The 1958-61 Famine Effects on Middle-Aged Adult Mortality in China: Debilitation versus Selection

Yong Li
Johns Hopkins University

Extended Abstract

Introduction

The 1958-61 famine in China is regarded as the world's largest famine in human history. Though complete and accurate population statistics is still lacking for that time, it is estimated approximate 30 million people died and about equal number of births were lost or postponed (Ashton et al. 1984, Hill 1988, Peng 1987, Kane 1988). The long-term effects of 1958-61 famine on the health of people who were born during famine and who were exposed to the famine in their developmental stages (e.g. childhood and adolescence) are still unknown. Since 1980s, China conducted and published three national Censuses and annual population statistics based on sampling surveys. The availability of mortality data in 1980s and 1990s make the assessment of the long-term effects of the 1958-61 famine on current Chinese adult mortality possible.

Theoretical focus

The focus of this study has two folds. First, it is to show whether there exists a long-term effect of the 1958-61 famine on current adult mortality in China, as a direct test of the fetal origins hypothesis of adult mortality (Barker 1998). Second, it is to study and distinguish two kinds of mechanisms through which famine may impose its effect: debilitation versus selection. The debilitation effect is related to the positive relationship between early life conditions and adult mortality, reflecting the aggregate change of mortality due to risk change at individual level. The selection effect is related to the negative relationship between early life conditions and adult mortality, reflecting the aggregate change of mortality due to change of population composition.

It is well-known that early life conditions can have strong effect on health and mortality in later life (e.g. Elo and Preston 1992, Hayward and Gorman 2004). Through starvation-incurred severe nutritional deficiency, famine can debilitate the health of survivors, including those born during famine. It may be particularly harmful for fetuses, infants, children and adolescents who are at the critical periods of development, therefore affect their health in the long run. Recent development in life course perspective of chronic epidemiology incorporates both fetal origins hypothesis and social origins of chronic diseases as to understand the etiology and evolution of chronic diseases in adulthood (Kuh and Ben-Shlomo 2004). Fetal origins hypothesis proposes that nutritional deficiency in prenatal and early postnatal period increases the risk of adult diseases and mortality through the complex fetal programming process (Barker 1998). A number of epidemiological studies based on birth records and retrospective surveys have shown a strong individual association between various prenatal and infancy growth markers and adult diseases and mortality (e.g. in Kuh and Ben-Shlomo 2004).

Instead of using indirect measures of fetal conditions, studying the famine effects on people born during the famine serves a direct test of fetal origins hypothesis. Previous studies of famines in European countries found both positive and negative results in terms of the association between early life exposure to famine and health and survival in later life. Cohorts born in Finland during the severe 1866-68 famine were found no differential in survival after age 17 with cohorts born before and after the famine (Kannistor et al. 1997). A group of men and women who were in utero during the 1941-44 Leningrad siege was found no difference in mean blood pressure and glucose-insulin metabolism with people born at the same time who were not subject to famine conditions in utero (Stanner et al 1997). However, studies on Dutch Hunger Winter 1944-45 have shown that the offspring who were exposed to maternal malnutrition during early gestation have elevated coronary heart disease in later life (Roseboom et al. 2000), and those who were undernourished at any stage of gestation are linked with reduced glucose tolerance and insulin resistance controlling for low birth weight (Ravelli et al. 1998). Paralleled studies on two World Wars in the twentieth century also found compelling

evidence that in general people who were born in and who were adolescents during wartime have elevated adult mortality relative to neighboring cohorts (Horiuchi 1983; Caselli and Capocaccia 1989, Tango and Kurashina 1987, Anderson and Silver 1989, Wilmoth et al. 1990, Willenkens and Scherbov 1992). To explain this, fetal nutrition deficiency is an important candidate among various proposed causal factors. Thus, the focus on the possible debilitation effect of the 1958-61 famine in China follows the above line of research. Comparatively, such kind of study has been rare in the developing countries where famine and malnutrition is more prevalent historically and where significant transition towards chronic diseases is occurring.

Elo and Preston (1992) mentioned two processes that operate in the opposite direction of the positive association between childhood conditions and adult mortality: selection and acquired immunity. Given the enormity of the 1958-61 famine, an equally compelling argument is that the selection process may be at play. It may be the case that famine survivors is a selected group who were in general more robust (both physically and mentally) than those who failed to go through the harshness of the disaster, particularly for children and adolescents. Therefore, for the young famine survivors, their mortality in adulthood would be lowered relative to what would be the case under non-famine condition in which more of the frail individuals survive, holding all other conditions constant.

The above asserted positive correlation between genetic susceptibility to death in childhood (infectious diseases) and in adulthood (degenerative diseases) has been questioned from the view of evolutionary biology that the forces of evolution have paid little attention to post-reproductive mortality (Rose 1991, cited in Elo and Preston 1992, p. 195). However, dying from starvation may be quite different from dying of infection in childhood. In the study of Finland 1866-68 famine, Kannisto et al (1997) found the survival from birth to age 17 years was significantly lower in cohorts born before and during the famine than the cohorts born after the famine. Though at the subsequent ages, there are no observed differential mortality between the famine-born cohorts and other five cohorts born before and after the famine. Noymer and Garenne (2000) studied the

effect of 1918 Spanish Influenza on sex differentials in morality in the United States. They found strong sex-specific selection effect: huge male excess of deaths in the 1918 epidemic reduced male death rates in subsequent years and changed sex differential of mortality (as measured by the expectation of life at birth or by the age-standardized death rate) for 15 years. Studies on this subject, though limited, seemly suggest that after a great shock or huge disaster, the selection process do exist but only manifest itself for a relatively short period (e.g. less than 20 years). In the case of 1958-61 famine in China, the survivors who were young at the time (say, less than age 15) were at their 20s and 30s in 1980 and up to 40s and 50s in 2000 during the period mortality data are available in China. Thus, it is of interest to examine whether a possible selection effect of the famine exists and manifests through the middle-aged adult mortality in China.

The development of life course studies on chronic epidemiology as to understand the etiology and evolution of chronic diseases is equally needed for developing countries. In China, public health research and policies have geared towards prevention and control of chronic diseases. The burden of chronic diseases is increasingly heavy in China. In 2000, chronic diseases already accounted for two thirds of total deaths in both urban and rural areas. With the advance of socioeconomic development in China, a transition from the traditional diet with high fiber and low fat and low energy to the modern diet with low fiber and high fat and high energy is occurring, particularly in urban areas (Ji et al. 2004). Along with other life style and behavioral changes, the interaction between recently increased nutritional intake and past history of malnutrition among people who were exposed to the famine in utero or during childhood may not as good as one would expect. It may be even harmful as the thrifty phenotype hypothesis (Hale and Barker 1992) asserts the imbalance between fetal under-nutrition and postnatal over-nutrition triggers metabolic disorders and associated chronic diseases in adulthood. Therefore, it might be possible to compare the differentials of famine effects between urban and rural areas within China, as a partial examination of the thrifty phenotype hypothesis as well.

Data and research method

To investigate the famine effects, it requires mortality data available at the single-year age interval through which the relevant birth cohorts can be determined as precisely as possible. The major data consist of mortality data from three China Censuses in 1982, 1990 and 2000, two large 1 per cent sampling surveys in 1987 and 1995, and annual population statistics available in 1994-2002 at the national level. The detailed mortality data are also available for each of 31 provinces in 1990 and 2000 Census, and for some provinces in 1987 and 1995 large sampling surveys. All the data are stratified by sex and urban-rural residence though the definition of urban and rural area has not been consistent through all the sources. We use urban area refers to cities and towns while rural area refers to only counties.

We are going to employ a number of and different approaches of methods to facilitate better observation and explain better what we might observe. The three Censuses provide the best quality of mortality data for China at both national and provincial level in three cross-sectional time points. Firstly, we employ a very useful exploratory tool developed by Horiuchi (1983, Horiuchi and Coale 1990), also called the life-table aging rate (LAR, Horiuchi 1997, 1998), for each of three Censuses. The LAR is defined by $k(x) = \frac{d \log(\mu(x))}{dx}$ where μ(x) is the mortality rate at exact age x. The LAR actually reflects the age-specific growth rate of mortality. Therefore, an unusually high value suggests relatively elevated mortality at the ages. Plotting the age pattern of *k(x)* will suggest a high-mortality cohort existence between the trough and the peak. Figure 1 shows the LAR pattern of 3-year age groups for China in 2000. The age group 39-41 in 2000 corresponds to the famine cohorts 1959-61. The statistically significant increase of LAR for that age group implies increased mortality for famine cohorts relative to neighboring cohorts. We also calculate LAR for each province and six larger geographic regions. Increased LAR for famine cohorts are observed across and quite consistent with the famine coverage across the country. These results will be available in the final version of this paper.

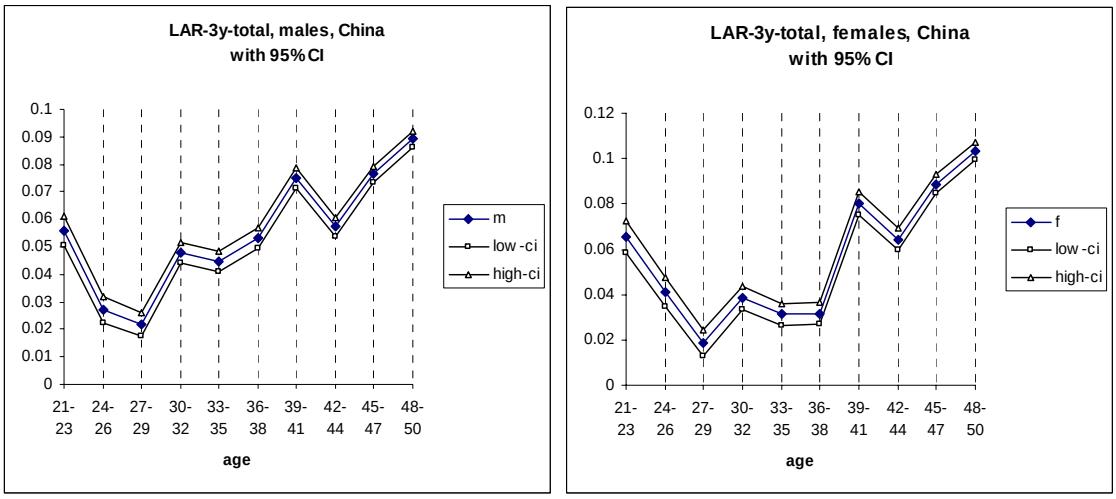

Figure 1 LAR pattern for 3-year age groups, by sex, China, 2000 Census

Secondly, in order to simultaneously model age, period and cohort effects, we conduct formal age-period-cohort (APC) analysis using time series age-specific mortality data at national level in the available 12 years, ranging from 1981 to 2002. Since APC model has the well-known identification problem that is due to the linear dependency between age, period, and cohort. Following Wilmoth's exploratory approach (1988, 1989), we make sequential models by fitting data by age and period first, then fitting age-period model residuals by age-period interaction, finally fitting remaining residuals by cohort. We plan to employ Wilmoth's complete iterative fitting procedure in the next step.

$$log\ (m_{ij}) = \alpha_i + \beta_j + \sum_{m=1}^{\rho} \varphi_m \gamma_{im} \delta_{jm} + \theta_k + \varepsilon_{ij}$$

where m_{ij} is the age-specific death rate. The age and period parameters are α_i and β_j respectively. A set of age-period interaction terms $\gamma_{im}\,\delta_{jm}$ $(m=1 \ldots \rho)$ are introduced into the linear APC model at the same time constrained the cohort parameters θ_k to express the deviations from the overall pattern of change by age, period, and age-period interaction. The set of age-period interactions are determined by the first ρ terms of the singular value decomposition (SVD) from the residuals of the additive model based on age and period only. The relative magnitude of singular value φ_m, which expresses the variance explained by each term of the SVD, is used to choose the proper number of

interaction terms ρ. At this point, the preliminary results from fitting exploratory sequential models are consistent with the LAR findings in 2000, suggesting famine cohorts in 1959-61 had increased adult mortality relative to the average level, particularly for males.

Furthermore, because our focus is not on how to partition the linear trend between period and cohort, rather on the deviation of irregular cohort effects beyond the regular continuous change over time, we also follow Holford's (1983) suggestion to estimate identifiable parameters of APC model: non-linear deviation. The deviations for age are the residuals from a linear regression of the estimated age effects α_i (obtained using any arbitrary constraint for identifiability) on the age group index i. The deviations for period and cohort are similarly obtained. Positive deviation suggests mortality is higher than expected from the linear trend. Negative deviation suggests mortality is lower than expected from the linear trend. It is also shown that the curvatures are also identifiable in any APC model (Clayton and Schifflers 1987), which are the *second differences of the parameter estimates, e.g. a curvature of age effects is* $\alpha_3 - 2\alpha_2 + \alpha_1 = (\alpha_3 - \alpha_2) - (\alpha_2 - \alpha_1)$. Positive value implies a sudden downward bending of the mortality (convex curve). Negative value implies a sudden acceleration of the mortality (concave curve). Therefore, through constructing and observing the non-linear deviation and curvatures, we can examine whether there are expected deviation in related to increased mortality for famine cohorts or in related to depressed mortality for cohort born before/after the famine.

Thirdly, we compare the mortality of China to a number of mortality references, including model life tables and real mortality data in a number of western countries at the time their adult survival was similar to the level of China in each of its three Census years. After matching level of adult survival between certain adult age ranges (e.g. age 20 to 50) between China in 2000 and the reference country (e.g. Sweden in 1950), the ratio of age-specific mortality between the two countries during this age range would reveal the relatively higher mortality in certain ages and relatively lower mortality in other ages for China comparing to the reference. We have done such work in a number of countries including Sweden, France, Japan, and Taiwan, to compare with China 2000 Census. The

results of comparisons are generally consistent in pointing out the relatively higher mortality around famine cohorts and lower mortality for about 10-year cohorts before famine. In the next step, we are going to repeat such work in using more countries and expand the work to China 1990 and 1982 Censuses as well. The consistency across Census years would be an even stronger piece of evidence in supporting our debilitation and maybe selection hypothesis of the famine effects.

Expected findings

We expect the following findings, as preliminary results have suggested some of them but still need to be further confirmed:

I. Famine cohorts in 1958-61 (more concentrated in 1959-61) had relatively elevated adult mortality when they were reaching the adulthood in their 30-40s during 1990s.
II. The famine effects are stronger among males than females due to the toll of chronic diseases hit male earlier in adulthood than females.
III. The famine effects are differential across provinces due to the mix effects of differential coverage and severity of famine, and the interaction between early poor nutrition and later over nutrition in more advanced urban areas.
IV. Compared with reference mortality level (from a number of countries), Chinese cohorts born before famine (e.g. under age 15) had relatively lower adult mortality in 1990s due to selection effect, while cohorts born during the famine had relatively higher adult mortality in 1990s due to debilitation effect of famine.
V. In general, both debilitation and selection effect exist though their effects may be different between sex and across age groups.

Reference

Anderson, B. A., Silver, B.D. (1989). "Patterns of cohort mortality in the Soviet population" Population and Development Review 15(3): 471-501.

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

Banister, J., Preston, S.H., (1981). "Mortality in China." Population and Development Review 7(1): 98-110.

Banister, J. (1987). China's Changing Population, Stanford University Press, Stanford, California.

Banister, J., Hill, K. (2004). "Mortality in China 1964-2000." Population Studies vol.58(No.1): pp. 55-75.

Barker, D. J. P. (1998). Mothers, Babies and Health in Later Life, Churchill Livingstone.

Barker, D. J. P. (1999). "The Fetal Origins of Type 2 Diabetes Mellitus." Annals of Internal Medicine 130: 322-324.

Barker, D. J. P., Ed. (2001). Fetal Origins of Cardiovascular and Lung Disease. Lung Biology in Health and Disease, Marcel Dekker, Inc. New York.

Caselli, G., Vallin, J., Vaupel, J.W., Yashin, A., (1987). "Age-specific mortality trends in France and Italy since 1900: period and cohort effects." European Journal of Population 3(1): 33-60.

Caselli, G., Capocaccia, R. (1989). "Age, Period, Cohort and Early Mortality: An Analysis of Adult Mortality in Italy." Population Studies 43(1): 133-153.

Caselli, G. (1990). The Influence of Cohort Effects on Differentials and Trends in Mortality. Measurement and Analysis of Mortality: New Approaches. J. Vallin, D'Souza, S., Palloni, A., Oxford: Clarendon Press.

Clayton, D., Schifflers, E., (1987). "Models for temporal variation in cancer rates II: Age-period-cohort models." Statistics in Medicine 6: 468-481.

Coale, A. J. (1984). Rapid Population Change in China, 1952-1982. Committee on Population and Demography, Report No. 27, National Academy Press, Washington D.C.

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

Hales, C., Barker, DJP, (1992). "Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis." Diabetologia 35: 595-601.

Hales, C., Barker, DJP, (2001). “The thrifty phenotype hypothesis” British Medical Bulletin 60: 5-20.

Hill, K. (1988). Demographic Trends in China from 1950 to 1982, World Bank discussioin papers: 22. The World Bank, Washington, D.C.

Holford, T. R. (1983). "The Estimation of Age, Period and Cohort Effects for Vital Rates." Biometrics 39(2): 311-324.

Horiuchi, S. (1983). "The Long-Term Impact of War on Mortality: Old Age Mortality of the First World War Survivors in the Federal Republic of Germany." Population Bulletin of the United Nations 15: 80-92.

Ji CY, Sun JL, Chen TJ. 2004. [Dynamic analysis on the prevalence of obesity and overweight school-age children and adolescents in recent 15 years in China] [Article in Chinese]. Zhonghua Liu Xing Bing Xue Za Zhi (Chinese Journal of Epidemiology) 25(2):103-8.

Kane, P. (1988). Famine in China, 1959-61: Demographic and Social Implications, St. Martin's Press, New York.

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

Kermack, W. O., McKendrik, A.G., McKinlay, P.L., (1934). "Death rates in Great Britain and Sweden: some regularities and their significance." Lancet March 31: 698-703.

Kuh D, Ben-Shlomo Y. (2004). A life course approach to chronic disease epidemiology. 2nd ed. Oxford: Oxford University Press.

Noymer, A., Garenne M. (2000). "The 1918 Influenza Epidemic's Effects on Sex Differentials in Mortality in the United States." Population and Development Review 26(3): 565-581.

Okubo, M. (1981). Increase in mortality of middle-aged males in Japan. Research Paper Series, No. 3. Tokyo, Japan: Nihon University, Population Research Institute.

Peng X. (1987). Demographic consequences of the Great Leap Forward in China's provinces. Population and Development Review 13:639-70.

Population Census Office China (1984). A Census of One Billion People: Papers for International Seminar on China's 1982 Population Census, March 26-31, 1984, Beijing, China, Westview Press, Boulder, Colorados.

Population Census Office People's Republic China, Ed. (1985). 1982 Populatioin Census of China: Results of Computer Tabulation, China Statistics Press.

Ravelli, A., et. al. (1998). "Glucose tolerance in adults after prenatal exposure to the Dutch famine." Lancet 351: 173-177.

Ravelli, A. C. e. a. (1999). "Obesity at the age of 50 y in men and women exposed to famine prenatally." American Journal of Clinical Nutrition 70: 811-816.

Roseboom, T. J., van de Meulen, J.H.P., Osmond, C. et al. (2000). "Coronary heart disease after prenatal exposure to the Dutch famine, 1944-45." Heart 84: 595-598.

Stanner, S. A. e. a. (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: 1342-1348.

Stein, Z., Susser, M., (1975). "Fertility, fecundity, famine: food rations in the Dutch famine 1944/5 have a causal relation to fertility, and probably to fecundity." Human Biology 47: 131-154.

Stein, Z., Susser, M., Saenger, G., Marolla, F. (1995). Famine and Human Development: The Dutch Hunger Winter of 1944-45, Xoford University Press, Oxford.

Susser, E., et. al. (1998). "Schizophrenia after prenatal exposure to famine." Lancet.

Tango, T., Kurashina, S. (1987). "Age, Period and Cohort Analysis of Trends in Mortality from Major Diseases in Japan, 1955 to 1979: Peculiarity of The cohort Born in The Early Showa Era." Statistics in Medicine 6: 709-726.

Willekens, F., Scherbov, S. (1992). "Analysis of Mortality Data from The Former USSR: Age-Period-Cohort Analysis." World Health Statistics Quarterly 45: 29-49.

Wilmoth, J. (1988). Ph.D. Dissertation: On the Statistical Analysis of Large Arrays of Demographic Rates, Princeton University.

Wilmoth, J. (1989). Fitting Three-Way Models to Two-Way Arrays of Demographic Rates, Research Report No. 89-140. Population Studies Center, University of Michigan, Ann Arbor, April 1989.

Wilmoth, J. (1990). "Variation in Vital Rates by Age, Period, and Cohort." Sociological Methodology 20: 295-335.

Wilmoth, J., Vallin, J., Caselli, G. (1990). "When Does a Cohort's Mortality Differ from What we Might Expect?" Population: An English Selection 2: 93-126.