	(1.31)	(2.47)	(1.78)	(2.48)
Excess death rate	-1.85**	-0.74	-0.85	-3.29*	-1.20	-2.46**
	(0.71)	(1.01)	(0.79)	(1.73)	(1.01)	(1.03)
Cohort59-61	0.02	0.04	-0.01	0.11	0.02	0.03
	(0.04)	(0.06)	(0.05)	(0.07)	(0.07)	(0.08)
Observations	1,205	807	670	535	622	583
Panel B: childhood/puberty exposure						
Cohort43-58*EDR	7.57***	10.65***	9.15**	6.25	8.30**	6.72*
	(2.91)	(2.77)	(3.70)	(4.53)	(3.87)	(3.88)
Excess death rate	-12.52***	-16.40***	-14.44***	-12.59***	-13.43***	-11.61***
	(2.81)	(2.93)	(3.76)	(4.38)	(3.52)	(3.91)
Cohort43-58	-0.04	-0.13	-0.11	0.08	0.06	-0.14
	(0.10)	(0.12)	(0.13)	(0.16)	(0.12)	(0.13)
Observations	3,160	1,956	1,878	1,282	1,650	1,510
Controls are the same as in column 5 of Table III	Yes	Yes	Yes	Yes	Yes	Yes

Notes: For the prenatal/infant famine-exposed cohort, cohort59-61 is the treatment group and cohort63-65 is the comparison group; for the childhood/puberty famine-exposed cohort, cohort43-58 is the treatment group and cohort39-42 is the comparison group. The table presents coefficients of ordinary least square estimation of equation (1) for prenatal/infant and puberty exposure. Excess death rate is a continuous measurement of famine intensity (definition described in section 2.1). Income per capita is log-transformed, and the self-perceived environmental conditions are averaged within each community. Robust standard errors clustered by primary sampling unit (community) are reported in parentheses.

*** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

Table A6. Exposure to Famine and Diabetes in Later Life

	Full sample	Non-migrants	Rural	Urban	Female	Male
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: prenatal/infant exposure						
Cohort59-61*EDR	-2.36	-3.44	-4.71**	0.10	-4.47	-0.09
	(1.72)	(3.78)	(2.15)	(1.59)	(3.10)	(1.35)
Excess death rate	1.04	0.40	1.52	-1.61	3.17	-1.19
	(1.33)	(2.56)	(1.95)	(1.63)	(2.52)	(1.18)
Cohort59-61	-0.02	0.02	0.01	-0.00	-0.00	-0.04
	(0.04)	(0.11)	(0.06)	(0.06)	(0.07)	(0.04)
Observations	1,205	807	670	535	622	583
Panel B: childhood/puberty exposure						
Cohort43-58*EDR	11.46***	12.04***	18.51***	5.51	17.80***	3.07
	(3.57)	(4.30)	(5.94)	(3.73)	(4.33)	(5.21)
Excess death rate	-15.22***	-15.20***	-20.37***	-9.50***	-21.33***	-7.21
	(3.62)	(4.12)	(6.93)	(3.60)	(4.20)	(5.36)
Cohort43-58	-0.06	-0.15	-0.17	0.01	-0.25	0.14
	(0.14)	(0.15)	(0.26)	(0.15)	(0.20)	(0.21)
Observations	3,160	1,956	1,878	1,282	1,650	1,510
Controls are the same as in column 5 of Table III	Yes	Yes	Yes	Yes	Yes	Yes

Notes: For the prenatal/infant famine-exposed cohort, cohort59-61 is the treatment group and cohort63-65 is the comparison group; for the childhood/puberty famine-exposed cohort, cohort43-58 is the treatment group and cohort39-42 is the comparison group. The table presents coefficients of ordinary least square estimation of equation (1) for prenatal/infant and puberty exposure. Excess death rate is a continuous measurement of famine intensity (definition described in section 2.1). Income per capita is log-transformed, and the self-perceived environmental conditions are averaged within each community. Robust standard errors clustered by primary sampling unit (community) are reported in parentheses.

*** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.