Famine exposure and adult height: disentangling stunting from survival selection

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Abstract

We estimated the stunting effect of famine exposure on the adult height of female survivors of the Chinese famine of 1959-61, using variations in famine exposure across cohorts and regions to construct a difference-in-difference estimator. A methodological challenge was that individuals who survive severe famine may be more robust and have greater potential for growth in height than those without famine exposure, and this selection may mask the stunting effect. To disentangle the stunting from the selection effect, we adopted a quantile regression approach and adjusted for the birth size of babies born to these women as a proxy of potential for growth in height of the women. We found that famine exposure caused a substantial stunting effect among women who were born around or before the onset of the Chinese famine. We also found that estimates at the 90th percentile of adult height always exhibited a larger stunting effect than those at the 10th percentile. Adjustment for offspring birth size magnified the stunting effect estimates at the 10th percentile but not at the 90th percentile, which suggested that attenuation bias caused by selection for survival occurred mainly at the lower quantile of adult height. Therefore, estimating the impact on the upper quantile of adult height with quantile regression may serve as a better approach to capture the potential stunting effect than conventional mean regression.

Keywords: Famine, stunting, survival selection, sample selection

1. Introduction

Since Barker and his colleagues proposed the "fetal origins hypothesis," which states that malnutrition during the "critical" period of life in utero may permanently increase the risk of chronic diseases in adulthood (Barker 1993; 1997; Barker and Clark 1997; Barker et al. 2002), increasing effort has been devoted to studying the potential causality between early-life nutrition and adult health and human development. In particular, recent research into the long-term consequences of external nutritional shock in early life has shed new light on early origins of adult outcomes. For example, based on the 1960-80 decennial U.S. census data, Almond (2006) found that, compared to surrounding birth cohorts, American cohorts with prenatal exposure to the 1918 influenza pandemic typically displayed lower socioeconomic status (SES) and increased rates of physical disability as well as accelerated adult mortality; the magnitude of the impact of pandemic exposure was also positively associated with pandemic severity. The adverse long-term impact of prenatal exposure to the 1918 Influenza Pandemic was confirmed by a similar study in Brazil (Nelson 2009). Furthermore, using rainfall as an external determinant of agricultural output, Maccini and Yang (2009) linked historical rainfall across cohorts and regions with individual adult outcomes and found that higher early-life rainfall led to improved social achievement and better adult health for women in rural Indonesia. This pattern was interpreted to mean that higher rainfall boosted agricultural output and improved food availability, which consequently improved nutrition status among infants. Female infants in particular benefitted from improved agricultural output in this society with a preference for sons, which in turn positively impacted their adult health and human development.

Famines have been widely used as a quasi-experimental method to study the early origins of health and human development, most notably the Dutch Famine.[1] Extensive evidence has shown that children in utero during the Dutch famine were smaller in body size at birth and had a higher risk of diabetes, obesity, and cardiovascular diseases as well as lower human capital in their adulthood (Elias et al. 2005; Lumey and Van Poppel 1994; Roseboom et al. 2000; Stein et al. 1975).

Another quasi-experimental event, the Chinese famine of 1959-61 (hereafter abbreviated Chinese famine), provides a context to study the social and health impacts of early-life famine on survivors but has rarely been studied until recently due to a lack of publicly available data. Compared with the Dutch famine, the Chinese famine was more severe, lasted longer, and exposed several birth cohorts to extreme nutritional shock in utero and/or in infancy, creating a unique opportunity to study the consequences of early-life malnutrition in an Eastern, developing country exposed to different famine conditions (Huang et al 2010). Using the famine as a unique experiment, recent studies have found that early-life exposure to the Chinese famine was associated with stunted growth (Chen and Zhou 2007; Gorgens, Meng, and Vaithianathan 2007; Meng and Qian 2009; Huang et al 2010); higher risk of adult schizophrenia (Clair et al 2005); reduced labor supply, income, and educational achievement (Almond et al 2007; Chen and Zhou 2007; Meng and Qian 2009; Shi 2008); as well as less attractiveness in the marriage market

[1] The Dutch famine occurred during the last 5-6 months of World War II, when the Germans placed an embargo on all food transports to the western Netherlands. Food stocks rapidly depleted in the cities of the western Netherlands, where adult rations dropped to 580 kilocalories by the end of February 1945. The Dutch famine ended in May 1945, when the Allies liberated the western Netherlands. About 18,000 people died during the famine; approximately 10,000 deaths were attributed to malnutrition (Stein et al 1975).

(Brandt, Siow, and Vogel 2008). Furthermore, adult female survivors with famine exposure in utero or during infancy were more likely to experience stillbirths and miscarriages than other women (Cai and Wang 2005).

For a typical study that aims to quantify the long-term impact of famine exposure in early life, a major methodological challenge surrounds sample selection caused by survival selection. It is likely that famine survivors represent a selected group of robust individuals because fragile individuals are more susceptible to the life-threatening effects of hunger and malnutrition during the famine. Therefore, even though famine exposure has adverse long-term impacts on adult health, famine survivors may not necessarily exhibit worse health conditions than regular cohorts without famine exposure. In particular, this is likely to be the case when famine caused very high mortality and severe survival selection, leaving only very robust individuals surviving. In such a scenario, the selection effect may substantially mask the adverse impact of famine exposure. This attenuation bias may explain the lack of correlation between early-life famine exposure and adult health among survivors of the Finish famine of 1866-68[2] and the Siege of Leningrad[3], both of which were accompanied by widespread death by starvation. In a recent paper, Bozzoli, Deaton, and Quintana-Domeque (2009) reviewed evidence about stunting and selection from studies including those on the Chinese famine and developed a model in which adult height is a

[2] The 1866-68 Famine was caused by a series of poor harvests and a tardy and insufficient public policy reaction (Lefgren 1973). Over 100,000 people died from the Famine, out of a total population of 1.8 million. In 1868, deaths peaked at 77.6 per thousand, almost three times the death rate in normal years.

[3] During the siege of Leningrad, German troops prevented supplies from reaching Leningrad from 8 September 1941 to 27 January 1944, resulting in severe food shortage and 63,000 deaths from hunger-related causes out of a population of 2.9 million residents (Sparen et al 2004).

function of both the "scarring" effect and selection. When mortality is sufficiently high, selection can dominate scarring, leaving an even taller population of survivors.

Despite longstanding awareness that survival selection may attenuate estimates of long-term impact of famine exposure, few researchers have addressed this methodological concern. Meng and Qian (2009) adopted a quantile regression approach and estimated the impact of famine exposure at the upper percentiles of the outcome distributions. The rationale is that survivors typically come from the top of the distribution of important characteristics, such as height, and those from the lower parts of the distribution are at greater risk of death. Therefore, by applying quantile regression and estimating the famine impact at upper quantiles instead of the conventional mean regression carried out by ordinary least squares (OLS), the bias due to selection is partially eliminated (Meng and Qian, 2009). Gorgens, Meng, and Vaithiannathan (2007) disentangled the stunting effect from selection effect by using information conveyed by the height of children born to famine survivors. They argued that genetic determinants of survival, not exposure to famine, are more likely to be transmitted to children; therefore, selection bias can be mitigated by controlling for attributes of children, such as potential height, that can stand as a proxy of the parents' genotype (Gorgens, Meng, and Vaithiannathan 2007).

In this study, we used data from a large sample of Chinese women born from the late 1950s to the mid 60s across a wide area to examine the effect of early-life famine exposure on adult height. We used the variation in famine exposure across both birth cohorts and regions to construct a difference-in-difference estimator, which is similar to the analytic strategy of Chen and Zhou (2007). We further addressed the potential selection bias by combining the approaches of quantile regression (Meng and Qian 2009) and "genotype" adjustment (Gorgens, Meng, and

Vaithiannathan 2007). Results from the mean regression with the difference-in-difference estimator suggest a significant stunting effect for the 1959 birth cohort, whose members would otherwise be 1.5 centimeters taller on average in the absence of famine. Results from quantile regression suggested that the stunting effect estimated at the 90^{th} percentile of adult height is larger than that estimated at the 10^{th} percentile; among those in the 1959 birth cohort, the famine impact estimated at the 90^{th} percentile is a 3.4 centimeter reduction in adult height, compared to a reduction of 0.21 centimeters estimated at the 10^{th} percentile. Similarly, among the pre-famine birth cohorts (1956-1958 combined), the stunting effect estimated at the 90^{th} percentile is a 3.5 centimeter reduction in adult height, compared to a reduction of 0.21 centimeters estimated at the 10^{th} percentile. Controlling for birth weight and length of children born to mothers in the famine cohorts did not alter estimates at the 90^{th} percentile but substantially altered estimates at the 10^{th} percentile. For example, for the 1959 birth cohort, the stunting effect estimated at the 10^{th} percentile with adjustment for the birth size of children was 2.73 centimeters, compared to 0.21 centimeters, the stunting effect estimated at the 10^{th} percentile without adjustment for the birth size of children.

Our study contributes to the literature in several major ways: First, it provides more reliable estimates of the potential stunting effect of exposure to the Chinese famine compared with previous relevant studies by utilizing a larger sample and adjusting for birth size of children that may serve a better proxy of genotype of parents than height of children at different ages. Most previous studies were based on data from the China Health and Nutrition Survey (CHNS), which included a very small sample of famine cohorts; for example, only 66 individuals from the 1961 cohort were included in the rural sample of CHNS across eight sampled provinces (Chen and Zhou 2007). To pursue the data analysis at the county level, Meng and Qian (2009)

combined the cohorts into categories, including prefamine and during-famine groups, limiting a deeper examination of the potential heterogeneity of famine impact by the timing of exposure. Furthermore, the offspring of the sampled individuals in the CHNS were between 3 and 28 years old when their height was measured; such a wide range of ages subjects the height to dramatic environmental impact during their developmental period, making it a less ideal proxy of genetic endowment. In contrast, the birth sizes of babies born to the women of the famine cohorts was measured within 24 hours of delivery in our sample, and may serve as a better proxy of the genotype of parents.

Second and more importantly, in this study we have not simply treated the quantile regression and genotype adjustment approaches as additive but have combined the two in a way that enables each to test the validity of the other as an independent approach to address the sample selection problem caused by selected survival. Logically, we expected that the genotype adjustment would alter the estimates of famine impact at upper percentiles of adult height less than estimates at lower percentiles, if individuals from upper quantiles were less likely to have died from famine and experienced survival selection compared with individuals from lower quantiles. Controlling for birth size of the children born to the famine cohorts barely changed the estimates of famine impact at the 90^{th} percentile but dramatically magnified famine impact estimated at the 10^{th} percentile, providing strong evidence to support the two approaches as tools in addressing selection issues in general.

The remainder of this paper is organized as follows. Section 2 contains the background material on the Chinese famine as well as a novel index we proposed to measure the famine intensity. Section 3 describes the data and sample we used for the empirical analysis. Section 4

includes the analytic strategy that we adopted to identify the causal effect of famine exposure and adult height as well as the approach we applied to address the sample selection problem caused by potential survival selection. Section 5 presents empirical results, including a test of the validity of analytic models. Section 6 discusses the results and relevant issues, and Section 7 concludes.

2. The Chinese famine of 1959-61

The Chinese famine was one of the worst catastrophes in Chinese history with tens of millions of deaths estimated to occur during the 3 years of famine (Ashton et al 1984; Smil 1999). Although the causes of the famine remain under debate, the weather, specifically a drought, has traditionally been blamed; this notion has been perpetuated by the Chinese, who continue to refer to it as "three years of natural disasters (*san nian zi ran zai hai*)" despite more evidence suggesting that policy failures played a major role (Lin 1990; Lin and Yang 2000). In 1958, Mao launched the "Great Leap Forward Campaign," during which millions of peasants were mobilized to assist in heavy industry and to promote iron production in particular. In rural areas, mess hall communes were built, and private kitchens were prohibited. Farmers were provided free meals and tremendous food waste was recorded during the harvest year of 1958 right before the famine (Peng 1987; Johnson 1998; Chang and Wen 1998; Yang 1996; Yang and Su 1998; Lin 1990; Lin and Yang 2000; Li and Yang 2005). Famine suddenly hit China in 1959, and grain output dropped sharply during the next 3 years. During the worst year-1961-only 70% of the 1958 record was reached (Chen and Zhou 2007). Daily consumption per capita decreased dramatically to 1,500 calories, far below average energy requirements of 2100 calories per day (Ashton el al 1984; Chen and Zhou 2007). Because of the inflexible nature of the centrally planned procurement system, the Chinese government failed to adjust the procurement and

transfer of food to thousands of heterogeneous counties as a necessary response to the food shortage. Overprocurement occurred in many regions where food stores ran out, and large numbers of people subsequently died of starvation (Meng and Qian, 2009). As a result, mortality dramatically increased and fertility rates dropped sharply during 1959-61. The famine led to an estimated 30 million excess death and another 30 million lost births (Ashton et al 1984; Chen and Zhou 2007; Peng 1987).

The severity of famine varied across regions and affected rural areas disproportionately (Chen and Zhou 2007; Huang et al 2010). Urban residents were less impacted by the famine because of preferential treatment by governments through a grain ration system, even though rations were cut modestly during famine years in some regions (Chen and Zhou, 2007). Dramatic variations in famine intensity also occurred across regions, evident in the substantial difference in mortality rates across regions in the year 1960. The highest rates were recorded in Anhui (68.6 per thousand) and Sichuan (54.0 per thousand); the lowest rates were in provinces in Northeast China (Liaoning, Heilongjiang, and Jilin, about 11 per thousand). The variation in famine severity was more likely to be determined by the degree of willingness of local leaders to follow the radical central government policies, not local grain production. In fact, high productivity areas were more likely to experience severe famine (Meng, Qian, and Yared 2010).

Although famines are normally accompanied by migration away from famine areas, the Chinese famine led only to mild mobility, primarily caused by the restriction imposed by the residence registration (*hukou*) system (Chen and Zhou 2007; Meng and Qian 2009). *Hukou* regulation was initiated in 1951, formalized, and then strictly reinforced in both cities and rural areas by 1958 (Chan and Zhang 1999). Under the *hukou* regulation, every citizen was required to register one and only one permanent residency. Regulation on migration was firmly controlled

by a public security system, which monitored and controlled not only the rural influx to the cities but also all intra-rural and intra-urban movement (Chan and Zhang 1999).

3. DATA

We analyzed data from the China-U.S. Collaborative Project for Neural Tube Defect Prevention conducted collaboratively by the U.S. Centers for Disease Control (CDC) and Beijing University Medical Science Center since the early 1990s (Berry et al 1999). The Project was designed to evaluate the efficacy of taking folic acid during pregnancy to prevent neural tube congenital defects. This program covered the major regions of three provinces: Hebei, Zhejiang, and Jiangsu. All pregnant women and women planning to marry were required to register with the pregnancy-monitoring system that documented prenatal care and delivery. At the time of registration, women participated in an examination that included physical measurements and biochemical tests. In addition, information about their reproductive history, personal and family health history, and socioeconomic status was collected. Characteristics of the delivery, body measurements of the infants, and the presence of birth defects were also collected by medical professionals within 24 hours after birth (Berry et al 1999). We restricted our study to the sample of 52,316 rural women born from 1956-1964. Table 1 reports the average height of these women and the birth size of babies born to these women by single-year birth cohort surrounding the famine years. Adult height was rounded up or down to the nearest centimeter. The most frequently measured height was 160 cm (19.7%), followed by 158 cm (14.4%), 156 cm (7.8%), 155cm (7.6%), 162 cm (6.9%), 157 cm (5.5%); the remainder of the values represented less than 5% of the total (Figure 1). Birth length was measured with standard length boards. Although the data on numerical birth length was characterized by considerable heaping, in particular, at 50

centimeters (Figure 2), birth weight data were less heaped and approximately normally distributed with a mean slightly varying across maternal cohorts (Figure 3).

4. Analytic Strategy

4.1 Measuring the intensity (severity) of the famine in China

An empirical challenge confronting previous researchers on the Chinese famine involved capturing the heterogeneity in famine severity across regions. Chen and Zhou (2007) measured famine severity with excess mortality rate at the province level by calculating the difference between death rate in 1960, the worst famine year with the highest mortality, and the average death rate during 1956-58. A drawback of this measure was the inability to capture the variation in famine intensity within a province, which varied substantially by county (Meng, Qian, and Yared 2009).

To calculate famine intensity at the county level, we used the relative size of famine birth cohorts compared to near cohorts, using the 1% sample of China's 1990 population census. As Table 2 shows, for all three provinces in the sample, the cohorts born during famine years (1959-61) were significantly smaller in size compared to earlier and later cohorts with variations in shrinkage magnitude. Given that the famine, not factors such as infectious pandemic or fertility-regulating policy, was the major force driving this demographic change during this period, the shrinkage and its magnitude reflects famine-caused mortality and postponement of pregnancy. We therefore generated a cohort size shrinkage index (CSSI) to derive a measure of famine intensity at the county level. We collapsed the data by county and birth year. The 1990 Census did not indicate whether the county of residence was the birth county of respondents, but fortunately, the migration rate was very low before 1990 (Lin, Wang, and Zhao 2004).

Nevertheless, we restricted the sample to individuals who had stayed in the county of residence for five and more years. For each county, we first calculated the mean cohort size of individuals born during the 3 years immediately before the famine (1956-1958) and the 3 years immediately after the famine (1962-1964), labeled as N_{normal}, as well as the mean cohort size of individuals born during the famine years (1959-1961), labeled as $N_{famine.}$ We then calculated the $CSSI_r$ as the difference between N_{normal} and N_{famine} divided by N_{normal}; the larger the $CSSI_r$, the more intense (severe) the famine in the county. Among the 35 counties in our data, $CSSI_r$ ranged from 0.25 to 0.78, with a mean of 0.41.

We further compared this cohort size shrinkage index with excessive mortality at the provincial level, which was calculated as the difference between average death rates during the three famine years (1959-61) and the average death rates during 1956–58 and 1962–64. A Pearson test suggested a correlation of 0.85 between the cohort size shrinkage index and excessive mortality rates at the provincial level, suggesting that the two measures shared similar validity as a proxy of famine intensity across provinces. However, the cohort size shrinkage index may capture not only excessive mortality rate but also famine-caused fertility change, including fertility reduction during famine years and fertility rebound after the famine. Another advantage over a mortality-based indictor is its capacity to capture variation in famine intensity at a more disaggregated level than the province level, at which level most previous studies measured famine intensity.

4.2 Basic model: difference in difference (DID) estimator

To estimate the causal effect of early exposure to famine on adult height, we used the variation in famine intensity across counties and cohorts to construct a difference-in-difference (DID) model, similar to that in a previous study (Chen and Zhou 2007):

$$H_{irk} = H_0 + \alpha_k Cohort_k + \phi_r County_r + \sum_{k=1956}^{1963} \beta_k (CSSI_r \times Cohort_k) + \gamma CSSI_r + \varepsilon_{irk} \quad (1)$$

Where H_{irk} is the adult height of a woman born in county r and year k (birth cohort k), α_k is the cohort fixed effect, ϕ_r is the fixed regional effect at the county level, and $CSSI_r$ is the cohort size shrinkage index in county *r* (an indicator of famine intensity in the county). β_K is the coefficient of the interaction between the cohort size shrinkage index and the birth cohort dummy variables, which measures the effect of the early life famine exposure on adult height. The 1964 birth cohort was the reference group.[4] We hypothesized that women who were born or were in utero during the famine years would be, on average, shorter than the reference group without famine exposure because of the stunting effect of the famine. We also expected that the magnitude of the stunting effect would vary across cohorts because of the different timing and duration of famine exposure. We reported standard error clustered at county level to adjust for the potential correlation due to clustering.

[4] The earliest birth cohort without famine exposure is the 1963 cohort, not the 1962 birth cohort, because some individuals born in 1962 were conceived during the famine year of 1961. A concern of using the 1963 birth cohort as reference group is that some individuals in the 1963 birth cohort were conceived in 1962, immediately after the famine, and the severe famine exposure and malnutrition may have lagged effects on women who were pregnant in 1962. Therefore, we used the 1964 birth cohort as the reference group.

4.2 Addressing sample selection

We further considered the potential attenuation bias caused by survival selection (sample selection) in the foregoing model specification. In Equation (1), potential adult height was treated as completely random and specified as part of error term ε_{irk} . Equation (1) is valid only when ε_{irk} is independent of the interaction between $CSSI_r$ and $Cohort_k$; otherwise, the estimation of β_k will be biased. This independence assumption is unlikely to be the case because results from previous studies have suggested that individuals who survived a severe famine with high mortality tend to have greater adult potential height than their counterparts without severe famine exposure (Gorgens, Meng, and Vaithianathan 2007; Bozzoli, Deaton, and Quintana-Domeque 2009); in other words individuals born in a famine year and a region with higher CSSI are expected to systematically achieve a greater adult height potential than the rest due to selective survival.

One approach proposed to address the potential bias caused by sample selection is genotype adjustment by which the body sizes of offspring were used as a proxy of potential height of the famine cohorts (Gorgens, Meng, and Vaithianathan 2007).[5] The underlying assumption is that children inherit their parents' genotype instead of their phenotype, such as famine exposure. In this study, we used the birth weight and birth length of babies born to the famine cohorts, adjusting for the sex of the newborn, as a proxy of genetic endowment of the

[5] Gorgens, Meng, and Vaithianathan (2007) used the height of their children aged 0-33 year as a proxy. To control properly for the age effect on height, they relied on cubic splines modeling the population average height-age relationship.

mother. [6] We respecified the relationship of famine exposure across regions and cohorts with adult height as follows:

$$H_{irk} = H_0 + \alpha_k Cohort_k + \phi_r County_r + \sum_{k=1956}^{1963} \beta_k (CSSI_r \times Cohort_k) + \gamma CSSI_r + \varphi Birthsize_{irk} + \varepsilon_{irk} \quad (2)$$

Another approach, which is less intuitively straightforward, is to estimate the impact of the famine on the upper quantiles of distribution of outcomes, such as the 90th percentile of adult height, with a quantile regression in the form below (Equation 3), instead of conventional mean regression as specified in Equations (1)–(2).

$$Q_{H_{irk}}(\tau | Cohort_k, county_r, CSSI_r) = \mu_0(\tau) + \alpha_k Cohort_k + \phi_r County_r + \sum_{k=1956}^{1963} \beta_k (CSSI_r \times Cohort_k) + \gamma CSSI_r \quad (3)$$

Here $Q_Y(\tau | Z)$ denotes the τ-*th* conditional quantile of Y given covariates Z, where 0<τ<1. Next we present heuristic arguments for why the estimation of model (3) is robust to selection bias when τ is chosen close to 1. First, we may view τ, the percentile of subject's height, as an index for some latent characteristic, say propensity for surviving from famine, denoted by Aτ. Let S be an indicator for famine survivor (i.e., S=1 if survived and 0 otherwise), H be a random variable for adult height, and Z denote a vector of covariates including cohort, country, and CSSI. Assume that mortality was low among infants with tall potential adult height, that is, survival

[6] We acknowledged that birth weight and birth length are decided by a complex interaction between genetics and environmental factors, primarily including fetal nutritional status. We assumed that nutritional status in the fetus is randomly distributed among children born to mothers of different cohorts, but we could not rule out the possibility that women with famine exposure may feed their children better than women without famine exposure (Gørgens et al 2007).

selection rarely occurs among individuals from upper percentiles of height. When τ is large, say equal to 0.9, we would expect Pr(S=0, A0.9) to be small, and hence, Pr(H≤h|Aτ, Z) may be well approximated by Pr(H≤h|S=1, Aτ, Z) provided that 0≤Pr(H≤h, A0.9, Z)-Pr(H≤h, S=1, A0.9, Z)≤Pr(S=0, A0.9) and 0≤Pr(A0.9, Z)-Pr(S=1, A0.9, Z)≤Pr(S=0, A0.9). This result shows that fitting model (3) using the available sample (with S=1) would only produce small biases when τ is large. Following similar arguments, with a smaller τ, say 0.1, one may speculate a greater extent of survival selection among subjects with A0.1 reflected by a larger Pr(S=0, A0.1). This would lead to a bigger discrepancy in the height quantile between the observable population which only consists of famine survivors and the latent population without survival selection, which is more relevant to the evaluation of the stunting effect of famine. Therefore, β_k estimated at $\tau = 0.9$ is less likely to be biased by sample selection and therefore provide a more reliable estimate of famine impact than that estimated at $\tau = 0.1$.

In this study, we addressed the sample selection bias by combining the two foregoing approaches, in which we controlled for genotype in the quantile regression. This analytical strategy worked well in the sense that neither approach was simply additional to the other, but also calibrated the validity of each other as a solution in addressing sample selection. Logically, if the famine impact estimated at upper quantiles (say, the 90th percentile) should be less biased by survival selection than that estimated at lower quantiles (say, the 10th percentile), then adjusting for genotype should alter the estimates at upper quantiles less than it alters the estimates at lower quantiles.

5. Empirical Results

5.1Average effect of early exposure to the Chinese famine of 1959-61 on adult height

A significant stunting effect of the famine was identified among women born in 1959 from the mean regression with the difference-in-difference estimator as specified in Equation 1(Mean regression, Table 3). The coefficient (-3.681) refers to the change in adult height with a one unit

change in the cohort size shrinkage index (CSSI), which is hypothetical. We multiplied the mean of the CSSI, 0.41, by this coefficient, producing an impact of -1.51 cm (-3.681*0.41); this suggests that, on average, the 1959 birth cohort would be 1.51 centimeters taller in the absence of famine. Other cohorts did not exhibit any significant stunting effect.[7]

The results of quantile regression suggested that stunting effects estimated at upper percentiles were larger than that those estimated at lower percentiles (see Table 3). For the prefamine cohorts (1956-58 birth cohorts combined), a reduction of 2.41 centimeters (-5.883*0.41) was estimated at the 90th percentile (τ=0.9), compared to a reduction of 0.15 centimeters (-0.365*0.41, p >0.1) estimated at the 10th percentile (τ=0.1). For the 1959 birth cohort, a larger stunting effect of a 3.43 (-8.362*0.41) centimeter reduction in adult height was estimated at the 90th percentile, compared to a 0.20 centimeters (-0.493*0.41, p >0.1) reduction estimated at the 10th percentile.

Estimates from both mean regression and quantile regression controlling for sex-adjusted birth length and birth weight of children born to the women were presented in Table 4. In the mean regression, this control for genotype barely altered the estimates of the stunting effect presented in Table 3. Results from quantile regression suggested that adjustment for genotype did not alter estimates at upper percentile (τ=0.9); however, adjustment for genotype substantially altered the estimates at the lower percentile (τ=0.1). Controlling for sex-adjusted birth weight and length substantially increased the stunting effect estimated at the 10th percentile

[7] Results from alternative analysis in which the pre-famine cohorts were further divided into three cohort groups (1956 cohort, 1957 cohort, and 1958 cohort) suggested a stunting effect for the 1958 cohort with marginal significance (P<0.1).

-2.67 centimeters (-6.501*0.41, $p<0.1$) for the 1959 birth cohort (Table 4), compared with -0.20 centimeter (-0.493*0.41, $p>0.1$) estimates without controlling for genotype (Table 3).

5.2 More on robustness

A key assumption in the difference-in-difference model (DID) refers to "common trends," which in the current study means that the regions should have shared the same cohort trends if no famine had occurred and that changes causing cohort size shrinkage should not be systematically related to other omitted factors that also impact adult height. Previous studies on the Chinese famine intensively discussed and excluded this possibility of such omitted variable bias.[8]

We pursued a test of the assumption of our DID estimator, similar to that in Chen and Zhou (2007). We used a sample of 1964-1969 birth cohorts who were born after the Chinese 1959-1961 famine. Logically, if the model captures the famine impact rather than other "omitted" factors, we should not observe any significant interaction terms between birth cohort at CSSI, which was supposed to capture the famine impact among these cohorts who were not affected by the famine. We did a similar difference-in-difference analysis as described above for

[8] Several potential "omitted" variables were discussed in previous studies on Chinese famine. For example, it was speculated that poor regions with less grain reserves may be more susceptible to famine; on the other hand, they may experience a faster economic growth after famine, which may cause a mean reversion bias for the DID estimator (Meng and Qian 2009). However, a study on the causes of the famine revealed a striking finding that regions with more grain production were more severely hit by the famine and had higher famine-caused mortality (Meng, Qian, and Yared 2010). In addition, it was also speculated that the government may invest more in the most adversely affected regions as a compensation or treatment (Meng and Qian 2009). However, because the government had not acknowledged the existence of the famine for political reasons until recently, such compensation was never attempted (Meng and Qian 2009).

these cohorts (the cohort 1964 was used as reference group). As shown in Appendix Table 5, none of the coefficients of the interaction terms are statistically significant, which suggests that the assumption for our DID estimator is not likely to be violated.

6. Discussion

In the present study, we examined the long-term effect of early exposure to the Chinese famine of 1959-61 on adult height, using a large sample of female survivors. Because the famine caused substantial and excessive mortality, it was expected that an attenuation bias caused by selective survival was likely to mask or even dominate the potential stunting effect of famine when using a conventional mean regression strategy. We addressed this concern with a combination of different approaches, each proposed separately in previous studies (Gorgens, Meng, and Vaithianathan 2007; Meng and Qian 2010). We applied the quantile approach and estimated the stunting effect at the upper percentile of height because survival selection is more likely to occur among individuals from lower percentiles of height than individuals from upper percentiles of height. We further controlled for the birth size of babies born to the famine cohorts because children inherit their parent's genotype but not famine exposure experiences and therefore children's birth size may serve as a proxy of potential adult height of the famine cohorts.

We found that the stunting effect estimated at the 90^{th} percentile of adult height was more adverse and larger than the stunting effect estimated at the 10^{th} percentile, which was expected because survival selection should be milder among individuals from higher quantiles of potential adult height than among individuals from lower quantiles. Thus, the selection effect was less likely to suppress the stunting effect at higher quantiles of adult height. This corroborates

findings from CNHS data by Meng and Qian (2009) that the famine impact estimated at the 90th percentile doubles the magnitude of the effect estimated from mean regression and it is also much larger than the effect estimated at the 10th percentile.

It is worth noting that the differences in estimates from different percentiles may not be caused exclusively by different intensity of survival selection among individuals across percentiles of adult height. For example, differences may be caused fully or partially by the heterogeneous effect such that individuals in the upper quantiles of height are more likely to be affected by famine exposure, which has been argued unlikely to be the case, given that height indicates nutritional and health status and taller individuals are more resilient following health shock (Meng and Qian 2009). Reasonably, if some heterogeneous effect exists, famine impact at the 90th percentile should be smaller than that at the 10th percentile, instead of vice versa.

Our study generated an opportunity to test directly whether the different famine impacts across quantiles of adult height were likely to be caused by survival selection or heterogeneous effect; we found that genotype adjustment affects the estimates of famine impact differently across quantiles of adult height. Logically, if differentials in famine impact across quantiles are driven solely by heterogeneous effect, they should be robust to adjustment for potential height. In contrast, if differentials in famine impact across quantiles are driven by survival selection such that individuals from lower quantile of adult height were more likely to have died from famine than those from upper quantile, then adjustment for genotype should substantially alter the famine impact at lower quantiles but not at upper quantiles. We found this pattern in this study on the Chinese famine, therefore empirically ruling out potential heterogeneous effects.

Despite the significant and substantial stunting effect among the 1959 birth cohort and prefamine birth cohorts, we did not find any significant reduction in height among other famine-born cohorts. We expect that the lack of stunting effect among the 1960 and 1961 birth cohorts may have occurred because survival selection was more severe among these cohorts than among prefamine and 1959 birth cohorts. The highest excessive mortality during the famine period was in 1960. A previous study (Huang et al forthcoming) also found that the largest babies were born to women who themselves were born in 1960 in regions that experienced the most severe famine. As Bozzoli, Deaton, and Quintana-Domeque (2009) argued, when mortality is sufficiently high, the selection can dominate scarring, leaving an even taller population of survivors; this dominant selection pattern is consistent with the pattern for the 1960 and 1961 birth cohorts. The severe survival selection may have involved even infants at the upper percentile of potential adult height, making it difficult to identify any stunting effect even when estimated at upper quantiles (the sign of the coefficients indicates a stunting effect at the 90^{th} percentile among the 1960 and 1961 cohorts, but the effect is not statistically significant). In addition, because of measurement error for birth size and its nature as a weak proxy of maternal potential height, adjustment for genotype cannot dramatically address the survival selection.

We did not directly address other issues that may be relevant, in particular, the potential measurement error and endogeneity of famine intensity. Meng and Qian (2009) created instruments for famine intensity with the interaction between non-famine production and birth year;[9] results from two stage least squares (2SLS) estimates suggested that correcting for

[9] Two instrument variables were used in their study: the interaction terms between the grain suitability of the county of birth and birth cohort dummy variables and the interaction terms of the 1997 per capita grain sown in the county of birth and birth cohort dummy variables.

measurement error led to a larger stunting effect in both conventional mean regression and quantile regression. With a focus on anthropometric consequences of famine exposure instead of other social outcomes, such as education and occupational achievements, our analysis was exempted from concerns about potential impact of the complicated social changes following the Chinese famine, such as the Great Cultural Revolution of 1966-1976. Furthermore, although a reduced cohort size for individuals born in famine years may have benefited survivors with more access to public school and less labor market competition from peers and potentially offset the famine impact (Meng and Qian 2009), this effect should less concern the achievement of adult height.

Caution should be exercised in interpreting the empirical results of this study. For its original purpose of birth defect intervention effectiveness, the China-U.S. Collaborative Project for Neural Tube Defect Prevention focused mainly on fecund women. Possibly, this selection may favor older women who were, in general, healthier and more fecund. In addition, because of the lack of fathers' information, we were unable to model birth size as a function of both fathers' and mothers' characteristics; husbands' characteristics may not be totally random but instead "matched" to their wives. For example, taller women tend to marry taller men. Finally, the birth length in our data may be subject to measurement error as a result of heaping, as about 50% of the children were recorded at 50 centimeters long. The lack of sufficient variation in birth length makes the birth size a weak proxy of potential adult height.

Our study linking early life exposure to the Chinese famine of 1959-61and adult height is policy relevant in that investing in children's health in early life may have positive returns to later life health, especially in developing countries. There are 34.4 million people starving or at

risk of starvation who need assistance from agencies such as UNHCR, and of these, almost half are children (United Nations 2010). The international community has not always responded as generously and as timely as desired. Demonstrating important long term impacts on survivors will further aid advocacy for greater attention to famine prevention and mitigation.

7. Conclusion

Using the Chinese 1959-61 famine as a natural experiment, this study examined the anthropometric consequences of malnutrition in early life. Along with evidence from other studies, our results suggest that the nutritional deprivation in an early "critical period" may cause a serious stunting effect, which underscores the importance of antipoverty measures targeting children and early nutritional intervention in developing countries.

This study also addressed a methodological issue that survival selection may bias the estimates of the stunting effect among famine survivors as a selected group of individuals with above-average robustness, which is a common empirical challenge confronting studies of long-term impacts of a "shock" experience in early life, in particular when the "shock" caused high mortality and severe survival selection. Showing that attenuation bias caused by survival selection may vary across quantiles, with the lower quantiles being more subjected to the attenuation bias, our analysis suggests that an approach to estimating "shock" impact at the upper quantile of outcome variables with quantile regression may serve as a better analytic strategy than the conventional mean regression-based method, along with evidence presented elsewhere (Meng and Qian, 2009). An inclusion of genotype information in the analysis is also beneficial, especially when considerable survival selection may occur at the upper quantiles of distribution of outcomes.

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TABLE 1-Adult height and birth size of children among the sampled rural Chinese women by birth cohorts

Birth year of the women	Adult height of the women				Birth size of the offspring			
					Female children		Male children	
	Mean (Std Dev)	10^{th} (τ=0.10)	Median (τ=0.50)	90^{th} (τ=0.90)	weight	length	weight	length
Pre-famine								
1956	158.4	153.0	158.0	163.0	3223.3	49.3	3220.5	49.2
(n=449)	(4.2)				(482.6)	(2.6)	(462.4)	(2.7)
1957	158.0	152.0	158.0	164.0	3220.5	49.2	3370.5	49.3
(n=643)	(4.5)				(462.4)	(2.6)	(473.6)	(2.5)
1958	157.9	152.0	158.0	163.0	3243.2	49.1	3369.3	49.7
(n=952)	(4.4)				(44.2)	(2.6)	(459.4)	(2.2)
During-famine								
1959	157.9	152.0	158.0	163.0	3259.9	49.4	3411.9	49.7
(n=1,152)	(4.2)				(45.8)	(2.2)	(493.8)	(2.4)
1960	158.2	152.0	158.0	163.0	3306.1	49.4	3403.1	49.6
(n=2,090)	(4.3)				(438.7)	(2.3)	(471.2)	(2.4)
1961	158.4	153.0	158.0	164.0	3290.7	49.5	3402.0	49.8
(n=3,113)	(4.3)				(450.4)	(2.5)	(470.8)	(2.5)
Post-famine								
1962	158.7	153.0	159.0	164.0	3309.8	49.4	3444.0	49.9
(n=10,378)	(4.3)				(457.3)	(2.4)	(458.1)	(2.3)
1963	158.9	154.0	159.0	164.0	3311.2	49.4	3425.5	49.8
(n=14,381)	(4.3)				(451.8)	(2.3)	(458.2)	(2.3)
1964	158.9	154.0	159.0	164.0	3304.7	49.4	3429.3	49.8
(n=14,573)	(4.2)				(446.2)	(2.4)	(460.4)	(2.3)

Source: China-U.S. Collaborative Project for Neural Tube Defect Prevention

Table 2 Cohort size shrinkage index and excessive death rate index by province

Province	Cohort size shrinkage index	Excessive death rate	Mortality rate by year (1 in 1,000)								
			1956	1957	1958	**1959**	**1960**	**1961**	1962	1963	1964
Beijing	0.22	1.67	7.7	8.2	8.1	**9.7**	**9.1**	**10.8**	8.8	8.1	8.3
Tianjin	0.33	1.80	8.8	9.4	8.7	**9.9**	**10.3**	**9.9**	7.4	7.3	7.8
Hebei	0.34	3.12	11.3	11.3	10.9	**12.3**	**15.8**	**13.6**	9.1	11.2	10.9
Shanxi	0.26	0.95	11.6	12.7	11.7	**12.8**	**14.2**	**12.2**	11.3	11.4	14.0
Nei Mongol	0.20	0.47	7.9	10.5	7.9	**11.0**	**9.4**	**8.8**	9.0	8.5	11.8
Liaoning	0.32	5.55	6.6	9.4	6.6	**11.8**	**11.5**	**17.5**	8.5	7.9	9.3
Jilin	0.23	2.22	7.5	9.1	9.1	**13.4**	**10.1**	**12.0**	10.0	9.4	12.6
Heilongjiang	0.19	1.75	10.1	10.5	9.2	**12.8**	**10.6**	**11.1**	8.6	8.6	11.5
Shanghai	0.22	0.62	6.8	6.0	5.9	**6.9**	**6.8**	**7.7**	7.3	7.0	6.1
Jiangsu	0.42	5.10	13.0	10.3	9.4	**14.6**	**18.4**	**13.4**	10.4	9.0	10.1
Zhejiang	0.35	1.88	9.5	9.3	9.2	**10.8**	**11.9**	**9.8**	8.6	7.9	9.2
Anhui	0.63	21.07	14.3	9.1	12.3	**16.7**	**68.6**	**8.1**	8.2	7.9	8.6
Fujian	0.37	3.68	8.4	7.9	7.5	**7.9**	**15.3**	**11.9**	8.3	7.4	8.6
Jiangxi	0.35	2.37	12.5	11.5	11.3	**13.0**	**16.1**	**11.5**	11.0	9.8	10.9
Shandong	0.40	7.87	12.1	12.1	12.8	**18.2**	**23.6**	**18.4**	12.4	11.8	12.0
Henan	0.47	10.22	14.0	11.8	12.7	**14.1**	**39.6**	**10.2**	8.0	9.4	10.6
Hubei	0.42	5.02	10.8	9.6	9.6	**14.5**	**21.2**	**9.1**	8.8	9.8	10.9
Hunan	0.54	8.80	11.5	10.4	11.7	**13.0**	**29.4**	**17.5**	10.2	10.3	12.9
Guangdong	0.35	3.37	11.1	8.4	9.2	**11.1**	**15.2**	**10.8**	9.4	7.6	8.3
Guangxi	0.46	10.90	12.5	12.4	11.7	**17.5**	**29.5**	**19.5**	10.3	10.1	10.6
Sichuan	0.61	28.63	10.4	12.1	25.2	**47.0**	**54.0**	**29.4**	14.6	12.8	13.9
Guizhou	0.52	16.38	7.5	8.8	13.7	**16.2**	**45.4**	**17.7**	10.4	9.4	10.5
Yunnan	0.38	3.15	15.2	16.3	21.6	**18.0**	**26.3**	**11.8**	10.9	14.1	15.2
shannxi	0.25	0.13	9.9	10.3	11.0	**12.7**	**12.3**	**8.8**	9.4	10.6	15.6
Gansu	0.45	10.48	10.8	11.3	21.1	**17.4**	**41.3**	**11.5**	8.3	10.4	15.6
Qinghai	0.52	12.65	9.4	10.4	13.0	**16.6**	**40.7**	**11.7**	5.4	8.4	15.5
Ningxia	0.41	2.00	10.6	11.1	15.0	**15.8**	**13.9**	**10.7**	8.5	10.2	13.4
Xinjiang	0.22	2.63	14.2	14.0	13.0	**18.8**	**15.7**	**11.7**	9.7	9.4	16.3

Note: Excess death rates are calculated as the difference between average death rates in the famine years (1959-61) and the average of death rates in 1956–58 and 1962-64. Sources: mortality rates by years were adapted from Lin and Yang (2000), Table 3, P147.

Cohort size shrinkage index was calculated as the difference between average size of cohorts born in the famine years (1959-61) and the average size of cohorts born in 1956–58 and 1962-64 divided by the average size of cohorts born in 1956–58 and 1962-64, in the 2000. Source: the 1990 census of Chinese population.

TABLE 3- Estimate of the impact of famine exposure on adult height among Chinese women

	Mean regression	Quantile regression		
		τ=0.10	τ =0.50	τ =0.90
Pre-famine-born				
CSSI×1956-58©	-0.794	-0.365	-0.176	-5.883**
	(1.308)	(2.235)	(0.700)	(2.034)
During-famine-born				
CSSI×1959	-3.681**	-0.493	-6.776**	-8.362**
	(1.298)	(3.338)	(1.474)	(2.574)
CSSI×1960	1.464	3.692	0.052	-3.615
	(1.374)	(2.319)	(0.920)	(2.359)
CSSI×1961	0.602	0.108	0.090	-0.442
	(1.749)	(1.343)	(0.439)	(1.993)
Post-famine-born				
CSSI×1962	0.184	0.145	0.071	-0.788
	(1.094)	(0.302)	(0.129)	(1.369)
CSSI×1963	0.454	0.162	0.074	-0.160
	(1.045)	(0.266)	(0.125)	(0.463)

Note: © The pre-famine born cohorts (1956, 1957 and 1958 birth cohorts) are combined;

†p<0.1, ** p<0.01; CSSI, Cohort size shrinkage index

TABLE 4-Esimate of the impact of famine exposure on adult height among Chinese women, controlling for birth weight and length of their children

Reference (1964 cohort)	**Mean regression**	**Quantile regression** τ =0.10	τ =0.50	τ =0.90
Pre-famine-born				
CSSI×1956-58©	-1.132	-0.944	-0.997	-5.381*
	(1.237)	(2.731)	(1.280)	(2.529)
During-famine-born				
CSSI×1959	-3.747**	-6.501†	-4.033**	-8.295**
	(1.178)	(3.680)	(1.598)	(3.234)
CSSI×1960	1.221	0.602	1.105	-2.155
	(1.290)	(2.363)	(1.664)	(2.335)
CSSI×1961	0.479	-0.026	1.208	-2.238
	(1.646)	(2.451)	(1.224)	(2.038)
Post-famine-born				
CSSI×1962	0.206	1.446	0.812	-2.355
	(1.046)	(1.729)	(0.759)	(1.356)
CSSI×1963	0.433	1.239	0.591	-2.040
	(0.998)	(1.332)	(0.587)	(1.197)

Note: © The pre-famine born cohorts (1956, 1957 and 1958 birth cohorts) are combined;

† p<0.1, *p<0.05, ** p<0.01; CSSI, Cohort size shrinkage index

APPENDIX

TABLE 5- Test the Difference-in-Difference Model

Reference (1964 birth cohort)	Mean regression	Quantile regression		
		τ =0.10	τ =0.50	τ =0.90
CSSI×1965	-1.002	-0.144	-0.070	-0.077
	(0.603)	(0.139)	(0.060)	(0.102)
CSSI×1966	0.320	-0.047	0.011	0.072
	(0.222)	(0.137)	(0.072)	(0.091)
CSSI×1967	0.221	-0.064	0.037	0.115
	(0.477)	(0.147)	(0.059)	(0.092)
CSSI×1968	-0.227	-0.068	-0.014	0.051
	(0.206)	(0.130)	(0.059)	(0.080)
CSSI×1969	-0.421	-0.047	-0.050	-0.072
	(0.253)	(0.138)	(0.053)	(0.096)

†p<0.1, ** p<0.01; CSSI, Cohort size shrinkage index

Sources: As for Table 1

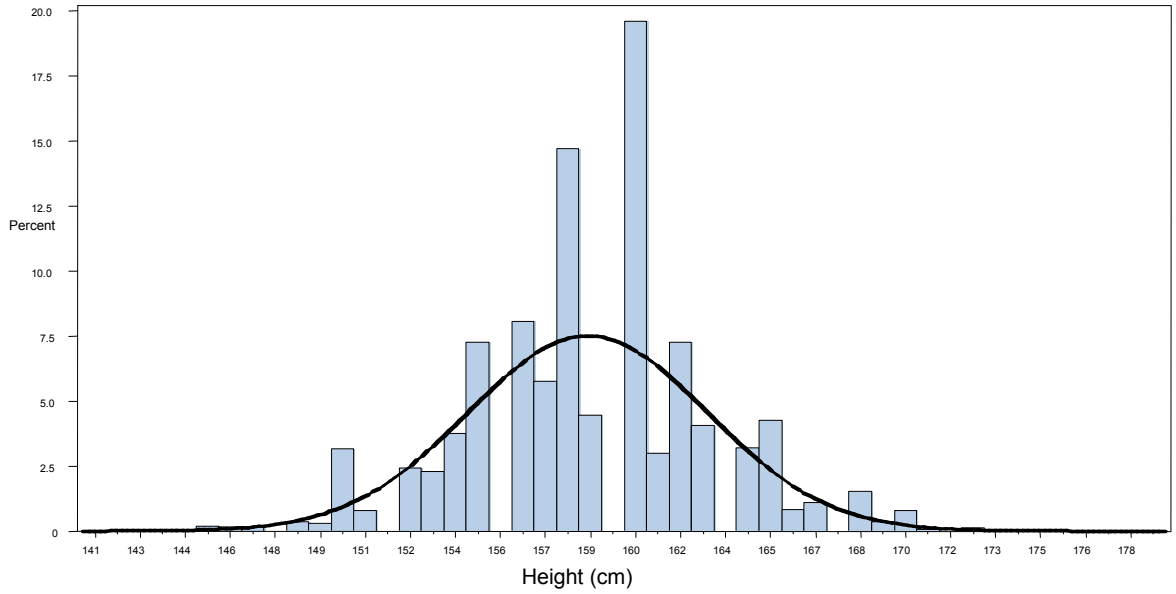
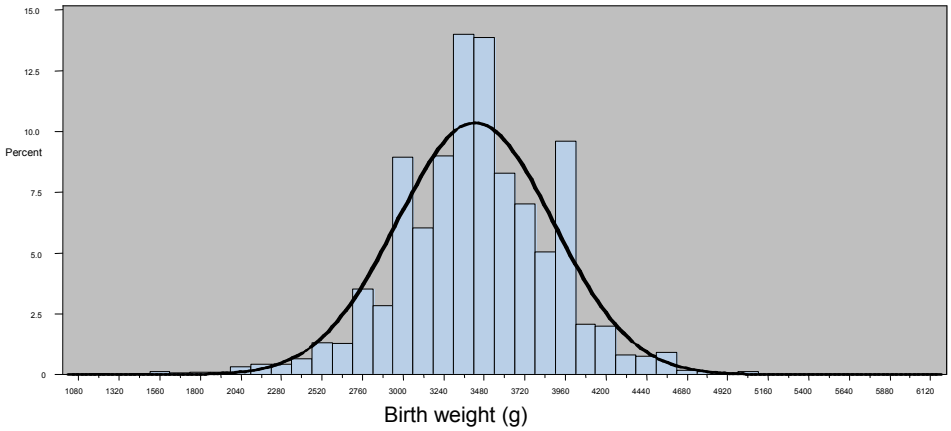
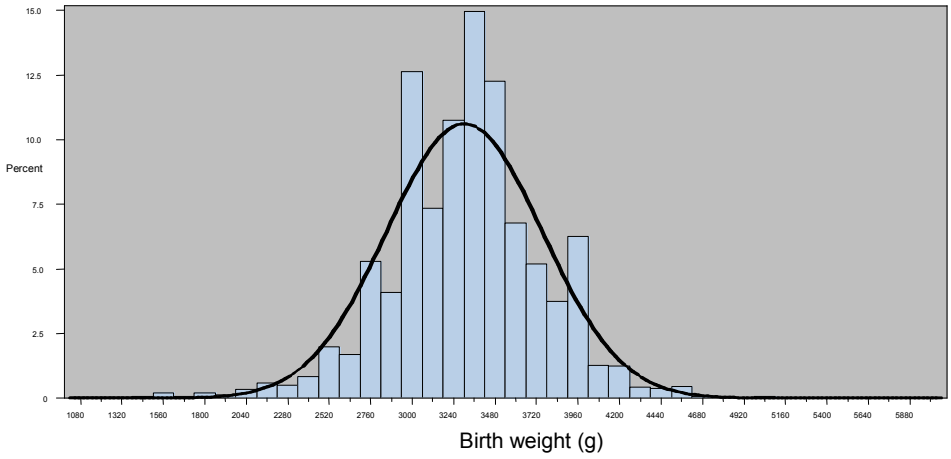


Figure 1-Adult height of women born during 1956-1964



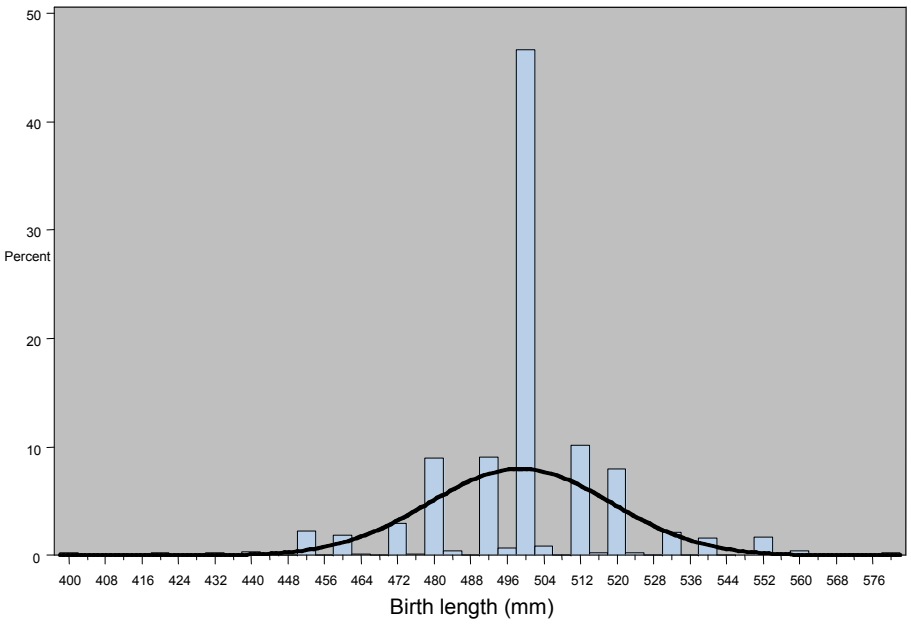
Panel a, Birth weight of male children



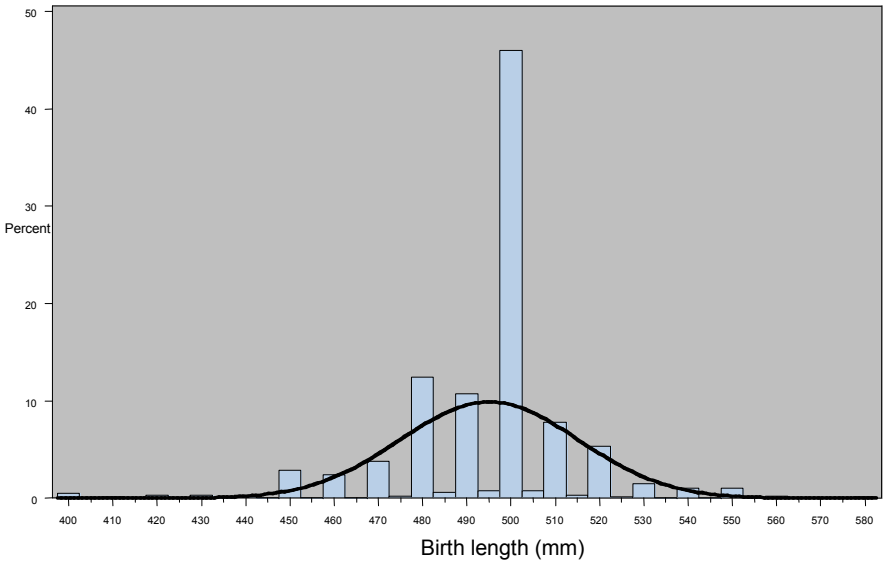
Panel b, Birth weight of female children

Figure 2-Birth weights of children born to women participants

born during 1956-64 in the Folic Acid Intervention Study



Panel a. Birth length of male children



Panel b. Birth length of female children

Figure 3-Birth lengths of children born to women participants

born during 1956-64 in the Folic Acid Intervention Study

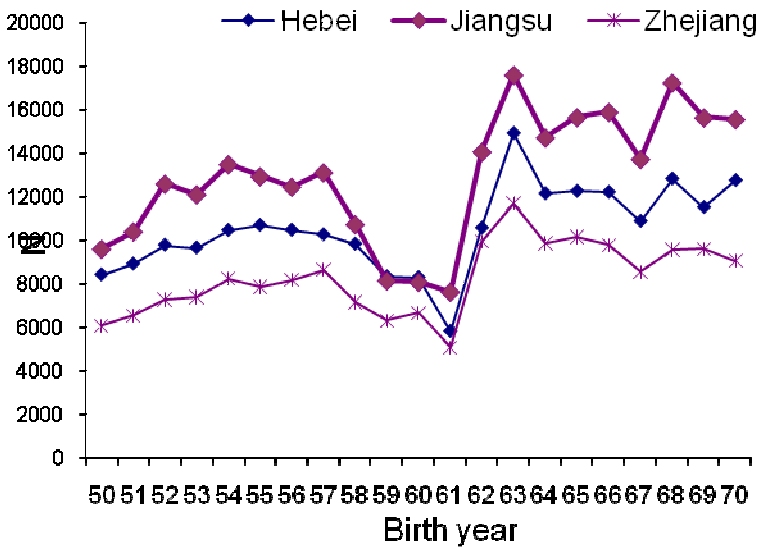


Figure 4-Cohort size by year of birth in the sampled provinces, the 1990 Census of Chinese Population