The Long Term Consequences of Famine on Survivors: Evidence from a Unique Natural Experiment using China's Great Famine

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Abstract

This paper estimates the long run impact of famine on survivors in the context of China's Great Famine. To address problems of measurement error of famine exposure and potential endogeneity of famine intensity, we exploit a novel source of variation in regional intensity of famine derived from the unique institutional determinants of the Great Famine. To address attenuation bias caused by selection for survival, we estimate the impact on the upper quantiles of the distribution of outcomes. Our results indicate that in-utero and early childhood exposure to famine had large negative effects on adult height, weight, weight-for-height, educational attainment and labor supply. (O1 Development, I0 Health, J1 Demography)

1 Introduction

The impact of famine has been a question of long standing interest amongst economists. In the twentieth century, more individuals perished from famine than from both World Wars combined.[1] In 1959-61, an estimated 16-30 million people died in China's Great Famine alone.[2] Both the fear of future famines and the lingering consequences of those that have already passed are ever relevant as we begin the new millennium. Hundreds of millions of people alive today have been

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[1]See Sen (1981) and Ravallion (1997).

[2]See Coale (1981), Yao (1999), Peng (1987), Ashton et al. (1984) and Banister (1987).

affected by a famine at some point during their lifetime. And as recently as 2002, the United Nation's *World Food Programme* calculated that up to 38 million Africans were living under the threat of famine. In China's case, survivors who were exposed to the Great Famine during early childhood or in-utero have only just passed the middle of their life.

While much attention has been paid to the effects of famine on mortality, there has been surprisingly little coverage on the effect for the millions of survivors. The net effect of famine exposure on survivors is not *a priori* obvious for either health or labor market outcomes. On the one hand, severe food deprivation can only have adverse effects on the health of any given individual. Besides the direct mechanism of starvation, malnutrition also decreases the human body's ability to resist disease. On the other hand, it has been observed that conditional on survival, children who are malnourished exhibit rapid "catching-up" if proper care and nutrition is given.[3] Furthermore, studies have argued that when the famine is severe and mortality rates are high, survivors tend to be comprised of a small number of selected individuals who have naturally stronger constitutions and are better able to resist the negative effects of famine.[4] In the extreme scenario, these few survivors may be resilient to the extent that they do not exhibit symptoms of the adverse effects. For labor market outcomes, one must in addition consider the impact of famine in reducing labor supply, which could increase wages for survivors. This could potentially offset negative effects of the famine on survivors' health and work capacity.

This study addresses these questions by examining the net effect of childhood exposure to China's Great Famine on adult health and labor supply outcomes thirty years after fact. Existing studies of the impact of famine on survivors provide somewhat ambiguous results. Epidemiological studies on the long run impact of the Dutch Famine (1944-1945) find that famine is positively correlated with psychological disorders in adulthood (Neugebauer et al., 1999; Brown et al., 2000; Hulshoff et al., 2000); obesity (Ravelli et al., 1999); and glucose intolerance (Ravelli et al., 1998). In contrast, Stanner et al. (1997) studies a sample of approximately 600 survivors of the Leningrad siege (1941-1944) and find no effects of exposure. And Luo et al. (2006) find little or no difference in outcomes between survivors and a control group of individuals who were never exposed to China's Great Famine.

All studies of the long-run impact of famine face several empirical difficulties. First, it is difficult to measure the intensity of exposure. Researchers are generally not able to collect data at the time of the famine. The migration typically induced by famine makes it difficult to determine an individual's level of exposure after the fact. Even if researchers can learn of

[3] Outside of the famine context, there is evidence that conditional on survival, the negative impact of adverse childhood nutritional shocks is mitigated by rapid "catch-up" (Krueger, 1969; Hoddinott and Kinsey, 2001).

[4] For example, see Gorgens et al. (2002. revised 2007) and Deaton (2008).

a survivors' location during the famine, the constructed measure of exposure intensity is likely to be measured with error. Second, famine typically occurs in concurrence with other events such as conflict, which can confound causal interpretation. Alternatively, one may suspect that famine is more likely to strike in less agriculturally productive regions. In that case, the correlation between famine intensity and survivor outcomes may reflect the effect of being born in a particular region rather than the causal impact of famine on survivors. Finally, selection for survival can attenuate the estimated impact of famine. Survivors are typically from the top of the distribution of factors that determine survival (Gorgens et. al., 2002, revised 2007; Deaton, 2008). Those from the lower parts of the distribution die. Since these factors are likely to be correlated with health and labor market outcomes later in life, comparing the mean of the distribution of survivors to the mean of the distribution of the control group will underestimate the true impact of famine.

Several recent studies have carefully constructed empirical strategies to address some of these issues for studying the long run effects of China's Great Famine. Gorgens et al. (2002, revised 2007) argue that the observed similarity between survivors and unexposed individuals is driven by attenuation bias from selection; and that genetic determinants for survival are more likely to be transmitted to children than exposure to famine. Under this assumption, they show that selection bias can be mitigated by controlling for attributes of children. Their study does not address the problem that exposure to famine may be endogenous to other factors. To establish causal identification, Chen and Zhou (2002, revised 2007) uses a differences-in-differences (DD) strategy. They compare exposed cohorts to unexposed cohorts between provinces where famine era mortality rates were high to those where they were low. They find that individuals who were one or two years of age during the famine were shorter in stature on average. Also using a DD strategy, Almond et al. (2006) focus on the effect of in-utero exposure. They compare children born to parents in Hong Kong who were never exposed to those born to parents who immigrated from mainland China and were exposed. They find that in-utero famine exposure has adverse effects on the adult labor supply and marriage outcomes for men. Their results are consistent with the findings of Brandt, Siow and Vogel (2008), who uses a decomposition approach and finds that the famine cohort is less attractive on the marriage market relative to the cohorts that immediately preceded and followed. These latter three studies do not attempt to address problems from selection.

The principal contribution of this paper is to improve upon existing studies of the impact of famine for survivors by attempting to simultaneously address the problems from of measurement error, endogeneity and selection. We use the average county level cohort size of survivors born during the famine to retrospectively measure exposure. This provides us with a continuous measure of famine intensity at a more disaggregated level than previous studies that have relied on province level variation. Next, to address the issue of positive selection for survival, we estimate the effect of exposure on the upper quantiles of the distribution of outcomes. Assuming that survivors are from the top of the distribution of outcomes, these estimates will more accurately reflect the true effect of exposure to famine. To the best of our knowledge, this is the first study to apply quantile analysis or to use survivor cohort size for studying the impact of famines. Finally, to address the problems of measurement error and omitted variable bias (OVB), we instrument for famine intensity with a unique source of variation derived from the institutional causes of what's often called the "Great Leap Forward Famine". The Great Famine is widely believed to be caused by over-procurement. In a companion paper, Meng and Qian (2009) point out the surprising positive correlation between normal grain production and famine intensity: regions that historically produced more grain suffered more during the famine. In that paper, we show how the grain procurement design at the time could have caused this pattern. In this paper, we use this institution driven variation in cross-sectional famine intensity (measured as non-famine grain production or as geographic and climatic suitability for rice or wheat production) in combination with birth cohort variation to instrument for exposure. The more grain a region normally produces, the more intense the famine in that region; and the younger an individual was at the time of the famine, the more vulnerable she was to exposure. Only the combination of the two sources of variation can be interpreted as plausibly exogenous.

The analysis uses data from several existing sources: the *1990 Population Census*, the *1989 China Health and Nutritional Survey*, the *1997 China Agricultural Census*, and the FAO GAEZ data set on crop suitability. Our study focuses on agricultural households who suffered most from the famine and was relatively undisturbed by the Cultural Revolution (1966-1976). Our results indicate that in-utero exposure to famine on average caused a 1.7% (2.8 cm) reduction in height, a 2.3% (1.4 kg) reduction in weight and a 8.6% (6.8 years) reduction in educational attainment. For those exposed during early childhood, the famine on average reduced height by 1.6% (2.7 cm), weight by 5% (3 kg), WFH by 1.2% (0.004 kg/cm) and labor supply by 13.9% (12.6 hours per week).

The adverse effects are consistent with results from other studies of the famine. However, by mitigating the attenuation bias caused by measurement error and selection, our estimated impact of early childhood exposure is larger in magnitude than most previous studies. The results can also shed some light on the impact of severe childhood malnutrition.[5] As recently as

[5]Recent studies on long run effects of health shocks during childhood include studies by Almond and Mazumder (2005), Almond et al. (2005), Berhman and Rosenzweig (2005), Black et al. (2005), Bleakley (2002), Case et al.

2004, World Development Indicators reported that, worldwide, 30% of children under the age of five are estimated to be severely malnourished.[6] Our results show that these deprivations can have large adverse long run effects even amongst a sample of "strong" individuals selected to survive severe famine. And this could in turn affect long run economic development.[7] This study also makes a methodological contribution. It shows that estimation of the impact on the upper quantiles of the distribution of outcomes is one way for addressing attenuation bias caused by selection for survival. This method is straightforward and can be easily employed for impact evaluations of other events where similar patterns of selection for survival is present.

There are several advantages to studying the long run impact of famine in the China context. The institutional causes of the Great Famine provides an unusual natural experiment. It is unlike any other famine in history with the exception of the Ukrainian famine (1932-33) in that migration was strictly restricted. This allows us to measure regional famine intensity with the cohort size of survivors with reasonable accuracy. Second, it is a relatively good setting for isolating the effect of famine. Compared to other famines, it occured in a time of relative prosperity and political stability. Unlike notable famines such as the Ethiopian famine, the Chinese famine was not a direct outcome of civil war or other types of conflict. Famine stricken regions are not known to have received any special treatment from the government which would confound an estimate of the impact of famine. For political reasons, the Chinese government actually denied the extent of the famine domestically and internationally for three decades. Finally, as a study of the long run effects of childhood malnutrition, the interpretation of the effect of the region-cohort level shocks from the Great Famine are not confounded by omitted variables related to parental heterogeneity.[8]

The paper is organized as follows. Section 2 discusses the background, the empirical strategy and the conceptual framework. Section 3 describes the data. Section 4 presents the results. Section 5 interprets the results. Section 6 offers conclusions.

(2004), Glewwe et al. (2001).

[6] Prevalence of child malnutrition is the percentage of children under five years of age whose height-for-age is more than two standard deviations below the median for the international reference population for ages 0 to 59 months. The reference population adopted by the WHO in 1983 is based on children from the United States, who are assumed to be well-nourished.

[7] The correlation between improved health status and economic factors has been found in studies by Fogel (1994), Fogel and Costa (1997), and Smith (1999). Bloom et al. (2001) find a correlation between longer life expectancy and higher economic growth rates. Weil (2005) finds that 26% of the cross-country variation in income can be explained by differences in health.

[8] If "bad" parents invest less in children's nutrition and education, then the correlation between the outcomes will reflect parental preferences rather than the causal impact of malnutrition on educational attainment.

2 Background

Officially, the cause of the famine, called the "three years of natural disasters" (*san nian zi ran zai hai*), was a fall in grain output due to bad weather. However, recent studies have argued that very little can be explained by weather.[9] Today, it is widely accepted that although there was a fall in output, the extent of the famine was largely driven by a set of misguided policies.[10] Specifically, over-procurement of grain from rural areas is a major contributor to the famine. Over-procurement in 1959 led to a decrease in nutrition intake in rural areas, which in turn led to a decrease in rural workers' physical capacity to produce grain. The reduction in work capacity along with the consumption of inputs such as seeds in the winter of 1959 prolonged the famine. In 1960, the central government had decreased procurement. The famine ended in 1961, when grain reserves were distributed to aid stricken regions. Production recovered to pre-famine levels in few subsequent years. The government is not known to have targeted any subsidies or compensation programs towards famine stricken regions after the event. This is not wholly surprising given that the government has typically tried to minimize people's perceptions of the famine's severity (Li and Yang, 2005). Figure 1 plots average county level cohort sizes by birth year from the 1990 Population Census. The most severe reductions in cohort size are for those born close before and during the famine. The reduction in cohort size is several orders of magnitude larger for agricultural populations relative to non-agricultural populations.

Meng and Qian (2009) expanded on previous studies on the causes of the famine. They argue that procurement targets were set in such as way that a drop in grain production could have generated a large famine even if production exceeded subsistence; and that this would be excarebated by the local over-reporting of production which occured under pressure from the central government. The details of their arguments are not relevant for the purposes of this paper. Hence, we do not discuss them for the sake for brevity. The important point for this paper that they notice a strong negative correlation between grain production capacity and famine intensity. They use historical data to show that this pattern is consistent with a procurement system where grain procurements (or over-reporting of production) are proportional to past production.

This rather surprising pattern of the distribution of famine is consistent with the observation of journalist, Jasper Becker (1996), who noted that unlike previous famines in China, the Great

[9] For example, see Li and Yang (2005), and Meng and Qian (2009).

[10] The suspected causes of the famine include labor and acreage reductions in grain production (e.g., Peng, 1987; Yao, 1999), implementation of radical programs such as communal dining (e.g., Yang, 1996; Chang and Wen, 1997), reduced work incentives due to the formation of the people's communes (Perkins and Yusuf, 1984), and the denial of peasants' rights to exit from the commune (Lin, 1990). Lin (1990) argues that the removal of exit rights destroyed reduced work incentives for shirkers, and hence decreased overall grain production. See Li and Yang (2005) and Meng and Qian (2008) for a detailed discussion.

Famine was most severe in grain rich regions rather than the traditional famine belt. Figure 2, which we take from Meng and Qian (2009), plots the coefficients of the interaction terms of grain suitability and birth cohort dummies from a regression of county level cohort size in 1990 on these interactions, birth county and birth year fixed effects.[11] Using cohort size in 1990 as a measure of survival, this shows that for agricultural populations who were affected by the grain procurement system, grain suitability in the county of birth as predicted by natural conditions is negatively correlated with survival for those born closely before or during the famine. For non-agricultural populations living in the same counties but who were not affected by the grain procurement system, grain suitability is uncorrelated with survival for non-agricultural populations. We will use this peculiar mechanically-generated pattern in regional famine intensity to create our instrumental variables.

2.1 Empirical Strategy

Our strategy exploits cross sectional and cohort variation in famine exposure. Individuals living in regions that had intense famines will suffer more deprivation. Exposure to famine is likely to have different effects depending on the age of the individual. The elderly and the very young are most likely to perish. This could be because they are biologically more vulnerable or because households decide to allocate more food to individuals who can work and bring in more food or income. Because we can only observe survivors thirty years after the fact, our study focuses on those who were in-utero or very young children at the time of the famine.

We treat exposure during early childhood separately from exposure in-utero. Malnutrition is likely to have different effects at different stages of development. For very young children, exposure to famine will cause severe malnutrition and decrease the quality of care received from adults. For those who were in-utero, exposure to famine is transmitted through malnutrition of the mother and during infancy. Moreover, the attributes that determine survival is different for the two groups. Both groups will be selected based on characteristics that determine one's

[11] They estimated the following equation for the population of agricultural households and non-agricultural households seprately. Figure 2 plots the vectors of coefficents, $\hat{\beta}_t$.

$$\ln pop_{ct} = \sum_{t=1943}^{1966} \beta_t (\ln grain\_suitability_c) \times biryr_t) + \alpha + \gamma_c + \delta_t + \varepsilon_{it}$$

$\ln pop_{ct}$, the natural logarithm of the cohort size of individuals born in county c and birth year t is a function of: the interaction terms between the per capita area suitabile for grain cultivation in birth county c, $grain\_suitability_c$, and dummy variables for being born in birth year t, $biryr_t$; and birth cohort dummy variables; county fixed effects, γ_c; and birth year fixed effects, δ_t. The reference group is comprised of individuals born in during 1930-1942. This group and all of its interactions are dropped. β_t is the correlation between grain suitability and cohort size for those born in year t. The coefficients and standard errors are reported in Appendix Table A1.

own survival. For the in-utero group, parents' decisions to have children when there is a famine also play a role. Overall fertility rates were extremely low during the famine, hence children born during the famine are likely to be very different on average than children born prior to the famine.

Our main estimating equation is the following.

$$\begin{aligned} Y_{ct} &= \beta_{54-58}(\ln fampop_c \times born54\_58_t) + \beta_{59-61}(\ln fampop_c \times born59\_61_t) \\ &\quad + \gamma_v + \rho_t + \varepsilon_{vt} \end{aligned} \tag{1}$$

The outcome for individuals born in county c during year t is a function of: the interaction between the intensity of the famine in that county measured as the natural logarithm of the average cohort size of individuals born during 1959-61, $\ln fampop_c$, and a dummy variable for whether that individual was born during 1954-58, $born54\_58_t$; the interaction between the intensity of famine and a dummy variable for whether she was born during the famine, $born59\_61_t$; county fixed effects, γ_c, and birth year fixed effects, ρ_t. Individuals born before 1954 and after 1961 form the reference group. They and their interaction terms are dropped. Standard errors are clustered at the province$\times$year level. Each observation is a county-birth year cell. The outcomes are the means of each cell. The cell size is used to weigh the regressions by population. The coefficients and standard errors are numerically identical to regressions with individual level data.

Like differences-in-differences, changes across cohorts that affect different regions similarly are controlled for by the comparison across regions. Cohort invariant differences between regions are controlled for by the comparison across cohorts. For example, if regions with bad institutions are more prone to famines and institutions do not change over short periods of time, then differences in institutions will be controlled for by region fixed effects.

There are several reasons for the OLS estimate to under-state the true impact of exposure to famine. First, cohort size may measure famine intensity with error. Since measurement error is most likely random, this will attenuate the OLS estimate. Second, selection bias suggests that the mean of the outcomes in the control group are below the mean of the group of individuals who are "naturally equivalent" to the treated group. Alternatively, OLS may also over-state the true effect. If famine was more severe where local politicians, seeking promotion, delivered more grain to the upper levels of government, then these same politicians may also have implemented other policies that had adverse effects on the population.

To address the problems of measurement error and endogeneity, we instrument for famine intensity with the interaction between non-famine grain production and birth year. Three facts are exploited: 1) the grain procurement system caused the famine to be more severe in regions that typically produced more grain; 2) children who were younger at the onset of the famine were more vulnerable to disease and malnutrition; and 3) children born after the famine were not exposed. We proxy for non-famine grain productivity with the per capita area suitable for grain production as predicted by geographic and climatic suitability, and observed per capita area of grain sown in 1997. This allows us to avoid misreporting issues related to historical production numbers (which are only available at much more aggregate levels). The instruments are the interaction terms between the grain suitability of the county of birth and birth cohort dummy variables; and the interaction terms of the per capita grain sown area in the county of birth and birth cohort dummy variables. Only the interaction terms can be interpreted as exogenous. Using both sets of proxies for non-famine grain production as instruments produce similar 2SLS coefficients as when using only one set. But using both increases the precision of the estimates. For brevity, we only report the 2SLS estimates from when both sets of the instruments are used.

We estimate two first stage equations for the two endogenous independent variables: $\ln fampop_c \times born_t$, where $t = \{54-58\ ,59-61\}$.

$$\begin{aligned}
\ln fampop_c \times born_t \;=\; & \sum_{t=54-58}^{59-61} \beta_t \ln(grain_pcsuit_c) \times born_t \\
& + \sum_{t=54-58}^{59-61} \beta_t \ln(grain_pcprod97_c) \times born_t \\
& + \alpha + \gamma_c + \delta_t + \varepsilon_{ct}
\end{aligned} \tag{2}$$

The key identification assumption is that suitability for grain production and the adult outcomes of famine survivors in 1990 are not jointly determined by some omitted variable. The most likely problem is that areas that produce more grain normally would recover from the famine sooner. This may be because in non-famine years, there is more food. Individuals born in those regions are better nourished prior to the famine and may be better able to recover. And after the famine, when production recovered, the local population will once again receive better nutrition. The first possibility is not very likely since for many years before and after the famine, agricultural households were left with little surplus (Perkins, 1966). Alternatively, because these regions are typically richer, they may have better institutions or public goods (e.g. provision of health and education infrastructure) that mitigates the negative effects of famine and aids in the subsequent recovery. If this is true, then the most likely direction of the bias is towards zero. Survivors in places with better schools and health care are likely to recover better and faster.

We are able to partially check whether this assumption is true to the extent that the institutions that can mitigate the effects of famine for agricultural and non-agricultural households are provided for at the county level. We can compare the effect of grain suitability for agricultural households who were under the procurement system to non-agricultural households living in the same county who were not under the grain procurement system. Figure 2 shows that grain suitability had no effect on cohort sizes for the latter. These results should be interpreted loosely since most public goods provision in China are specific to rural or urban households. However, the clear distinction between the effect of grain suitability on agricultural and non-agricultural households lends credibility to our strategy.

Note that despite being isolated from the full extent of the famine, non-agricultural populations cannot be used as a comparison group for rural areas in the second stage because they are subject to different policies regarding the access to labor market and schooling opportunities. These differences also differed by cohort and hence can produce confounding results.[12]

The instrumental variables cannot address attenuation bias caused by selection. "Survival of the fittest" is a general concern in estimating the impact of severe deprivations on survivors. For China's Great Famine, findings from Gorgens et al. (2002) indicate that individuals with higher stature were more likely to survive. This means that the estimated impacts on average height may be attenuated by selection bias. And if the underlying determinants of other outcomes such as labor supply are correlated with the latent indicators of health measured by these outcomes, then the estimated impact on those other outcomes will also be attenuated. Assuming that it is the strongest (or tallest) that survive, the attenuation bias will be smaller in magnitude for individuals on the higher quantiles of the distribution of outcomes. Those individuals have comparable control groups. For individuals in the lower quantiles, the strategy compares individuals in the control group with those in the treatment group who would be higher on the distribution absent the famine-induced selection.

Therefore, we can address the problem of selection by estimating the impact of famine on the upper quantiles of the distribution of outcomes. We calculate the sample means of the different quantiles for every birth year-county cell and repeat the same estimation. If height, weight and weight-for-height are correlated with the determinants for survival, then estimates on the upper quantiles (e.g. 90th percentile) will be less attenuated by selection relative to

[12] Another difference is the Cultural Revolution (1966-1976) which targeted non-agricultural populations (Unger, 1982; Meng and Gregory, 2005; Giles et al., 2006). The Cultural Revolution caused widespread closings of schools for approximately three years (1966-1969). Children who survived the famine will be in school during the Cultural Revolution. Hence, comparing the famine cohort between urban and rural areas would compare outcomes for two different treatments rather than a treatment and a control. We therefore restrict the sample to individuals living in rural areas. Our empirical strategy will be robust to the occurrence of school disruptions in rural areas as long as school closings were not correlated to famine intensity.

estimates for the lower quantiles. If those determinants are also correlated with education and labor supply, a similar logic implies that estimates at higher quantiles will also be more accurate for those outcomes. Since famine intensity, the treatment variable, is only measured at the birth county and birth year level, this aggregation does not change the independent variables. The only difference between the analysis on the quantiles and the analysis on the mean is in the outcome variables. This strategy is similar to Quantile Regressions and Quantile Instrumental Variables in spirit. It has the advantage that we are able to control for fixed effects.

Using this method to address selection assumes that differences in the estimated effects of famine exposure across quantiles is due to selection bias rather than to heterogenous treatment effects. We have no way of testing this assumption directly. A more detailed discussion is provided later in the paper.

2.2 Conceptual Framework

Exposure to famine at young ages affects adult health and labor market outcomes through two main channels. First, it adversely affects childhood health, which is a product of genetic endowment, fetal health (in-utero nutrition), nutrition and other forms of investment (e.g., health care). The famine potentially also reduced the quality and/or quantity of other forms of investment into children by reducing the health status of parents. Childhood health can in turn affect adult outcomes directly and indirectly (Kuh and Wadsworth, 1993), as poor childhood health can affect adult health directly, which consequently can affect work capacity and labor supply. Barker (1995) and Ravelli et al. (1998) have found that nutrition in-utero can affect health status in middle age, through its impact on chronic conditions such as coronary heart disease and diabetes, in a phenomenon widely known as the "Barker Hypothesis".[13] Poor childhood health could also decrease educational attainment by decreasing returns to education or by increasing the costs of school attendance (Curie and Madrian, 1999; Miguel and Kremer, 2004). This may in turn affect labor supply and/or wages later in life.[14] Second, exposure to famine could potentially have a positive effect by reducing the cohort size of exposed individuals,

[13] Experimental work by Ozanne and Hales (2004) using laboratory mice find that lab mice that are underfed in-utero but who are well-fed after birth catch up rapidly. However, they die earlier than mice that are also well-fed in-utero.

[14] Poor health in children has been associated with lower education and/or labor market outcomes in the U.S. (Case et al., 2004; Doblhammer, 2002), Canada (Currie and Stabile, 2004), Great Britain (Case et al., 2002; Kuh and Wadsworth, 1993; Marmot et al., 2001) and many developing countries (Behrman, 1996; Bleakley, 2002; Brinkley, 1994; Glewwe and Jacoby, 1995; Glewwe et al., 2001; Miguel and Kremer, 2004; and Strauss and Thomas, 1998). See Curie and Madrian (1999) and Curie and Hyson (1998) for a review of studies linking health to educational attainment and labor market outcomes. The latter focuses on the effects of low birth weight. Smith (1999) shows a strong correlation between reported health and income of adults in the U.S. Reduced height has been associated with lower education and labor market outcomes in many countries (Maccini and Yang, 2005; Perisco et al., 2002; Strauss and Thomas, 1998; Schultz, 2001; Schultz, 2002; Strauss and Thomas, 1998).

hence reducing labor market competition and competition for family resources.[15] This paper will estimate the net effect of exposure to famine: the sum of the adverse effect of malnutrition and the potentially positive effects from smaller cohort sizes.

In addition to the health channels that we describe above, famine also affects those exposed in utero by affecting parents' decisions to bear children. The extremely low fertility rates during the famine suggests that very few parents were able to or choose to have children. Because we cannot observe parents of survivors, we cannot investigate the attributes driving this selection. Nor will we be able to reliably predict how these attributes would affect the impact of famine exposure. Therefore, absent wild speculation, we cannot say more about the differential impacts between exposure in-utero and exposure during early childhood. The most we can do is to allow the effects for in-utero exposure to differ from the effects of early childhood exposure.

3 Data

This paper constructs a panel of birth cohorts by matching several existing data sets: the 1% sample of the 1990 Population Census, the 1989 China Health and Nutritional Survey (CHNS), the 1% sample of the 1997 Agricultural Census, and GIS data on suitability for grain cultivation which we construct using data from the Food and Agricultural Organization's GAEZ database.

The 1990 Population Census contains 32 variables including birth year, region of residence, and how long an individual has lived in a region of residence. We use this data to count the number of "missing people" from the 1959-61 cohort. The average cohort size across these three birth years for each county is our measure of famine intensity. The large sample size of the Census gives us more precise measures than other smaller data such as the CHNS. We restrict the sample to individuals who report as living in the same county for five or more years and interpret their county of residence as their birth county. For this, we assume that there is little migration between when the famine occured and 1990. Our assumption is supported by studies on migration in China, which find that strict migration controls were well enforced until the early to mid 1990s.[16]

[15] Easterlin (1980) discusses how the size of a generation affects the personal welfare of its members through family and market mechanisms. See Becker and Lewis (1973), Becker and Tomes (1976), Galor and Weil (2000), Hazan and Berdugo (2002) and Moav (2005) for theoretical discussions of the quantity-quality tradeoff; and see Angrist et al. (2006), Black et al. (2004), Qian (2006), Rosenzweig and Zhang (2006), and Schultz (2005) for recent empirical evidence on the quantity-quality tradeoff.

[16] Migrants had no access to government-controlled food rations, housing, schools and medical care once they left their registered homes. The first wave of rural migration did not occur until the early to mid 1990s, during the urban construction boom, and most of those migrants were young adult men. Consequently, it is highly unlikely that the results of this paper are confounded by migration. Using data from China's Ministry of Agriculture RCRE's National Fixed Point Survey for 1986-90, we find that the probability of having a household member work away from the home village is very low and similar for regions that suffered very different levels of famine intensity. They are not reported in the paper. Also see West and Zhao (2000) and De Brauw and Giles (2006)

Our outcome data come from the 1989 CHNS. It uses a random cluster process to draw a sample of approximately 2,520 rural households with a total of 10,534 individuals across seven provinces that vary substantially in geography, economic development, public resources, and health indicators. The survey includes a physical examination of all individuals as well as information on labor supply, work intensity and wages. For this study, we use height, weight and weight-for-height (WFH). The CHNS also report diastolic and systolic blood pressure. We use these to create a dummy variable for whether an individual has hypertension (>140/90mmHg). These data have the advantage that they are measured by the surveyor and hence avoids measurement problems from self reported data. The CHNS also reports education level, which we translate to a continuous variable for the years of educational attainment. The wage data is not used in this study because majority of rural workers are involved in non-wage earning production and because wages in China during this period do not reflect marginal productivity. Instead, we use labor supply to measure the physical capacity to work. It is calculated as the sum of the number of hours per week spent in wage labor, agricultural labor (e.g. farming, gardening, tending livestock, fishing), and home production.

We create two proxies for regional grain productivity. The first measure is the per capita area suitable for cultivating rice and wheat, the two main crops of the procurement targets. This is calculated using the model and data provided by the United Nations Food and Agriculture Organization's (FAO) Global Agro-Ecological Zones (GAEZ), 2002 database. The data are the result of over twenty years of research and are the product of a joint collaboration between the FAO and the International Institute for Applied Systems Analysis (IIASA). Their measure is based purely on the biophysical environment of a region and it is not influenced by which crops were actually adopted in an area. Factors that are easily affected by human actions, such as soil pH, are not parameters in their model. Nunn and Qian (2008) provide a detailed description of the construction of this data and how to calculate suitability measures at the regional level. We use the same method. The GAEZ database provides data on suitability at a 50km×50km grid cell level. We are able to choose the level of inputs that the calculation is based on. Great Leap Forward policies required collectives to not use chemical fertilizers. And to the best of our knowledge, use of heavy machinery such as tractors was extremely rare. Hence, our chosen level of inputs allows for rain-fed irrigation but no heavy machinery or chemical fertilizers. We assign a cell with a value of one indicating that it is "suitable" if it can produce 40% or more of the maximum output. This threshold was arbitrarily chosen. The measure of suitability is the fraction of cells in a county that is suitable. We multiply this by the total area of the county and

for detailed discussions on migration.

then divide by county population in 1990 to obtain per capita area suitable for grain production. Moderately changing the threshold will not affect the 2SLS estimates. A higher threshold (e.g. suitable defined as cells that can produce 60% or more of the maximum output) will mean that the suitable land can produce more grain per grid. However, this will be offset by the fact that now there will be fewer grids in a regions that are classified as suitable. Our second proxy for non-famine production is the per capita area of rice and wheat sown as reported in the 1997 Agricultural Census. This is the only source of county level data that uses a consistent measure across regions. For each county, we divide the area reported as sown for rice or wheat by county population in 1997.

The data are collapsed and matched by county of residence and birth year. All regressions will be weighted by the county-birth year cell size. To mitigate potentially confounding effects from the Cultural Revolution, which was primarily an urban disturbance and rural-urban migration, we excludes cities. The CHNS data and the 1990 Population Census matched for seventeen counties within seven provinces. The number of individuals in each county-birth year cell is retained so that we can weight our regressions by population. Figure 1 plots the cohort sizes of agricultural and non-agricultural populations by birth year. The vertical line indicates the beginning of the famine. It shows a significant decrease in cohort size for those born closely before and during the famine in both urban and rural areas. For those born before the famine, the decrease most likely reflects increased mortality due to the famine. For those born during the famine, the decrease is likely to reflect a combination of increased mortality and reduced fertility. This only reflects a part of the estimated 16.5-30 million death during the famine because we cannot observe the mortality of the elderly in the 1990 data. The elderly, like the very young, are more vulnerable to health shocks and experienced higher mortality rates relative to other age groups. But because this cohort would be approximately 100 years old in 1990, they do not appear in the 1990 Census.

For ease of interpretation, we measure famine intensity as the logarithm of the average cohort size of those born during 1959-61. Figure 3A is a histogram of this measure. It shows that there was much variation in famine intensity. For illustrative purposes, we can also measure 1959 cohort sizes as a fraction of the average cohort size of those born in 1952-54. Figure 3B is a histogram of this normalized measure of famine intensity. In some counties, there were no births during 1959, resulting in this ratio being zero. In other counties, there was little difference in the 1959 cohort size relative to previous cohort sizes. The geographic dispersion of famine can be observed in Figure 4, where we map the main famine measure, average cohort size for 1959-61, by county. Lighter shades reflect more intense famines. This map highlights two facts. First, although southern provinces are more suitable for grain production, neighboring counties within each province can have very different famine intensities. Second, the famine was most severe in regions that are best suited for grain production (e.g. southern provinces in China).

Table 1 shows the summary statistics of our outcome variables by birth year. All of these variables are from the 1989 CHNS data, except for years of schooling, which is obtained from the 1990 population census data. Height is a commonly used measure of the stock of nutritional investments during the fetal and childhood stages of life (Fogel et al., 1982; Fogel, 1994; Steckel, 1986; Micklewright and Ismail, 2001). Average height is approximately 160 cm, four centimeters less than the average height of the same cohort in Japan. Weight, weight-for-height and BMI are crude measures of the body's inability to retain body mass after recovering from a severe nutritional shock. This could be due to the inability to absorb nutrition after suffering severe shocks to the gastrointestinal system such as severe repeated diarrhea during early childhood (Cutler, Deaton and Lleras-Muney, 2006). The sample means for both of these outcomes are similar to comparable cohorts from Japan. Individuals have on average 5.3 years of education. The main economic outcome we examine is total hours worked per week. Adults in the sample work over 73 hours per week, on average. Comparing the outcomes of individuals born prior to or during the famine in columns (2), (3) and (4) to those born after shown in column (6) do not show cohorts affected by the famine as systematically worse off than those not affected. This most likely reflects the strong secular trends in improvement in nutrition, sanitation and public health during this period.[17]

4 The Long Run Impact of Exposure to Famine

4.1 OLS

The estimates from equation (1) are shown in Table 2. Recall that a positive coefficient reflects adverse effects of the famine since famine reduced cohort size. To address the attenuation bias from positive selection for survival, we estimate this equation on the 90th percentiles, the means and the 10th percentiles of the distribution of outcomes. For almost all outcomes, the estimates are larger in magnitude and more precisely estimated for the 90th percentile shown in Panel A. For example, columns (1)-(3) show that for the early childhood cohort, the estimated correlation between famine exposure for the 90th percentile in Panel A are over twice the magnitude as the

[17] Although mst of the outcome variables are extracted from the CHNS data where the sample size of each county-cohort cell is quite small, we weight them using the population census county-birthyear cell size in the regression. On average, each birth-year-birth-county cell contains 9,276 individuals. For those born before the famine (1952-54), there are on average 10,249 individuals per birth year county. And for those born during the famine (1959-61), there are on average 6,614 individuals per county per birth year.

estimated correlation for the sample mean shown in Panel B. The difference is larger when compared to the 10th percentile shown in Panel C.

We focus the discussion of the results on the 90th percentile in Panel A. For the in-utero cohort, the estimates show that exposure to a famine that decreased average famine cohort size by 1% reduced height by 0.024%, weight by 0.053%, and educational attainment by 0.174%. These estimates are statistically significant at the 5% and 1% levels. The coefficients for WFH, BMI and labor supply have similar signs but are not statistically significant. For the early childhood cohort, exposure to a famine that decreased the famine cohort size by 1% is associated with a 0.027% reduction in height, a 0.1% reduction in weight, a 0.026% in WFH and a 0.01% decrease in BMI. These estimates are statistically significant at the 1% and 5% level.

4.2 First Stage Estimates

The estimates from equation (2) are shown in Table 3. Columns (1) and (4) show the correlation between the first set of instruments and the endogenous variables. Columns (2) and (5) show the correlation between the second set of instrument and the endogenous variables. Columns (3) and (6) shows the first stage estimates using all of the instruments. The instruments are strongly significant and inclusion of both sets of instruments in columns (3) and (6) does not change the signs or greatly affect the first stage coefficients.

4.3 Two Stage Least Squares

Table 4 shows the 2SLS estimates estimated for the 90th, mean and 10th percentiles of the distribution of outcomes.[18] Like the OLS estimates, the effects of the famine are more adverse and more precisely estimated for the 90th percentile. Once again, we focus our discussion on those results in Panel A. For the in-utero cohort, exposure to a famine that reduces famine cohort size by 1% decreases height by 0.047%, weight by 0.065%, WFH by 0.018%, BMI by 0.008%, and educational attainment by 0.238%. The estimates are statistically significant at the 1% and 5% level with the exception of those for WFH and BMI, which are significant at the 20% level. For the early childhood cohort, exposure to a famine that decreased the famine cohort by 1% caused a 0.045% reduction in height, a 0.138% reduction in weight, a 0.033% reduction in WFH, a 0.007% reduction in BMI, and a 0.387% reduction in labor supply. These estimates are statistically significant at the 5% and 1% level with the exception of the estimate for BMI, which is significant at the 20% level.

[18] *We obtain 2SLS estimates with similar magnitudes using only the instruments constructed from per capita suitability. Including the second set of instruments improves the precision of the estimates. For brevity, the paper reports only the latter.*

4.4 Selection or Heterogeneous Effects?

Based on previous studies, we entered this study with the presumption that there is non-random selection for survival (Gorgens et al., 2002; Deaton, 2008). The finding that the estimated effects are more adverse and larger in magnitude for individuals on the 90th percentile of the distribution of outcomes is consistent with this. However, the same pattern could exist if there are heterogenous effects of exposure to famine such that individuals on the upper quantiles of the distribution are more adversely affected. To the best of our knowledge, there is no evidence from the medical or epidemiological literature arguing this case. And without a concrete prediction of the pattern of heterogenous effects across quantiles, we will not be able to directly test for them or to rule them out empirically. However, we can look for suggestive evidence in the data based on a few reasonable, albeit crude assumptions.

If those that survived were on average the "strongest" or "most able" individuals of the population, and these attributes are correlated with the outcomes of interest, then we would expect the estimated effect of exposure to vary for the lower quantiles but to stabilize for the upper quantiles. In contrast, if the results on the 90th percentile are driven solely by heterogenous effects, and the occurrence of heterogenous effects is symmetric for both tails of the distribution, then the effects should be very different for individuals on the lower *and* upper quantiles relative to those on the mean. Figures 5A and 5B plot the estimated effects and their 95% confidence intervals for exposure on labor supply for the early childhood cohort. The coefficients and standard errors for these estimates are shown in Appendix Table A2. The OLS and 2SLS estimates exhibit similar patterns. The estimated impact of exposure is monotonically increasing with quantile. However, the rate of increase is much lower above the 30th percentile. This does not rule out the presence of heterogenous effects. But if the pattern of outcomes from heterogenous effects are relatively more symmetric than that from selection, then the results are consistent with presence of selection.

4.5 Average Effects

In Table 5, we calculate the average effect for those exposed to famine during early childhood. The 2SLS estimates for the effect on the 90th percentile from Table 4 Panel A columns are shown in rows (A) and (B) of Table 5. We use only the estimates that were statistically different from zero at the 5% or 1% levels. The estimates reveal the effect of exposure to a famine which reduced the population of the 1959 cohort size by 1%. The average effect of famine exposure is the product of the 2SLS estimate and the average percentage reduction of the 1959 cohort size. We measure the latter as one minus the ratio of the average 1959-61 cohort size cohort size to the 1952-54 cohort size. Hence, if we assume that 1959-61 cohort sizes would be equal to 1952-54 cohort sizes, then we calculate that the famine reduced the 1959-61 cohort size by 36%. Row (C) shows that on average, exposure to famine reduced height of the in-utero cohort by 1.7%, weight by 2.3% and educational attainment by 8.6%. Row (D) shows that for the early childhood cohort, exposure on average decreased height by 1.6%, weight by 5%, WFH by 1.2% and labor supply by 13.9%. The average effect in levels are shown in Row (F) and (G). They are the products of the sample means for the 90th percentile shown in Row (E) and the average percentage effects. They show that in-utero exposure on average reduced height by 2.8 cm, weight by 1.42 kg, and educational attainment by 0.6 years. For the early childhood cohort, exposure on average decreased height by 2.7 cm, weight by 3.03 kg, WFH by 0.004 kg/cm, and labor supply by 12.7 hours per week.

5 Interpretation

The main finding of this study is that in-utero and childhood exposure to famine significantly reduces adult health outcomes and labor supply. There are several caveats to the interpretation of the results. Our strategy attempts to capture the effect of exposure to the overall famine. As an estimate of the impact of childhood malnutrition, our results should be interpreted as the lower bound of the magnitude of adverse effects. While we are able to address part of the attenuation bias caused by selection for survival, we are not able to disentangle potentially offsetting effects from being part of a small cohort.

The second caveat arises from our using the results on the 90th percentiles to calculate the average effects of exposure. As we discussed earlier, we cannot empirically rule out the presence of heterogenous effects. This does not affect the validity of our estimates as the estimated impact for each quantile. But it means that caution should be born in mind when extrapolating the estimated effects of the higher quantiles for other segments of the population. A casual look at Figures 5A and 5B suggests that one conservative interpretation of our calculated average effects is that they are relevant for individuals above the 30th percentile.

One result which we examine and for brevity did not report in the paper is the effect on hypertension, the most commonly used indicator of heart disease. It can be used to investigate the Barker Hypothesis which predicts that individuals who suffer severe malnutrition will recover and appear healthy until the middle of their life, when they will be more likely to suffer from conditions such as coronary heart disease. We did not find any evidence that exposure to famine increased the rates of hypertension for either those who were exposed as children or those that were exposed in-utero. In addition to the 1989 survey, we used a later wave of the CHNS in

1997, when those born during 1954-61 would be 36-43 years of age. One possible explanation is that the effects of famine are non-linear in age so that we may only observe the effects as the survivors reach their 50s and 60s. We plan to use future waves of the CHNS to investigate this hypothesis. Similarly, we plan to use a similar empirical strategy with mortality inferred from comparing the 2000 and 2010 Chinese Population Censuses to examine the effect of famine exposure on life expectancy.

6 Conclusion

This paper attempts to overcome significant empirical difficulties to estimate the causal impact of childhood exposure to famine. Our estimates show that exposure in-utero and as very young children can have severe adverse consequence on adult health, educational attainment and labor supply. The instrumental variables strategy we use comes from the specific institutional context of China's Great Famine and cannot be easily used to evaluate the impact of other famines. However, examining the impact on the upper quantiles of the distribution of outcomes to address positive selection for survival can be easily applied to any context that experienced similar patterns of selection. It is important to note that China's Great Famine is far from the most severe in terms of mortality as a fraction of total population. Applying this method to famines with higher mortality rates and more selection should make a larger difference than in our context.

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Table 1: Descriptive Statistics

	Birth Cohort									
	All (1)		Before 1955 (2)		1955-1958 (3)		1959-61 (4)		After 1961 (5)	
Variable	Obs	Mean	Obs	Mean	Obs	Mean	Obs	Mean	Obs	Mean
Height	337	159.546	171	159.192	59	159.367	39	158.802	68	161.020
		(0.326)		(0.456)		(0.765)		(1.085)		(0.671)
Weight	335	55.485	169	55.673	59	55.636	39	54.644	68	55.369
		(0.346)		(0.493)		(0.908)		(1.107)		(0.636)
WFH	334	0.347	169	0.349	58	0.350	39	0.343	68	0.343
		(0.002)		(0.002)		(0.004)		(0.006)		(0.003)
BMI	334	21.768	169	21.920	58	21.935	39	21.634	68	21.324
		(0.101)		(0.140)		(0.245)		(0.377)		(0.182)
Years of Education	333	5.347	178	4.929	50	5.577	34	6.522	71	5.667
		(0.110)		(0.158)		(0.247)		(0.272)		(0.215)
Total Hours Worked Per Week	313	73.919	174	74.505	49	71.974	31	69.220	59	76.272
		(1.883)		(2.760)		(4.389)		(5.775)		(3.390)
Cell Size	337	92.763								
		(2.070)								
1952-54 Average Cohort Size	337	102.485								
		(2.749)								
1959-61 Average Cohort Size	337	66.144								
		(2.091)								

Note: Observations are county-birth year cells.

Table 2: The OLS Estimates of the Effect between Famine Exposure

Coefficients of the interaction terms between the logarithm of 1959-61 cohort size and birth year categorical variables

	Dependent Variables					
	LnHeight	LnWeight	WFH	BMI	LnEduYrs	LnTotWk
	(1)	(2)	(3)	(4)	(5)	(6)
A. 90th Percentile						
Sample Mean (Not Logged)	165.291	61.191	0.376	23.500	6.797	90.802
LnFampop x Born 1959-61	0.024	0.053	0.017	0.008	0.174	0.042
	(0.014)	(0.033)	(0.012)	(0.006)	(0.090)	(0.179)
LnFampop x Born 1955-58	0.027	0.101	0.026	0.009	-0.065	0.098
	(0.008)	(0.022)	(0.008)	(0.004)	(0.124)	(0.119)
Observations	337	335	334	334	333	313
B. Mean						
Sample Mean (Not Logged)	159.546	55.485	0.347	21.768	5.347	73.919
LnFampop x Born 1959-61	0.012	0.037	0.007	0.000	-0.048	0.033
	(0.009)	(0.023)	(0.007)	(0.000)	(0.064)	(0.158)
LnFampop x Born 1955-58	0.010	0.051	0.011	0.000	0.004	-0.005
	(0.005)	(0.017)	(0.005)	(0.000)	(0.096)	(0.098)
Observations	337	335	334	334	333	313
C. 10th Percentile						
Sample Mean (Not Logged)	153.809	50.100	0.320	20.023	4.132	57.633
LnFampop x Born 1959-61	-0.004	0.001	0.000	-0.002	-0.339	0.009
	(0.010)	(0.026)	(0.007)	(0.004)	(0.068)	(0.188)
LnFampop x Born 1955-58	-0.007	0.010	0.000	-0.001	0.012	-0.219
	(0.006)	(0.023)	(0.006)	(0.003)	(0.095)	(0.137)
Observations	337	335	334	334	333	313

All regressions control for birth year and birth county fixed effects. Regressions are population weighted. Standard errors are clustered at the province-year level.

Table 3: The First Stage Estimates of the Effect of Grain Suitability and Grain Sown Per Capita on Famine Exposure

Coefficients of the interaction terms between area suitable for grain cultivation and birth year categorical variables, and between the logarithm of per capita grain sown and birth year categorical variables

	Dependent Variables					
	Ln Fampop x Born 1959-61			Ln Fampop x Born 1955-58		
	(1)	(2)	(3)	(4)	(5)	(6)
Suitable Area x Born 1959-61	-2.699		-2.833	0.007		0.003
	(0.034)		(0.033)	(0.007)		(0.007)
Suitable Area x Born 1954-58	0.002		0.001	-2.680		-2.831
	(0.002)		(0.003)	(0.019)		(0.018)
Ln Sown Area x Born 1959-61		-46.007	-47.333		-0.898	-0.998
		(5.848)	(6.002)		(1.292)	(1.327)
Ln Sown Area x Born 1954-58		-0.090	-0.160		-49.910	-51.171
		(0.603)	(0.612)		(4.279)	(4.317)
Observations	11734	11826	11734	11734	11826	11734

All regressions control for birth year and birth county fixed effects. Regressions are population weighted. Standard errors are clustered at the province-year level.

Table 4: The 2SLS Estimates of the Effect of Famine Exposure

Coefficients of the interaction terms between the logarithm of 1959-61 cohort size and birth year categorical variables

	Dependent Variables					
	LnHeight	LnWeight	WFH	BMI	LnEduYrs	LnTotWk
	(1)	(2)	(3)	(4)	(5)	(6)
A. 90th Percentile						
Sample Mean (Not Logged)	165.291	61.191	0.376	23.500	6.797	90.802
LnFampop x Born 1959-61	0.0470	0.0645	0.0178	0.0083	0.2379	0.1119
	(0.0168)	(0.0390)	(0.0130)	(0.0068)	(0.1409)	(0.1394)
LnFampop x Born 1955-58	0.0454	0.1376	0.0325	0.0067	0.0926	0.3868
	(0.0114)	(0.0257)	(0.0083)	(0.0044)	(0.1213)	(0.1596)
Observations	337	335	334	334	333	313
B. Mean						
Sample Mean (Not Logged)	159.546	55.485	0.347	21.768	5.347	73.919
LnFampop x Born 1959-61	0.0242	0.0483	0.0071	0.0000	-0.0718	0.0180
	(0.0118)	(0.0289)	(0.0082)	(0.0001)	(0.0849)	(0.1316)
LnFampop x Born 1955-58	0.0241	0.0661	0.0135	0.0000	0.0984	0.2401
	(0.0078)	(0.0173)	(0.0049)	(0.0000)	(0.0992)	(0.1430)
Observations	337	335	334	334	333	313
C. 10th Percentile						
Sample Mean (Not Logged)	153.809	50.100	0.320	20.023	4.132	57.633
LnFampop x Born 1959-61	-0.0054	0.0063	-0.0051	0.0083	-0.5264	-0.1794
	(0.0136)	(0.0363)	(0.0086)	(0.0068)	(0.1164)	(0.1658)
LnFampop x Born 1955-58	-0.0023	0.0078	0.0000	0.0067	-0.0204	-0.0349
	(0.0089)	(0.0292)	(0.0073)	(0.0044)	(0.1265)	(0.1794)
Observations	337	335	334	334	333	313

All regressions control for birth year and birth county fixed effects. Regressions are population weighted.

Standard errors are clustered at the province-year level.

Table 5: The Effect of the Great Famine

Average effects of the famine calculated with the sample mean, 2SLS estimates on the 90th Percentile, and the average estimated intensity of famine

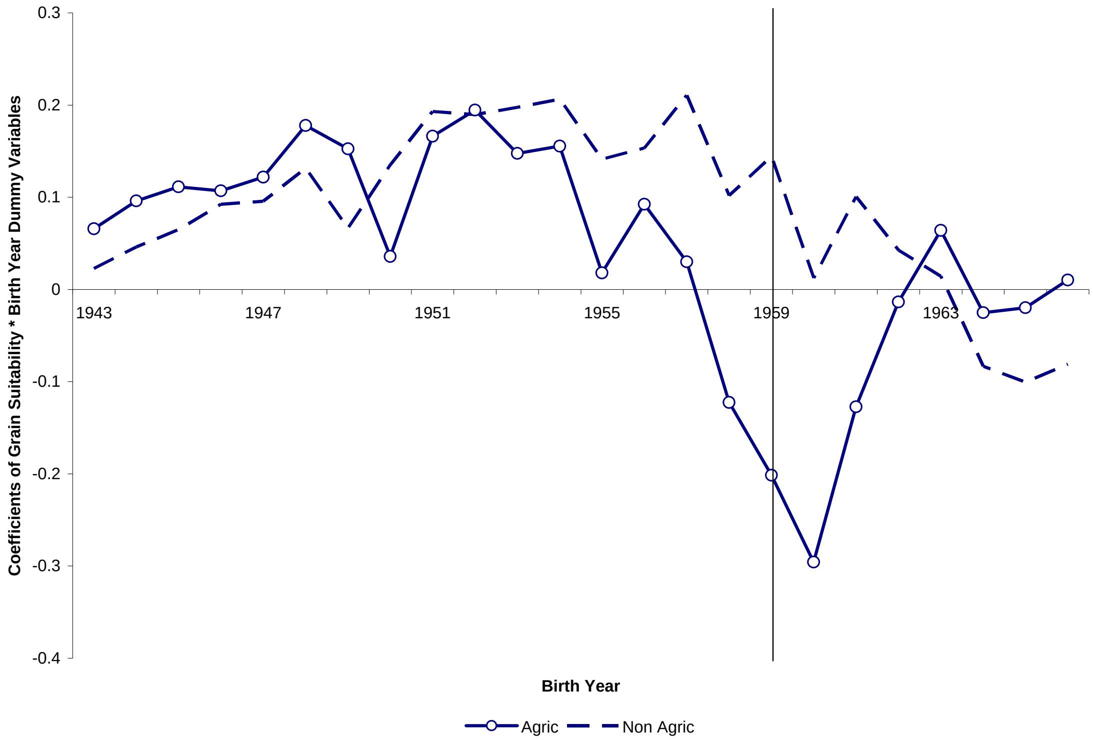
		Variables				
		Height	Weight	WFH	Edu Yrs	Total Work Hrs
		(1)	(2)	(3)	(4)	(5)
A	Coefficient for LnFampop x Born 1959-61	0.0470	0.0645		0.2379	
B	Coefficient for LnFampop x Born 1955-58	0.0454	0.1376	0.0325		0.3868
	% Effect of famine = 2SLS Coefficient x (1 - 1959-61 cohort size/1952-54 Cohort Size)					
C	In Utero Cohort: A x -0.36	-0.017	-0.023		-0.086	
D	Early Childhood Cohort: B x -0.36	-0.016	-0.050	-0.012		-0.139
	Level Effect of famine = % Effect x Sample Mean					
E	Sample Mean for 90th Percentile	165.29	61.19	0.38	6.80	90.80
F	In Utero Cohort: E x C	-2.80	-1.42		-0.58	
G	Early Childhood Cohort: E x D	-2.70	-3.03	-0.0044		-12.64

Notes: Estimates in Rows (A) and (B) show 2SLS estimates on the 90th percentile from Table 4 Panel A. These calculations assume that absent the famine, 1959-61 cohort sizes would be equivalent to the average size of 1952-54 cohorts.

Figure 1: Population by Rural/Urban and by Birth Year

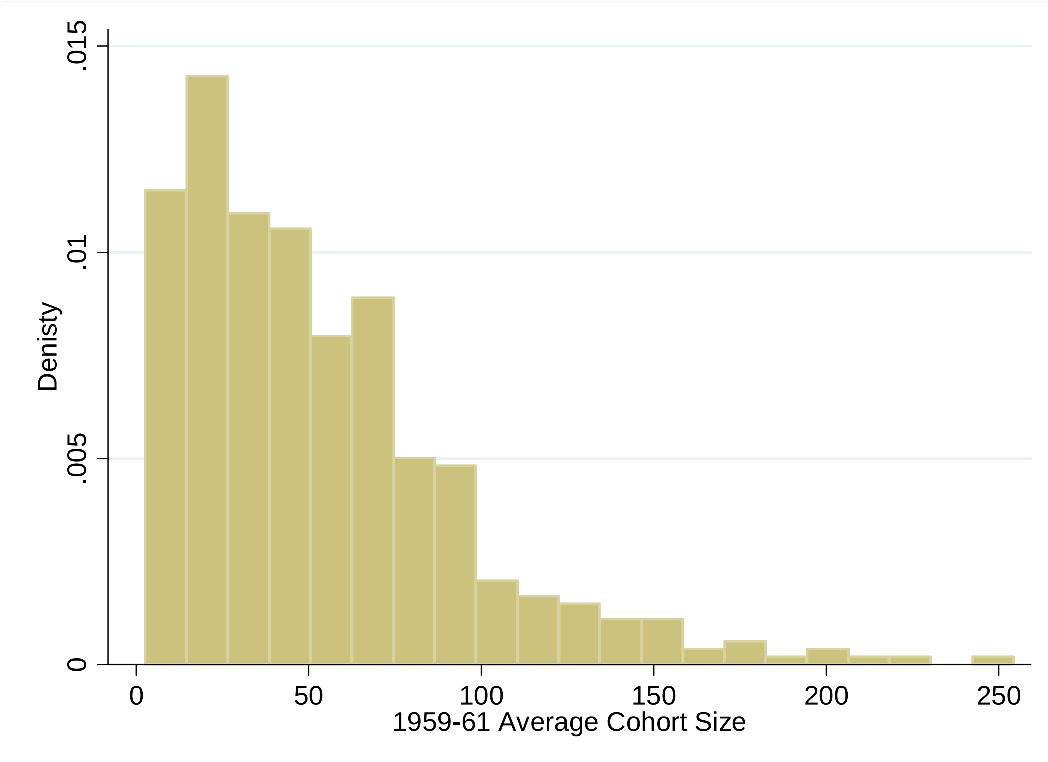
Source: Authors' Calculation

Figure 2: The Correlation between Suitability for Grain Cultivation and Cohort Sizes for Agricultural and Non-Agricultural Populations in All Counties in China
Coefficients of the interactions of the fraction of land suitable for grain and birth year dummy variables



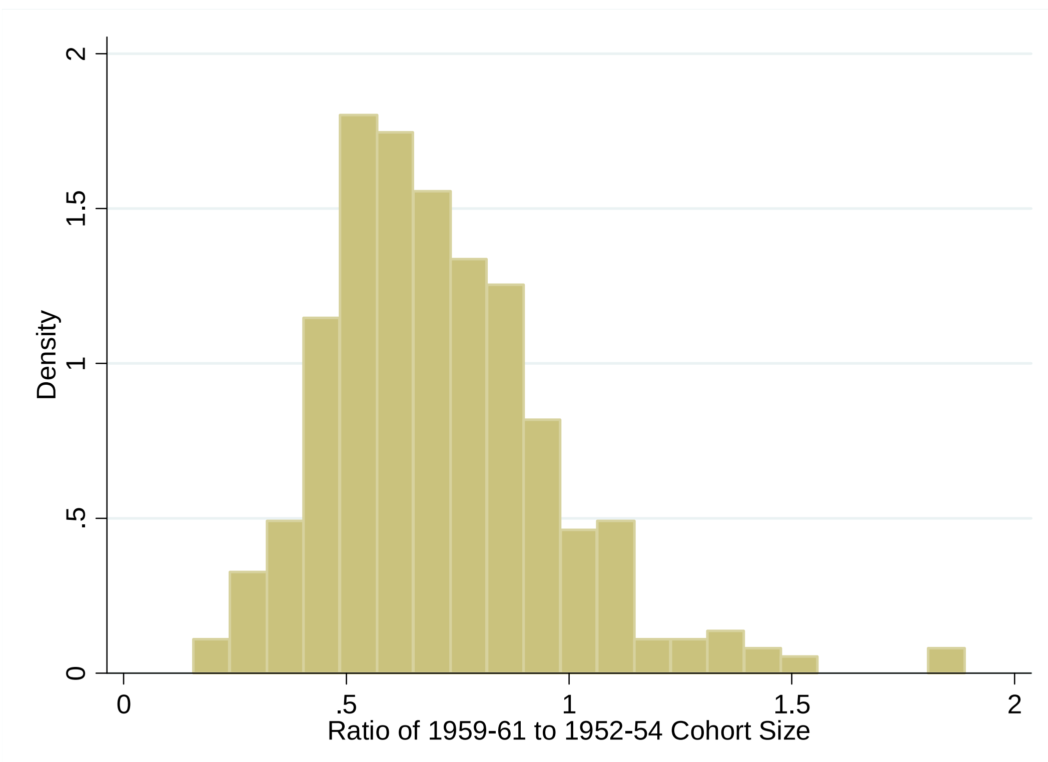
Source: Meng and Qian (2009) Note: This sample excludes cities. Counties in China contain both agricultural and non-agricultural households.

Figure 3A: Famine Intensity across Counties Measured as 1959-61 cohort size



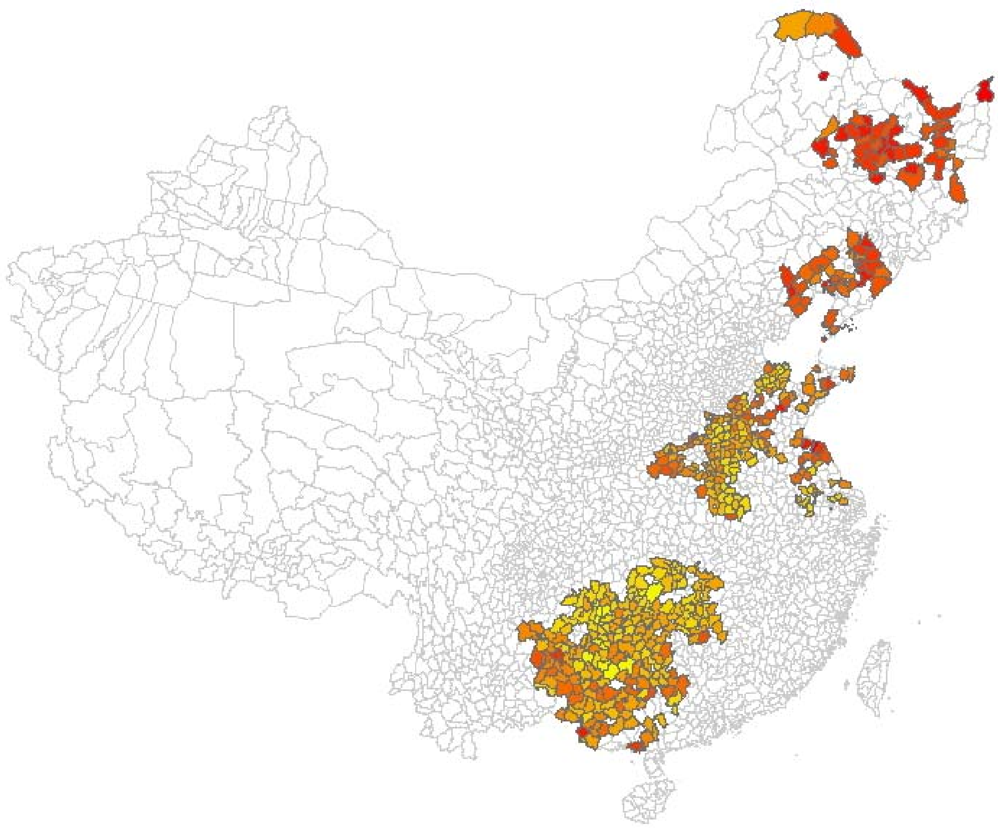
Source: Authors' calculation

Figure 3B: Famine Intensity across Counties the Ratio of 1959-61 cohort size to 1952-54 Cohort Size



Source: Authors' calculations

Figure 4: Map of County-Level Famine Intensity



Note: *Lighter* shading reflects greater famine intensity; Source: Authors' calculation

Figure 5A: Estimated OLS Effect of Exposure on Labor Supply by Quantile
Coefficients of the interaction terms between the logarithm of 1959-61 cohort size and a dummy variable for being born during 1954-58; and their 95% Confidence Intervals

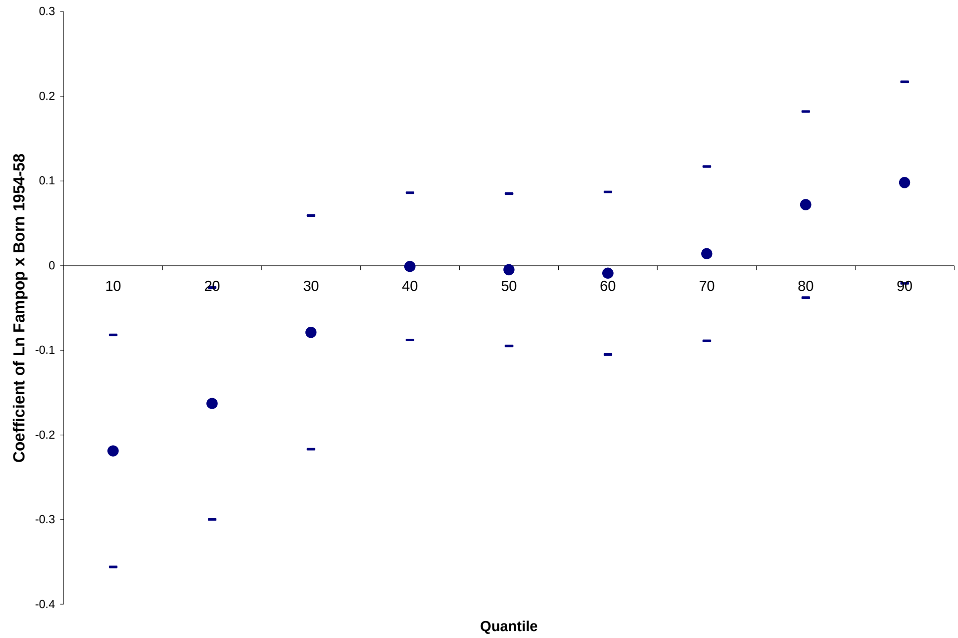


Figure 5B: Estimated 2SLS Effect of Exposure on Labor Supply by Quantile
Coefficients of the interaction terms between the logarithm of 1959-61 cohort size and a dummy variable for being born during 1954-58; and their 95% Confidence Intervals

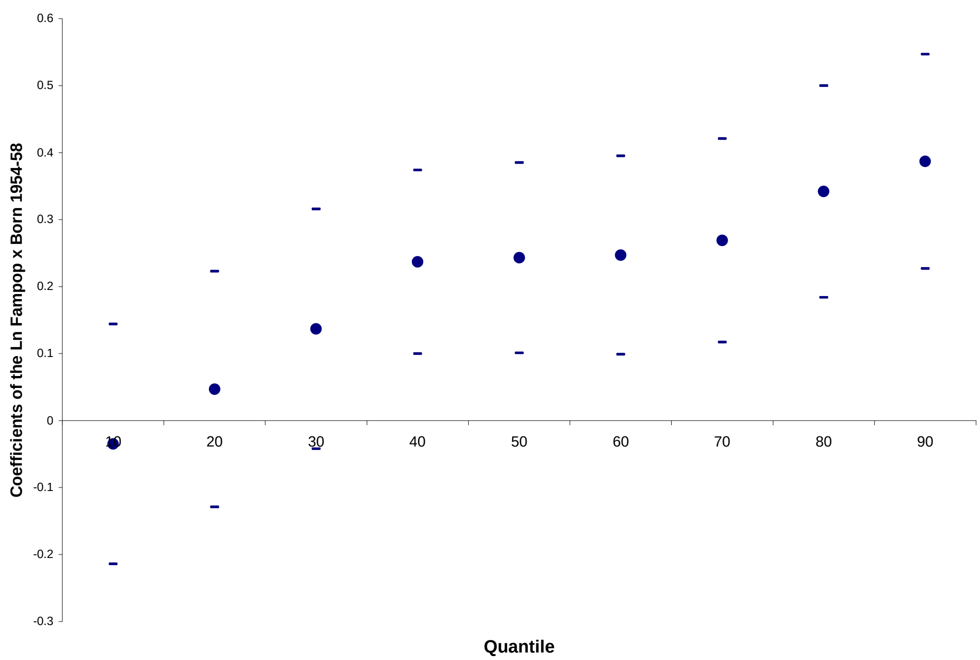


Table A1: The Correlation between Grain Suitability and Per Capita Grain Sown with Cohort Size

Coefficients of the interaction terms between of grain suitability and birth year dummies, and between per capita grain sown and birth year dummies

	Dependent Variable: Ln Population	
	National Sample	
	Agric	Non Agric
	(1)	(2)
Suitability * Born 1943	0.066	0.023
	(0.031)	(0.052)
Suitability * Born 1944	0.096	0.046
	(0.028)	(0.067)
Suitability * Born 1945	0.111	0.065
	(0.036)	(0.075)
Suitability * Born 1946	0.107	0.093
	(0.029)	(0.071)
Suitability * Born 1947	0.122	0.096
	(0.039)	(0.069)
Suitability * Born 1948	0.178	0.132
	(0.032)	(0.066)
Suitability * Born 1949	0.152	0.066
	(0.033)	(0.063)
Suitability * Born 1950	0.036	0.135
	(0.041)	(0.068)
Suitability * Born 1951	0.166	0.193
	(0.040)	(0.067)
Suitability * Born 1952	0.195	0.190
	(0.037)	(0.064)
Suitability * Born 1953	0.148	0.198
	(0.037)	(0.068)
Suitability * Born 1954	0.155	0.206
	(0.031)	(0.069)
Suitability * Born 1955	0.018	0.141
	(0.039)	(0.066)
Suitability * Born 1956	0.093	0.153
	(0.034)	(0.068)
Suitability * Born 1957	0.030	0.211
	(0.038)	(0.072)
Suitability * Born 1958	-0.122	0.102
	(0.047)	(0.074)
Suitability * Born 1959	-0.202	0.144
	(0.067)	(0.069)
Suitability * Born 1960	-0.296	0.012
	(0.074)	(0.063)
Suitability * Born 1961	-0.127	0.101
	(0.062)	(0.062)
Suitability * Born 1962	-0.013	0.043
	(0.037)	(0.057)
Suitability * Born 1963	0.064	0.014
	(0.037)	(0.060)
Suitability * Born 1964	-0.025	-0.083
	(0.027)	(0.074)
Suitability * Born 1965	-0.020	-0.101
	(0.026)	(0.089)
Suitability * Born 1966	0.010	-0.081
	(0.029)	(0.127)
Observations	46212	35175

Regression controls for birth year and birth county fixed effects, and is population weighted. Standard errors are clustered at the province x year level. This Table is taken from Meng and Qian (2009).

Table A2: The OLS Estimates of the Correlation between Famine Exposure by Quantile

Coefficients of the interaction terms between the logarithm of 1959-61 cohort size and birth year categorical variables

	Dependent Variable: Ln Total Hours Worked Per Week								
Quantiles	10%	20%	30%	40%	50%	60%	70%	80%	90%
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
A. OLS									
LnFampop x Born 1959-61	0.009	0.040	0.078	0.111	0.061	0.024	0.001	0.036	0.042
	(0.188)	(0.180)	(0.178)	(0.179)	(0.151)	(0.135)	(0.145)	(0.170)	(0.179)
LnFampop x Born 1955-58	-0.219	-0.163	-0.079	-0.001	-0.005	-0.009	0.014	0.072	0.098
	(0.137)	(0.137)	(0.138)	(0.087)	(0.090)	(0.096)	(0.103)	(0.110)	(0.119)
Observations	313	313	313	313	313	313	313	313	313
B. 2SLS									
LnFampop x Born 1959-61	-0.179	-0.134	-0.076	0.048	0.058	0.057	0.065	0.095	0.112
	(0.166)	(0.162)	(0.152)	(0.151)	(0.137)	(0.140)	(0.136)	(0.138)	(0.139)
LnFampop x Born 1955-58	-0.035	0.047	0.137	0.237	0.243	0.247	0.269	0.342	0.387
	(0.179)	(0.176)	(0.179)	(0.137)	(0.142)	(0.148)	(0.152)	(0.158)	(0.160)
Observations	313	313	313	313	313	313	313	313	313

All regressions control for birth year and birth county fixed effects. Regressions are population weighted.

Standard errors are clustered at the province-year level.