California Center for Population Research
University of California - Los Angeles

Does Famine Have a Long-Term Effect on Cohort Mortality? Evidence from the 1959-1961 Great Leap Forward Famine in China

Shige Song

CCPR-043-07

December 2007

California Center for Population Research
On-Line Working Paper Series

Does Famine Have a Long-Term Effect on Cohort Mortality? Evidence from the 1959-1961 Great Leap Forward Famine in China

Abstract

Using individual mortality records from three cohorts of newborns (1954-1958, 1959-1962, and 1963-1967) from a large national fertility survey data conducted in 1988 in China, I examine the effect of being conceived or born during the 1959-1961 Great Leap Forward Famine on postnatal mortality. The results show strong evidence of a short-term (period) effect of the famine, caused directly by starvation or severe malnutrition during the period of the famine. After controlling for period mortality fluctuation, however, the famine-born cohort does not show higher postnatal mortality than either the pre-famine or the post-famine cohort – as would have been expected from the "fetal origins" hypothesis. Aggregate-level cross-temporal comparisons using published cohort population counts from China's 1982 Census, 1990 Census, 1995 micro Census, 2000 Census, and 2005 micro Census lead to the same conclusion. The relevance of these new findings for the "fetal origins" hypothesis and the selection effect hypothesis is discussed.

Keywords: selection effect, fetal origins, famine, China, mortality, Great Leap Forward.

INTRODUCTION

Famine is a catastrophic event in human societies, not only causing great suffering but also leading to major social disruptions. Both natural and human events may be implicated. Among the three well-studied famines in modern history, the 1866-1869 Finnish famine was caused by three successive crop failures. It resulted in a death rate of 8% and an infant mortality rate of 40% (Kannisto, Christensen and Vaupel 1997). The 872-day Nazi siege of Leningrad during World War II created a famine that killed at least 30% of all the inhabitants in the city (Stanner et al. 1997). The Dutch hunger of 1944-1945 was caused by a combination of environmental factors (an early and harsh winter in 1944) and human maneuver (being at the center of the battlefield and the Nazi food embargo). More than 10,000 Dutch people died directly from starvation, and many more died with malnutrition as a contributing factor (Stein 1975). As for the cause of the 1959-1961 Chinese famine, most scholars agree that a combination of natural disaster and policy mistakes, especially those during the Great Leap Forward in 1958, together led to the three-year famine that caused over 30 million excess deaths (Ashton et al. 1984; Kung and Lin 2003; Lin and Yang 1998, 2000; Peng 1987).

Due to the tragic nature of famine, the first thing that catches people's attention is "how many people died?" Dying directly from starvation and malnutrition during the famine might not be the only consequences, however. According to Barker (1992), poor conditions experienced during the fetal period, especially malnutrition, may have serious negative consequences for an individual's health and longevity as adult. To be more specific, a baby's nutritional conditions before birth and during infancy could cause permanent changes influencing its susceptibility to various health problems later in life, including cardiovascular diseases, diabetes, hypertension,

raised serum cholesterol, abnormal blood clotting, autoimmune diseases, and even suicide. If Barker is correct, the serious consequences of the famine do not end with the famine itself; rather, the whole generation of famine survivors, especially those who were conceived or born during the famine, will face elevated risk for many kinds of illnesses during their entire lives.

The fetal origins hypothesis was not designed specifically to deal with the possible long-term aftermath of famines, and its use is not limited to the famine context. However, for several reasons famine does provides an ideal setting to test the fetal origins hypothesis. In most cases, it is relatively easy to identify cohorts conceived or born during the famine and to distinguish them from cohorts conceived or born either before or after the famine. Thus, we can transform the analytical task of the fetal origins hypothesis from comparing individuals with different fetal nutritional conditions to comparing fetuses conceived or born during the famine versus those conceived or born prior to or after that. Obtaining adequate measures of fetal nutritional conditions has proven to be difficult. In practice, researchers often use children's birth weight as a proxy, but birth weight is less than satisfactory because it is heavily influenced by genetic factors. In addition, birth weight is only available in a small number of studies, thus cannot be used in a broader context. Additionally, using famine as the "treatment" indicating poor fetal nutritional conditions minimizes effects of unobserved heterogeneity. In non-experimental studies, fetal nutritional conditions are not randomly assigned, but are dependent on both observed and unobserved factors. A positive association between fetal conditions and adult health outcomes may be nothing more than a statistical artifact due to failure to control for observed or unobserved factors such as genetic propensities or parental knowledge about child care. Famine works like a natural experiment. The assignment of individuals to the famine cohort

or the non-famine cohort does not depend on individual- or household-level characteristics, observed or unobserved, and is completely exogenous (although, of course, not everybody in the famine cohorts experiences famine to the same degree).

Past research on famine provides mixed support for the fetal origins hypothesis. On the one hand, Kannisto et al. (1997) show that the 1866-1869 Finnish famine increased the mortality rate of those born before and during the famine from birth to age 17, but did not affect adult mortality (after age 17). Stanner et al. (1997) studied the Leningrad famine and report no difference in glucose intolerance, dyslipidemia, hypertension, or CVD in adulthood between those exposed and those not exposed to the famine. On the other hand, results from the Dutch hunger study show that exposure to the famine was associated not only with reduced birth weight and increased perinatal and infant mortality, but also with obesity later in life (Ravelli et al. 1998; Susser and Stein 1994). Recently, with the increasing availability of survey data in China, researchers have started to investigate the impact of the 1959-1961 famine from various perspectives. They have demonstrated that the famine increased the likelihood of stillbirth and miscarriage (Cai and Wang 2005), schizophrenia (St Clair et al. 2005) and overweight and obesity at adulthood (Luo, Mu and Zhang 2006), and led to reduced adult height, labor supply, and earning power (Chen and Zhou 2007), providing some support for the fetal origins hypothesis.

An alternative perspective on the relationship between famine and health and longevity comes from demographic insights regarding selection effects caused by differential mortality: famine may weed out “frail” members of the population and leave the healthier and stronger ones; insofar as this is so, cohorts exposed to famine would be expected to have *lower* long-term

mortality and morbidity than unexposed cohorts. Elo and Preston (1992:195) mentioned this possibility but dismissed it on the ground that the probability that "genetic susceptibilities to death in childhood [are] positively correlated with genetic susceptibilities to death in adulthood" is not high. Genetic susceptibility need not be the only factor at play, however. For example, the relationship between both infant and adult mortality/morbidity and socioeconomic status has been well established – higher socioeconomic status reduces both infant and adult mortality (Gortmaker and Wise 1997; Hobcraft, McDonald and Rutstein 1984; Lleras-Muney 2005; Preston and Taubman 1994). It would not be surprising to see that the excess mortality caused by famine also has a socioeconomic gradient. If this is the case, those exposed to famine will end up with higher proportion of individuals with higher socioeconomic status, compared to pre- and post-famine cohorts, which in turn will result in lower average cohort mortality and morbidity afterwards.

In the present research, I focus on an important aspect of the 1959-1961 Chinese famine that yet has not been fully explored – the possible long-term mortality consequences of the famine – and directly test the fetal origins hypothesis and selection effect hypothesis. Although the fetal origins hypothesis does not directly address the issue of mortality, mortality may be the one best single indicator that can be used to test this hypothesis in a population-based study because the illnesses that are hypothesized to have fetal origins account for almost all major causes of death in adulthood (Kannisto et al. 1997). If the fetal origins hypothesis correctly specifies the causal pathway that connects adult health status with fetal development, children conceived or born during the famine (the famine cohort) would inevitably have had higher mortality than children not conceived or born in the famine (both the pre- and post-famine

cohorts). By contrast, if the selection effect hypothesis holds, the exposed cohorts would have had a lower mortality than the unexposed cohort. The exposed group includes the cohorts born before the famine (who experienced famine as infants or young children) and the cohorts born during the famine (who experienced famine as fetuses or infants); while the unexposed group refers to cohorts born after the famine.

By comparing cohort mortality difference between those born before the famine (1954-1958), during the famine (1959-1962)[1], and after the famine (1963-1967), while controlling for period mortality fluctuations between the famine period and the non-famine periods, I separate the short-term effect (period effect) from the estimated long-term effect (cohort effect) of the famine on mortality.

Results based on a sample of 343,973 Chinese children born between 1954 and 1967 selected from a large nationally-representative sample survey show that, after controlling for the short-term mortality differential between the famine period and non-famine period, the cohorts conceived or born during the three-year famine do not suffer from elevated postnatal mortality up until their late 20s. Supplemental analysis using aggregate population counts from Chinese Census reports extend the age coverage to the mid-40s and 50s and show similar patterns.

The paper proceeds as follows: I begin with a brief description of the 1959-1961 Chinese Great Leap Forward famine. Then I introduce the data, variables, statistical methodology, and

---

[1] The famine started in 1959 and ended in 1961 and affected people throughout China. I included as famine cohorts people born between 1959 and 1962 because most people born in 1962 were conceived in 1961, the height of the famine.

the main findings. In the last section, I discuss the relevance of the new findings to the test of the fetal origins hypothesis and the selection effect hypothesis, as well as discuss potential shortcomings and possible next steps.

THE "GREAT LEAP FORWARD" FAMINE AND ITS LONG-TERM MORTALITY CONSEQUENCES

It is widely accepted among China researchers that the three-year famine in 1959-1961 was triggered by a series of events, including several waves of accelerated agricultural collectivization, the nation-wide establishment of the commune system, and especially the "Great Leap Forward" movement begun in 1958 (Ashton et al. 1984; Kung and Lin 2003; Lin 1990; Lin and Yang 1998). The goal of the Great Leap Forward was to accelerate the pace of industrialization and urbanization in China by mobilizing rural surplus labor to participate in labor-intensive non-agricultural productive activities, such as making iron and steel using backyard blast furnaces (Peng 1987). The results, however, turned out to be disastrous. Much of the iron and steel produced in backyards was useless, grain production declined partly because of a shortage of agricultural labor, and the rapidly expanding urban nonagricultural population resulting from the rural-to-urban migration exacerbated the food supply problem. Beginning from 1959, the second year of the Great Leap Forward movement, China experienced a severe famine affecting the whole country. Over 30 million people died from starvation or severe malnutrition and about 33 million births were either lost or postponed during the three-year period from 1959 to 1961 (Ashton et al. 1984; Yao 1999), making this the largest famine in human history. The serious demographic consequences of the Great Famine put the Great Leap

Forward to an end and sparkled policy debates among the highest leaders that eventually triggered the Cultural Revolution several years later (MacFarquhar 1974). Facing the serious socioeconomic consequences, the government had to send millions of unemployed peasant workers back to villages, and began to strictly implement the *hukou* system, an internal passport system designed to restrict rural-to-urban labor migration, on a national scale (Wu and Treiman 2004). In a sense, the Great Leap Forward Famine is a watershed event in the early history of the PRC, marking the transition from a period of rapid recovery from war losses, economic prosperity, and political stability to a period of economic stagnation and political turmoil.

Past research on the Great Leap Forward Famine focuses primarily on *what happened during the famine*: how many people died during that three-year period who otherwise would have lived (Ashton et al. 1984; Peng 1987), how many miscarriages and stillbirths took place during that three-year period of time that otherwise would have been live births (Cai and Wang 2005), and who or what was responsible for this misfortune (Kung and Lin 2003; Lin 1990; Lin and Yang 1998, 2000; Riskin 1998). Only recently have researchers begun to expand their scope of observation from the three years between 1959 and 1961 to a much broader time period, from those who died during the famine to those who survived, and from the short-term consequences of the famine to the long-term consequences (Chen and Zhou 2007; Luo et al. 2006; St Clair et al. 2005).

The distinction between “short-term” and “long-term” effects is crucial. The biological mechanism of the short-term effect is straightforward: people are more likely to die if they do not have enough calories and nutrients. They face a higher mortality hazard during the period of the famine because of malnutrition or even starvation; once the famine ends and food supply returns

to normal, the elevated mortality hazard should also drop to the usual level. All cohorts, young or old, are subject to the influence of the short-term effect. In this sense, the short-term effect of the famine can be characterized as "period effect." Neither the fetal origins hypothesis nor the selection effect hypothesis is about period or short-term effects. Instead, they both predict that, among the surviving population, some cohorts will have higher than average mortality risk later in life because of the famine. They differ regarding the underlying mechanism through which the famine exerts its influence and in predictions about which particular cohort will have higher mortality. In the absence of secular mortality trends, while the fetal origins hypothesis emphasizes the importance of "biological programming" or "biological imprints" of prenatal malnutrition on fetuses and predicts that cohorts conceived or born during famine will have higher mortality, the selection effect hypothesis emphasizes the importance of differential (excess) mortality caused by the famine and predicts that the cohort born after the famine will have higher mortality relatively to cohorts born before and during the famine because the latter consists of famine survivors, who are generally stronger and healthier.

In order to test the two hypotheses regarding long-term mortality consequences of famine, short-term mortality fluctuations must be controlled for. Failure to do so creates biased estimates that lead to incorrect conclusions. Since the famine-born cohort has been exposed to the famine while the post-famine cohort has not, the famine cohort will definitely have higher overall mortality than the post-famine cohort, simply because of the excess deaths caused by malnutrition and starvation during the period of the famine. Only after the short-term mortality consequence of the famine has been effectively controlled for is it possible to evaluate the long-term mortality consequence of the famine caused by biological programming at the fetal stage.

To be more specific, the fetal origins hypothesis predicts that, in the absence of secular mortality trend and after controlling for short-term mortality fluctuations, *the cohort conceived or born during the famine (1959-1962) has higher mortality than both cohorts born before and after the famine.* Alternatively, the selection effect hypothesis predicts that, after controlling for short-term mortality fluctuations, *both the cohort born before and during the famine will have lower mortality than the cohort born after the famine*.

DATA AND METHODS

I conduct two sets of analyses in the present research. In the first set of analyses, using individual-level retrospective records of ever-married women's birth histories collected in 1988 and multilevel generalized piecewise survival model with multiple clocks (cohort and period), I aim to assess patterns of cohort mortality differences between the famine cohort and the non-famine cohort, from birth to their mid 20s-30s. In the second set of analyses, I use aggregate cohort-specific population counts from the 1982 Census, the 1990 Census, the 1995 mini Census (1% sample survey), the 2000 Census, and the 2005 mini Census (1% sample survey) to compare long-term relative mortality trends among the pre-famine, famine, and post-famine cohorts. This second set of analyses extends the age range of the studied population from birth to people in their mid forties and fifties, and helps to establish the robustness of the individual-level results.

The 1988 Two-Per-Thousand Fertility Survey

To assess mortality consequences of the Great Famine in China, I use data from the National Survey of Fertility and Contraception in China, also known as the two-per-thousand fertility survey. It was conducted in 1988 by the State Family Planning Commission of China. It covers

29 provinces and a total of 2.1 million household members, making it the largest fertility survey in the world. The two-per-thousand survey administered three types of questionnaires, designed to collect information about communities, households, and ever-married women. The ever-married women questionnaire collected detailed information on each woman's complete pregnancy/birth history, including the time the pregnancy ended, result of the pregnancy (live birth, stillbirth, miscarriage, or abortion), and time of death of each live-born baby that did not survive until 1988, along with mother's characteristics such as education, place of residence, and ethnicity.

The data structure is inherently multilevel in the sense that each woman may have multiple pregnancies/births. The data set contains information on 433,250 ever-married women with 1,478,206 pregnancies. I focus on three cohorts: those born during 1954-1958, those born during 1959-1962, and those born during 1963-1967. The 1959-1962 birth cohort is the famine cohort (conceived or born during the famine), the other cohorts are adjacent and act as comparison groups. The analysis sample includes 122,352 women with 343,973 births.

It is worth noting that, due to the nature of the data, the chance of a newborn to be included in the analysis depends partly on the mother's longevity. Since the primary respondent is the ever married woman, she and her children are eligible for inclusion in the sample only if she was still alive in 1988. This may create sample selection bias. However, there is no compelling reason to believe that the influence of differential mortality of mothers is systematically different on mortality patterns of the pre-famine, famine, and the post-famine cohorts.

The following variables are used in the present analysis:

Birth cohort has three categories: the pre-famine cohort (1954-1958), the famine cohort (1959-1962), and the post-famine cohort (1963-1967). Although the famine was between 1959 and 1961 in most places, the cohort born in 1962 was mostly conceived in 1961 and is treated as part of the famine cohort. Difference between birth cohorts is at the center of the fetal origins hypothesis as well as the selection effect hypothesis when using non-experimental design: According to the fetal origins hypothesis, the mortality level of post-famine cohort should be significantly lower than that of the famine cohort, controlling for short-term mortality fluctuations; according to the selection effect hypothesis, the post-famine cohort will have a higher mortality level than both the pre-famine and the famine cohort, controlling for short-term mortality fluctuation. About 22% of all cases (live births) belong to the pre-famine cohort, 23% belong to the famine cohort, and 55% belong to the post-famine cohort, indicating that the famine occurred during a period of rapid fertility increase; the trend was temporarily interrupted by the famine but resumed immediately after the famine was over.

Period and *age* are the two main time variables that structure the duration dependencies. They indicate when (at what age and during which year) the death occurred. Those who have survived to 1988 are considered to be censored and assigned values corresponding to the year 1988. Both variables are coded as continuous variables and enter the model as piecewise linear spline functions. Children born in 1954-1967 were followed until 1988, when they were 21-34 years old.

Mother's education has four categories: illiterate and semi-illiterate (66%), primary school graduate (23%), lower middle school graduate (8%), and upper middle school graduate and above (4%). Mother's education is the only socioeconomic status (SES) indicator used in the

present analysis. Children of better educated mothers are expected to have lower mortality, controlling for all else.

Type of residence has three categories. It indicates whether a child's mother lived in a city (16%), a town (22%), or a village (62%). People living in cities were better protected from the famine than those living in towns and villages, and those living in towns were better protected than those living in villages (Peng 1987). With the strict *hukou* system in place since the late 1950s, migration that crossed the urban-rural boundary was rather limited and did not become common until after the beginning of the economic reform in 1978, thus I treat mother's place of residence as equivalent to children's place of growing up.

The following control variables are also included: (1) *ethnicity* (1 = Han, 0 = other); (2) *birth order (range = 1-13, median = 2);* and (3) *sex* (1 = male, 0 = female). In China, members of ethnic minorities tend to live in remote rural areas and generally have shorter life expectancy (Poston Jr and Shu 1987). Birth order has been shown to have a significant effect on mortality in other societies (Kaplan, Mascie-Taylor and Boldsen 1992; Preston, Hill and Drevenstedt 1998), and men are known to have higher mortality than women in general.

Table 1 presents summary statistics of variables used in the present research.

Census and Micro Census

A second data source used in the present research is the published cohort-specific population counts of the three Censuses (1982, 1990, and 2000) and the two micro Censuses (1995 and 2005) of China (National Population Sample Survey Office 1997, 2007; Population Census Office under the State Council and Department of Population Statistics and Statistics 1985, 1993, 2001). For each census year, I generate the population counts of the pre-famine cohort (1954-

1958), the famine cohort (1959-1962), and the post-famine cohort (1963-1967). Then within each census year, I calculate the ratio of the pre-famine cohort size to the famine cohort size and the ratio of the post-famine cohort size to the famine cohort size. Comparing the cross-temporal trend of these two ratios gives some ideas of the relative mortality of the three cohorts.

Statistical Models

Postnatal mortality is modeled as two-level piecewise Gompertz proportional hazard model:[2]

$$\ln h_{ij}(t) = \gamma T(t) + \beta_0 + \beta_1 Z_{ij}(t) + \beta_2 C_{ij} + \delta_j \qquad (1)$$

The log hazard of j^{th} woman's i^{th} birth experiencing postnatal mortality, $\ln h_{ij}(t)$, is modeled as function of: (1) baseline duration dependencies $T(t)$, (2) a vector of mother- or child-level covariates Z, (3) birth cohort C, and (4) a mother-specific random component δ_j. The baseline duration dependences are modeled as a set of piecewise liner spline functions that are flexible enough to approximate complicated functional forms of any kind. More than one baseline duration dependency can be added to the model to represent effects of different "clocks". Here I compare two different baseline duration dependency specifications, age only ($\gamma_1 T(a)$) and age and period ($\gamma_1 T(a) + \gamma_2 T(p)$), to evaluate the extent to which the estimated cohort difference in postnatal mortality is confounded by period mortality fluctuation. Although age and period are perfectly collinear with cohort, the linear spline functions $T(a)$ and $T(p)$ are not because they

[2] I tested the proportional hazard assumption using graphical method described by Hess (1995). The assumption holds reasonably well for the main covariates, including birth cohorts. These figures are available upon request.

are nonlinear transformations of age and periods; thus model identification is possible (Lillard 1993; Wu 2003). The random component δ_j is assumed to follow a normal distribution with zero mean and constant variance σ_δ.

The above mortality model only deals with live births. Not every pregnancy ends with a live birth because of stillbirth, miscarriage, and abortion. This will not create bias in coefficient estimation for postnatal mortality as long as the process of stillbirth, miscarriage, and abortion is *independent from* the process of postnatal mortality. But such independence should not be taken for granted; in order to check the robustness of the results, I jointly estimate the survival model as depicted in Equation (1) and a multinomial logistic regression model of stillbirth and miscarriage[3], while allowing the possibility that the random components in the postnatal mortality model and in the stillbirth/miscarriage model are nonzero. The two-level multinomial logistic regression model for the stillbirth/miscarriage process is:

$$\ln \frac{\mathrm{P}(y_{ij}=s)}{\mathrm{P}(y_{ij}=1)} = \alpha_0^{(s)} + \alpha_1^{(s)} X_{ij} + \alpha_2^{(s)} C_{ij} + \mu_j^{(s)}, s = 2,3 \qquad (2)$$

Where the j^{th} woman's i^{th} pregnancy has three possible outcomes: live birth ($y_{ij}=1$), stillbirth ($y_{ij}=2$), or miscarriage ($y_{ij}=3$). Live birth is treated as the reference category. The log odds of

[3] The mechanism of abortion is quite different from stillbirth and miscarriage, based on preliminary analysis and results reported by Cai and Wang (2005). Unlike stillbirth and miscarriage, abortion is mainly a result of *choice* as opposed to uncontrollable forces (such as famine). It was strictly restricted by the Chinese government in the 1950s and 1960s, and thus very rare at the time (Scharping 2003:45). I exclude abortion from the analysis.

having either a still birth or miscarriage as opposed to a live birth is modeled as function of a vector of women- or pregnancy-level covariates X , birth cohort C, plus a women specific random component $\mu^{(s)}$. The two random components, $\mu^{(2)}$ and $\mu^{(3)}$, are assumed to follow a bivariate normal distribution with mean zero and constant variance σ_u^s (s=2, 3) and correlation $\rho_u^{(2,3)}$. Random components of the joint model of Equation (1) and (2) follow a trivariate normal distribution with mean zero, constant variances σ_u^2, σ_u^3 ,and σ_δ, and correlations $\rho_u^{(2,3)}$, $\rho_{u\delta}^{(2)}$, and $\rho_{u\delta}^{(3)}$. This joint model can be identified without covariate exclusion because a high percentage of women have multiple pregnancies/births.

All three types of models discussed above are estimated using open source statistical software aML via the full information likelihood method (Lillard and Panis 2003). Since modeling mortality and stillbirth and miscarriage jointly makes little difference in coefficient estimates and certainly does not change the substantive conclusions, I do not present results from the multinomial logistic regression and the joint model. These results are available upon request.

ANALYSIS

In the first set of analyses, I compare two multilevel generalized Gompertz survival models to assess cohort differences in postnatal mortality. If the famine cohort (1959-1962) shows significantly higher mortality than both the pre-famine (1954-1958) and the post-famine (1963-1967) cohorts, this can be seen as evidence supporting the fetal origins hypothesis. On the other hand, if the post-famine cohort demonstrates significantly higher mortality than both the pre-famine cohort and the famine cohort, then the selection effect hypothesis is supported. It is worth

noting that the above predictions are based on the assumption that there have been no secular mortality trends; things become more complicated in the presence of such trends, as we will see.

At the time of interview, children of the selected primary respondents (ever-married women) in the 1988 two-per-thousand fertility survey were between 21 and 34 years old. Past research (based on Finnish data) has shown that if a mortality difference between the famine cohort and the non-famine cohort exists, it most likely appears before age 17 (Kannisto et al. 1997). Given this, we should expect to see higher long-term mortality in cohorts conceived during the famine than in other cohorts even in the relatively young sample analyzed here. However, we cannot rule out the possibility that fetal origin effects on mortality occur at later ages, which is a limitation of the fertility survey data. To address this possibility, a second set of analyses, based on aggregate cohort population counts, extends the age range of the selected cohorts to between 38 and 51.

Describing the Overall Trend of Cohort Mortality

Figure 1 shows the cross-cohort trend in the child (below 18) mortality rate in China, calculated from the 1988 National Survey of Fertility and Contraception. An overall trend of declining mortality is clear: the cohort mortality rate declines from 0.025 in the 1948 birth cohort to 0.005 in the 1970 birth cohort. There are only a few exceptions to this secular long-term trend, among which the most noticeable one occurs between 1957 and 1958, where mortality increases from 0.013 to 0.014. Does this suggest the presence of a long-term influence of the Great Famine on cohort mortality?

It is interesting to note that the sudden increase in mortality occurred between the 1957 and the 1958 cohort, not between the 1958 cohort and the 1959 cohort, when the famine began to

strike. The one-year gap between the increase in cohort mortality and the beginning of the famine seems to suggest that the most dangerous risk factor for cohort mortality is not being conceived or born during the famine but being born right before the famine and being exposed as an infant.

To make this clear, I plot in Figure 2 the mortality rate between ages 0-1 years, ages 1-2 years, and ages 2-3 years for a subset of cohorts. Mortality rates of all three age groups have an irregular bell shape, first increasing then decreasing, with very flat tails. What is most interesting for the purpose of the present study is this: the 1958 cohort has the highest mortality for the 0-1 age group, the 1957 cohort has the highest mortality for the 1-2 age group, and the 1956 cohort has the highest mortality for the 2-3 age group. This does not seem likely to be mere coincidence because these cohorts all reach their peak mortality at the beginning of the Great Leap Forward Famine: 1959.

Figure 3 shows the smoothed empirical hazard function of postnatal mortality for the three selected birth cohorts. Clearly, cohort differences in mortality are more significant at younger ages than at older ages. There is virtually no cohort difference in mortality after age 15, which is consistent with the pattern observed for Finland by Kannisto et al. (1997). Also, mortality is the highest among the oldest cohort (the pre-famine cohort) and the lowest among the youngest cohort (the post-famine cohort), leaving the famine cohort in the middle, showing a clear secular trend of decline in postnatal mortality from older cohorts to younger cohorts.

The empirical hazard functions plotted in Figure 3 provide helpful information about the shape of the baseline hazard function and serve as the important first step; while age is certainly the most important time dimension that structures mortality, it may not be the only one. As

indicated by Figure 2, time period (beginning of the famine vs. other) may also have played an important role in generating the observed mortality patterns, and needs to be accounted for appropriately; in addition, there may be other factors that need to be controlled for. To partial out the influence of time period as well as other factors from the estimated cohort mortality difference, I carry out a series of multivariate analyses in the next section.

Postnatal Mortality: Are There Long-Term Effects?

Table 2 reports results from two two-level piecewise Gompertz hazard models of postnatal mortality. Model 1 includes age splines as the only duration dependence. The estimated cohort difference in the mortality rate is likely to be confounded by period mortality variation (as depicted by Figure 3). Model 1 is treated as the baseline model against which a second model is compared. Model 2 includes both age and period in the baseline duration dependence in the form of linear spline functions. Contrasting Model 2 with Model 1 sheds some light on the extent to which the omission of period mortality variation biases the estimated cohort difference in mortality. A likelihood ratio test rejects the hypothesis that adding the linear spline functions representing period effects does not significantly change the model fit ($P < .001$), making Model 2 the preferred model. I start my discussion with the baseline model – Model 1.

As Model 1 shows, people growing up in cities have a lower risk of mortality than people growing up in villages (hazard ratio = 0.41); people growing up in towns also have a lower risk of mortality than people growing up in villages but not as low as those growing up in cities (hazard ratio = 0.80). Mother's age at childbirth also matters: a one year increase in mother's age at childbirth decreases the hazard of children's mortality by 7%. Mother's education also decreases children's hazard of mortality significantly. Compared to illiteracy, primary school

education reduces children's mortality hazard by 24%, junior high school education reduces the mortality hazard by 39%, and senior high school and above reduces children's mortality hazard by 52%. The mortality hazard increases with parity. Compared to the first-born, the second child's hazard of mortality increases by 30%, the third child's hazard increases by 49%, the fourth child's hazard increases by 83%, and the hazard for the fifth child or higher increases by 128%. Compared to ethnic minorities, the hazard of mortality is 34% lower for Han children. Compared to girls, boys' hazard of mortality is 6% higher. The standard deviation of the random component is 0.74 ($P < .01$), indicating significant variations in mother-level unobserved heterogeneity.

Some experimentation shows that a three-node (four-piece) linear spline approximates the baseline hazard function (age-dependence) reasonably well.[4] Controlling for age-dependence, the covariates, and mother-specific unobserved heterogeneity, the mortality hazard of the pre-famine cohort is 21% greater than that of the famine cohort, and the hazard of the famine cohort is 29% greater than that of the post-famine cohort, supporting the pattern revealed in Figure 3. However, from Model 1, it is difficult to identify any long-term effect of the Great Leap Forward Famine on postnatal mortality: the fact that the pre-famine cohort has higher mortality than the famine cohort clearly does not support the fetal origins hypothesis; the fact that the famine

[4] I follow the strategy suggested by Lillard and Panis (2003) for determining the optimal specification of linear spline function: start with a model with four or five evenly spread nodes, combine adjacent nodes with roughly equal slopes, and shift individual nodes to find a pattern that captures the essence of the baseline duration pattern in the data.

cohort has higher mortality than the post-famine cohort can hardly be attributed to the effect of fetal origins, given the secular trend of mortality reduction revealed in Figure 1.

Adding period dependence into the baseline hazard function significantly improves the model fit, and completely changes the estimated cohort mortality differences. On the one hand, adding a four-node (five-piece) piecewise linear spline representing period mortality differences has little impact on the age dependence pattern; and it has little or no impact on the effects of mother's age, mother's education, place of residence, birth order, ethnicity, or sex of the child. On the other hand, including period duration dependence dramatically changes the estimated cohort difference in the mortality hazard. Without including the period duration dependence, the post-famine cohort has a much lower mortality hazard than the famine cohort (hazard ratio = 0.71); adding the period duration dependence completely reverses the relationship, now *both the pre-famine (hazard ratio = 1.20) and the post-famine cohorts (hazard ratio = 1.09) have a higher mortality hazard than the famine cohort*.

The above comparison shows that the "cohort effect" estimated in Model 1 (as well as in Figure 3) has been confounded by the presence of a strong "period effect," the fact that the hazard of mortality is much higher than usual during the period of famine, as demonstrated in Figure 2. Both the pre-famine cohort (as young children) and the famine cohort (as fetuses or infants) have been exposed to the famine, but the post-famine cohort has not. Without controlling for the mortality difference between the famine period (1959-1961) and non-famine period, the excessively high mortality in the pre-famine and the famine cohorts directly caused by malnutrition and starvation during the three-year period of famine would have been counted as

part of the long-term cohort difference in mortality, leading to biased results. In the present case, the bias is large enough to completely reverse the true pattern.

Period duration dependence of postnatal mortality is displayed in Figure 4. The log hazard of mortality starts high in 1954 but keeps declining gradually until 1958 when it starts to rise suddenly. The hazard reaches its peak in 1961, which is also the peak of the famine, and then begins to decline. The decline is most rapid between 1961 and 1962; it gradually slows down between 1962 and 1965, and even further after 1965. Presumably the trend of postnatal mortality between 1954 and 1967 would have been a smooth and reasonably linear curve if there had not been a famine between 1959 and 1961.

Including period duration dependence adjusts for period differences in postnatal mortality, and yields a more accurate estimate of long-term cohort differences in postnatal mortality. Model 2 suggests that while the Great Leap Forward Famine had a strong short-term influence on postnatal mortality (as depicted in Figure 4), its influence ended when the famine did. The fact that the post-famine cohort has significantly higher mortality than the famine cohort, despite the strong secular trend of cross-cohort mortality reduction, is a strong and unambiguous indication that *there is no long-term or lasting fetal origins effect of the famine on mortality, and the only long-term effect of the famine on mortality is due to selection*. Note that these results pertain to people in their late 20s and early 30s. The selection effect might be expected to become even stronger as people age, which is what the census results show (see below). The fact that controlling for period differences does not change the estimated cohort difference between the pre-famine cohort and the famine cohort is not surprising because they

both were exposed to the three-year famine period and hence both suffered from the excess mortality caused by malnutrition and starvation.

In summary, the fact that both the pre-famine cohort and the post-famine cohort have higher mortality than the famine cohort provides unambiguous evidence against the fetal origins hypothesis. Also, the fact that post-famine cohort has higher mortality than the famine cohort clearly indicates the effect of selection by differential mortality. In this case, the selection effect is strong enough to compensate for the very strong secular trend of mortality reduction, which drastically reduces the mortality level of younger cohorts. For cohorts that are not adjacent but are separated by several years, as in the case of the pre-famine cohort versus the post-famine cohort, the effect of the secular trend of mortality decline is much stronger and thus more difficult to offset. As a result, in this particular case, the secular trend wins out and produces the pattern that the pre-famine cohort has higher mortality than the post-famine cohort.

Evidence from Census Population Counts

As noted above, because the oldest children in the 1988 fertility survey data were age 35, it could be that the survey sample was not old enough to reveal long-term consequences of adverse fetal origins on mortality. To assess this possibility, I compiled information on cohort population counts from published tabulations for three censuses and two micro-censuses (1% sample surveys) spanning the period from 1982 to 2005, when the oldest respondents in the main analysis were 51 years old.

For each census/micro census year, Figure 5 shows the ratios between: (1) the population counts of the pre-famine cohort and the famine cohort, and (2) the population counts of the post-famine cohort and the famine cohort. If these three cohorts have roughly parallel mortality rates

as they age, the two lines would be approximately parallel to the horizontal axis. According to the fetal origins hypothesis, both lines – the one for the pre-famine cohort and the one for the post-famine cohort – should show an upward trend: as the famine cohort dies faster, the proportion of the non-famine cohorts in the population should increase with time. According to the selection effect hypothesis, however, the post-famine cohort should show a downward trend since the excess mortality in the famine cohort selects for stronger individuals, whereas there is no such selection in the post-famine cohort. Under this hypothesis, there is no clear prediction regarding the relative mortality rates for the pre-famine and famine cohorts, both of which were subject to selection by the conditions prevailing during the famine.

In Figure 5, the two lines representing the pre-famine cohort and the post-famine cohort both show a gradual downward trend, indicating that over time the size of each of these cohorts was shrinking faster than for the famine cohort. This means that the famine cohort has a lower mortality rate than both the pre-famine and the post-famine cohort, *contrary to the expectations of the fetal origin hypothesis*, but in agreement with the results reported in Table 2. The negative slope for the post-famine cohort indicates that the difference in mortality between the famine and the post-famine cohort increases with age, consistent with the selection hypothesis. One possible explanation for the negative slope for the pre-famine cohort is that the famine cohort is more highly selective than the pre-famine cohort with regard to the short-term excess mortality caused directly by the famine. When the famine started, individuals in the famine cohort were mostly infants, who were more vulnerable and less resistant to the adverse nutritional environment caused by the famine than individuals of the pre-famine cohort, who were mostly young children at that time. Also, the famine period had much higher stillbirth and miscarriage rates than both

the pre- and post-famine periods (Cai and Wang 2005), which further increased the selectivity of the famine cohort compared to the pre- and post-famine cohorts.

DISCUSSION

The Great Leap Forward Famine between 1959 and 1961 in China was unparalleled in modern human societies with regard to its duration as well as the magnitude of its effects. The famine created a cohort of malnourished children who are different from both their older and younger siblings in many ways. Such a "natural experiment" provides a rare opportunity to re-examine important scientific questions regarding early life conditions and adult mortality and morbidity. Using retrospective information from a large representative fertility survey conducted in 1988 in China and aggregate population counts from census and micro census, I compared cohort mortality patterns of three cohorts of newborns. Multilevel survival models showed that, contradicting to Barker's fetal origins hypothesis, there is no long-term fetal origins effect on mortality, *after controlling for short-term period mortality fluctuations*. Similar findings have emerged from studies of famines in other countries (Kannisto et al. 1997; Stanner et al. 1997). Instead, the results support predictions made by the selection effect hypothesis that the post-famine cohort has the highest mortality.

The distinction between long-term and short-term effects is crucial, because they represent drastically different causal pathways. Famines generate excess mortality because of the harsh environments they create, especially with regard to food and nutrition. Once the famine ends and the food supply returns to normal, the elevated mortality rate also will return to normal. The fetal origins hypothesis predicts that, in addition to the short-term excess mortality generated by starvation and malnutrition during the period of the famine, exposure to famine as a fetus has

independent long-term effects on mortality and morbidity, because of biological programming. With regard to postnatal mortality, these two different effects of the famine can be most conveniently conceptualized as a period effect and a cohort effect. This is the core assumption of the present research.

Like Kannisto et al. (1997), this study did not find independent long-term impacts of the famine on mortality, as are predicted by the fetal origins hypothesis. Two different processes seem to be in place in generating the observed cohort mortality pattern. The first is the secular trend in mortality reduction since the establishment of the socialist regime in 1949, mainly due to improvements in food supply, public health facilities, and hygiene conditions (Banister and Hill 2004; Kantha 1990; Popkin et al. 1993). Although the three-year famine temporarily interrupted this process, once the famine was over, the process resumed immediately. The second process explaining these findings is a selection effect caused by differential excess mortality during the three-year famine. Many "frail" members of cohorts that experienced the famine died during that three-year period. Survivors of these cohorts are stronger and healthier and have longer life expectancies than cohorts that never experienced the famine. These two forces have opposite impacts on cohort mortality and work against each other in creating the observed cohort mortality patterns.

In contrast to the lack of evidence for biological programming, the present research found strong evidence for "social programming": regardless of birth cohorts or time periods studied here, urban children have much lower mortality; mother's education significantly decreases children's mortality; and ethnic majority members have much lower mortality. None of these findings is new to social scientists, but putting them side by side with the estimated fetal origins

effect makes the starkest comparison: social factors make a far larger contribution to postnatal mortality differentials than do biological factors.

The present research faces several limitations. As mentioned before, the data used in the present study includes only information on children of women who have survived to 1988, which constitutes only a subset of all women and a subset of all children. This is the problem shared by all retrospective studies and suggests caution in interpreting the results. However, there is no compelling reason to believe that differential mortality of mothers led to systematic differences in children's characteristics in the three different birth cohorts. In other words, children in the sample may have different mortality from children excluded from the sample, but as long as the same exclusion mechanism has been applied to all three cohorts, the exclusion will not bias the estimated cohort mortality difference shown here. The 1988 two-per-thousand data cover a relatively short period of life course (from birth to age 21-34). Even with the auxiliary analysis using aggregate census population counts, these analyses can only extend the age range to 38-51. The possibility that the mortality rate for the famine cohort rises after age 51 cannot be ruled out. However, results from analyses of the 1866-1868 Finnish famine show that the elevated mortality of the famine cohort appears before age 17, if it appears at all (Kannisto et al. 1997). This lends some confidence to my results. Finally, the lack of cause-specific mortality rates makes it impossible to pursue further in-depth analysis. These results cannot rule out the possibility that some cause-specific mortality rates do show patterns that agree with Barker's hypothesis. Better data are required to address this issue, and more in-depth research is needed.

Despite these limitations, results presented here demonstrate convincingly the presence of a short-term effect of famine on postnatal mortality, caused directly by food deprivation and

malnutrition. After the crisis was over, however, there was no lingering long-term effect of biological programming, as would have been predicted by the fetal origins hypothesis. It is thus reasonable to conclude that the only long-tem mortality consequence the famine produced, that the post-famine cohort has higher mortality than the famine cohort, was due to a selection effect. For the purpose of understanding postnatal mortality differentials, socioeconomic indicators are more efficient predictors than biological ones, even in extreme situations such as the Great Leap Forward Famine.

REFERENCES

Ashton, B., K. Hill, A. Piazza, and R. Zeitz. 1984. "Famine in China, 1958-61." *Population and Development Review* 10(4):613-645.

Banister, J. and K. Hill. 2004. "Mortality in China 1964-2000." *Population Studies* 58(1):55-75.

Barker, D.J.P. 1992. "The Fetal Origins of Diseases of Old Age." *European Journal of Clinical Nutrition* 46(3):S3-9.

Cai, Y. and F. Wang. 2005. "Famine, Social Disruption, and Involuntary Fetal Loss: Evidence from Chinese Survey Data." *Demography* 42(2):301-322.

Chen, Y. and L.A. Zhou. 2007. "The Long-Term Health and Economic Consequences of the 1959-1961 Famine in China." *Journal of Health Economics* 26:659-681.

Elo, I.T. and S.H. Preston. 1992. "Effects of Early-Life Conditions on Adult Mortality: A Review." *Population Index* 58(2):186-212.

Gortmaker, S.L. and P.H. Wise. 1997. "The First Injustice: Socioeconomic Disparities, Health Services Technology, and Infant Mortality." *Annual Review of Sociology* 23(1):147-170.

Hess, K.R. 1995. "Graphical Methods for Assessing Violations of the Proportional Hazards Assumption in Cox Regression." *Statistics in Medicine* 14(15):1707-1723.

Hobcraft, J.N., J.W. McDonald, and S.O. Rutstein. 1984. "Socio-Economic Factors in Infant and Child Mortality: A Cross-National Comparison." *Population Studies* 38(2):193-223.

Kannisto, V., K. Christensen, and J.W. Vaupel. 1997. "No Increased Mortality in Later Life for Cohorts Born During Famine." *American Journal of Epidemiology* 145(11):987-994.

Kantha, S.S. 1990. "Nutrition and Health in China, 1949 to 1989." *Progress in Food and Nutrition Science* 14(2-3):93-137.

Kaplan, B.A., C.G. Mascie-Taylor, and J. Boldsen. 1992. "Birth Order and Health Status in a British National Sample." *Journal of Biosocial Science* 24(1):25-33.

Kung, J.K. and J.Y. Lin. 2003. "The Causes of China's Great Leap Famine, 1959-1961." *Economic Development and Cultural Change* 52(1):51-73.

Lillard, L.A. 1993. "Simultaneous-Equations for Hazards - Marriage Duration and Fertility Timing." *Journal of Econometrics* 56(1-2):189-217.

Lillard, L.A. and C.W.A. Panis. 2003. *Aml User's Guide and Reference Manual, Version 2*. Los Angeles: EconWare.

Lin, J.Y. 1990. "Collectivization and China's Agricultural Crisis in 1959-1961." *The Journal of Political Economy* 98(6):1228-1252.

Lin, J.Y. and D.T. Yang. 1998. "On The Causes of China's Agricultural Crisis and the Great Leap Famine." *China Economic Review* 9:125-140.

—. 2000. "Food Availability, Entitlements and the Chinese Famine of 1959-61." *The Economic Journal* 110(460):136-158.

Lleras-Muney, A. 2005. "The Relationship Between Education and Adult Mortality in the United States." *Review of Economic Studies* 72(1):189-221.

Luo, Z., R. Mu, and X. Zhang. 2006. "Famine and Overweight in China." *Review of Agricultural Economics*.

MacFarquhar, R. 1974. *The Origins of the Cultural Revolution*. New York: Columbia University Press.

National Population Sample Survey Office. 1997. *1995 National 1% Population Sample Survey Data*. Beijing: China Statistical Publishing House.

—. 2007. *2005 National 1% Population Sample Survey Data*. Beijing: China Statistical Publishing House.

Peng, X. 1987. "Demographic Consequences of the Great Leap Forward in China's Provinces." *Population and Development Review* 13(4):639-670.

Popkin, B.M., K. Ge, F. Zhai, X. Guo, H. Ma, and Z. Namvar. 1993. "The Nutritional Transition in China: A Cross-Sectional Analysis." *European Journal of Clinical Nutrition* 47:333-346.

Population Census Office under the State Council and Department of Population Statisticsand N.B.o. Statistics. 1985. *1982 Population Census of China (Results of Computer Tabulation)*. Beijing: China Statistical Publishing House.

—. 1993. *Tabulation on the 1990 Population Census of the People's Republic of China*. Beijing: China Statistical Publishing House.

—. 2001. "Major Figures on 2000 Population Census of China." Beijing: China Statistics Press.

Poston Jr, D.L.and J. Shu. 1987. "The Demographic and Socioeconomic Composition of China's Ethnic Minorities." *Population and Development Review* 13(4):703-722.

Preston, S.H., M.E. Hill, and G.L. Drevenstedt. 1998. "Childhood Conditions That Predict Survival to Advanced Ages among African-Americans." *Social Science and Medicine* 47(9):1231-1246.

Preston, S.H.and P. Taubman. 1994. "Socioeconomic Differences in Adult Mortality and Health Status." *Demography of Aging*:279–318.

Ravelli, A.C., J.H. van der Meulen, R.P. Michels, C. Osmond, D.J. Barker, C.N. Hales, and O.P. Bleker. 1998. "Glucose Tolerance in Adults after Prenatal Exposure to Famine." *Lancet* 351(9097):173-177.

Riskin, C. 1998. "Seven Questions about the Chinese Famine of 1959–61." *China Economic Review* 9(2):111-124.

Scharping, T. 2003. *Birth Control in China, 1949-2000: Population Policy and Demographic Development*. London ; New York: Routledge.

St Clair, D., M. Xu, P. Wang, Y. Yu, Y. Fang, F. Zhang, X. Zheng, N. Gu, G. Feng, and P. Sham. 2005. "Rates of Adult Schizophrenia Following Prenatal Exposure to the Chinese Famine of 1959-1961." *Journal of American Medical Association* 294(5):557-562.

Stanner, S.A., K. Bulmer, C. Andres, O.E. Lantseva, V. Borodina, V.V. Poteen, and J.S. Yudkin. 1997. "Does Malnutrition In Utero Determine Diabetes And Coronary Heart Disease In Adulthood? Results from the Leningrad Siege Study, A Cross Sectional Study." *British Medical Journal* 315(7119):1342-1348.

Stein, Z. 1975. *Famine and Human Development: The Dutch Hunger Winter of 1944-1945*. New York: Oxford University Press.

Susser, M.and Z. Stein. 1994. "Timing in Prenatal Nutrition: A Reprise of the Dutch Famine Study." *Nutrition Reviews* 52(3):84-94.

Wu, L.L. 2003. "Event History Models for Life Course Analysis." Pp. 477-502 in *Handbook of the Life Course*, edited by J.T. Mortimer and M.J. Shanahan. New York: Kluwer Academic/Plenum Publishers.

Wu, X. and D.J. Treiman. 2004. "The Household Registration System and Social Stratification in China: 1955-1996." *Demography* 41:363-384.

Yao, S. 1999. "A Note on the Causal Factors of China's Famine in 1959-1961." *The Journal of Political Economy* 107(6):1365-1369.

Table 1. Descriptive Statistics of All Variables, Selected Cohorts from the Two-Per-Thousand Fertility Survey of China

	Mean	Standard Deviation	Percentage (%)
Mother's Characteristics			
Type of Residence			
City			16
Town			22
Mother's Education			
Primary School			23
Lower Middle School			8
Upper Middle and Above			4
Ethnic Majority			90
Child's Characteristics			
Birth Order			
2^{nd} Birth			27
3^{rd} Birth			19
4^{th} Birth			12
5^{th} and Above Birth			12
Male			52
Cohort			
Pre-Famine			22
Post-Famine			55
Mother's Age	24.88	4.20	

Note: N. of mothers = 122,352; N. of children = 371,105.

Table 2. Two-Level Generalized Piecewise Linear Hazard Model for Postnatal Mortality in China

	Model 1	Model 2
Duration on Age		
0-1 years (slope)	-2.92	-2.90
1-10 years (slope)	-0.32	-0.28
10 years and above (slope)	0.01	0.04
Duration on Calendar Time		
1954-1957 (slope)		-0.03[a]
1957-1961 (slope)		0.11
1961-1964 (slope)		-0.24
1964-1967 (slope)		-0.03
Type of Residence		
Cities	-0.88	-0.90
Towns	-0.22	-0.23
Birth Cohort[b]		
Pre-Famine	0.19	0.19
Post-Famine	-0.34	0.08
Mother's Age	-0.08	-0.07
Mother's Education[c]		
Primary School	-0.27	-0.26
Junior High	-0.50	-0.48
Senior High and Above	-0.73	-0.73
Birth Order[d]		

	2nd	0.26	0.26
	3rd	0.40	0.38
	4th	0.60	0.59
	5th and Above	0.82	0.81
Ethnic Han		-0.42	-0.42
Male		0.06	0.06
Intercept		0.76	0.76
σ		0.74	0.74
Log Likelihood		-412155	-411665

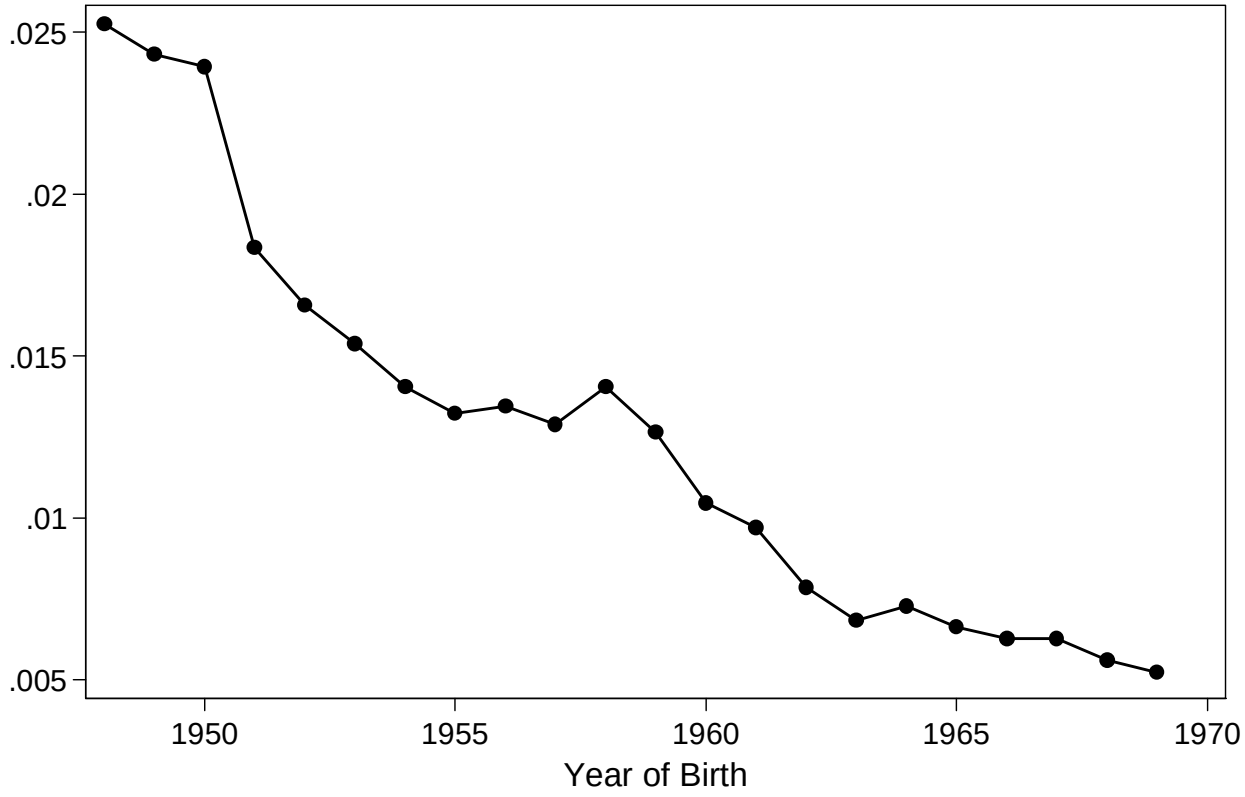
[a] $p < .05$; all other coefficients reported in the table have significance level of $p < .001$.

[b] The reference category is "famine cohort".

[c] The reference category is "illiterate or semi-illiterate".

[d] The reference category is "the 1st birth".

Figure 1. Overall Trend in Cohort Mortality (age ≤ 18) in China



Note: Cohort mortality rate is calculated using the 1988 National Survey of Fertility and Contraception in China. I exclude the very early cohorts (1941-1946) with small cohort size and the very late cohorts (1971-1988) to make sure the cohorts are comparable.

Figure 2. Single-Year Mortality Rate for People Born between 1954 and 1967

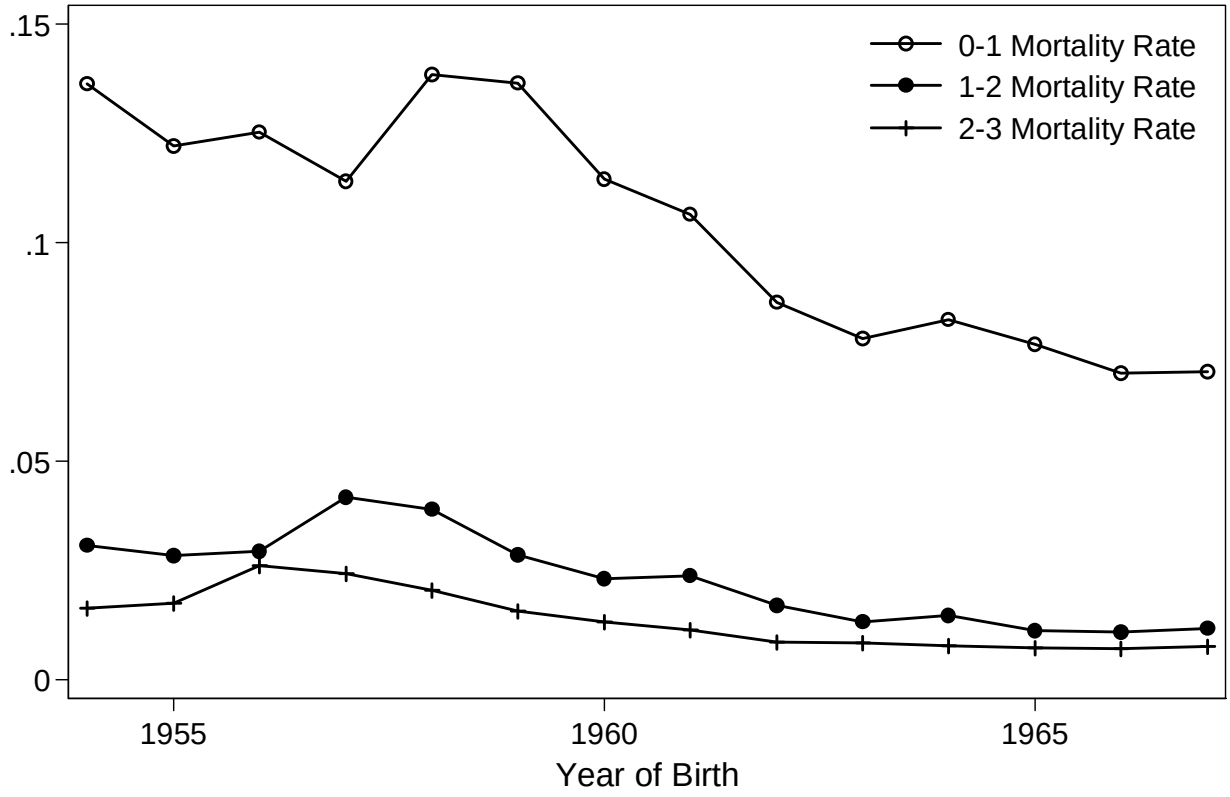


Figure 3. Cohort Comparison of Smoothed Hazard Function of Postnatal Mortality in China

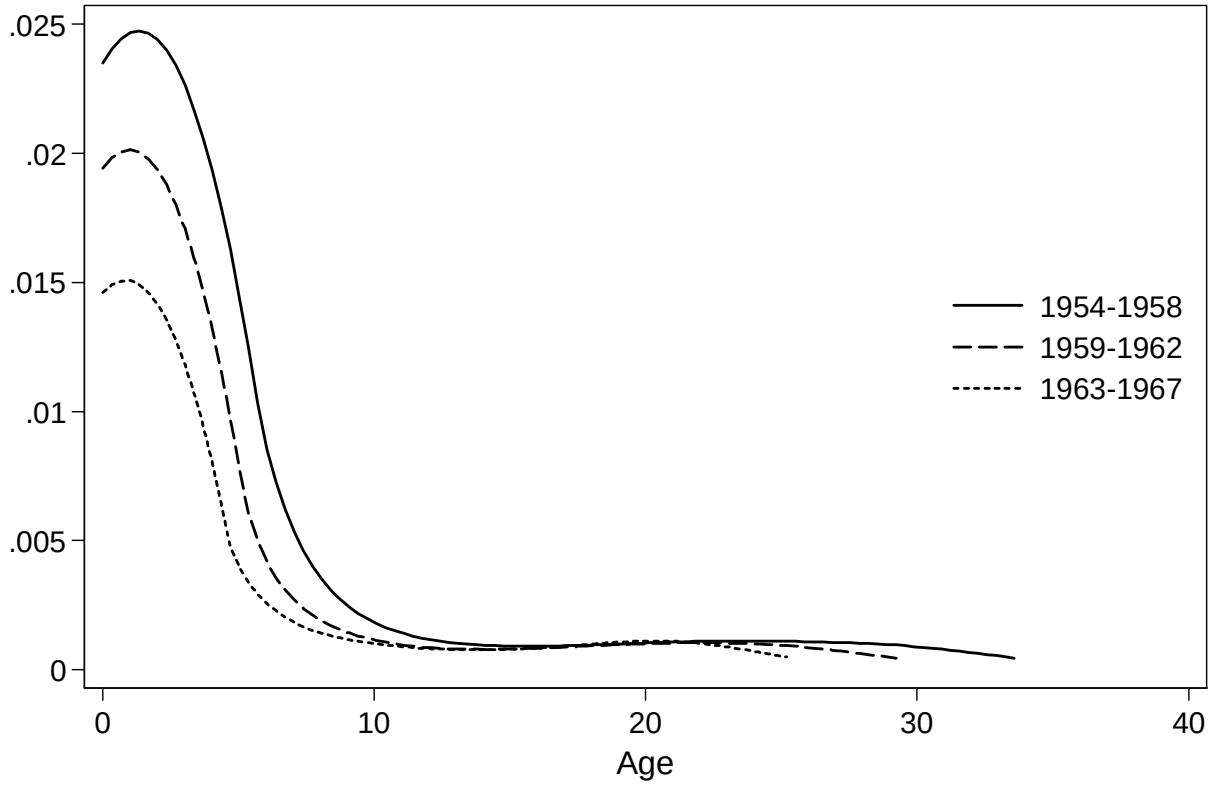


Figure 4. Period Dependence of the Baseline Log Hazard of Postnatal Mortality in China

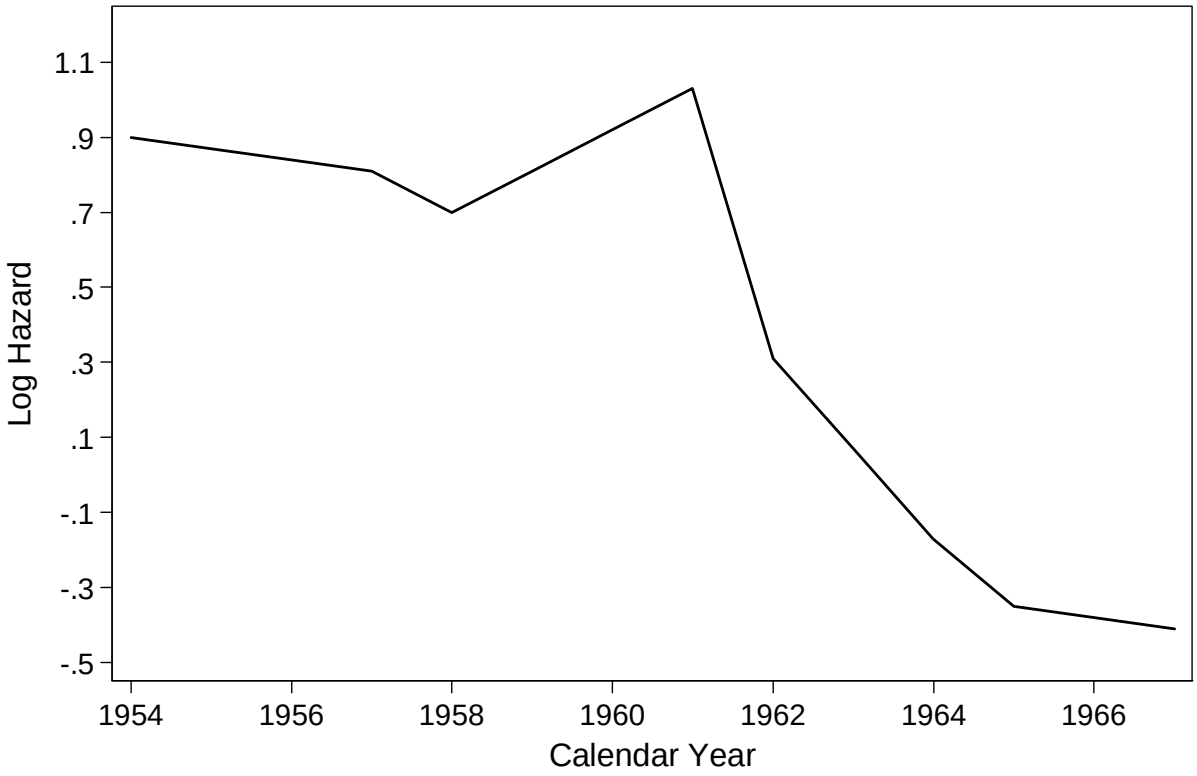


Figure 5. Changes in Relative Cohort Size of the Pre-Famine Cohort, Famine Cohort, and Post-Famine Cohort, Using the Famine Cohort as the Baseline