Early Childhood Malnutrition and Adult Obesity: Evidence from the 1959-61 China Famine[+]

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Abstract

Developing countries today face the paradoxical dual burden of malnutrition and obesity. It has been hypothesized that early childhood malnutrition leads to a higher risk of adult obesity, though evidence is mixed. I study the health outcomes and health behaviors of adult men and women who were born during the 1959-61 China Famine. I find that women who as infants were exposed to famine have on average a higher body mass index (BMI) of 0.84 kg/m^2 (3.7% higher) and are more likely to be obese (5 percentage points more) than women who were not exposed to famine. The effect of early childhood famine exposure increases along the BMI distribution. I do not find significant effects on obesity for men. I also find no evidence that the increase in BMI is differentially greater for the famine cohorts who are exposed to a food-rich environment in adult life than for the famine cohorts who are not. Using detailed individual-level data on food intake and physical activities, I show that the increase in BMI for famine-exposed women is not due to higher caloric or fat intakes nor to more sedentary lifestyles. A biological rather than a behavioral mechanism appears to underlie the association between early childhood malnutrition and adult obesity.

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I Introduction

Developing countries today face a dual burden of undernutrition and overnutrition.[1] Undernutrition affects over one billion people worldwide and causes more than one-half of all child deaths each year (Caulfield et al., 2004; FAO, 2009). In China, despite rapid economic growth in the past two decades, the prevalence of undernutrition remains high, with 14% of preschool children being stunted and 7% of adults being underweight (NSS, 2005).

At the same time, the developing world faces the growing problem of obesity. About one-third of the world's population today is overweight, and almost one in ten is obese (Kelly et al., 2008).[2] In China, more than 25% of adults are overweight, and obesity is increasing at a faster rate than that in developed countries such as the U.S. and U.K. (Popkin, 2008).

Obesity is a growing concern because it imposes serious health and economic costs on both the individual and the society. Defined by the WHO as "the disease in which excess body fat has accumulated to such an extent that health may be adversely affected," obesity is a key risk factor of cardiovascular diseases, type 2 diabetes, musculoskeletal disorders, and certain types of cancers (Must et al., 1999; WHO, 2000). These conditions cause premature death and substantial disability (Fontaine et al., 2003; Flegal et al., 2005). In China, the total medical cost attributable to overweight and obesity has been estimated at 21.1 billion yuan (about US $2.7 billion), or 3.7% of national health expenditures in 2003 (Zhao et al., 2008).

Obesity is not just a result of overeating or a lack of physical activity, as is popularly believed. Although obesity is indeed a consequence of an energy imbalance, where energy intake exceeds energy expenditure over a considerable period of time, there are actually many different genetic, physiological, social, environmental, and behavioral factors that interact to affect a person's susceptibility to weight gain (WHO, 2000). Of particular interest to this study and to developing countries is the hypothesis that early childhood malnutrition can lead to adult obesity.

According to the thrifty genotype hypothesis, people who suffer from undernutrition *in utero* develop "thrifty genes," which alter the body's physiology and metabolism in order to promote survival in times of food shortage (Neel, 1962, 1982; Barker, 1992). Such thriftiness,

[1] Undernutrition is defined as "the result of prolonged low levels of food intake." It is generally applied to energy deficiency, but may also be related to vitamin and mineral deficiencies. Overnutrition is defined as the "excessive food intake in relation to energy requirements." (FAO, 2009)

[2] An individual with a body mass index (BMI) of 25 kg/m^2 or above is considered overweight, while an individual with a BMI of 30 kg/m^2 or above is considered obese.

however, may become maladaptive if undernutrition is followed by nutritional abundance in later life, leading to higher risks of obesity, type 2 diabetes, and coronary heart diseases.[3]

This idea can be illustrated using an animal study. Ozanne and Hales (2004) set up two treatments for four groups of mice. The first treatment is to expose the mice to undernutrition during their fetal and early postnatal life. The second and subsequent treatment is to give the mice a fattening diet as opposed to a standard laboratory diet when they become adults. The researchers find that mice that were undernourished in early life have a shorter lifespan than mice that were not. Among mice that were undernourished in early life, the fattening diet in adult life has an additional impact of reducing their lifespan even more.[4]

It is not difficult to see the parallel in many developing countries. Adults who are now experiencing higher dietary energy intakes and more sedentary lifestyles were born when rates of poor maternal nutrition, low birth weight, and infant growth faltering were high. As developing countries undergo economic growth, the long-term adverse effects of early childhood malnutrition may be compounded by the adoption of diets and lifestyles of developed countries.

In this paper, I study the health outcomes and health behaviors of adult men and women who were born during the 1959-61 China Famine and who experienced China's rapid economic growth and nutrition transition beginning in the 1980s. I focus on obesity as the outcome, since obesity is a key risk factor of morbidity and mortality. I ask three questions: First, are individuals who were exposed to famine during early childhood (whom I call the "famine cohorts") more likely to be obese when they are adults? Second, are the adverse effects of famine exposure greater for the famine cohorts who are exposed to a food-rich environment in adult life, as compared to the famine cohorts who are not? Third, what are the mechanisms underlying the association between early childhood malnutrition and adult obesity?

Using data from the China Health and Nutrition Survey (CHNS), I find that women who as infants were exposed to famine have on average a higher body mass index (BMI) of 0.84 kg/m^2 (3.7% higher) and are more likely to be obese (5 percentage points more) than women who were not exposed to famine. The effect of famine exposure increases along the BMI distribution, and appears to decrease with age. I do not find significant effects on obesity for men.

[3] The idea that later-life nutritional environments may affect the expression or non-expression of thrifty genes is known as the thrifty phenotype hypothesis, which emphasizes gene-environment interaction and can be thought of as an update to the thrifty genotype hypothesis (Hales and Barker, 1992).

[4] Jones and Friedman (1982) present a similar study with obesity in mice as the outcome. They find that mice that were undernourished *in utero* gained more weight than mice that were not. The introduction of a high-fat diet in adult life accelerated the weight gain in the mice that were undernourished *in utero.*

I also find no evidence that the increase in BMI is differentially greater for the famine cohorts who are exposed to a food-rich environment in adult life than for the famine cohorts who are not. Using detailed individual-level data on food intake and physical activities, I show that the increase in BMI for famine-exposed women is not due to higher caloric or fat intakes nor to more sedentary lifestyles. A biological rather than a behavioral mechanism appears to underlie the association between early childhood malnutrition and adult obesity.

The rest of my paper is structured as follows: Section II discusses related literature. Section III gives the background and conceptual framework. Section IV describes the data, and Section V presents the empirical strategy and results. Section VI concludes.

II Related literature

There is increasing evidence that malnutrition in early life affects long-term health outcomes (Barker et al., 1989; Barker, 1992; Hertzman and Wiens, 1996; Godfrey and Barker, 2000; Hertzman and Power, 2003; Heymann et al., 2005). However, the effect of early childhood malnutrition on adult obesity is less clearly established.

The first famine study that links intrauterine malnutrition to later life obesity is on the 1944-45 Dutch Hunger Winter.[5] Ravelli et al. (1976) find that 19-year-old men who were exposed to the famine *in utero* during the first two trimesters are 80% more likely to be overweight than those who were not exposed to famine. On the other hand, those who were exposed during the last trimester or during the postnatal period are 40% less likely to be overweight than those who were not exposed. In a later study, Ravelli et al. (1999) find that 50-year-old women who were exposed to famine in early gestation have a higher BMI and waist-to-hip ratio than those who were exposed in late gestation or those who were not exposed. They do not observe significant effects for 50-year-old men.

Studies of another famine, one that occurred during the 1941-44 Leningrad Siege, have led to opposite findings.[6] Stanner et al. (1997) study a small sample of survivors who were born

[5] The Dutch Hunger Winter took place between October 1944 and May 1945, and is a result of Nazi Germany's embargo of all food transports to the western Netherlands and the early onset of an unusually harsh winter. Adult food rations dropped to a mere 580 calories per day by the end of February 1945. A total of 18,000 people died during the famine (Stein et al., 1975).

[6] The Leningrad Siege began in September 1941 when German troops surrounded the city. The siege lasted for 900 days and about 800,000 people died. In January 1942, the city's food rations reached an all-time low of 125 grams of bread per person per day (Barber and Dzeniskevich, 2005).

in Leningrad around the time of the Nazi German siege and they find no association between prenatal famine exposure and adult obesity.

How does one reconcile these different findings? First, it must be noted that the economic and nutrition conditions in the two countries after the famines were quite different. Individuals born in Amsterdam around the time of the Dutch famine grew up in a time of increasing economic affluence, while the living conditions of those born in Leningrad remained relatively poor (Stanner et al., 1997; Ravelli et al., 1999). This suggests that the effects of early childhood malnutrition on adult obesity may depend on later life environmental context, specifically, on whether individuals born during the famine grew up in an environment of nutritional abundance, as was the case in the Netherlands but not in Leningrad. Results of the Dutch Hunger Winter studies also suggest that there may be a gender difference in the effects of famine exposure. The effects of famine exposure may also decrease with age, as suggested by the significant effects observed in 19-year-old men but not in 50-year-old men.[7]

I investigate the effects of early childhood famine exposure in the China context. Recent studies have looked at the long-term health and economic outcomes of famine survivors (Luo et al., 2006; Chen and Zhou, 2007; Gorgens et al., 2007; Almond et al., 2007; Shi, 2007; Mu and Zhang, 2008; Meng and Qian, 2009). Most related to my study are the work by Luo et al. (2006) and Meng and Qian (2009). Using the 1991 to 2000 China Health and Nutrition Surveys (CHNS), Luo et al. (2006) find that famine exposure leads to an increase in probability of overweight by 0.086 for rural women, and they find no effect for urban women or for men.[8] Meng and Qian (2009), on the other hand, use the 1989 CHNS and find that individuals who were exposed to famine during early childhood have a lower height by 1.6% (2.7 cm), a lower weight by 5% (3 kg), and a lower weight-for-height by 1.2% (0.004 kg/cm).[9] They do not distinguish the effects between men and women.

In my study, I track individuals to an older age than in the previous studies. I highlight the gender difference in the effect of early childhood famine exposure, and I allow for differential treatment effects for underweight, normal weight, and overweight individuals. I

[7] On the other hand, there are studies showing that the effect of early childhood malnutrition on chronic disease markers is amplified with age (Law et al., 1993).
[8] Luo et al. (2006) use a difference-in-difference strategy, dividing provinces into more severe and less severe famine regions, and dividing individuals into a treatment group (those born during the famine in 1959-62) and a control group (those born after the famine in 1963-66).
[9] Meng and Qian (2009) use the county-level average cohort size of the 1959-61 birth cohorts as a measure of famine intensity.

discuss potential biases that may arise from migration and sample selection. I also test the thrifty phenotype hypothesis explicitly, by allowing for the manifestation of the effects of early childhood famine exposure to depend on later life economic environment. Lastly, I go beyond studying the long-term effect of famine exposure on adult obesity to testing the mechanisms behind the effect.

III Background and Conceptual Framework

One can think of individuals in this study as being exposed to two treatments: The first treatment being famine exposure during their prenatal and early childhood period, and the second treatment being exposure to energy-dense diets and sedentary lifestyles when the individuals are in their mid-twenties to mid-forties.

III.1 1959-61 China Famine

The China Famine began in the winter of 1959 and affected all regions of China. National grain output decreased by 15% in 1959 and by a further 16% in the following two years. The declining trend in grain production came to a halt in 1962, and only by 1966 had total grain production recovered to the 1958 output level (Li and Yang, 2005). The dotted line in Figure 1 shows that per capita food energy availability fell from over 2,100 calories per day in 1958 to about 1,500 calories in 1960. As comparison, the minimum dietary energy requirement for an individual is 1,900 calories per day (FAO, 2008).[10] The 1,500 number represents the national average for both rural and urban areas. In reality, food availability was much worse in rural areas than in urban areas (Chen and Zhou, 2007).[11] Historically, famine victims die either from starvation or from infectious diseases. Although little is known about the actual causes of deaths during 1959-61, popular accounts of the famine emphasize death by starvation rather than infectious diseases (O Grada, 2007). An estimated total of 16.5 to 30 million people died prematurely during the famine, making it the worst famine in human history (Li and Yang, 2005).

[10] The minimum dietary energy requirement (MDER) is defined as the amount of energy needed for light activity and for maintaining a minimum acceptable weight given attained height (FAO, 2008). The MDER varies by country and from year to year. 1,900 calories is the MDER for China for 2003-05.

[11] This is because of the government's preferential treatment towards urban residents through grain rationing and the maintenance of state-controlled stockpiles (Chen and Zhou, 2007).

It is generally believed that the radical economic policies of the Great Leap Forward were to be blamed for the famine (Lin, 1990; Li and Yang, 2005).[12] During the Great Leap Forward which began in 1958, the Chinese Communist government diverted agricultural resources to industry and imposed an excessive grain procurement burden on farmers. These policies together with bad weather, wasteful use of food supplies in communal kitchens, an urban bias in food distribution, and continued grain exports during the early years of the crisis all contributed to the famine (Ashton et al., 1984; Peng, 1987; Lin, 1990; Li and Yang, 2005).

Although almost all regions of China were affected by the famine, there was large regional variation in famine intensity, due to the variance in population density, exposure to bad weather, and provincial response to food shortage (Peng, 1987; Chen and Zhou, 2007). Appendix Figure A1 shows the geographic variation in famine intensity. We see that the central part of China, from Gansu in the north to Guangxi in the south, suffered the most, while provinces in the far north such as Heilongjiang and Jilin were the least affected. Figure 2 shows the variation in death rates across time and across provinces.[13] We see that the death rates in the pre-famine years (1954-58) and post-famine years (1962-66) are about 9 to 13 per thousand, and there is little variation across provinces. By contrast, in 1960, which is the peak year of the famine, the death rates vary greatly from 10.52 per thousand in Heilongjiang to 52.33 in Guizhou.

In the spirit of Almond et al. (2007) and Shi (2007), I use the province-level excess death rate (hereafter denoted as EDR) as a measure of famine intensity. The EDR is calculated as the crude death rate minus the average death rate during the pre-famine years of 1954 to 1958.[14] Appendix Table A1 shows the EDR by province by year. I assign EDR to individuals according to their province and year of birth. This means that an individual born in Guangxi in January 1960 is assigned the EDR of Guangxi in 1960, even though this individual also experienced famine *in utero* (in 1959) and in his/her second year of birth (in 1961).[15] EDR can thus be thought of as a proxy of the nutrition environment faced by an individual during his/her prenatal and early childhood period.

[12] The Great Leap Forward is the name given to China's Second Five Year Plan which was scheduled to run from 1958 to 1963, but actually ended in 1961 because of the famine and social catastrophe it triggered. The original aim of the Great Leap Forward is to transform China from a primarily agrarian economy to a modern Communist society through collectivization and industrialization (Yang, 2008).

[13] I present only the death rates for the nine provinces sampled in the China Health and Nutrition Survey (CHNS).

[14] As a robustness check, I use a second definition of excess death rate, calculated as the crude death rate minus the average death rate during the pre- and post-famine years (1954-58 and 1962-66 average). The regression results are essentially the same.

[15] Although I have month of birth information for individuals, there is a lack of data on the exact timing of famine in different provinces, making it difficult to strictly distinguish prenatal and postnatal effects of the famine.

III.2 China's economic growth and nutrition transition

Individuals born around the time of the famine were born to a very poor country. China's per capita real GDP in the 1950s is lower than that of India in the 1940s, and lower than that of almost all African countries in 1980 (Maddison, 2009). However, China has experienced rapid economic growth in the past two decades, with an annual growth rate of 8.2% from 1990 to 2006 (Maddison, 2009). Figure 3 shows a ten-fold increase in per capita real GDP from about 2,100 yuan (about US $300) in 1980 to over 22,000 yuan (about US $3,200) in 2008.

With rapid economic growth comes a drastic shift in food consumption patterns. Historically, the Chinese diet has been plant-based. Over the past two decades, the consumption of cereals and vegetables has decreased by more than 20%, while energy intake from animal sources has more than tripled (Wu, 2006; Li et al., 2008). Figure 4 shows the drastic increase of fat consumption from about 30 g per day in 1980 to almost 100 g in 2003.[16] Daily caloric intake increases from about 2,000 calories in the late 1970s to almost 3,000 calories in 2004. The increase in energy and fat intake is also accompanied by a decrease in transport-related physical activities and an increase in leisure time inactivity (Li et al., 2008).

Individuals who were born during the famine were in their mid-twenties when China started experiencing the nutrition transition. My study therefore focuses on the nutrition environment faced by individuals in their early childhood period and then in their early adult years.[17]

III.3 Mechanisms behind the effect of early childhood famine exposure on adult obesity

Conceptually, there are various possible mechanisms behind the effect of early childhood famine exposure on adult obesity. From a behavioral viewpoint, given that weight gain is a function of energy consumption and energy expenditure, famine cohorts may be more likely to be obese if famine exposure leads to increased caloric or fat intakes or decreased physical activities.[18] People born during the famine may have lower incomes and hence can afford

[16] As comparison, the recommended fat intake is 65 g per day for an adult man.
[17] Postnatal and adolescent years are also critical periods of catch-up growth and weight gain, and the effects of nutrition during those periods on adult obesity warrant further studies.
[18] High-fat, energy-dense diets and sedentary lifestyles have been shown to be strongly and positively associated with obesity (Popkin et al., 1995; Prentice and Jebb, 1995).

cheaper and fattier foods only. They may be less educated and therefore less health-conscious, paying less attention to the amount and kinds of food they eat and to the amount of exercise they engage in. Famine exposure may also lead to poorer health in general, so that people cannot be as active as they would like and are forced to engage in less physical activities. From a biological viewpoint, early childhood famine exposure may have affected the individual's metabolism or central endocrine regulatory mechanisms, so that given the same amount of energy intake and energy expenditure, the individual is still more likely to gain weight.[19] I test whether the effect of famine exposure on adult obesity is operating through these various channels, by first studying the effect of famine exposure on diet and exercise behaviors, and then by testing whether the effect of famine exposure on adult obesity is mediated by income, education, or diet and activity patterns.

IV Data

I use data from the China Health and Nutrition Survey (CHNS), which is a panel dataset with seven survey waves (1989, 1991, 1993, 1997, 2000, 2004, and 2006). The survey is designed to examine the effects of health, nutrition, and family planning policies in China, and it contains detailed information about household and individual economic, demographic, and social characteristics. I use data from the physical examination and nutrition modules in particular. Anthropometric measurements are taken by trained interviewers using standardized equipment. Dietary information is collected by trained nutritionists using a 24-hour recall method and verified by examining changes in household food inventory. These objective measurements help prevent self-reporting bias, especially in the case of height, weight, and food intake data.

The CHNS covers nine provinces in China: Guangxi, Guizhou, Heilongjiang, Henan, Hubei, Hunan, Jiangsu, Liaoning, and Shandong.[20] These provinces are highlighted by dark borders in Appendix Figure A1. They vary substantially in geography, economic development,

[19] The biological mechanisms underlying body weight regulation are not well understood. Studies have shown that a range of signaling mechanisms within the intestine, the adipose tissue and the brain are involved in maintaining the body's energy balance and hence body weight (WHO, 2000). Studies by Barker (1992) and Law et al. (1992) suggest that fetal and infant malnutrition leads to hormonal changes which in turn affect abdominal obesity, while Ravelli et al. (1976) suggest that metabolic tissues such as the hypothalamus are reprogrammed as a result of early-life malnutrition, leading to changes in appetite and weight control.

[20] Heilongjiang was introduced in the 1997 survey to replace Liaoning which was not able to participate. Since 2000, Liaoning has returned to the study.

public resources, and health indicators.[21] At the sub-provincial level, there are 54 counties and 235 communities in the CHNS. The primary sampling unit is the community, which refers to urban neighborhoods, suburban neighborhoods, towns, and rural villages.

I restrict my sample to individuals born between 1954 and 1966, which are five years before and after the famine. The narrow birth window helps reduce bias from confounding factors.[22] I also restrict my sample to individuals with a rural *hukuo* (household registration), since the famine affected mostly rural areas. Focusing on the rural sample also prevents confounding from the 1966-76 Cultural Revolution, which caused social and economic disruptions mainly in urban areas. My final sample consists of 1,674 households, 2,700 individuals, and 11,098 observations pooled from all seven survey waves. Each individual is observed an average of 4.1 times. Table 1 gives the summary statistics.

The main outcome variable is body mass index (BMI). BMI is the most common measure of obesity, and is defined as the weight in kilograms divided by the square of the height in meters.[23] An individual with a BMI less than 18.5 kg/m^2 is classified as underweight. An individual with a BMI of 25 kg/m^2 or above is considered overweight, and an individual with a BMI of 30 kg/m^2 or above is considered obese.[24] It is important, however, to note that the classification of obesity by BMI cutoffs is relatively arbitrary. Health risks are greatly increased above this level of fatness but it does not imply that individuals with BMIs below 25 or 30 kg/m^2 are free from such risks. In fact, the risks of associated morbidity and mortality begin at relatively low levels of BMI (WHO, 2000). I therefore focus on a continuous measure of BMI as the main outcome variable. I also use waist and hip circumferences as alternate measures of obesity. Waist circumference measures "central" obesity and is an approximate index of intra-

[21] Geographically, Jiangsu, Liaoning, and Shandong are coastal regions, while the other provinces are inland. In terms of economic development, Jiangsu, Liaoning, and Shandong are considered the richer provinces, while Guangxi and Guizhou are among the poorest (Chen and Zhou, 2007).

[22] As a robustness check, I further restrict my sample to those born between 1956 and 1964, which are three years before and after the famine. I get very similar regression results (available from author).

[23] For example, the average man in my sample weighs 61 kg and is 1.66 m tall, and hence has a BMI of 22.1 (61 / (1.66 * 1.66)) kg/m^2. A BMI of 21-23 kg/m^2 is thought to be optimal for adults, in the sense that mortality and morbidity risks are minimized in this range (WHO, 2000).

[24] These BMI cutoffs are independent of age and gender. The cutoff values of 25 and 30 kg/m^2 are derived from Caucasian populations. When applied to Asian populations, they underestimate the true risks of morbidity and mortality. The Working Group on Obesity in China has recommended cutoff values of 24 and 28 kg/m^2 for the Chinese population (Zhou et al., 2002). I use these Chinese cutoffs in my study.

abdominal fat mass and total body fat. Waist circumference is unrelated to height (unlike BMI) and is strongly correlated with cardiovascular risk and risks of other chronic diseases.[25]

V Empirical Strategy and Results

V.1 Effects of early childhood famine exposure on adult obesity

Before turning to regression results, it is helpful to look at a descriptive figure. Figure 5 shows the average BMI of men and women by year of birth and by famine severity. The solid lines denote BMIs for individuals born in severely affected regions, whose EDRs in 1960 exceed the national average. The dotted lines denote less affected regions. We see that for both men and women, the average BMIs in less affected regions are generally higher than the average BMIs in severely affected regions. In severely affected regions, the spike in average BMI for women during the famine years stands out in the figure. Men born in severely affected regions during 1959-62 also see an increase in BMI, though the increase is less pronounced than that for women.

To study the effects of early childhood famine exposure on adult obesity, I estimate the following equation:

$$(1) \qquad y_{ijct} = \alpha + \beta \cdot EDR_{jc} + \lambda_j + \mu_c + \Gamma \times X_{ijct} + \varepsilon_{ijct}$$

where y_{ijct} is the outcome variable for individual *i* born in province *j* in year *c* and observed in survey wave *t*. Famine exposure is captured by the excess death rate at the province and year of birth level (EDR_{jc}). β is the coefficient of interest. λ_j is a vector of province dummies and μ_c is a vector of year of birth dummies. X_{ijct} is a vector of individual characteristics, namely, age and ethnicity dummies.[26] Standard errors are clustered at the province-birth year level.

Given that an individual is observed multiple times and that the observations of each individual are clearly non-independent, I estimate equation (1) using generalized least squares (GLS) with individual random effects. As a robustness check, I also run OLS regressions using

[25] A waist circumference of 94 cm or above is considered moderate risk for men (the cutoff is 80 cm for women) and a waist circumference of 102 cm or above is considered severe risk for men (the cutoff is 88 cm for women) (WHO, 2000).

[26] I include age dummies in additional to year of birth dummies because an individual may be observed multiple times, that is, at different ages in the survey waves. Ethnicity dummies include Han Chinese majority and other ethnic minorities such as Miao, Buyi, Man, and Tujia.

cross-sectional data, that is, by using single survey waves.[27] I run all regressions separately for men and women.

Table 2 shows the baseline results. The outcome variables are different measures of obesity: BMI, waist circumference, hip circumference, and the probability of being overweight or obese. There are no statistically significant effects for men. Women, on the other hand, experience positive and significant effects of famine exposure on all measures of obesity. To interpret the magnitudes of the coefficients, I multiply the coefficients by 13.26, which is the mean EDR in 1960, the peak year of the famine.[28] For women, early childhood famine exposure leads to an average increase in BMI of 0.84 kg/m^2 (3.7%), increases in waist and hip circumferences of 1.57 cm (2.0%) and 1.74 cm (1.9%), and increases in the probability of being overweight and obese by 14.6 percentage points and 5.3 percentage points.[29]

Very similar results are obtained in the cross-sectional analysis, as shown in columns (3) to (9) in Table 3. Apart from being a robustness check, the regression estimates also suggest that the effect of early childhood famine exposure decreases with age.[30] In the 1989 survey, individuals in my sample were 23 to 35 years old, with an average age of 29. By the 2006 survey, they were 40 to 52 years old, with an average age of 46. The estimated effect of early childhood famine exposure on BMI for women decreases from 1.21 kg/m^2 (0.091 * 13.26) in the 1989 cross-section to 0.90 kg/m^2 (0.068 * 13.26) in the 2006 cross-section, although the difference is not statistically significant. I repeat the analysis restricting my sample to individuals who are observed in all seven survey waves and get very similar results.[31]

One may wonder whether the effect of early childhood famine exposure is uniform along the BMI distribution, or whether it is concentrated on underweight, normal weight, or overweight individuals. Welfare implications can be quite different if the BMI increase is observed mostly

[27] As discussed below, the two methods give very similar results. I choose to use multiple waves in order to maximize the number of observations.

[28] 13.26 per thousand is the population-weighted average EDR for the nine provinces in my sample. The national average EDR in 1960 is 13.50.

[29] In subsequent tables, I only report the effects of early childhood famine exposure on BMI, as similar patterns are observed for all five measures of obesity.

[30] *A priori*, one may expect the effects of early childhood famine exposure to remain constant, or to dissipate or worsen as one ages. As discussed in Section II, studies of the Dutch Hunger Winter suggest that the effect of famine exposure may be mitigated over time. On the other hand, there are studies showing that the effect of early childhood malnutrition on chronic disease markers is amplified with age (Law et al., 1993).

[31] One may worry that the over-time decrease in the effect of early childhood famine exposure is due to differential attrition. For example, if the least healthy (or the most obese) famine cohorts are more likely to attrite, then they are no longer observed in the later survey waves, and the estimated average effect of famine exposure will decrease. I treat attrition as a dependent variable and regress the probability of attrition on EDR. I find no statistically significant relationship between the two.

for underweight individuals as opposed to overweight persons, since an increase in BMI may actually be a good thing for underweight individuals. I run quantile regressions to study the differential effects of early childhood famine exposure at various BMI percentiles. Figure 6 and Appendix Table A2 show that famine exposure does lead to an increase in BMI of 0.52 to 0.69 kg/m^2 (2.4% to 3.1%) for normal weight men at the 50^{th} and 60^{th} percentiles (with BMIs of about 22 kg/m^2). For women, the effect of famine exposure is felt by almost all except the underweight individuals.[32] The effect of famine exposure increases along the BMI distribution, from an increase in BMI of 0.37 kg/m^2 (1.8%) at the 20^{th} percentile (normal weight women with BMIs of about 20 kg/m^2) to an increase of 1.13 kg/m^2 (4.0%) at the 95^{th} percentile (obese women with BMIs over 28 kg/m^2).

V.2 Robustness Checks

Migration

Ideally, EDRs should be assigned according to individuals' province of birth. The CHNS, however, reports only the province of residence for all individuals. An identifying assumption therefore is that province of residence is a good proxy for province of birth. To estimate the potential bias underlying this assumption, I make use of the fact that the CHNS does collect province of birth information, though only for household heads and their spouse, who constitute 46% of my sample.[33] In my sample, 2.9% of rural household heads and 7.3% of the spouses are migrants, that is, they report a province of birth different from their province of residence.[34] Inter-province migration is uncorrelated with famine exposure.[35] Column (2) of Table 4 shows that when I use province of birth information where available to assign EDR, I get very similar results as before.

I also restrict my sample to those who first appear in the CHNS in the early survey waves 1989, 1991, and 1993, since migration in China was limited up till the early 1990s.[36] Such individuals constitute 74% of my sample. The coefficient estimate for women as shown in

[32] Underweight individuals are those with a BMI less than 18.5 kg/m^2, which is at about the 5^{th} percentile and below.
[33] The province of birth information has not been used in other China famine papers that use the CHNS data.
[34] The majority of migrants are from Shandong (31%), Liaoning (20%), and Jilin (17%). About 77% of migrants end up in Heilongjiang.
[35] The correlation between migrant status and EDR is -0.03. A regression of migrant status on EDR gives a coefficient of 0.0009 (insignificant).
[36] China's *hukuo* system is very successful at restricting migration before the 1990s, and especially so for rural residents (Chen and Zhou, 2007). Under the *hukuo* system, migrants have no access to government-controlled food rations, housing, schools, and medical care if they leave their registered hometowns (Meng and Qian, 2009).

column (3) of Table 4 is statistically significant and slightly larger than the baseline estimate in column (1).

Sample selection

Another potential concern is selection bias. There may be selective fertility and selective mortality as a result of the famine. Figure 1 shows that the birth rate decreases sharply during the famine, from 34 per thousand in 1957 to a historic low of 18 per thousand in 1961. The birth rate rebounds immediately after the famine, reaching a rate even higher than that in pre-famine years, suggesting delayed births. Selective fertility is a concern if it is women with more fat reserves who can conceive and give birth during the famine. If women with more fat reserves are of a particular physiological or metabolic type, and this in turn is inherited by their children, then the children born during the famine are more likely to be obese, regardless of whether they have been malnourished *in utero* or in early childhood.

Ideally, I want to control for the mothers' BMI or weight at pregnancy. I do not have this retrospective information. The CHNS also does not link adults to their parents, unless the parents are still living in the same household as the adults and hence are observed in the household roster, though this is the case for only 7.6% of the adults. Therefore I cannot control for the parents' current BMI or weight either. The best alternative is to control for the parents' survival as of the survey year, information that I do have for ever-married women in the CHNS sample.[37] Parents' survival serves as a crude proxy for the parents' health status or health endowments that were presumably passed onto the individual. Column (4) of Table 4 shows that the effect of early childhood famine exposure is essentially unchanged when one controls for parents' survival.

Another concern is the selection into childbearing and into survival based on socioeconomic status (SES). If richer parents are the ones more likely to give birth during the famine years, they may also be more likely or more able to provide more food to their children right after the famine as compensation, so that it is the rapid catch-up growth in the first few years of life that leads to adult obesity, and not because of early childhood malnutrition *per se*. I argue that selection into fertility based on SES is less of a concern in the China context. As pointed out by Chen and Zhou (2007), China in the 1950s had just gone through a series of land reform and agricultural collectivization, so that there was no private ownership of land and other

[37] In my sample, 70.6% of mothers and 56.9% of fathers of the 1954-66 female cohorts are still alive at the time of survey.

productive assets, and SES was almost equalized within and across communes, especially in the rural areas.

Other robustness checks

As further robustness checks, I include additional control variables to the baseline equation (1). I control for whether the individual has a household member who is currently an official cadre. Since households with members who are official cadres are more likely to have political power and hence preferential access to food during the famine, these households may suffer less from famine exposure. I do not have retrospective information on official cadre membership in the 1950s, so I use the contemporary measure as a proxy.[38] I also control for the number of children (or number of pregnancies) in the regressions for women, since having gone through pregnancies may affect one's BMI. I get very similar results as the baseline estimate (available from author).

I perform another robustness check by restricting my sample to the 1959-66 birth cohorts. In the baseline specification, the pre-famine cohorts (those born in 1954-58) are assigned EDRs of the pre-famine years and are thus treated as a control group, but in reality, they experienced the famine when they were two to seven years old. By restricting my sample to the 1959-66 birth cohorts, I use only the post-famine cohorts (those born in 1962-66) as the control group. Column (5) of Table 4 reports a slightly smaller but still strongly significant coefficient on EDR.

I also run my analysis on individuals with an urban *hukuo*. Urban residents had favorable food entitlements during the famine years and hence were not subject to severe starvation. However, given rural-urban migration in recent years, some of the current urban residents may not have been born in cities. Column (6) of Table 4 shows that there is a positive effect of famine exposure on BMI for urban women, although it is significant only at the 10% level. The slightly larger coefficient relative to the baseline estimate for rural women in column (1) suggests that the effect of famine exposure may be amplified for individuals who were exposed to famine when young but are now living in an urban environment with access to energy-dense foods and with a more sedentary lifestyle. I test this explicitly in Section V.3.

Other health outcomes

[38] 7.4% of individuals in my sample have a household member who is currently an official cadre.

Apart from obesity, I also look at other health outcomes, namely, self-reported health status, whether the individual has been diagnosed with high blood pressure, and the measured systolic and diastolic blood pressure. One reason we care about obesity is because it is a key risk factor for chronic diseases such as cardiovascular diseases and type 2 diabetes. One would like to study mortality and different types of morbidity directly, but individuals who were born during the famine are less than 50 years old now, and most of the chronic diseases do not hit until a later age.

I restrict my sample to those aged 45 and above, and find no significant effects of early childhood famine exposure on self-reported health status or blood pressure (results in Appendix Table A3).[39] This is not surprising, given that the proportion of men and women diagnosed with high blood pressure in my sample is only 3% and 7%. It will be worthwhile to revisit this and to study mortality outcomes in 10 or 20 years time.

V.3 Interaction between early childhood famine exposure and adult life economic environment

Having established the effect of early childhood famine exposure on adult obesity, I now test whether the long-term effect of famine exposure is greater for the famine cohorts who are exposed to a food-rich environment in adult life than for the famine cohorts who are not. I estimate the following equation:

(2) $$y_{ikct} = \alpha + \beta \cdot EDR_{jc} + \phi \cdot income_{kt} + \gamma \cdot EDR_{jc} * income_{kt} + \lambda_j + \mu_c + \Gamma \times X_{ikct} + \varepsilon_{ikct}$$

The main difference between equations (1) and (2) is the introduction of the interaction term: $EDR_{jc} * income_{kt}$. The coefficient of interest is now γ. $Income_{kt}$ is the demeaned community-level per capita household income for community *k* in survey year *t*.[40] It is calculated by first averaging the per capita household incomes of all households in community *k* and in survey year *t*. The community-level income is then demeaned by subtracting the average income for all communities in that survey year. $Income_{kt}$ ranges from -6,899 yuan to 24,807 yuan in my sample, with a mean of -615 yuan.

The community-level income captures the extent to which an individual is exposed as an adult to an environment of energy-dense foods and sedentary lifestyles. I use the community-

[39] The oldest individuals in my sample are only 52 years old (those born in 1954 and observed in the 2006 survey wave).
[40] As described in Section IV, there are 235 communities sampled in the CHNS.

level income instead of the individual's own income because the latter is endogenous. More importantly, community-level income is not correlated with famine intensity, as shown by the flat fitted line in Appendix Figure A2, which plots community-level income in 1989 against EDR in 1960.

Table 5 shows the regression results for equation (2). We see that the coefficients on the interaction term between EDR and community-level income are not statistically significantly different from zero, with the exception of waist and hip circumference outcomes for men.[41] The effect of community-level income itself is generally positive, as what one might expect in developing countries where the prevalence of obesity is positively associated with income. The richer one's community is, the more likely one has access to richer diets and the more likely one adopts a sedentary lifestyle, hence increasing the likelihood of obesity.

The community-level income as constructed above is based on the per capita household income data from the CHNS. Given that the CHNS samples only an average of 27 households per community (while in reality there are on average 1,286 households per community), one may be concerned about measurement error. I use a set of community characteristics such as geographic features and community infrastructure to predict the community-level income for each community in each survey year, and re-estimate equation (2) using this predicted income measure.[42]

Results are shown in Appendix Table A4. Columns (1) and (4) show that the coefficients on the interaction term between EDR and predicted community-level income are statistically insignificant for both men and women and for both BMI and waist circumference outcomes. Similar results are found when I use province-level per capita GDP as of the survey year as an alternate measure of adult life economic environment.[43] Finally, I use community-level caloric intake as a direct proxy of food availability in adult years, and also find no significant effect for the interaction term between EDR and food availability. In sum, I find no evidence that the

[41] To interpret the magnitude of the coefficient for waist circumference, I note that a difference in community-level income of 2,895 yuan exists between communities at the 25th and 75th percentiles of the income distribution. From column (2) of Table 5, we see that famine exposure leads to an average increase in waist circumference of 1.47 cm (0.111 * 13.26) for men. For famine-exposed men, living in a community with an average per capita household income at the 75th as opposed to 25th percentile leads to a further increase in waist circumference of 0.99 cm (0.259 * 13.26 * 0.2895). Given that the mean waist circumference is 79.1 cm for men, 0.99 cm represents a 1.3% increase.

[42] The set of community characteristics includes, but is not limited to, whether the community is near a navigable river or near an open trade area; availability of telephone service, postal service, daily newspaper; presence of and distance to bus stops, train stations, public baths; whether it is an old revolutionary base or a minority village; distance from provincial capital; most common kind of local roads, etc.

[43] Province-level per capita GDP is obtained from China's National Bureau of Statistics. I use the province-level measure as a robustness check since it is measured at a broader geographic level and can help minimize endogeneity.

increase in adult obesity is differentially greater for the famine cohorts who are exposed to a food-rich environment in adult life than for the famine cohorts who are not.

V.4 Mechanisms behind the effect of early childhood famine exposure on adult obesity

I now turn to investigate the possible mechanisms underlying the association between early childhood famine exposure and adult obesity. First, from a purely mechanistic perspective, BMI is determined by a person's height and weight. The question then is whether the increase in BMI is due to a decrease in height or an increase in weight or both. From column (1) in Table 6, we see that the coefficients on EDR for height for both men and women are negative but not statistically significant. This is consistent with the findings of Gorgens et al. (2007), who find no effect of famine exposure on height but argue that the negative stunting effect of famine is offset by the positive effect of selective survival. Column (2) shows that most of the action on BMI comes through weight. For women, famine exposure leads to an average increase in weight of 1.58 kg (0.119 * 13.26), which is about a 2.9% increase (1.58 / 54.8).

Is the observed increase in weight due to higher caloric or fat intake? Studies have shown that weight changes attributable to diet composition are primarily due to disruptions in fat balance rather than carbohydrate or protein balance (Flatt, 1988; Horton et al., 1995). Hence fat and total caloric intakes matter more for obesity than carbohydrate and protein intakes.[44] Columns (3) and (4) in Table 6 show that famine exposure is not associated with higher dietary energy or fat intake. If anything, famine-exposed men have on average a lower caloric intake of 93 calories (-0.007 * 13.26 * 1000) per day. Other health behaviors which may affect body weight, such as smoking and drinking, are also not associated with famine exposure (see columns (7) and (8)).

If there is no observed difference in food intake between famine and non-famine cohorts, is there a difference in the amount of physical activities? Columns (9) to (11) show that there are no statistically significant associations between famine exposure and time spent on various kinds of physical activities. Television watching, which is frequently used as a proxy of inactivity, is also not associated with famine exposure (see column (12)).

[44] Excess dietary fat is readily stored in adipose tissue depots with a high efficiency of 96%. By contrast, the body has limited storage capacity for protein or glycogen (WHO, 2000).

As a final way to test whether the effect of early childhood famine exposure on adult obesity is mediated by diet and activity patterns, or by income and education, I re-estimate equation (1) controlling for dietary intake, physical activities, individual's educational attainment and per capita household income. Table 7 shows that the coefficients on EDR are almost unchanged. Given the same amount of caloric intake and energy expenditure, famine cohorts still have a higher adult BMI than non-famine cohorts. A biological rather than a behavioral mechanism appears to underlie the association between early childhood malnutrition and adult obesity. [45]

VI Conclusion

In this paper, I study the health outcomes and health behaviors of adult men and women who were born during the 1959-61 China Famine. I find significant effects of early childhood famine exposure on adult obesity for women but not for men. There may be different reasons behind this gender difference. Animal studies have shown that early-life malnutrition affects body weight and fat cell size differently in male and female animals, suggesting that sex hormones modulate the effects of early-life malnutrition (Jones and Friedman, 1982). Ravelli et al. (1999), observing a gender difference in the results of their Dutch Hunger Winter study, suggest that a biological mechanism might be at work.[46]

The gender difference in obesity outcomes may also be due to differential mortality by gender. Mu and Zhang (2008) show that male infant mortality exceeded female infant mortality during the China famine.[47] If it is the healthier and better nourished male infants who were born during the famine and who were able to survive, while the severely malnourished male infants simply died *in utero* or in their early years of life, it may explain why we do not observe significant effects of famine exposure for adult men.[48]

[45] Some biological factors thought to affect the development of obesity are insulin sensitivity, leptin action, growth hormone status, adipose tissue and muscle lipoprotein lipase activity, as well as capacities for fat and carbohydrate oxidation (WHO, 2000). The relationship between these biological factors and other environmental factors (including the nutrition environment in early childhood) remains an active area of research.

[46] Based on the gender difference in obesity outcomes, Ravelli et al. (1999) suggest that increased obesity after famine exposure in early gestation is due to altered functions of the central endocrine regulatory mechanisms rather than to abnormalities of adipocytes.

[47] Mu and Zhang (2008) point out that the male-to-female sex ratio for the pre-famine cohorts in rural China is about 109, and it decreases substantially to 100.3 for the 1961 birth cohort.

[48] One can also tell a gender bias story, where boys and girls were treated differently during and right after the famine. If boys were given preferential access to food and hence were less severely malnourished than girls, this

I also test the thrifty phenotype hypothesis and find no evidence that the increase in BMI is differentially greater for the famine cohorts who are exposed to a food-rich environment in adult life than for the famine cohorts who are not. Using detailed individual-level data on food intake and physical activities, I show that the increase in BMI for famine-exposed women is not due to higher caloric or fat intakes nor to more sedentary lifestyles.

The findings in this paper suggest that improving maternal and infant nutrition can reap long-term benefits in terms of reducing the obesity burden in the long run. Maternal and infant malnutrition are still very prevalent in the developing world today, yet there are few serious public health initiatives (for example, improvement of infant weaning foods) to reduce maternal and infant malnutrition in most developing countries (Popkin et al., 1996).

To interpret the magnitudes of the results in this paper, it is worthwhile to do a back-of-the-envelope calculation. From Table 2, we see that early childhood famine exposure leads to increases in the probability of being overweight and obese for women by 14.6 percentage points and 5.3 percentage points. Given that there are about 7 million preschool children in China who are suffering from undernutrition (NSS, 2005), and assuming that 3.3 million of them are girls (given a sex ratio of 1.11 at birth) and assuming that the effect of early childhood malnutrition found in my study is generalizable to this population, we can expect about 480,000 (3.3 million * 0.146) of these malnourished children who otherwise would be normal weight to become overweight when they grow up and 170,000 (3.3 million * 0.053) of them to become obese.[49]

What kind of health and economic burden does this translate to? The total healthcare costs due to overweight and obesity in China have been estimated at 13.0 billion yuan and 8.1 billion yuan (Zhao et al., 2008). Dividing by the total number of individuals who are overweight or obese, we obtain average healthcare costs of 45 yuan (13,000 / 290) per overweight individual and 90 yuan (8,100 / 90) per obese individual.[50] Multiplying these average costs by the number of overweight and obese individuals calculated above, we get that early childhood malnutrition leads to a total healthcare cost attributable to adult overweight and obesity of about 37 million yuan (45 * 0.48 + 90 * 0.17). This does not even include indirect economic costs of obesity nor other health and economic costs of early childhood malnutrition.

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may explain the gender difference in outcomes. However, given the higher male mortality rates during the famine, such a gender bias story seems unlikely.

[49] Here, I have assumed that the degree of undernutrition for the 3.3 million preschool children is comparable to the undernutrition faced by the 1959-61 famine cohorts, who had on average 1,500 calories per day.

[50] According to China's 2002 National Nutrition and Health Survey, 22.8% of Chinese (about 290 million) are overweight and 7.1% (about 90 million) are obese (Zhao et al., 2008).

In addition to making the case for improving maternal and infant nutrition, another policy implication of this study is that individuals who were malnourished at birth should be one of the target populations of obesity prevention and reduction efforts. Understanding that some individuals are more susceptible to the development of obesity than others will help inform and target the fight against the global obesity epidemic.

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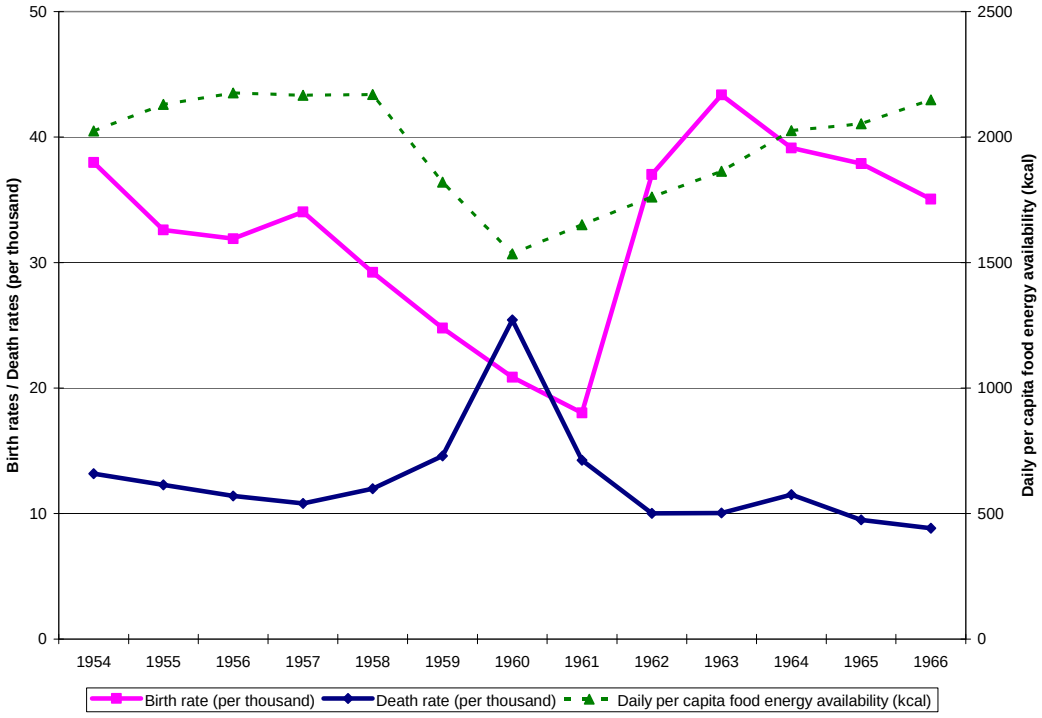
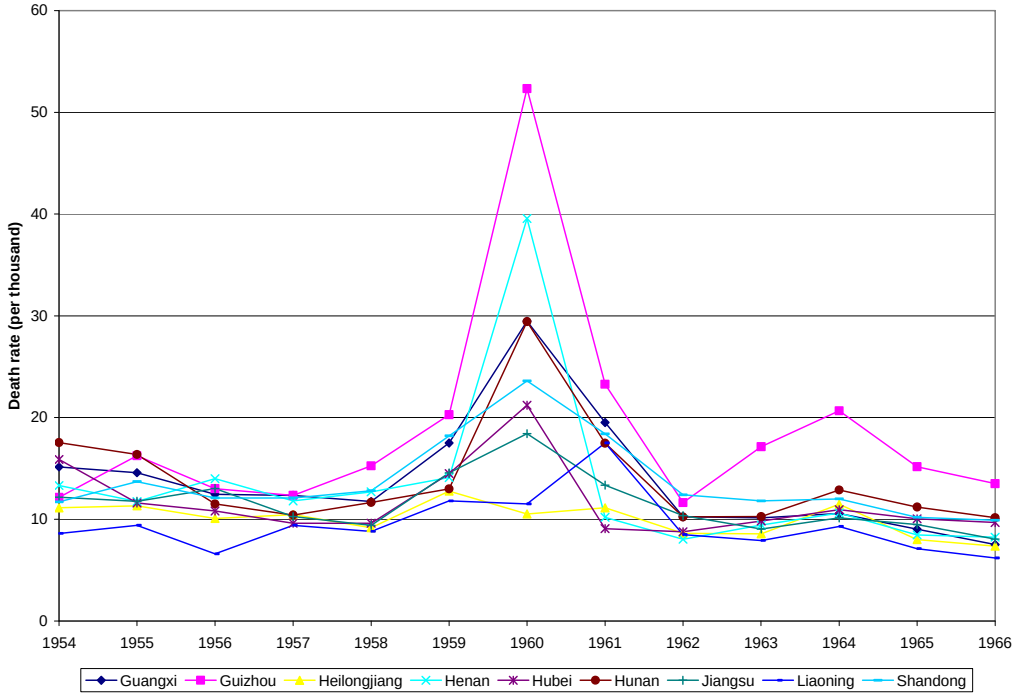
FIGURE 1: Birth Rates, Death Rates, and Food Energy Availability in China, 1954-1966



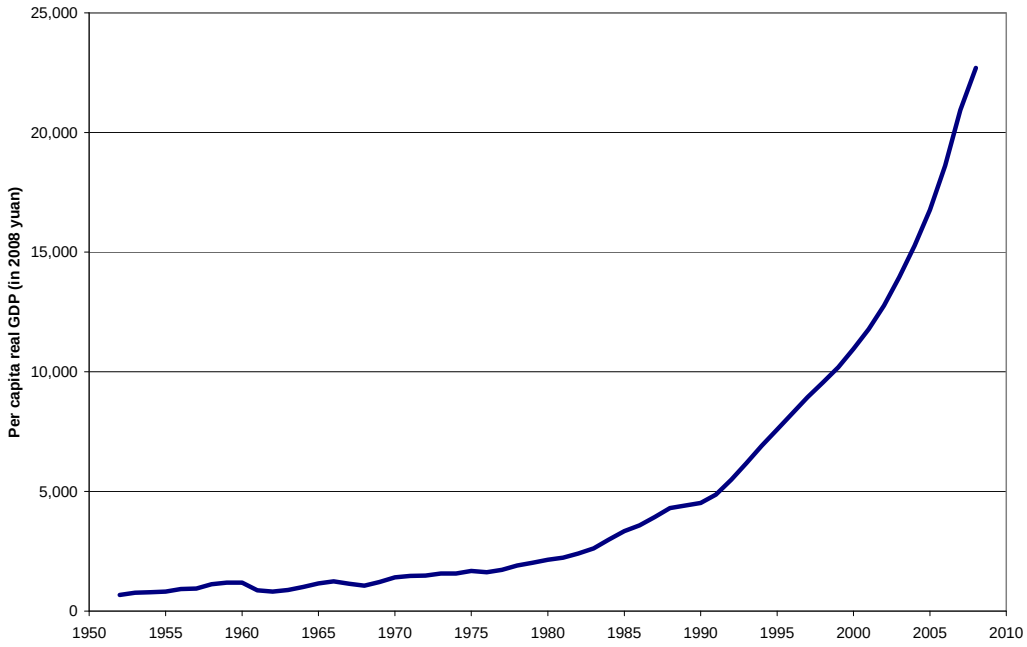
Source: Birth rates and death rates are from *China Statistical Yearbooks*. Daily per capita food energy data is from Table 5 in Ashton et al. (1984) and Table 3 in Wang et al. (1993).

FIGURE 2: Death Rates by Province by Year, 1954-1966



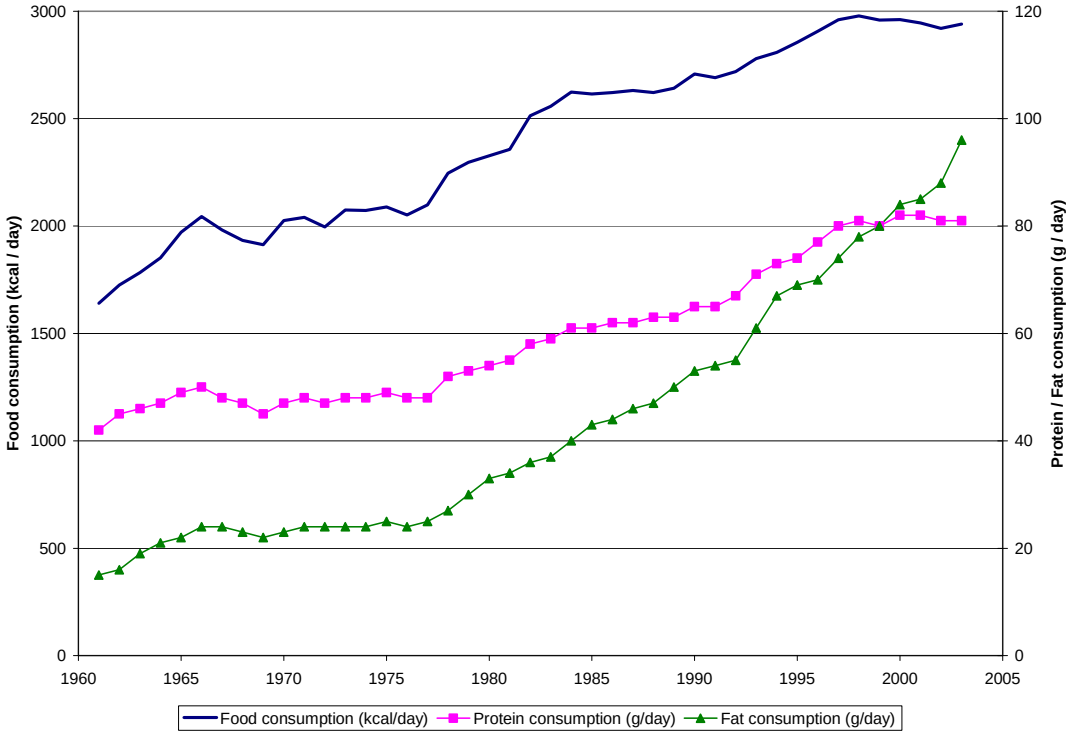
Source: *China Statistical Yearbooks*. See also Appendix Table A1.

FIGURE 3: Per Capita Real GDP in China, 1952-2008



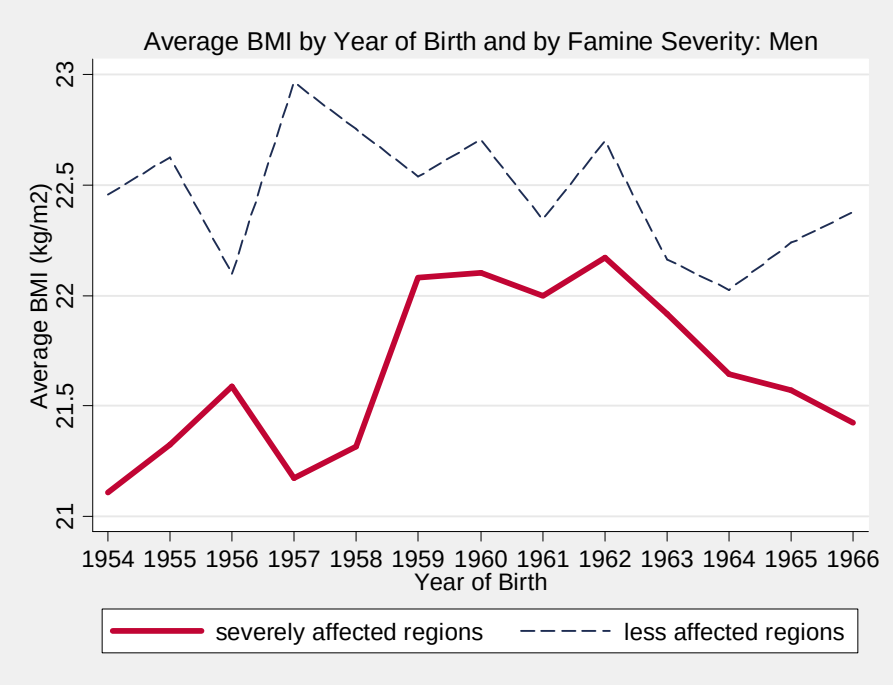
Source: *China Statistical Yearbooks*.

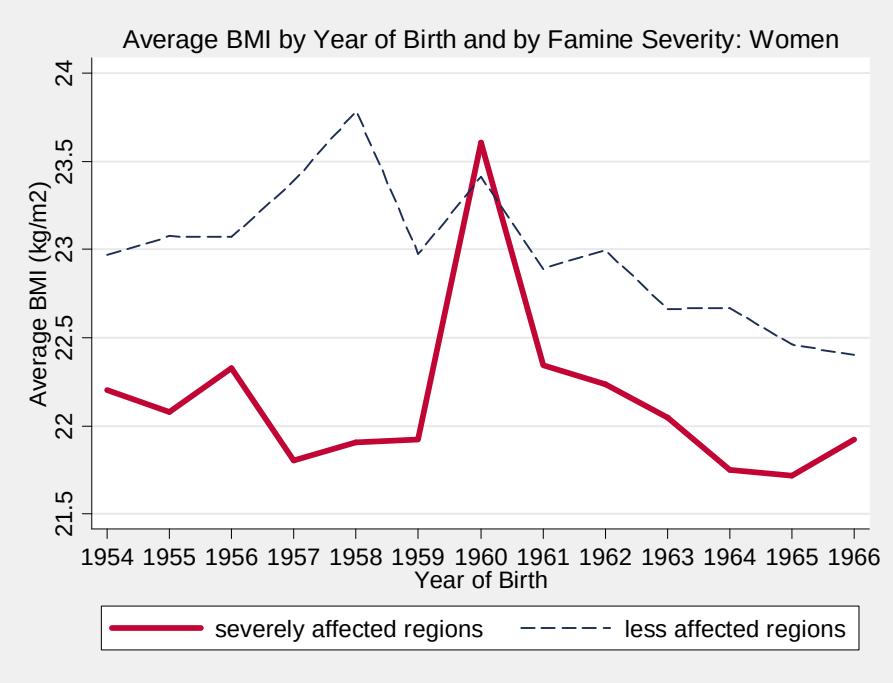
FIGURE 4: Dietary Intake in China, 1961-2003



Source: Food and Agriculture Organization (FAO) of the United Nations.

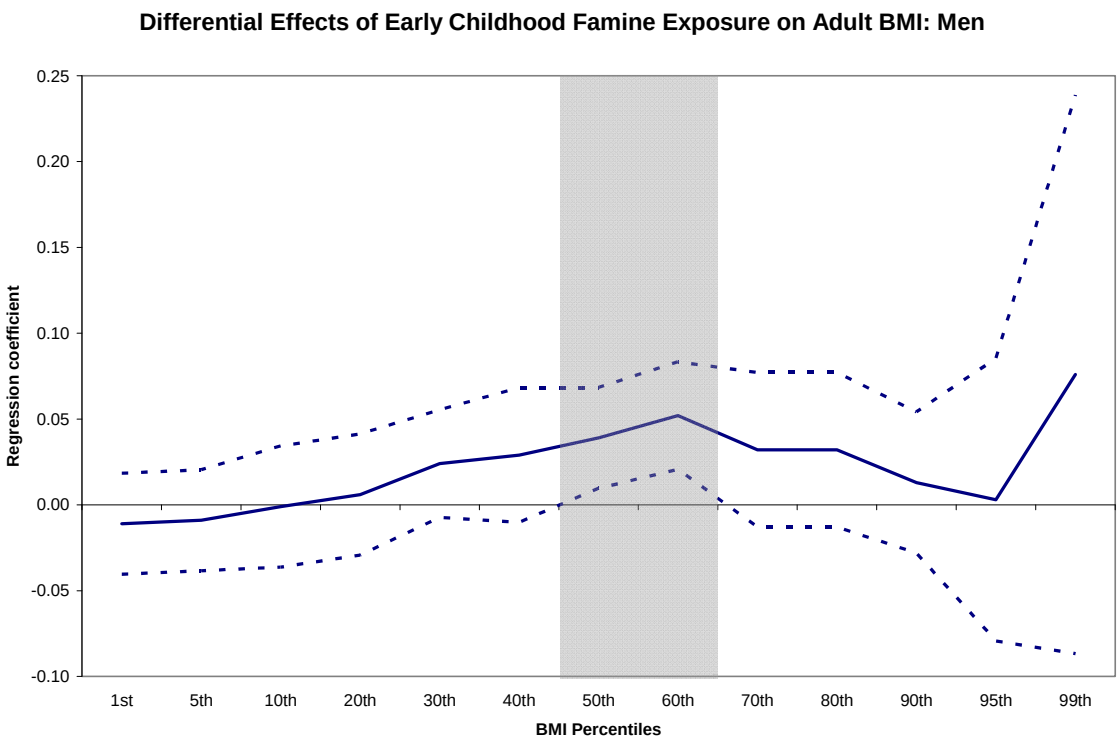
FIGURE 5: Average BMI by Year of Birth and by Famine Severity

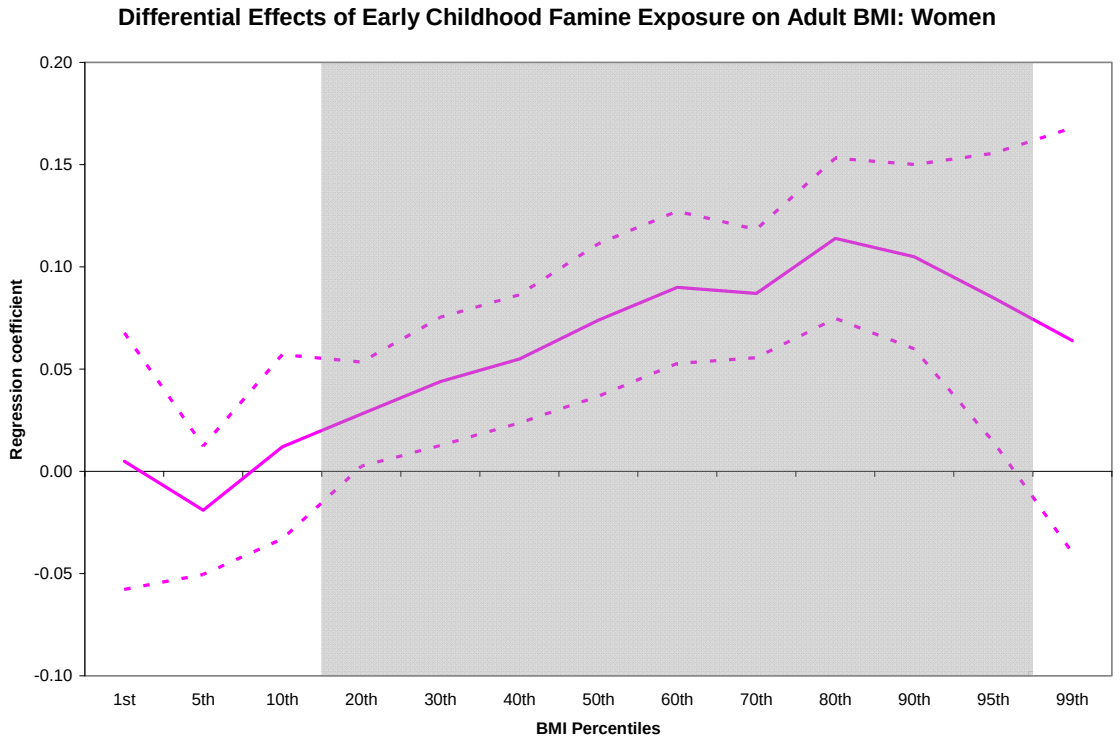




Note: The severely affected regions are Guangxi, Guizhou, Henan, and Hunan, whose excess death rates in 1960 are greater than the national average excess death rate. The less affected regions are Heilongjiang, Hubei, Jiangsu, Liaoning, and Shandong. It is important to note that the sample size is relatively small for each birth cohort in the China Health and Nutrition Survey. For example, there are 146 observations for 1959-born rural women in severely affected regions and 202 observations for 1959-born rural women in less affected regions.

FIGURE 6: Quantile Regression Results





Note: Regression coefficients are given in Appendix Table A2. The dotted lines denote the 95% confidence intervals. The shaded regions indicate that the regression coefficient is statistically significant at or above the 5% level.

TABLE 1: Summary Statistics

	Men		Women	
	Mean	(Std. Dev.)	Mean	(Std. Dev.)
Demographics				
Age	37.5	(7.1)	37.8	(7.1)
Famine cohort (born in 1959-61)	0.15	(0.36)	0.16	(0.37)
Physical characteristics				
BMI (kg/m^2)	22.1	(2.7)	22.6	(3.0)
Waist circumference (cm)	79.1	(10.2)	77.1	(9.9)
Hip circumference (cm)	91.1	(9.1)	92.0	(9.2)
Weight (kg)	61.0	(8.9)	54.8	(8.5)
Height (cm)	166.0	(6.0)	155.6	(5.6)
Dietary intake				
Calories (kcal/day)	2849	(889)	2477	(802)
Fat (g/day)	70.1	(38.5)	64.1	(35.7)
Carbohydrates (g/day)	451.0	(152.6)	398.9	(142.6)
Protein (g/day)	79.5	(26.7)	69.3	(23.9)
Ever smoked cigarettes (1=yes, 0=no)	0.70	(0.46)	0.03	(0.17)
Drank beer/alcohol last year (1=yes, 0=no)	0.68	(0.47)	0.09	(0.29)
Activity level				
Light physical activities (hours/week)	7.32	(14.87)	10.05	(16.56)
Moderate physical activities (hours/week)	10.70	(17.86)	7.63	(13.44)
Heavy physical activities (hours/week)	25.94	(22.84)	22.62	(20.79)
Watching television (hours/week)	4.29	(2.96)	4.32	(3.10)
Education and income				
Years of schooling	7.96	(2.89)	5.84	(3.76)
Primary school completion	0.90	(0.30)	0.70	(0.46)
Middle school completion	0.21	(0.41)	0.11	(0.32)
Per capita household income (in 2006 yuan)	4456	(5568)	4512	(5196)
Number of individuals	1292		1408	
Number of observations	5011		6087	

Note: Sample includes individuals with a rural *hukuo* born between 1954 and 1966. Observations are pooled from seven CHNS survey waves (1989-2006). Famine cohort is a dummy variable for being born in the years 1959-61. Physical activities refer to the activities during work time. Examples of jobs involving light physical activities are office work, watch smith, counter salesperson, and laboratory technician. Examples of jobs involving moderate physical activities are driver and electrician. Examples of jobs involving heavy physical activities are farmer, athlete, dancer, steel worker, lumber worker, and mason.

TABLE 2: Effects of Early Childhood Famine Exposure on Adult Obesity

Dependent Variables:	BMI	Waist circumference	Hip circumference	Overweight	Obese
	(1)	(2)	(3)	(4)	(5)
Panel A: Men					
Excess death rate (EDR)	0.026	0.089	-0.045	0.002	-0.000
	[0.021]	[0.068]	[0.049]	[0.003]	[0.001]
Mean of dependent variable	22.07 kg/m^2	79.13 cm	91.08 cm	0.205	0.030
Std. dev. of dependent variable	(2.67)	(10.16)	(9.06)	(0.404)	(0.170)
Estimated effect of famine exposure	0.35 kg/m^2	1.18 cm	-0.60 cm	0.027	0.000
Number of individuals	1288	1259	1259	1288	1288
Number of observations	5005	3546	3543	5005	5005
Panel B: Women					
Excess death rate (EDR)	0.063***	0.118*	0.131***	0.011***	0.004***
	[0.020]	[0.060]	[0.042]	[0.003]	[0.001]
Mean of dependent variable	22.59 kg/m^2	77.13 cm	92.04 cm	0.279	0.054
Std. dev. of dependent variable	(3.02)	(9.87)	(9.15)	(0.448)	(0.226)
Estimated effect of famine exposure	0.84 kg/m^2	1.57 cm	1.74 cm	0.146	0.053
Number of individuals	1403	1380	1380	1403	1403
Number of observations	6079	4360	4361	6079	6079

Note: Sample includes individuals with a rural *hukuo* born between 1954 and 1966. Overweight is a dummy variable that equals to one if BMI is greater than or equal to 24 kg/m^2. Obese is a dummy variable that equals to one if BMI is greater than or equal to 28 kg/m^2. All regressions include age and ethnicity dummies, as well as year of birth and province fixed effects. An individual random effects model is used, with robust standard errors clustered at the province-birth year level. * significant at 10%; ** significant at 5%; *** significant at 1%. Estimated effects of famine exposure are calculated by multiplying the coefficients on EDR by the mean EDR in 1960 (13.26 per thousand).

TABLE 3: Cross-Sectional Analysis of the Effects of Early Childhood Famine Exposure on Adult Obesity

	Dependent Variable: BMI								
	Baseline specification	Not including individual random effects	1989 cross-section	1991 cross-section	1993 cross-section	1997 cross-section	2000 cross-section	2004 cross-section	2006 cross-section
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Panel A: Men									
Excess death rate (EDR)	0.026	0.026	0.036*	0.023	0.016	0.055	-0.003	0.016	0.002
	[0.021]	[0.021]	[0.020]	[0.028]	[0.025]	[0.035]	[0.023]	[0.044]	[0.039]
Number of individuals	1288	1288	702	702	693	677	772	727	732
Number of observations	5005	5005	702	702	693	677	772	727	732
Panel B: Women									
Excess death rate (EDR)	0.063***	0.063***	0.091***	0.080*	0.087*	0.061**	0.060**	0.043	0.068*
	[0.020]	[0.020]	[0.030]	[0.046]	[0.044]	[0.025]	[0.024]	[0.034]	[0.035]
Number of individuals	1403	1403	794	847	868	827	927	895	921
Number of observations	6079	6079	794	847	868	827	927	895	921

Note: Sample includes individuals with a rural *hukuo* born between 1954 and 1966. All regressions include ethnicity dummies as well as year of birth and province fixed effects. Age dummies are also included in columns (1) and (2), which report results using the pooled CHNS sample. Column (1) estimates an individual random effects model, with robust standard errors clustered at the province-birth year level. Column (2) does not include individual random effects. Columns (3) to (9) report results using single survey waves, with standard errors clustered at the province-birth year level. * significant at 10%; ** significant at 5%; *** significant at 1%.

TABLE 4: Robustness Checks

	Dependent Variable: BMI					
	Baseline specification	Use province of birth	Restrict to 1989-1993 survey waves	Control for parents' survival	Restrict to 1959-1966 cohorts	Urban sample
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Men						
Excess death rate (EDR)	0.026	0.025	0.010	-	0.013	-0.016
	[0.021]	[0.021]	[0.026]	-	[0.022]	[0.022]
Number of individuals	1288	1275	950		787	1161
Number of observations	5005	4977	4205		2954	3508
Panel B: Women						
Excess death rate (EDR)	0.063***	0.064***	0.083**	0.062***	0.059***	0.072*
	[0.020]	[0.020]	[0.040]	[0.021]	[0.018]	[0.037]
Mother alive				-0.007		
				[0.094]		
Father alive				-0.076		
				[0.083]		
Number of individuals	1403	1389	1039	1384	845	1191
Number of observations	6079	6037	5147	5972	3540	3713

Note: Sample includes individuals with a rural *hukuo* born between 1954 and 1966. Column (2) uses province of birth information (where available) to assign excess death rates. Column (3) restricts sample to individuals who were first observed in the early survey waves, 1989, 1991, and 1993. Column (4) controls for whether the individual's parents are still alive, information that is available for ever-married women only. Column (5) restricts sample to those born between 1959 and 1966, and column (6) analyzes individuals with an urban *hukuo*. All regressions include age and ethnicity dummies, as well as year of birth and province fixed effects. An individual random effects model is used, with robust standard errors clustered at the province-birth year level. * significant at 10%; ** significant at 5%; *** significant at 1%.

TABLE 5: Interaction between Early Childhood Famine Exposure and Adult Life Economic Environment

Dependent Variables:	BMI	Waist circumference	Hip circumference	Overweight	Obese
	(1)	(2)	(3)	(4)	(5)
Panel A: Men					
Excess death rate (EDR)	0.022	0.111*	-0.030	0.002	-0.000
	[0.020]	[0.057]	[0.044]	[0.003]	[0.001]
Community-level income	-0.006	1.688**	0.643	0.070**	-0.005
	[0.132]	[0.660]	[0.654]	[0.030]	[0.013]
EDR * Community-level income	0.014	0.259**	0.203**	0.002	0.002
	[0.037]	[0.107]	[0.085]	[0.005]	[0.002]
Number of individuals	1281	1255	1255	1281	1281
Number of observations	4924	3501	3498	4924	4924
Panel B: Women					
Excess death rate (EDR)	0.064***	0.120**	0.134***	0.011***	0.004***
	[0.020]	[0.059]	[0.049]	[0.003]	[0.001]
Community-level income	0.264*	0.541	0.911*	0.065**	0.029**
	[0.153]	[0.553]	[0.525]	[0.028]	[0.015]
EDR * Community-level income	0.007	0.046	0.078	-0.001	-0.001
	[0.050]	[0.167]	[0.196]	[0.008]	[0.004]
Number of individuals	1401	1379	1379	1401	1401
Number of observations	6079	4360	4361	6079	6079

Note: Sample includes individuals with a rural *hukuo* born between 1954 and 1966. Community-level income is demeaned by the average community-level income for all communities in that survey year. It is measured in units of 10,000 yuan. All regressions include age and ethnicity dummies, as well as year of birth and province fixed effects. An individual random effects model is used, with robust standard errors clustered at the province-birth year level. * significant at 10%; ** significant at 5%; *** significant at 1%.

TABLE 6: Early Childhood Famine Exposure and Adult Health Behaviors

	Physical Characteristics		Diet, Smoking, and Drinking						Physical Activities			
Dependent Variables:	Height	Weight	Calories	Fat	Carbo-hydrates	Protein	Ever Smoked	Drank Alcohol	Light	Moderate	Heavy	Television Watching
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Panel A: Men												
Excess death rate (EDR)	-0.036	0.046	-0.007**	0.032	-0.017*	-0.006	-0.003	-0.002	0.064	0.088	-0.054	-0.019
	[0.052]	[0.071]	[0.003]	[0.021]	[0.010]	[0.010]	[0.003]	[0.004]	[0.137]	[0.135]	[0.198]	[0.026]
Mean of dep. variable	166 cm	61.0 kg	2847 kcal	69.9 g	451 g	79.5 g	0.70	0.69	7.3 hrs	10.6 hrs	25.9 hrs	4.3 hrs
Number of individuals	1288	1288	1297	1297	1295	1297	1302	1302	971	955	978	924
Number of observations	5008	5007	5079	5059	5013	5053	4576	4558	1414	1394	1451	1459
Panel B: Women												
Excess death rate (EDR)	-0.043	0.119**	0.004	0.001	0.010**	0.005	0.000	-0.000	-0.004	0.200	-0.076	0.003
	[0.047]	[0.061]	[0.003]	[0.018]	[0.004]	[0.011]	[0.001]	[0.002]	[0.151]	[0.147]	[0.190]	[0.024]
Mean of dep. variable	156 cm	54.8 kg	2473 kcal	63.9 g	399 g	69.2 g	0.03	0.09	10.0 hrs	7.6 hrs	22.4 hrs	4.3 hrs
Number of individuals	1403	1403	1413	1413	1413	1413	1412	1411	1053	1039	1057	1062
Number of observations	6085	6082	6155	6129	6130	6140	5421	5390	1605	1581	1645	1746

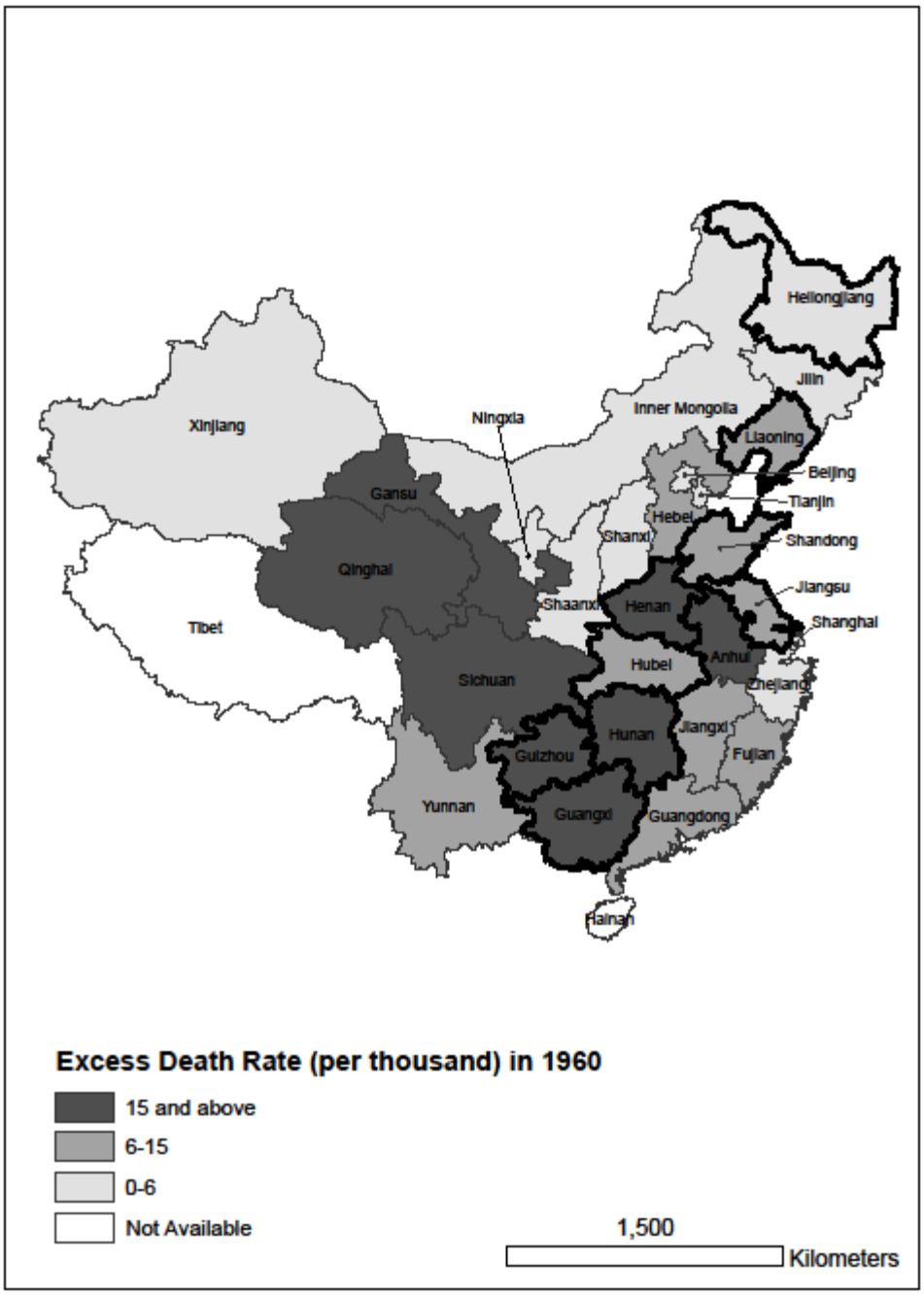
Note: Sample includes individuals with a rural *hukuo* born between 1954 and 1966. Calories are measured in units of 1,000 kcal. Fat and protein are measured in units of 10 g. Carbohydrates are measured in units of 100 g. The dependent variable in column (7) is a dummy variable that equals to one if the individual has ever smoked cigarettes. The dependent variable in column (8) is a dummy variable that equals to one if the individual drank beer or alcoholic beverages in the year before the survey. In columns (9) to (11), jobs that involve light physical activities include salesperson, laboratory technician, and teacher; jobs that involve moderate physical activities include student, driver, electrician, and metal worker; jobs that involve heavy physical activities include farmer, dancer, steel worker, and athlete. Physical activities and television watching are measured in hours per week. All regressions include age and ethnicity dummies, as well as year of birth and province fixed effects. An individual random effects model is used, with robust standard errors clustered at the province-birth year level. * significant at 10%; ** significant at 5%; *** significant at 1%.

TABLE 7: Exploring Mechanisms behind the Effect of Early Childhood Famine Exposure on Adult Obesity

Women only	***Dependent Variable: BMI***							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Excess death rate (EDR)	0.063***	0.062***	0.062***	0.061*	0.068**	0.067***	0.063***	0.062**
	[0.020]	[0.020]	[0.020]	[0.033]	[0.030]	[0.021]	[0.020]	[0.030]
Calories		0.055*						-0.575
		[0.032]						[0.822]
Fat			0.008					0.068
			[0.007]					[0.076]
Carbohydrates			-0.047*					0.287
			[0.025]					[0.324]
Protein			0.029**					0.024
			[0.014]					[0.047]
Ever smoked				-0.591				-1.132**
				[0.447]				[0.536]
Drank alcohol				-0.033				-0.042
				[0.166]				[0.188]
Light physical activity					0.247			0.204
					[0.204]			[0.217]
Moderate physical activity					0.071			0.025
					[0.193]			[0.205]
Heavy physical activity					-0.199			-0.299
					[0.188]			[0.199]
Television watching					0.026			0.030
					[0.018]			[0.019]
Years of schooling						0.010		-0.004
						[0.015]		[0.023]
Per capita household income							0.016***	0.012*
							[0.006]	[0.006]
Number of individuals	1403	1402	1402	1062	1025	1347	1401	1008
Number of observations	6079	5991	5934	1809	1663	5916	6011	1622

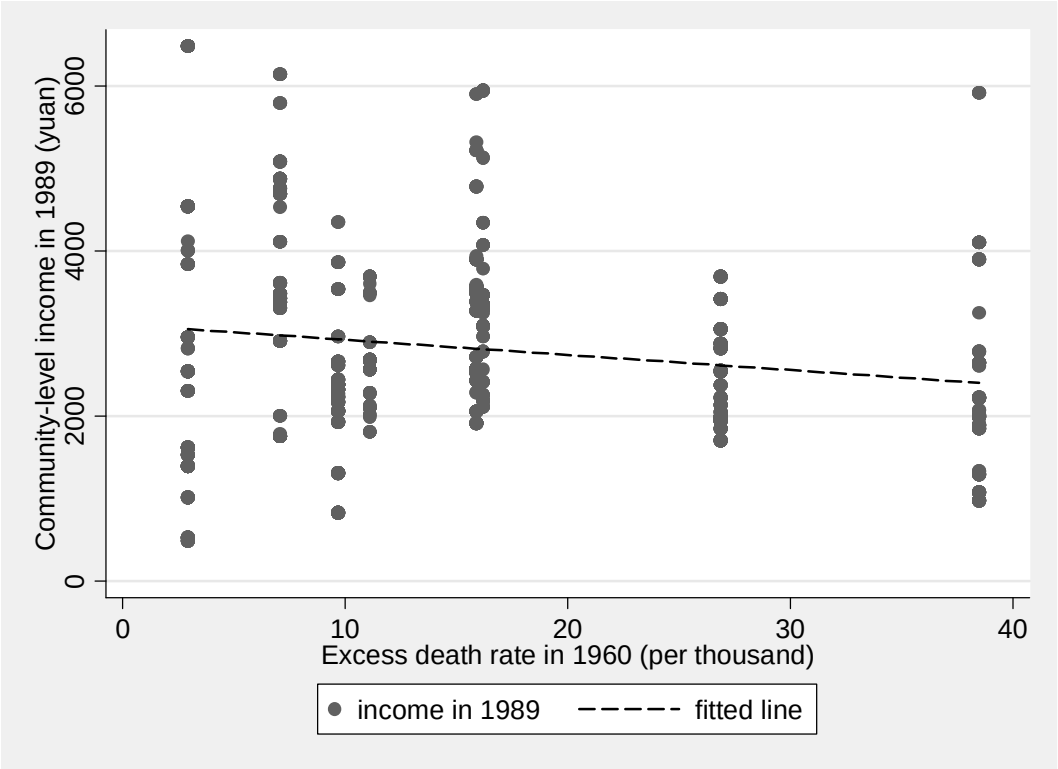
Note: Sample includes individuals with a rural *hukuo* born between 1954 and 1966. The dietary and physical activity variables are defined as in Table 6. All regressions include age and ethnicity dummies, as well as year of birth and province fixed effects. An individual random effects model is used, with robust standard errors clustered at the province-birth year level. * significant at 10%; ** significant at 5%; *** significant at 1%.

APPENDIX FIGURE A1: Excess Death Rates in China at Peak of Famine



Note: The excess death rates in 1960 are calculated by subtracting the average crude death rates in the pre-famine years of 1954-58 from the crude death rates in 1960. The nine provinces with dark borders are the provinces sampled in the China Health and Nutrition Survey.

APPENDIX FIGURE A2: Community-level Income in 1989 and Excess Death Rate in 1960



Note: Excess death rate in 1960 is measured at the province level. There are eight provinces in the 1989 China Health and Nutrition Survey. The community-level income is the average per capita household income for each community. The dotted line is from a linear regression of community-level income on excess death rate.

APPENDIX TABLE A1: Crude and Excess Death Rates in Select Provinces, 1954-1966

Province	**1954**	**1955**	**1956**	**1957**	**1958**	**1959**	**1960**	**1961**	**1962**	**1963**	**1964**	**1965**	**1966**	**1954-58 Average**
	Crude death rates													
Guangxi	15.15	14.58	12.46	12.35	11.74	17.49	29.46	19.50	10.25	10.13	10.55	9.03	7.50	13.26
Guizhou	12.15	16.24	13.01	12.35	15.26	20.28	52.33	23.27	11.64	17.14	20.66	15.16	13.49	13.80
Heilongjiang	11.12	11.33	10.08	10.45	9.17	12.76	10.52	11.13	8.62	8.56	11.47	8.00	7.36	10.43
Henan	13.32	11.75	14.00	11.80	12.69	14.10	39.56	10.20	8.04	9.43	10.61	8.45	8.24	12.71
Hubei	15.87	11.60	10.81	9.61	9.60	14.49	21.21	9.08	8.77	9.83	10.94	10.04	9.68	11.50
Hunan	17.54	16.36	11.51	10.41	11.65	12.99	29.42	17.48	10.23	10.26	12.88	11.19	10.15	13.49
Jiangsu	12.16	11.76	13.02	10.26	9.40	14.55	18.41	13.35	10.36	9.04	10.13	9.48	8.05	11.32
Liaoning	8.60	9.40	6.60	9.40	8.80	11.80	11.50	17.50	8.50	7.90	9.30	7.10	6.20	8.56
Shandong	11.70	13.70	12.10	12.10	12.80	18.20	23.60	18.40	12.40	11.80	12.00	10.20	9.90	12.48
Nation	*13.18*	*12.28*	*11.40*	*10.80*	*11.98*	*14.59*	*25.43*	*14.24*	*10.02*	*10.04*	*11.50*	*9.50*	*8.83*	*11.93*
	Excess death rates (Crude death rates minus 1954-58 average)													
Guangxi	1.89	1.32	-0.80	-0.91	-1.52	4.23	16.20	6.24	-3.01	-3.13	-2.71	-4.23	-5.76	
Guizhou	-1.65	2.44	-0.79	-1.45	1.46	6.48	38.53	9.47	-2.16	3.34	6.86	1.36	-0.31	
Heilongjiang	0.69	0.90	-0.35	0.02	-1.26	2.33	0.09	0.70	-1.81	-1.87	1.04	-2.43	-3.07	
Henan	0.61	-0.96	1.29	-0.91	-0.02	1.39	26.85	-2.51	-4.67	-3.28	-2.10	-4.26	-4.47	
Hubei	4.37	0.10	-0.69	-1.89	-1.90	2.99	9.71	-2.42	-2.73	-1.67	-0.56	-1.46	-1.82	
Hunan	4.05	2.87	-1.98	-3.08	-1.84	-0.50	15.93	3.99	-3.26	-3.23	-0.61	-2.30	-3.34	
Jiangsu	0.84	0.44	1.70	-1.06	-1.92	3.23	7.09	2.03	-0.96	-2.28	-1.19	-1.84	-3.27	
Liaoning	0.04	0.84	-1.96	0.84	0.24	3.24	2.94	8.94	-0.06	-0.66	0.74	-1.46	-2.36	
Shandong	-0.78	1.22	-0.38	-0.38	0.32	5.72	11.12	5.92	-0.08	-0.68	-0.48	-2.28	-2.58	
Nation	*1.25*	*0.35*	*-0.53*	*-1.13*	*0.05*	*2.66*	*13.50*	*2.31*	*-1.91*	*-1.89*	*-0.43*	*-2.43*	*-3.10*	

Note: Death rates taken from *China Statistical Yearbooks*. The shaded columns denote famine years.

APPENDIX TABLE A2: Quantile Regressions of Early Childhood Famine Exposure and Adult Obesity

	Dependent Variable: BMI												
BMI Percentiles:	1st	5th	10th	20th	30th	40th	50th	60th	70th	80th	90th	95th	99th
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
Panel A: Men													
Excess death rate (EDR)	-0.011	-0.009	-0.001	0.006	0.024	0.029	0.039***	0.052***	0.032	0.032	0.013	0.003	0.076
	[0.015]	[0.015]	[0.018]	[0.018]	[0.016]	[0.020]	[0.015]	[0.016]	[0.023]	[0.023]	[0.021]	[0.042]	[0.083]
BMI (kg/m^2)	17.50	18.48	19.05	19.87	20.48	21.08	21.70	22.32	23.03	24.04	25.61	27.08	30.10
Estimated effect of famine exposure	-0.15	-0.12	-0.01	0.08	0.32	0.38	0.52	0.69	0.42	0.42	0.17	0.04	1.01
No. of individuals	1288	1288	1288	1288	1288	1288	1288	1288	1288	1288	1288	1288	1288
No. of observations	5005	5005	5005	5005	5005	5005	5005	5005	5005	5005	5005	5005	5005
Panel B: Women													
Excess death rate (EDR)	0.005	-0.019	0.012	0.028**	0.044***	0.055***	0.074***	0.090***	0.087***	0.114***	0.105***	0.085**	0.064
	[0.032]	[0.016]	[0.023]	[0.013]	[0.016]	[0.016]	[0.019]	[0.019]	[0.016]	[0.020]	[0.023]	[0.036]	[0.053]
BMI (kg/m^2)	17.36	18.42	19.07	20.05	20.79	21.48	22.19	22.93	23.78	24.92	26.67	28.21	31.62
Estimated effect of famine exposure	0.07	-0.25	0.16	0.37	0.58	0.73	0.98	1.19	1.15	1.51	1.39	1.13	0.85
No. of individuals	1403	1403	1403	1403	1403	1403	1403	1403	1403	1403	1403	1403	1403
No. of observations	6079	6079	6079	6079	6079	6079	6079	6079	6079	6079	6079	6079	6079

Note: Sample includes individuals with a rural *hukuo* born between 1954 and 1966. All regressions include age and ethnicity dummies, as well as year of birth and province fixed effects. Bootstrap standard errors are reported in parentheses. * significant at 10%; ** significant at 5%; *** significant at 1%. Estimated effects of famine exposure are calculated by multiplying the coefficients on EDR by the mean EDR in 1960 (13.26 per thousand).

APPENDIX TABLE A3: Early Childhood Famine Exposure and Other Health Outcomes

Dependent Variables:	Self-reported health status	Diagnosed with high blood pressure	Systolic blood pressure	Diastolic blood pressure
	(1)	(2)	(3)	(4)
Panel A: Men (aged 45 and above)				
Excess death rate (EDR)	0.009	0.001	0.116	-0.062
	[0.010]	[0.002]	[0.111]	[0.098]
Mean of dependent variable	2.23	0.03	120.1 mmHg	79.8 mmHg
Number of individuals	523	523	506	506
Number of observations	901	901	862	862
Panel B: Women (aged 45 and above)				
Excess death rate (EDR)	-0.004	-0.003	0.279	0.130
	[0.006]	[0.002]	[0.179]	[0.128]
Mean of dependent variable	2.41	0.07	119.0 mmHg	77.3 mmHg
Number of individuals	601	600	590	590
Number of observations	1106	1102	1070	1070

Note: Sample includes individuals with a rural *hukuo* born between 1954 and 1966. Sample is further restricted to those aged 45 and above. Self-reported health status ranges from 1 (excellent) to 4 (poor). The dependent variable in column (2) is a dummy variable that equals to one if the individual has ever been diagnosed with high blood pressure. All regressions include age and ethnicity dummies, as well as year of birth and province fixed effects. An individual random effects model is used, with robust standard errors clustered at the province-birth year level. * significant at 10%; ** significant at 5%; *** significant at 1%.

APPENDIX TABLE A4: Robustness Checks for the Interaction between Early Childhood Famine Exposure and Adult Life Economic Environment

Dependent Variables:	***BMI***			***Waist circumference***		
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Men						
Excess death rate (EDR)	0.024	0.020	0.002	0.092	0.099	-0.097
	[0.019]	[0.019]	[0.058]	[0.068]	[0.081]	[0.239]
Y	0.204	0.095	0.012	4.984***	-2.165***	-0.133**
	[0.201]	[0.121]	[0.009]	[1.152]	[0.833]	[0.054]
EDR * Y	0.033	0.014	0.001	0.101	-0.020	0.008
	[0.059]	[0.026]	[0.002]	[0.180]	[0.080]	[0.010]
Where Y is:	Predicted community-level income	Province-level per capita GDP	Community-level caloric intake	Predicted community-level income	Province-level per capita GDP	Community-level caloric intake
Number of individuals	1281	1288	1284	1255	1259	1253
Number of observations	4924	5005	4866	3501	3546	3493
Panel B: Women						
Excess death rate (EDR)	0.064***	0.064***	0.033	0.114*	0.110*	0.058
	[0.021]	[0.021]	[0.037]	[0.060]	[0.066]	[0.200]
Y	0.488**	0.350***	0.016**	3.211***	-0.987*	-0.090
	[0.223]	[0.102]	[0.007]	[0.872]	[0.578]	[0.059]
EDR * Y	0.016	-0.002	0.001	0.030	0.016	0.002
	[0.055]	[0.014]	[0.001]	[0.181]	[0.064]	[0.009]
Where Y is:	Predicted community-level income	Province-level per capita GDP	Community-level caloric intake	Predicted community-level income	Province-level per capita GDP	Community-level caloric intake
Number of individuals	1401	1403	1402	1379	1380	1379
Number of observations	6020	6079	5993	4316	4360	4333

Note: Sample includes individuals with a rural *hukuo* born between 1954 and 1966. Predicted community-level income and province-level per capita GDP are measured in units of 10,000 yuan. Community-level caloric intake is measured in units of 100 calories. All regressions include age and ethnicity dummies, as well as year of birth and province fixed effects. An individual random effects model is used, with robust standard errors clustered at the province-birth year level. * significant at 10%; ** significant at 5%; *** significant at 1%.