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Mortality consequences of the 1959–1961 Great Leap Forward famine in China: Debilitation, selection, and mortality crossovers☆

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ABSTRACT

Using retrospective mortality records for three cohorts of newborns (1956–1958, 1959–1961, and 1962–1964) drawn from a large Chinese national fertility survey conducted in 1988, this article examines cohort mortality differences up to age 22, with the aim of identifying debilitating and selection effects of the 1959–1961 Great Leap Forward Famine. The results showed that the mortality level of the non-famine cohort caught up to and exceeded the level of the famine cohort between ages 11 and 12, suggesting both debilitating and selection effects. Multilevel multiprocess models further established a more direct connection between frailties in infancy and frailties at subsequent ages, revealing the underlying dynamics of mortality convergence between the famine and the non-famine cohorts caused by differential excess infant mortality. These results provide important new insights into the human mortality process.

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 causing an immediate rise in mortality, famine may also have a "long-term" or "sustained" mortality consequence. In a study of the 1974–1975 Bangladesh famine, Razzaque, Alam, Wai, and Foster (1990) showed that the famine-born cohorts continued to have a higher than usual mortality in the second year of life, one year after the famine was over, then followed by a lower than usual mortality between the ages 2 and 5. Kannisto, Christensen, and Vaupel (1997), in their study of the 1866–1869 Finnish famine, reported that the famine cohort had an higher than usual mortality up to age 17 but no cohort difference afterwards.

In both cases, the famine-born cohorts continued to show a higher mortality than the non-famine cohorts after the famine was over; then the cohort difference in mortality either disappeared (in the Finnish case) or even got reversed (in the Bangladeshi case). Such a pattern, known to demographers as "mortality crossover" (Nam, 1995), was considered as evidence for both "debilitation effect" – a lingering negative impact of poor nutrition and harsh environment on individual health, and "selection effect" – a result of selective frailty processes on cohort-level mortality difference. Debilitation and selection effects may occur in non-famine situations as well. For example, Caselli and Capocaccia (1989) reported a mortality pattern in Italy that was similar to the Bangladeshi case: cohorts that experienced unfavorable conditions early in life tended to show increased cohort mortality up to a certain age, followed by reduced cohort mortality afterwards. The main difference between the Bangladeshi and Italian studies was the timing of the turning point – the crossover point in Bangladesh occurred at age two, while in Italy it occurred at age 45.

Treating mortality crossover as suggestive evidence for debilitation and selection effects represents an important step toward a better understanding of long-term health consequences of famine. Doing so, however, raises several issues that need further discussion. First of all, the above reasoning implicitly assumes that debilitation and selection effects always work against each other – that debilitation increases mortality of the famine cohort while selection decreases it – in determining the observed cohort mortality trend, but not all researchers accept that assumption without reservation (Elo & Preston, 1992). Second, the nature and dynamics of selection effects, compared to that of debilitation effects, is much less well understood. For example, while debilitation effect has been suggested to be strong at the beginning of the

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post-famine era and then gets weaker over time, there has been little discussion regarding whether selection effect follows similar or different trends. Without such knowledge, the claim that mortality crossover indicates changes in relative strength of debilitation and selection effects in the post-famine period becomes much less convincing. This leads to the next point. If, for some reasons, no mortality crossover is observed after a famine, what can we say about debilitation and selection?

In this research, I examined long-term mortality consequences of the 1959–1961 Great Leap Forward Famine in China. I first reviewed conceptual and methodological issues in identifying debilitation and selection effects. Then I proposed a statistical approach to isolate selection effect, borrowing strength from the recent developments in multilevel multiprocess latent variable modeling methodology (Lillard, 1993; Panis & Lillard, 1995; Steele, Kallis, Goldstein, & Joshi, 2005). Using data from a large representative national sample survey conducted in 1988 in China, I (1) identified mortality crossover between the famine and the non-famine cohort; (2) demonstrated that famine may "reduce" mortality level of the famine cohort in the post-famine period by eliminating its frail members first; and (3) showed that both debilitation and selection effects declined with time, and debilitation effect declined faster than selection effect.

Identifying debilitation and selection effects after famine

Conceptualizing debilitation and selection

In the context of famine and other natural disasters, the "debilitation" or "scaring" effects refer to the possibility that certain negative conditions (e.g. diseases, malnutrition at birth, and growth retardation) experienced early in life may permanently impair the health of survivors and thus leave an imprint on their mortality risks at all subsequent ages (Almond, 2006; Elo & Preston, 1992). Selection effects, on the other hand, refer to the possibility that famine survivors (the surviving part of the famine cohort) tend to be unusually well endowed with some genetic or congenital traits that may reduce mortality risk later in life (Almond, 2006; Elo & Preston, 1992; Preston, Hill, & Drevenstedt, 1998). In short, in a cohort mortality comparison between the famine-born and the non-famine-born cohorts, debilitation effect tends to increase the mortality risk of the famine cohort, while selection effect tends to "reduce" the mortality risk of the famine cohort, relative to the non-famine cohort.

It is important to understand that debilitation and selection effects work at different conceptual levels and through different casual mechanisms. Debilitation is results of individual-level biomedical processes that can be identified and measured in laboratory settings or controlled experiments, and there have been extensive animal studies successfully identifying some types of debilitation effect (Rasmussen, 2001). The difficulty with measuring debilitation effects on human subjects is mainly ethical and legal: there is no justification to deliberately put pregnant women and newborn babies in harmful situations such as severe nutritional deprivation and psychological stress for extended period of time for research purpose. This is why many researchers consider famine as a good opportunity to identify debilitation effect of prenatal or early life exposure to malnutrition on human subjects, a "natural experiment". By resorting to simple cohort comparison, however, these researchers failed to account for an important difference between a natural experiment and a true randomized experiment: cohort attrition due to differential mortality, which is the source of selection effect (Lumey & Stein, 1997; St Clair et al., 2005).

Selection effect does not actually increase or decrease individual's mortality risk in the usual "treatment-effect" sense because it is not a result of processes operating at individual-level. Instead, it is a cohort-level phenomenon, a statistical artifact, produced by "unfair" comparisons in mortality level between a complete cohort (the non-famine cohort) and a positively selected subset of a cohort (the famine cohort) consisting of only the genetically strong and well-endowed individuals. Selection will not bias the result from cohort comparison if any of the following conditions are met: (1) there is no excess mortality, (2) excess mortality is not differentiated by health endowment, or (3) health endowment can be adequately measured and controlled for. Unfortunately, it has been a known fact that famine causes excess mortality (Bongaarts & Cain, 1981; Peng, 1987); the nature and characteristics of such excess mortality have not been well understood, but ecological analysis suggest the possibility of differential mortality; and health endowment is a theoretical construct that cannot be directly observed or easily measured.

In short, even though debilitation and selection effects operate at different levels and through different mechanisms, they cannot be easily separated from each other in most empirical research settings, due to the inherent difficulties in obtaining adequate measure of health endowment. Without such a separation, any statements regarding debilitation and selection effects based on observed cohort patterns and trends remain tentative.

Frailty, frailty model, and latent variable modeling framework

Health endowment and frailty are used exchangeably in empirical studies (Bhalotra & Soest, 2007; Lee, Rosenzweig, & Pitt, 1997; Schultz, 1984, 2003); and frailty is often associated with frailty model – an attempt to statistically address the issue of selection bias caused by unobserved heterogeneity in health endowment. By incorporating a multiplicative random component into the hazard function of ordinary survival models, frailty model aims to estimate the "true" individual-level hazard function that is free of selection bias, which may or may not be the same as the group-level hazard function (Vaupel, Manton, & Stallard, 1979; Vaupel & Yashin, 1985). More recent research has shown that frailty model is a special case of a more general statistical modeling framework: latent variable model (Skrondal & Rabe-Hesketh, 2004).

The latent variable approach toward health endowment or frailty has obvious appeals. By focusing on mortality instead of health measurements, such a statistical approach makes it possible to obtain important insights regarding the human mortality process in the absence of comprehensive health information, as in most social and demographic surveys. However, relying solely on individual's mortality indicator has its own problems. First, not all deaths can be attributed to frailty. For example, people may die of accidents, and young children may die of parental negligence, which have nothing to do with frailty. In these cases, mortality itself is not a good indicator of frailty; without additional information, it is not possible to separate frailty from other idiosyncratic factors that may also influence mortality. Second, death is a terminal event that cannot happen more than once, if respondents die in infancy, there is no information to directly identify their frailties in childhood, in adolescence, or in adulthood. This is, in some sense, analogous to the issue regarding the relationship between the propensity to work and observed wage in economics – for those who chose not to work, there would be no information to identify their earning potential, which inspired Heckman's proposal of jointly estimating a probit model for the propensity to work and a linear regression model for the observed wage (Heckman, 1979). The identification of such models, as pointed out by Little and Rubin (1987), among others, depends heavily on the existence of good