Mortality Consequences of the 1959-1961 Great Leap Forward Famine in China: Debilitation, Selection, and Mortality Crossovers

Shige Song

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Shige Song
Institute of Sociology, Chinese Academy of Social Sciences
5 Jiannei Dajie, Beijing 100732
P.R.China
Phone: 86-10-64460722
E-mail: sgsong@ccpr.ucla.edu

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Institute of Sociology, Chinese Academy of Social Sciences, E-mail:

sgsong@ccpr.ucla.edu, Telephone: 86-10-64460722, Fax: 86-10-85195566.

Running Head: Mortality Crossovers after Famine

Abstract

Using individual retrospective mortality records for three cohorts of newborns (1956-1958, 1959-1961, and 1962-1964) drawn from a large national fertility survey conducted in 1988 in China, I examined cohort differences in mortality up to age 22, aiming to identify debilitation and selection effects of the 1959-1961 Great Leap Forward Famine. The results revealed the presence of a mortality crossover between ages 11 and 12, when the mortality level of the non-famine cohort caught up to and exceeded the level of the famine cohort. The presence and timing of the mortality crossover suggests that both debilitation and selection effects influenced the post-famine cohort mortality pattern. In addition, the multilevel multiprocess models established a direct connection between frailties for infant mortality and mortality at subsequent ages, thus demonstrating the theoretical inevitability of mortality crossover after famine as the result of the convergence process caused by selection due to frailty.

Keywords: selection effect, famine, China, mortality crossovers, Great Leap Forward, non-proportionality, shared frailty, multilevel multiprocess model.

Introduction

Famine is a catastrophic event, and it has long been known to be associated with increased mortality. Some investigators claim that, in addition to an immediate rise in mortality level, famine may also have a long-term or sustained effect on cohort mortality, yet empirical evidence so far has been rather limited and mixed. So, what is really meant by a "long-term" or "sustained" effect of famine? And what is the appropriate time frame to identify such effects?

The pursuit of a long-term effect of famine hinges on the fact that famine represents a *natural experiment* from which the consequences of severe nutritional deprivation can be assessed. Past research suggests three alternative explanations. First, famine may have a debilitating effect on the famine cohort (those who were born during the famine or experienced famine at infancy or early childhood); negative effects of unfavorable living conditions typically associated with famine could continue after the famine is over, producing persistently higher than usual mortality (Razzaque et al. 1990; Preston et al. 1998). A second explanation is known as the "fetal origins" hypothesis, in which malnutrition experienced during the fetal period may cause permanent biological changes influencing one's susceptibility to various chronic diseases later in life, leading to an elevated mortality risk at older age (Barker 1992). The third explanation focuses on the effect of selection by frailty – heterogeneity in susceptibility to mortality – and argues that the excess mortality caused by famine reduces the overall level of frailty of the famine cohort by "weeding out" the frail members, making it stronger and healthier than non-famine cohorts. As a result, the mortality level of the non-famine cohort will be higher than the famine cohort after the famine is over (Vaupel et al. 1979; Razzaque *et al.* 1990).

In their study of the 1974-1975 Bangladesh famine, Razzaque et al. (1990) identified both a debilitation effect and a selection effect; the famine-born continued to have a higher than

usual mortality in the second year of life and one year after the famine was over, which was then followed by a lower than usual mortality between the ages of 2 to 5. Kannisto et al. (1997), in their study of the 1866-1869 Finnish famine, reported evidence for the debilitation effect (higher than usual mortality of the famine cohort up to age 17) but no indication of a fetal origins effect. Although they did not explicitly mention the selection effect, the famine cohort has a higher mortality than the non-famine cohort before age 17, but the mortality difference disappears afterwards, suggesting the effect of selection. Although not specific to a famine situation, Caselli and Capocaccia (1989) reported a mortality pattern in Italy that was similar to the Bangladesh case: cohorts who experienced adverse conditions early in life tended to show an increased (higher than usual) cohort mortality up to a certain age, followed by reduced cohort mortality (lower than usual) afterwards. The main difference between the Bangladeshi and Italian studies is the timing of the turning point—the turning point in Bangladesh occurred at age 2, in Italy it occurred at age 45.

Although neither Razzaque et al. (1990) nor Caselli and Capocaccia (1989) used the term "mortality crossovers" to refer to the pattern they discovered – that the mortality level of the disadvantageous group (the famine cohort or the cohort that experienced adverse early conditions) remained higher than the advantageous group (the non-famine cohort or the cohort that did not experience adverse early conditions) up to a certain age, then fell below the level of the advantageous group – they contributed interesting cases to an important line of demographic literature concerning racial and gender differences in cohort mortality (Pearl 1922; Spiegelman 1967; Coale and Kisker 1986; Nam 1995) in the context of famine. On the one hand, these new (famine-related) cases can shed light on some of the ongoing debates in the mortality crossover literature. On the other hand, knowledge accumulated by the past research on mortality

crossovers provides a good point of reference that can help to achieve a better understanding of famine-related mortality phenomena.

In the present research, I examine the mortality consequences up to age 22 of the 1959-1961 Great Leap Forward Famine in China, using data from a large representative national sample survey conducted by the Chinese government in 1988. Due to data limitations, I do not attempt to test the fetal origins hypothesis (the cohorts are too young to show the hypothesized effects). Instead, I focus on debilitation effects, selection effects, and their interactions. Based on past research on famine and mortality, I expected to see a trend in mortality convergence as well as the possibility of a mortality crossover between the famine cohort and the non-famine cohort within that age range (age 0-22). The first research objective was to identify that crossover point, if it existed. Based on my knowledge, identifying a post-famine mortality crossover has not been done in the Chinese context. The second objective was to establish a direct relationship between the frailty of infant mortality and the frailty of mortality at subsequent ages. A positive relationship between these frailties is considered to be the most forceful and direct evidence supporting the selection effect argument (Coale and Kisker 1986; Elo and Preston 1992). I first demonstrate the theoretical feasibility of such an analysis within the framework of multilevel multiprocess modeling (Lillard 1993; Panis and Lillard 1995; Steele et al. 2005) and then present the results in the context of China. Finally, I discuss the implications of the mortality crossover to the effort of identifying both the debilitation and selection effects of famine.

Mortality crossovers after famine: debilitation, selection, and frailty

Mortality crossover represents an extreme form of mortality convergence, and it can be caused by data error (overstatement of age by elderly cohorts), selection by frailty, or both, depending on specific situations (Nam 1995). In the context of famine, however, it is highly unlikely that an observed mortality crossover is caused by age overstatement, thus leaving selection as the most likely explanation. In general, the more severe the famine, the higher the excess infant mortality it incurs, the more highly selective the famine cohort becomes, and the more likely a mortality crossover between the famine and the non-famine cohort would be observed.

However, selection may not be the only force at play. Elo and Preston (1992) and Preston et al. (1998) discussed possible biomedical mechanisms through which a debilitation effect of adverse early conditions can continue into adulthood and thus have long-term mortality consequences. In the 1974-1975 Bangladesh famine study, Razzaque et al. (1990) reported a mortality crossover between the famine cohort and the non-famine cohort that occurred after the second year of life, a year after the end of the famine. The selection argument can explain the mortality crossover that eventually occurred, but it cannot explain the one year elevated mortality of the famine cohort between the end of the famine and the beginning of the crossover (after the second year of life). Razzaque et al. (1990) attributed this to the debilitation effect of famine, and Caselli and Capocaccia (1989) reported similar findings in their study of mortality patterns in Italy. However, since they studied a non-famine situation, the level of excess mortality of the disadvantageous group at early childhood was not as high as in a famine situation, and the selection effect was much weaker. Regarding outcomes, it took much longer (45 years) to observe a mortality crossover. Kannisto et al. (1997) did not report a mortality crossover between

the famine and the non-famine cohorts in Finland, but they did report a mortality convergence after age 17.

The above discussions suggest the possibility of disentangling the debilitation and selection effects by studying the timing of the mortality crossover. Although both are caused by famine, debilitation and selection influence post-famine cohort mortality differently. Without the effect of selection, the famine cohort would always have a higher cohort mortality than the non-famine cohort; without the effect of debilitation, a crossover in mortality between the famine and non-famine cohort will occur immediately after the end of the famine period.

Mortality crossover after a famine provides a good opportunity to study debilitation and selection. However, as Nam (1995) summarized, long-term mortality convergence may not necessarily end up with a crossover (especially when the period of observation is relatively short). How, then, can we identify the debilitation and selection effect when no mortality crossover is observed? Identifying debilitation in a famine context is relatively easy; if the famine cohort continues to have a higher than usual mortality after the famine is over, this provides good evidence for the debilitation effect. However, what about the selection effect?

Although mortality crossover provides a convincing case for the selection effect, it is worth emphasizing that the evidence it provides is *indirect* and inferential in nature. Selection is about frailty (Vaupel *et al.* 1979; Vaupel and Yashin 1985); the most direct and forceful evidence for the effect of selection will be a positive correlation between frailties early in life (e.g., infant mortality) and later in life (Coale and Kisker 1986; Elo and Preston 1992). Since frailty is unobserved and unmeasured, it is impossible to establish the abovementioned relationship at the individual level. This difficulty has led some investigators to explore alternative methods to establish the relationship, using aggregated indicators such as cohort

infant mortality as a measure of selectivity (Caselli and Capocaccia 1989). The weakness of this rationale is obvious; higher cohort infant mortality alone does not necessarily lead to a more selective cohort if the excess mortality is not selected by frailty, resulting in a dilemma.

Frailty can also be measured at the sibling level. Children with the same mother share a wide range of common characteristics, and, unlike the individual level frailty, the sibling level shared frailty can be identified and estimated in the form of random effects in the context of a multilevel modeling framework (Mason et al. 1983-1984; Raudenbush and Bryk 2002; Goldstein 2003), as long as there are a sufficient number of women in the sample who have multiple children. Shared frailty does not capture all of the individual level unobserved heterogeneity; instead, it captures those stable traits, biological and socioeconomic, inherited from mothers and shared among siblings. Shared frailty is an important determinant of mortality (Curtis et al. 1993; Willcox et al. 2006) and is probably more informative than individual level unshared frailty, putting identification issues aside.

Shared frailty can be identified and estimated for mortality that occurs at different ages (e.g., infant mortality, child mortality, and adult mortality). Furthermore, with recent methodological developments in the multilevel multiprocess statistical framework (Lillard and Waite 1993; Lillard 1993; Skrondal and Rabe-Hesketh 2004; Steele *et al.* 2005), it is possible to model multiple mortality processes simultaneously and to estimate the shared frailties as well as their correlations. A positive correlation between infant mortality frailty and mortality frailties at subsequent ages demonstrates that those who are less likely to die at infancy are also less likely to die at later ages. In the context of a famine, this means that survivors of the famine cohort tend to have a lower mortality risk after the famine.

Empirical context: the "Great Leap Forward" famine in China

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; Lin 1990; Lin and Yang 1998; Kung and Lin 2003). 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 non-agricultural population resulting from the rural-to-urban migration exacerbated the food supply problem. Beginning in 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 sparked policy debates among the highest leaders that eventually triggered the Cultural Revolution several years later (MacFarquhar 1974). Facing the serious socioeconomic consequences, the government was forced to send millions of unemployed peasant workers back to villages, and it 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 was 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 has focused 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 that otherwise would have been live births (Cai and Wang 2005), and who or what was responsible for this misfortune (Lin 1990; Lin and Yang 1998; Riskin 1998; Lin and Yang 2000; Kung and Lin 2003). 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, and from those who died during the famine to those who survived (St Clair et al. 2005; Luo et al. 2006; Almond et al. 2007; Chen and Zhou 2007; Gørgens et al. 2008; Song et al. 2008).

The present research contributes to this small but rapidly expanding literature by: (1) showing the presence of the debilitation effect (the "long-term" effect) of the famine; (2) identifying a post-famine crossover in cohort mortality between the famine and non-famine cohorts; and (3) demonstrating the underlying mechanisms of mortality convergence and discussing the implications of such a convergence on the effort of identifying long-term health consequences of the famine.

Research design

Cohort comparison is at the core of the present paper. To be more specific, I compared mortality risk up to age 22 between the pre-famine cohort (1956-1958), the famine cohort (1959-1961), and the post-famine cohort (1962-1964) using data from an extremely large nationally representative sample survey conducted in China in 1988. The comparison between the 1959-

1961 cohort and the 1962-1964 cohort is most revealing and thus will be the focus of this discussion. Since the Great Leap Forward Famine occurred in the middle of a secular mortality decline, I included the 1956-1958 cohort, who experienced the famine during early childhood, in the analysis to obtain an estimate of the effect of the secular trend on mortality decline.

I started the analysis by estimating 23 ordinary logistic regression models on single-year age-specific mortality risk from age 0-22 and then plotted the cohort pattern in mortality (in odds ratio) by age. Based on the revealed age pattern of the cohort mortality difference, I estimated a set of Cox hazard models that allowed the effect of birth cohort to vary between ages (time-varying coefficients). Lastly, I constructed a multilevel multiprocess model that jointly estimates a random-effect logistic regression model for infant mortality and random-effect proportional hazard model for mortality at later ages (the choice of age segments depends on the results from the first and second steps), allowing the frailties of the two equations to be freely correlated.

The 1988 two-per-thousand fertility survey

I used data from the National Survey of Fertility and Contraception in China, also known as the 1988 two-per-thousand fertility survey. It was conducted in 1988 by the State Family Planning Commission of China and covered all 29 provinces and a total number 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 about each woman's complete pregnancy/birth history, including the time of the end of the pregnancy, the result of the pregnancy, and the time of death of each live-born baby who did not survive to the time of the survey interview, along with characteristics of the mother such as education, place of registration, 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 focused on three cohorts: those born during 1956-1958, those born during 1959-1961 and those born during 1962-1964, which included 96,203 women with 207,046 births.

It is worth noting that, due to the retrospective 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 for 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 (1956-1958), the famine cohort (1959-1961), and the post-famine cohort (1962-1964). Cohort comparison in mortality risk, especially between the famine and the post-famine cohort, was the main analytical tool of the present study, used to clarify the debilitation and selection effects.

Mother's education is coded as a continuous variable with a mean of 2.14 years and a standard deviation of 3.43. Mother's education is the main socioeconomic status (SES) indicator used in the present analysis. Children of more highly educated mothers are expected to have lower mortality, all else being equal.

Place of registration (hukou) has three categories. It indicates whether a child's mother is permanently registered in a city (17%), a town (22%), or a village (60%) in the Chinese *hukou*

system. The mother's *hukou* status is a good indicator of her children's well-being because: (1) having a *hukou* in cities is a privilege and is more protective from adverse conditions than those having a *hukou* in towns and villages, and having a *hukou* in towns is more protective than having a *hukou* in villages (Peng 1987); and (2) children's *hukou* status goes with their mothers' *hukou* status (Wu and Treiman 2007). With the strict *hukou* system in place since the late 1950s and the early 1960s, migrations that crossed the urban-rural boundary were rather limited and did not become common until after the beginning of the economic reform in 1978. Thus, I treated the mother's place of registration as a good proxy for where her children grew up.

The following control variables are also included: (1) *ethnicity* (1 = ethnic minority, 0 = Han majority); (2) *birth order* (1^{st} – 5^{th} and more)*;* 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 expectancies (Poston Jr and Shu 1987). Birth order has been shown to have a significantly positive effect on mortality in other societies (Kaplan et al. 1992; Preston et al. 1998), and males are known to have higher mortality than females in general.

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

Statistical models

To establish basic cohort patterns of age-specific mortality risk, I estimated logistic regression models on mortality risk separately for each single-year age group, from age 0-22, which yielded 23 pairs of estimated cohort mortality difference (in odds ratio), after controlling for other covariates. This is a non-parametric approach toward cohort comparison because: (1) cohort mortality is not constrained to follow any known distribution over time, and (2) cohort difference in mortality is not constrained to follow any known distribution over time. The great flexibility

of this non-parametric approach does have limitations; there are many parameters, and it is difficult to tell to what extent they represent the true underlying cohort difference over time and to what extent they are simply due to random sampling errors.

For the rest of the analysis, I relied on various kinds of survival models. In its simplest form, a proportional hazard survival model is written as:

$$\ln h(t) = h^0(t) + \alpha X_t + \beta C \qquad (1)$$

where $\ln h(t)$ is the log-hazard of event occurrence at time t, $h^0(t)$ represents the baseline hazard, *C* denotes birth cohort, and X_t represents other covariates. The baseline hazard can be assumed as having a parametric form, such as Weibull, Gempertz, or log-logistic (parametric survival models), it can be represented by a piecewise liner spline (piecewise Gompertz model), or it can be integrated out through a partial likelihood method (Cox model). Both the piecewise Gompertz and Cox models are highly flexible in handling any forms of time dependency, while the piecewise Gompertz model has the added advantages of being easily extended to handle multilevel and multiprocess analysis (Lillard 1993). The latter is essential in estimating shared frailties and their correlations.

I started with the widely used Cox regression model, which is considered to be "semi-parametric" in the sense that it produces point estimates and a standard error of covariates using partial likelihood, but it does not assume the baseline hazard function to take any particular parametric forms. In its simplest form (as depicted in Equation (1)), covariates are assumed to have proportional effects on the baseline hazard, but non-proportional or time-varying effects can be easily incorporated by including interactions between covariates and the baseline hazard (Cleves et al. 2004).

I present here results from two Cox models. The first Cox model follows the proportional hazard assumption; the effect of all covariates, including birth cohort, remains constant over time (age). Based on this model, I tested the proportionality assumption using the Schoenfeld residuals method (Schoenfeld 1982; Grambsch and Therneau 1994). Then, I estimated the second Cox model that relaxes the proportional hazard assumption and allows the effect of all covariates that do not meet the proportionality assumption (including birth cohort) to vary over time (age).

As the last step, I jointly estimated a two-level logistic regression for infant mortality and two-level piecewise Gompertz models for mortality at subsequent ages, to tackle the issue raised by Elo and Preston (1992) and Coale and Kisker (1986) regarding the relationship between frailties at different ages. Let Y_{ij0} be the infant mortality indicator (mortality before age one) for the i^{th} child of the j^{th} woman, and let $\ln h_{ij}(t)$ be the log mortality hazard of the i^{th} child of the j^{th} woman at age t, where $t \geq 1$. A set of joint mortality models of the following form are estimated:

$$\begin{aligned} \text{logit}\left\{\Pr(Y_{ij0}=1)\right\} &= \alpha_{ij1}X + \beta_{ij1}C + \zeta_{j1} \\ \ln h_{ij}(t) &= h^{o}_{ij2}(t) + \alpha_{ij2}X + \beta_{ij2}C + \zeta_{j2} \end{aligned} \qquad (2)$$

where C denotes birth cohort and X represents a vector of the mother- and child-level covariates; the two mother-level (shared by all siblings) frailty terms ζ_{j1} and ζ_{jt} are assumed to follow a bivariate normal distribution. These frailty terms capture the residual effects of the mother's characteristics beyond the included covariates. The correlation between ζ_{j1} and ζ_{jt} thus captures the relationship between the shared frailties of infant mortality and the shared frailties of mortality at subsequent ages. The above model is a special case of the flexible multilevel

multiprocess modeling framework first proposed by Lillard (1993) and adopted and extended by various others (Panis and Lillard 1995; Upchurch et al. 2002; Steele et al. 2005), and it can be estimated using the open source software package aML (Lillard and Panis 2003).

Analysis

Describing the overall trend of cohort mortality: is there a mortality crossover between ages 0-22?

Figure 1 shows the cross-cohort trend in the child mortality hazard rates in China (up to age 18), calculated from the 1988 National Survey of Fertility and Contraception. The choice of age 18 as the cutoff point is based on the desire to maximize the number of included birth cohorts without losing comparability among them. An overall trend of declining mortality is clear; the cohort mortality hazard rate declines from 25 per thousand in the 1948 birth cohort to five per thousand in the 1970 birth cohort. There are only a few exceptions to this secular long-term trend, among which the most noticeable occurs between 1957 and 1958, where mortality increases from 13 per thousand to 14 per thousand. It is interesting to note that a sudden increase in mortality occurred in the 1958 cohort, whose members were at infancy when the famine struck, suggesting that the most detrimental event regarding cohort mortality is being exposed to the famine as an infant.

Infant mortality plays an important role in shaping cohort mortality patterns. This is most clearly demonstrated in Figure 2, which shows the non-parametric Kaplan-Meier survivor function for the three cohorts. For the pre-famine cohort, survival probability declined from 1 at age 0 to 0.80 at age 22, where 57% of the change occurred between ages 0-1. For the famine cohort, survival probability declined from 1 at age 0 to 83% at age 22, where 64% of change

occurred between ages 0-1; for the post-famine cohort, survival probability declined from 1 at age 0 to 88% at age 22, where 63% of the change occurred between ages 0-1.

Evidence from single-year age-specific logistic regression

Figure 3 plots single-year age-specific mortality odds ratio for the three cohorts, calculated from 23 logistic regression models from age 0 to age 22 (complete results tables are not presented for the sake of brevity but are available upon request from the author). Since the famine cohort is treated as the reference category in the logistic regressions, it appears in the figure as a horizontal line at the value of one. The figure reveals several important patterns. First, the pre-famine cohort has a higher mortality level than the famine cohort at almost all ages, with only a few exceptions (at age 10, 16, 18, 19, and 20). Second, the post-famine cohort has consistently lower mortality than the famine cohort from birth up to ages 11; after that, the post-famine cohort shows a consistently higher mortality than the famine cohort, with only one exception (at age 17).

In short, the contrast in age-specific mortality between the famine and the post-famine cohort, as depicted in Figure 2, suggests the presence of the mortality crossover point between ages 11 and 12.

Evidence from Cox regression models

Table 2 reports coefficients estimated from two Cox regression models. Model 1 restricts all included effects to be proportional to the baseline hazard and does not allow the coefficients to be changed over time. Statistical tests of the proportional hazard assumption based on Schoenfeld residuals show that the proportional hazard assumption does not hold for the

following covariates: place of registration, birth cohort, mother's education, birth order, and sex, making Model 1 a misspecified model.

A simple remedy is to treat these variables as time-varying, which can be easily done by (1) first splitting the duration spell into two parts, those between age 0 and ages 11 (including ages 11), and those between 11 and 22, and (2) including an interaction between the cohort dummy variable and the new duration spells. Technical details on how to handle these operations using Stata can be found in Cleves et al. (2004). The results are reported as Model 2 in Table 2.

Comparing Model 1 and Model 2 reveals that allowing time-varying coefficients drastically improves model fit, as revealed by the change in the log likelihood value. Based on Model 2, having a *hukou* in cities had much greater impact on mortality between ages 0-11 (hazard ratio = 0.40) than between 11-22 (hazard ratio = 0.66), as did the mother's education (hazard ratio = 0.95 between 0-11 and hazard ratio = 0.98 between ages 11-22) and birth order effect. In contrast, gender difference in mortality increased drastically between ages 0-11 and ages 11-22, from no significant gender difference (hazard ratio = 1.03) to a significant male disadvantage (hazard ratio = 1.27). The effect of ethnicity did not vary between ages: ethnic minority had significantly higher mortality than the Han majority (hazard ratio = 1.45); the effect of having a *hukou* in towns did not vary between ages either: people who had *hukou* in towns had a significantly lower mortality than people who had *hukou* in villages (hazard ratio = 0.79).

The pre-famine cohort had a significantly higher mortality than the famine cohort between ages 0-11 (hazard ratio = 1.13), but the difference diminished between ages 11-22 (hazard ratio = 1.07) and lost statistical significance. The most dramatic change was the difference between the post-famine and the famine cohorts. The post-famine cohort had a significantly lower mortality than the famine cohort at ages 0-11 (hazard ratio = 0.70), but a

significantly higher mortality at ages 11-22 (hazard ratio = 1.30), indicating the presence of mortality crossover between ages 11 and 12.

Evidence from multilevel and multi-process models

To rule out the possibility that the post-famine mortality crossover between ages 11 and 12 identified in earlier sections was merely incidental or caused by forces other than selection by frailty, I constructed two multilevel multiprocess models that jointly estimate an infant mortality model and (1) a morality model between age 1-11 and (2) a mortality model between ages 11-22. The goal was to replicate results from the Cox regression model (especially the mortality crossover) and to establish a relationship between the frailties for infant mortality and for mortality at subsequent ages. In addition, by comparing models with and without interaction between birth cohorts and a mother's education and place of registration in the infant mortality, I determined whether the effect of famine had a socioeconomic gradient.

Table 3 presents four multilevel multiprocess models. Models 3 and 4 jointly model infant mortality and mortality at ages 1-11, and Models 5 and 6 jointly model infant mortality at ages 12-22. The difference between Models 3 and 4, and between Models 5 and 6, is that Models 4 and 6 add to Models 3 and 5 interaction terms between birth cohort and a mother's education and place of registration in the infant mortality equation. As the results show, none of the interaction terms is statistically significant, and likelihood ratio tests show that the more parsimonious models (Model 3 and 5) are the preferred models, indicating no socioeconomic gradient in the infant mortality consequence of the famine. Thus, I will focus on Models 3 and 5.

It is worth noting that the common component shared by both Models 3 and 5, the random intercept logistic regression for infant mortality, yielded almost identical estimates. People with *hukou* in cities (odds ratio = 0.37) and towns (odds ratio = 0.78) had a much lower

infant mortality risk than people with *hukou* in villages; a mother's education significantly reduces infant mortality (odds ratio = 0.94); ethnic minorities have a much higher infant mortality than the Han majority (odds ratio = 1.55); boys have significantly higher infant mortality than girls (odds ratio = 1.11); and infant mortality increases with birth order. As for cohort differences, both pre-famine and post-famine cohorts have lower infant mortality than the famine cohort, but the difference between the pre-famine and the famine cohort is small and not significant (odds ratio = 0.99), while the difference between the post-famine and the famine cohorts is substantial and statistically significant (odds ratio = 0.69).

It is informative to compare coefficients estimated for mortality at ages 1-11 and at ages 12-22. First, people having *hukou* in cities and towns had lower mortality at both age groups than people having *hukou* in villages, but the difference was greater at ages 1-11 (hazard ratio = 0.37 for cities and 0.75 for towns) than at ages 12-22 (hazard ratio = 0.63 for cities and 0.83 for towns). A mother's education reduced mortality in both age groups, but the effect was much greater at ages 1-11 (hazard ratio = 0.94) than at ages 12-22 (hazard ratio = 0.98). In fact, the effect of the mother's education on mortality at ages 12-22 was not statistically significant. Ethnic minorities tended to have higher mortality than the Han majority in both age groups, but more so at ages 1-11 (hazard ratio = 1.58) than at ages 12-22 (hazard ratio = 1.21). Interestingly, gender difference in mortality risk was completely opposite between these two age groups; boys have significantly lower mortality at ages 1-11 (hazard ratio = 0.92), but significantly higher mortality at ages 12-22 (hazard ratio = 1.30) than girls. Another interesting finding is that, while mortality risk at ages 1-11 increased significantly with parity, there was no significant parity effect on mortality at ages 12-22. Finally, with regard to cohort comparisons, the pre-famine cohort had higher mortality than the famine cohort in both age groups (although the difference

was not significant for ages 12-22); the post-famine cohort had much lower motility (hazard ratio = 0.65) at ages 1-11, but a significantly higher mortality than the famine cohort (hazard ratio = 1.17) at ages 12-22 than the famine cohort. Despite the slight numerical difference, these results agree with the Cox models as reported in Table 2 and indicate the presence of mortality crossover between ages 11 and 12.

The unique contributions of multilevel multiprocess models are the estimated standard deviations and correlations of the three unobserved shared frailties (for infant mortality, mortality at ages 1-11, and mortality at ages 11-22) and two correlation coefficients (between infant mortality and mortality at ages 1-11, and between infant mortality and mortality at ages 11-22). In the present case, all three frailties were substantial and statistically significant, indicating a strong presence of mother-level clustering. A significantly positive correlation between frailties for infant mortality and mortality at ages 1-11 ($\beta = 0.27$) and at ages 12-22 ($\beta = 0.18$) reveals that, among these three cohorts of children, those who have a higher infant mortality would also tend to have a higher mortality at subsequent ages. In the famine context, the excess infant mortality that occurred in the famine cohort, as depicted in Figure 2, tended to be those who would also have a higher mortality at subsequent ages. In other words, members of the famine cohort who had survived infancy had, on average, a lower level of frailty for mortality between ages 1-22, which inevitably leads to mortality convergence between the famine and the post-famine cohorts and eventually a crossover in cohort mortality.

Discussion

The Great Leap Forward Famine between 1959 and 1961 in China is unparalleled in modern human society. It led to a tragic loss of over 30 millions human lives in a short period of time

and created a cohort of children being exposed to extreme nutritional deprivation at infancy or early childhood, making it the largest *natural experiment* of the effects of famine in human history. Here, I have attempted to contribute to the small but increasing literature concerning the long-term effects of famine on various health and life chance outcomes by (1) identifying a post-famine mortality crossover between the famine and the non-famine cohort between ages 11 and 12; (2) explaining how the debilitation and the selection effect of the famine together determine the timing of this mortality crossover; and (3) revealing the underlying causal mechanisms through which post-famine mortality convergence and eventually mortality crossovers are produced.

The omnipresence of selection effect deserves more attention in evaluating and interpreting other long-term effects of famine. Any conclusions about the long-term effects based on cohort comparison, directly or indirectly, are also based on the assumption that the famine and non-famine cohorts are comparable. The present study shows that this assumption does not hold in the case of cohort mortality because the famine cohort is composed of famine survivors who are genetically stronger and much less frail than the non-famine cohort. In other research settings where the primary outcomes are not mortality, cohort comparability should not be assumed automatically without thorough scrutiny because these outcomes might be correlated with frailty in one way or the other; when they do, the powerful force of selection might have led to completely erroneous conclusions. A good example of how selection by frailty distorts cohort difference in height is provided by Gorgens et al. (2008).

The present research also contributes to the demographic literature of mortality crossover. The main debate during the past century regarding mortality crossovers is whether the crossover is an artifact caused by age overstatement at advanced ages or a real population phenomenon

caused by selection by frailty (Spiegelman 1967; Coale and Kisker 1986; Nam 1995; Johnson 2000). It is difficult to reach a unanimous consensus based on evidence from ordinary (non-famine) times because (1) the group difference in mortality between the advantaged and the disadvantaged is not large enough, which makes the hypothesized crossover come at very advanced ages, if it comes at all, and (2) this makes it impossible to rule out age overstatement as an alternative explanation. The extreme condition provided by famine rules out the possibility of age overstatement (age misreporting is possible, but there is no reason to believe the pattern of misreporting differ drastically between ages 11 and 12), and it accelerates the pace of the process of mortality convergence. As a result, in the present case, a crossover in mortality between the famine and the non-famine cohort shows up between ages 11 and 12. In addition, the multilevel multiprocess analysis provides direct evidence regarding the relationship between frailties for infant and child mortality, showing the theoretical inevitability of a morality crossover as the result of the convergence process, caused by selection by frailty.

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

	Pre-Famine (1956-1958)	Famine (1959-1961)	Post-Famine (1962-1964)
Mother's Characteristics			
Type of Residence			
City (%)	18	19	14
Town (%)	21	22	22
Village (%)	60	59	64
Ethnic Majority			90
Han (%)	91	90	89
Non-Han (%)	9	10	11
Child's Characteristics			
Birth Order			
1^{st} Birth (%)	43	30	25
2^{nd} Birth (%)	32	26	25
3^{rd} Birth (%)	17	21	19
4^{th} Birth (%)	6	13	14
5^{th} and Above Birth (%)	2	9	17
Male (%)	51	52	52
Mother's Education	1.67	2.16	2.37
Mother's Age at birth	24.05	24.62	26.24

Note: No. of mothers = 90,203; No. of children = 207,046.

Table 2. Results from Cox regression model and with and without non-proportional covariate effects (time-varying coefficients), N=207,046.

	Model 1: Cox proportional hazard model	Model 2: Cox model with time-varying coefficients	
		Age ≤ 11	Age > 11
Type of residence[a]			
Cities	-0.89*** (0.02)	-0.92*** (0.02)	-0.41*** (0.08)
Towns	-0.23*** (0.01)	-0.23*** (0.01)	-0.23*** (0.01)
Birth cohort[b]			
Pre-famine	0.11*** (0.01)	0.12*** (0.02)	0.07 (0.07)
Post-famine	-0.32*** (0.01)	-0.35*** (0.01)	.26*** (0.06)
Mother's age	-0.06*** (0.02)	-0.06*** (0.02)	-0.06*** (0.02)
Mother's age (squared)	-0.00 (0.00)	-0.00 (0.00)	-0.00 (0.00)
Mother education	-0.05*** (0.00)	-0.05*** (0.00)	-0.02** (0.01)
Ethnic minority	0.37*** (0.02)	0.37*** (0.02)	0.37*** (0.02)
Male	0.04*** (0.01)	0.03* (0.01)	0.24*** (0.05)
Birth order			
2nd	0.27*** (0.02)	0.27*** (0.02)	0.13* (0.06)
3rd	0.42***	0.42***	0.34***

	(0.02)	(0.02)	(0.07)
4th	0.64***	0.65***	0.49***
	(0.03)	(0.02)	(0.08)
5th and above	0.91***	0.93***	0.50***
	(0.03)	(0.03)	(0.10)
Log likelihood	-381709.22	-381573.74	

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

[a]The reference category is “villages”

[b]The reference category is “famine cohort”

[c]The reference category is “the 1st birth”

Table 3. Results from joint multilevel infant and child mortality models.

	Model 3: infant and child mortality (1-11)	Model 4: infant and child mortality (1-11) with interactions	Model 5: infant and child mortality (12-22)	Model 6: infant and child mortality (12-22) with interaction
Fixed effects: infant mortality				
Intercept	0.73 *	0.77 *	0.73 *	0.77 *
	(0.32)	(0.32)	(0.32)	(0.32)
Place of registration				
City	-1.00 ***	-1.01 ***	-1.00 ***	-1.01 ***
	(0.06)	(0.03)	(0.06)	(0.03)
Town	-0.25 ***	-0.25 ***	-0.25 ***	-0.25 ***
	(0.04)	(0.02)	(0.04)	(0.02)
Birth cohort				
Pre-famine	-0.01	-0.01	-0.01	-0.01
	(0.03)	(0.02)	(0.03)	(0.02)
Post-famine	-0.37 ***	-0.38 ***	-0.37 ***	-0.38 ***
	(0.03)	(0.02)	(0.03)	(0.02)
Mother's age	-0.18 ***	-0.18 ***	-0.18 ***	-0.18 ***
	(0.03)	(0.03)	(0.03)	(0.03)
Mother's age (squared)	0.00 **	0.00 **	0.00 **	0.00 **
	(0.00)	(0.00)	(0.00)	(0.00)
Mother's education	-0.06 ***	-0.06 ***	-0.06 ***	-0.06 ***
	(0.01)	(0.00)	(0.01)	(0.00)
Ethnic minority	0.44 ***	0.44 ***	0.44 ***	0.44 ***
	(0.03)	(0.03)	(0.03)	(0.03)
Male	0.10 ***	0.10 ***	0.10 ***	0.10 ***
	(0.02)	(0.02)	(0.02)	(0.02)

Birth order				
2nd birth	0.25 ***	0.25 ***	0.25 ***	0.25 ***
	(0.02)	(0.02)	(0.02)	(0.02)
3rd birth	0.43 ***	0.43 ***	0.43 ***	0.43 ***
	(0.03)	(0.03)	(0.03)	(0.03)
4th birth	0.70 ***	0.70 ***	0.71 ***	0.70 ***
	(0.03)	(0.03)	(0.03)	(0.03)
5th and above	0.99 ***	0.99 ***	0.99 ***	0.99 ***
	(0.04)	(0.04)	(0.04)	(0.04)
Pre-famine × Mother's education		-0.01		-0.01
		(0.01)		(0.01)
Post-famine × Mother's education		0.01		0.01
		(0.01)		(0.01)
Pre-famine × Cities		0.13		0.12
		(0.08)		(0.08)
Pre-famine × Towns		-0.08		-0.08
		(0.08)		(0.08)
Post-famine × Cities		-0.02		-0.02
		(0.05)		(0.05)
Post-famine × Towns		0.00		0.00
		(0.05)		(0.05)
Fixed effects: child mortality				
Intercept	-4.45 ***	-4.45 ***	-13.03 ***	-13.03 ***
	(0.42)	(0.42)	(1.03)	(1.03)
Place of registration				
Cities	-1.00 ***	-1.00 ***	-0.47 ***	-0.46 ***
	(0.04)	(0.04)	(0.09)	(0.09)

Towns	-0.29 ***	-0.29 ***	-0.19 **	-0.19 **
	(0.03)	(0.03)	(0.06)	(0.06)
Birth cohort				
Pre-famine	0.38 ***	0.38 ***	0.14	0.14
	(0.03)	(0.03)	(0.08)	(0.08)
Post-famine	-0.45 ***	-0.45 ***	0.18 ***	0.18 ***
	(0.03)	(0.03)	(0.07)	(0.07)
Mother's age	0.04	0.04	0.01	0.01
	(0.03)	(0.03)	(0.08)	(0.08)
Mother's age (squared)	0.00 **	0.00 **	0.00	0.00
	(0.00)	(0.00)	(0.00)	(0.00)
Mother's education	-0.06 ***	-0.06 ***	-0.02	-0.02
	(0.00)	(0.00)	(0.01)	(0.01)
Ethnic minority	0.46 ***	0.46 ***	0.19 *	0.19 *
	(0.03)	(0.03)	(0.08)	(0.08)
Male	-0.08 ***	-0.08 ***	0.24 ***	0.24 ***
	(0.02)	(0.02)	(0.05)	(0.05)
Birth order				
2^{nd} birth	0.39 ***	0.39 ***	-0.03	-0.04
	(0.03)	(0.03)	(0.07)	(0.07)
3^{rd} birth	0.52 ***	0.51 ***	0.03	0.03
	(0.03)	(0.03)	(0.09)	(0.09)
4^{th} birth	0.70 ***	0.69 ***	0.08	0.09
	(0.04)	(0.04)	(0.10)	(0.10)
5^{th} and above	0.90 ***	0.89 ***	-0.03	-0.03
	(0.05)	(0.05)	(0.13)	(0.13)
Random effects				

σ_i	0.97 ***	0.97 ***	0.97 ***	0.97 ***
	(0.02)	(0.02)	(0.02)	(0.02)
σ_c	0.89 ***	0.89 ***	1.09 ***	1.09 ***
	(0.02)	(0.02)	(0.11)	(0.11)
ρ	0.27 ***	0.27 ***	0.18 *	0.18 *
	(0.03)	(0.03)	(0.08)	(0.08)
Log Likelihood	-171414.13	-171409.01	-81579.35	-81574.33

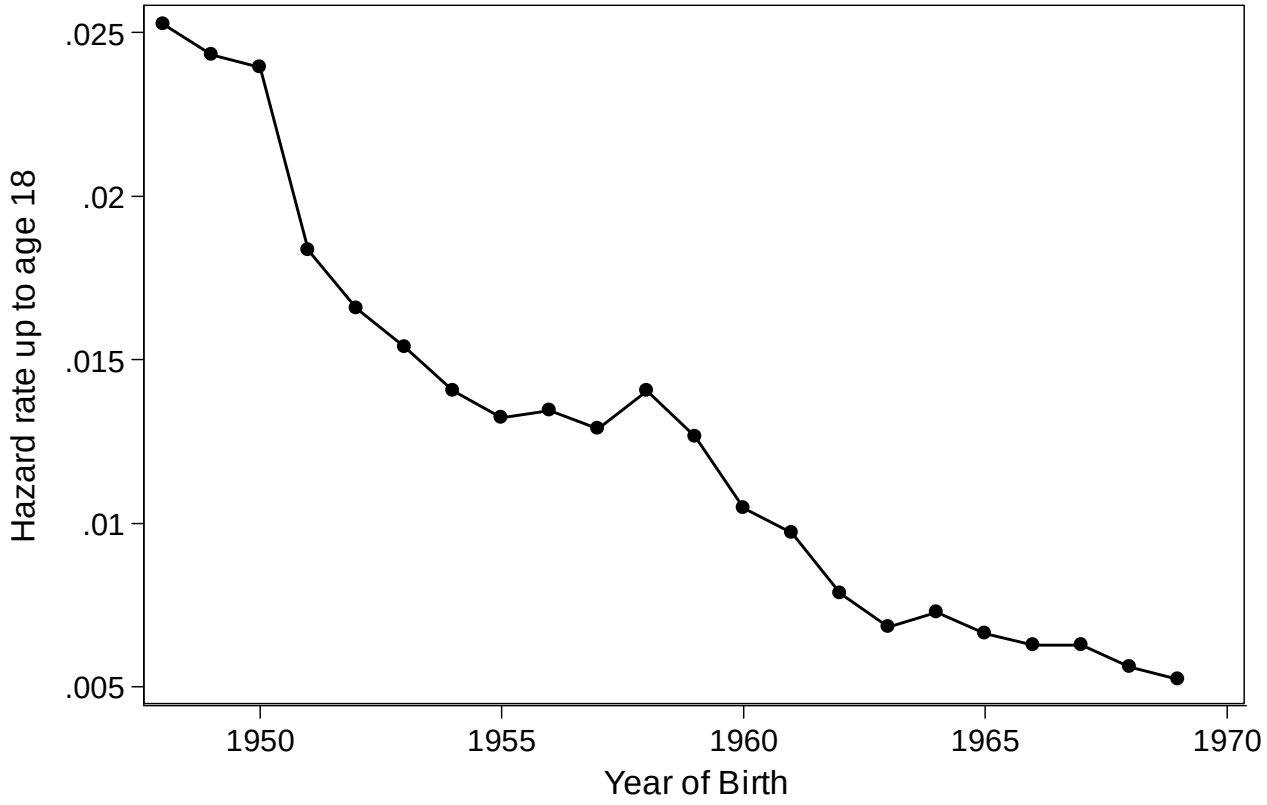
$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$

[a]The reference category is “villages”

[b]The reference category is “famine cohort”

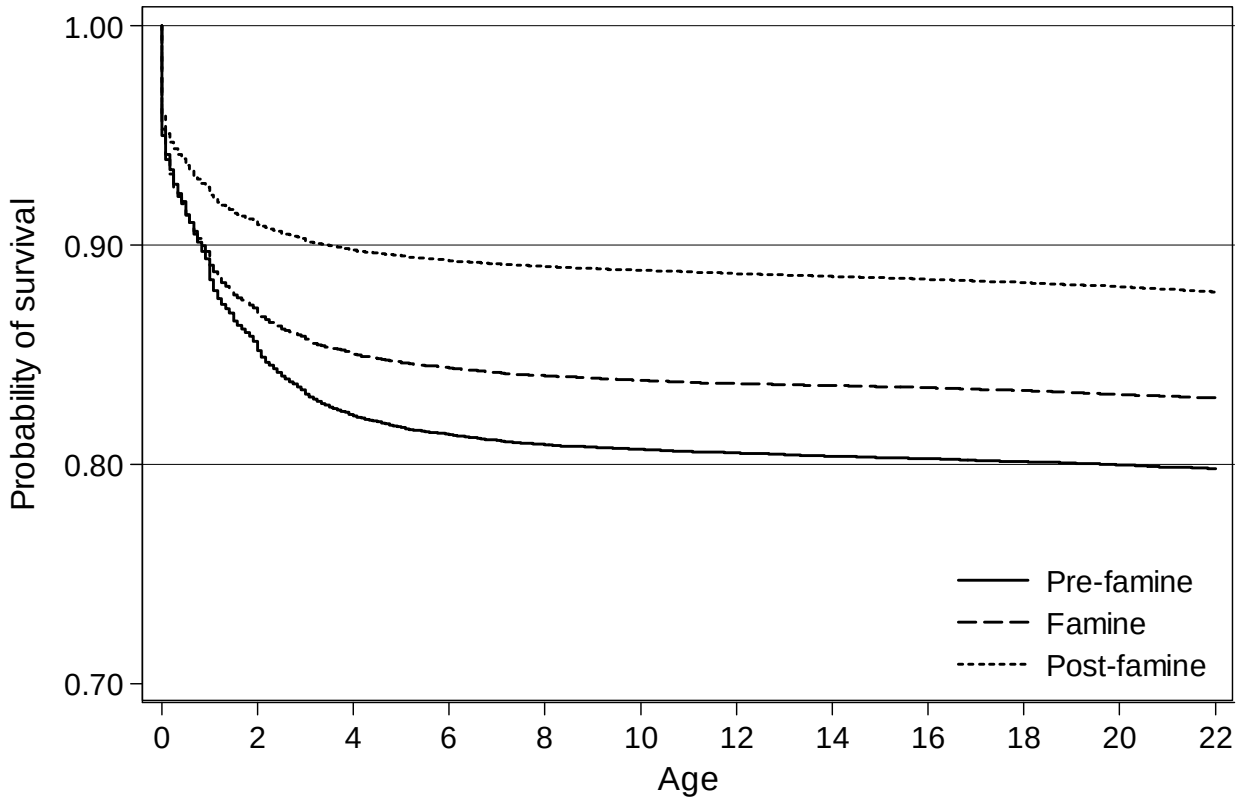
[c]The reference category is “the 1st birth”

Figure 1. Overall trend in cohort mortality hazard (age ≤ 18) in China.



Note: Cohort mortality rate is calculated using the 1988 National Survey of Fertility and Contraception in China. The very early cohorts (1941-1946) with small cohort size and the very late cohorts (1971-1988) were excluded to ensure that the cohorts were comparable.

Figure 2. Kaplan-Meier estimates of the survival function up to age 22 for three Chinese cohorts.



Note: Cohort mortality rate was calculated using the 1988 National Survey of Fertility and Contraception in China. The pre-famine cohort refers to those who were born in 1956-1958; the famine cohort refers to those who were born in 1959-1961; the post-famine cohort refers to those who were born in 1962-1964.

Figure 3. Predicted age-specific mortality odds ratios of the pre-famine (1956-1958), the famine (1959-1961), and the post-famine cohort (1962-1964).