Revisiting the Long-Term Health Effect of China's 1959-1961 Famine: An Instrumental Variable Approach*

Hongwei Xu

Institute for Social Research

University of Michigan

xuhongw@umich.edu

* Paper prepared for presentation at the 2015 Population Association of America Annual Meeting, San Diego (CA).

Abstract

Demographers have tested the provocative fetal-origin hypothesis by using famine as a natural experiment. Empirical identification of casual effects of prenatal famine exposure on later-life health is challenging. Existing evidence supporting the fetal-origin hypothesis from studies of famine is limited partly because of data limitation and/or methodological flaws. In fact, the disruption effect of prenatal exposure to famine is often overshadowed by a selection effect when cohort comparisons are carried out in population-based samples. Capitalizing on the biomarker data from the 2011 China Health and Retirement Longitudinal Study (CHARLS), this study proposes to adopt an instrumental variable strategy to identify long-term effects of prenatal exposure to China's 1959-61 famine on later-life cardiovascular and metabolic disease risks. Exploratory analysis using the conventional cohort comparison strategy reveals little significant disruption effect, except that for LDL cholesterol. Significant selection effect is present in cardiovascular disease risk as measured by resting pulse.

Introduction

The provocative fetal-origin hypothesis (Barker 1990, 1995) conjectures that exposure to an adverse environment, in particular malnutrition, during fetal period could "program" the fetus to develop particular metabolic characteristics, likely through environmental effects on the epigenome. Such developmental changes could persist over the life course and increase one's risks of cardiovascular and metabolic diseases in middle and later ages. More recently, the developmental origins hypothesis (Bateson et al. 2004; Gluckman et al. 2008; Gluckman et al. 2005) posits a nonlinear relationship between prenatal nutritional condition and later-life fitness due to different mechanisms at work depending on the nature of nutritional environment. Specifically, a disruption effect of prenatal development occurs when nutritional condition is too harsh or overly rich; whereas positive health effect may be achieved in case of mildly inadequate maternal nutrition, because the fetus may predict adverse postnatal environment based on such an early cue and thus adopt a life history strategy that involves phenotypic modifications and optimizes survival and reproductive success (Gluckman et al. 2008). Nevertheless, these predictive adaptive responses can lead to negative health consequences if there is a mismatch between prediction and subsequent reality.

However, empirical identification of these effects in human society is challenging. Early evidence in support of the fetal-origin hypothesis is largely derived from observational epidemiologic studies that fail to control for other prenatal confounders (Barker 1995; Barker and Osmond 1986). These studies also rely heavily on low birth weight as a crude proxy for fetal malnutrition, even though maternal malnutrition during gestation may induce later-life disease without affecting birth weight (Roseboom et al. 2001). The observed association between birth weight and future health outcomes could also reflect many unobserved joint determinants such as

genetic and environmental factors (Almond and Currie 2011; Paneth and Susser 1995; Song 2013). Later studies resort to famine as a natural experiment, because famine usually occurs beyond the control of most individuals and individual heterogeneity in the capacity to fight famine is substantially reduced. Nevertheless, empirical evidence from Western famines is highly ambiguous in support of the fetal-origin hypothesis, with positive and negative findings from the Dutch Hunger Winter of 1944-1945 (Stein et al. 1975; Susser, Hoek and Brown 1998), negative findings from the Leningrad Siege (Stanner et al. 1997) and the 1866-68 Finnish famine (Kannisto, Christensen and Vaupel 1997).

In fact, using famine as a natural experiment in itself does not free researchers from making erroneous inference, especially if the empirical strategy is flawed. To date, the dominant approach in famine studies involves comparisons between those born or conceived during the famine period (i.e., the famine cohorts) and those born before or after the famine period (i.e., the pre- and post-famine cohorts). This strategy can still be confounded by omitted variables that simultaneously affect cohort difference in prenatal famine exposure and that in later-life outcomes. Further, the disruption effect of prenatal exposure to famine is often overshadowed by a selection effect when cohort comparisons are carried out in population-based samples. That is, famines may eliminate frail members of the population, leaving the surviving famine cohorts dominated by the fittest ones. As a result, cohort comparison may reveal that the famine cohorts is surprisingly healthier than the none-famine cohorts (Li et al. 2011; Song 2009, 2010, 2013).

In this study, I adopt an alternative strategy to identify the long-term effects of prenatal malnutrition. This strategy hinges on an innovative instrumental variable (IV) in the context of China's Great Leap Forward (GLF) famine in 1959-1961. The GLF famine is an ideal setting to address the identification problem because of its relatively longer duration (three years), wider

geographic coverage (pandemic as opposed to endemic), and greater damage (16.5-30 million excess deaths with a mortality rate of over 3.0%), compared to Western famines (Song, Wang and Hu 2009). I will exploit geographic variation in famine severity among the famine cohorts only, thereby reducing the confounding selection effect in cohort comparison. I will geocode and match prefecture-level famine severity from historical demographic estimates to nationally representative data from the China Health and Retirement Longitudinal Study (CHARLS). The fasting blood sample from the CHARLS will allow me to construct clinical cardiovascular and metabolic disease measures. I will instrument famine severity by a measure of political radicalism that contributed to the GLF famine in the first place. This measure will be transcripted and geocoded from a 1958 newspaper archive.

Background of China's 1959-1961 Famine

From 1958 to 1961, the Communist Party of China (CPC) launched a massive campaign, known as the Great Leap Forward (GLF), mobilizing the entire country to adopt radical economic and social policies to rapidly transform China from a predominantly agrarian society to an industrialized socialist economy through a Soviet-style, high investment in heavy industry, supported by agricultural collectivization. To appeal to their superiors' radicalism and avoid being labelled as "anti-revolution", local cadres began to launch high-yield agricultural "satellites"[1] in June 1958, that is, fictitiously report a grain output of thousands or even ten thousands of catties per *mu*[2] (Bernstein 1984) to the *People's Daily*, the CPC's official newspaper (Kung and Chen 2011). The first satellite was launched on June 8, 1958, when the front-page headline of the *People's Daily* reported that a People's Commune in Suiping County

[1] The term was inspired by the Soviet Union's Sputnik satellite launched in 1957.
[2] One catty equals 1/2 kilogram, and one *mu* equals 1/6 acre.

of Henan Province achieved a significant breakthrough in an average wheat yield of 2,105 catties per *mu*. This exaggeration was soon topped on the next day as the *People's Daily* reported that another commune in Hubei Province harvested an average of 2,357 catties of wheat per *mu*. Picking up the high-profile cue from these reports by the *People's Daily*, other regions throughout the country exploded into a frantic race to over-report grain yields. From June to September, more than 800 false reports of abnormally high grain yield were published the *People's Daily*. In the end of 1958, the national grain production was reported to be 375 million metric tons (MMT), roughly doubled from that in 1957; however, verification in 1961 yielded a nearly half reduction to 200 MMT (Bernstein 1984; Li 1962).

Wild exaggerations of the growth in grain yield gave rise to a deception of the large harvest in 1958, which convinced the top political leaders that China was now confronted by a problem of storing and processing surplus grain rather than a problem of food shortage (Bernstein 1984; Yang 1996). This false vision of the agricultural production led the top political leaders to require excessive compulsory procurement (Ashton et al. 1984; Bernstein 1984). The total grain procurement in the 1958 grain year (April 1958 – March 1959) was 22.3 percent more than that in 1957 (Yang 1996:43). In addition, new policies were implemented to divert labor and resources from agriculture to fruitless projects such as the so-called backyard furnace movement in later 1958[3] and to reduce sown acreage in 1959 (Ashton et al. 1984). These movements together resulted in a sharp decline in grain production, and rural villages suffered from severe food shortage after forced grain requisitioning to support urban and industrial growth. Coupled with other manmade and natural devastating factors, the resulting great famine of 1959-1961, also known as the GLF famine, caused a total number of excess deaths that ranges

[3] The backyard furnace movement was characterized by melting any steel objects available, including pots and pans in the blast furnaces in the backyard of the people's communes. Most of the iron produced was of low quality and hence no value at all.

from 16.5 to 30 million, depending on the data sources, underlying assumptions, and methods of estimation employed (Ashton et al. 1984; Banister 1987; Coale 1984; Peng 1987; Yao 1999).

Research Plan

Survey Data and Dependent Variables

The survey data in this study will be drawn from the China Health and Retirement Longitudinal Study (CHARLS), a nationally representative longitudinal survey of adults aged 45 and older and their spouses if available. The national baseline survey was launched in 2011 and interviewed 17,708 respondents with a response rate of 80.5% (Zhao et al. 2014b). The CHARLS collected biomarker data from participants in fasting blood test, providing accurate clinical measures of cardiovascular and metabolic disease risks. This study will focus on cardiovascular and metabolic disease risks among the 690 CHARLS respondents of the famine cohorts (i.e., born during the famine period of 1959-1961 and aged 50-52 years at the time of interview), because these diseases are central to Barker's (1990, 1995) original formulation of the fetal origin hypothesis and they are posing serious new public health threats to the Chinese population (Popkin et al. 2010). Eleven dichotomous indicators of high disease risks will be created by applying CHARLS-recommended cut-points (Zhao et al. 2014a) to participants' physiological and blood test results.

Table 1 reports the prevalence of high disease risks among the 1959-61 famine cohorts along with the 1962-64 post-famine cohorts as a reference. The percentage of the famine cohorts at high risk ranged from 4.5% (resting pulse) to 26.7% (HDL cholesterol). Chi-squared tests revealed little significant difference between the famine and post-famine cohorts. Consistent with the fetal origin hypothesis, the famine cohorts had significantly higher metabolic risk with

respect to LDL cholesterol (10.4%) compared to the post-famine cohorts (7.5%). However, the famine cohorts also had significantly lower cardiovascular risk measured by resting pulse (4.5% versus 6.9%), indicating possible selection effect.

[Table 1 about here]

I will control for conventional demographic and socioeconomic characteristics available in the CHARLS data. Examples include gender, marital status, educational attainment, employment, family income and size, health behaviors (drinking, smoking, physical exercise), and rural-urban residence.

Measuring Famine Severity

The key predicator in this study is famine severity. Researchers are usually unable to collect the data at the time of the famine. Thus the intensity of individual's famine exposure is rarely available. In studies of the GLF famine, for example, a common solution is to use a period measure of total famine-caused excess mortality, regardless of cohort, during the famine years (Almond et al. 2010; Chen and Zhou 2007; Luo, Mu and Zhang 2006), or a cohort measure of famine-induced cohort size shrinkage among the famine cohorts relative to the pre- and post-famine cohorts (Huang et al. 2010a; Huang et al. 2010b; Meng and Qian 2009). Both types of measures are typically derived from one or more of China's population censuses conducted before (1953) and after (1964 and 1982) the famine, under the assumptions of accurate census data on fertility and mortality, stable secular trends in fertility and mortality in the counterfactual absence of the famine, and strictly restricted migration. These measures are then matched to the survey respondents based on their geographic location usually at the time of interview and at province level. This approach works well for estimating a national or provincial death toll, but

less so for gauging regional variation at sub-provincial level because it ignores the change in county-level population size due to local migration. In fact, despite the tightly controlled migration imposed by the household registration system, more than ten thousand of starving people managed to escape the famine in the mainland by sneaking into colonial Hong Kong (Burns 1987). On the other hand, famine severity varied substantially not only between provinces but across sub-provincial areas as well. The census-based approach may also not be sensitive enough to detect a minor increase in mortality in areas hit lighter by the famine. Therefore, the aforementioned indirect measures of famine severity are inevitably subject to measurement error, which could attenuate the estimate of the causal effect of famine exposure.

In this study, I will adopt the prefecture-level excess mortality rate estimated by Cao (2005) who used demographic data from local chronicles to adjust census-based estimation. Cao (2005) capitalized on local demographic chronicles from 1,050 sub-provincial level to improve census-based estimates. These chronicles were usually compiled by scholarly committees commissioned by local governments based on historical documents about local social, cultural, and demographic conditions. Completed two or three decades after the GLF famine, these chronicles are less susceptible to falsification for the sake of protecting the reputation of local officials who are no longer in power. These chronicles also documented local migration flows in response to the GLF famine, which would be completely missed if a simple linear interpolation is applied to the 1953 and 1965 census data. In essence, Cao first estimated average annual population growth rates before and after the GLF famine for each prefecture based the annual statistics of fertility, mortality, and migration during the period of 1953-1982 from local chronicles, as well as the 1953 and 1964 census data. He then applied these rates to calculate: (a) the actual population size in the end of 1958 and 1961, denoted by N_{1958}^{true} and N_{1961}^{true} respectively;

(b) the counterfactual number of new births during 1959-61, had no famine occurred, denoted by $N_{birth,1959-61}^{counterfactual}$; and (c) the counterfactual number of deaths during 1959-61, had no famine occurred, denoted by $N_{death,1959-61}^{counterfactual}$. The excess mortality rate (EMR) for the *i*th prefecture as calculated by Cao (2005) is:

$$EMR_i = \frac{(N_{1958}^{true} - N_{1961}^{true}) + (N_{birth,1959-61}^{counterfactual} - N_{death,1959-61}^{counterfactual})}{N_{1958}^{true}} \qquad (3)$$

where the first difference in the numerator is the (estimated) actual death toll during the famine; and the second difference is the (estimated) excessive population loss due to famine-induced fertility reduction.

As a robustness check, a cohort measure of famine severity will be analyzed as well. I will follow Huang and colleagues' (Huang et al. 2010a; Huang et al. 2010b) approach to derive a cohort size shrinkage index (CSSI) for each prefecture from the 1990 census data. Let $N_{nonfamine}^{i}$ denote the average cohort size of those born during the three years preceding the famine (1956-58) and the three years right after the famine (1962-64) in the *i*th prefecture, and N_{famine}^{i} denote the average cohort size of those born during the three famine years (1959-61). The CSSI for the *i*th prefecture is calculated as a ratio:

$$CSSI_i = \frac{N_{nonfamine}^{i} - N_{famine}^{i}}{N_{nonfamine}^{i}} \qquad (4)$$

where a larger value indicates a greater reduction in cohort size presumably induced by the GLF famine.

Constructing Instrumental Variable

The problems of omitted-variable bias and measurement error can be addressed by using an instrument variable (IV). An IV is an exogenous variable that correlates with the endogenous independent variable (i.e., famine severity) and but not with the error term (i.e., the IV affects the dependent variable only indirectly through its effect on the instrumented independent variable). A valid IV estimate is consistent in presence of measurement error and more importantly, address the omitted variable problem by effectively mimicking random assignments of respondents into the treatment and control groups in cross-sectional data (Angrist and Krueger 2001). Thus, the IV approach will free us from between-cohort comparisons that are susceptible to various unobserved confounding variables. Instead we will exploit variation within the famine cohorts.

The IV in this study is the frequency of wild exaggeration of grain yields in each prefecture as published in the *People's Daily*. As described above, local cadres demonstrate their loyalty to the top leaders through enthusiastic endorsement of the GLF movement in various forms, one of which was to falsely claim an unprecedentedly high grain yields thanks to the "novel" agricultural measures advocated in the movement.[4] Political loyalty was rewarded with career advancement and the associated increases in salary, occupation prestige, authority, and privileged access to bureaucratically controlled goods. At the provincial level, for example, seven party secretaries were promoted to be members of the CPC's Central Committee for their early support of the GLF movement (Goldstein, 1991). Unfortunately, such wild exaggeration backfired since the more frequently local cadres lied about grain yield, the greater the "harvest" appeared to be, leading to more excessive compulsory procurement required by the central

[4] Three commonly reported means to achieve high grain yields are deep plowing, intensive seeding, and heavy fertilizing.

government. In turn, local population was faced with increased food shortage and greater hunger threat. In other words, the frequency of exaggerating grain yields in summer 1958 should be positively associated with famine severity in the subsequent years, satisfying the instrument relevance requirement.

On the other hand, local cadres gradually stopped falsifying grain yields towards the end of 1958 as the harvest season ended. This devastating practice had been completely abandoned along with other radical measures by 1961 when the central government suspended the GLF movement as a whole. Therefore, it is unlikely that exaggeration of grain yields in 1958 could directly affect later-life health of the famine cohorts other than through its impact on famine severity, thereby satisfying the exclusion restriction for an IV.

Incidence of exaggerating grain yields will be transcripted from the archive issues of the *People's Daily* published in summer 1958. Frequency of exaggeration will be aggregated to prefecture level and matched to famine severity and survey data.

Statistical Model

Given that the dependent variables are binary, the nonlinear IV method, or Generalized Method of Moments (GMM), will be used to estimate the causal effect of famine severity on later-life high risks of cardiovascular and metabolic diseases among the famine cohorts. Standard IV validity and sensitivity tests will be performed.

References

Almond, D.and J. Currie. 2011. "Killing Me Softly: The Fetal Origins Hypothesis." *The Journal of Economic Perspectives* 25(3):153-172.

Almond, D., L. Edlund, H. Li, and J. Zhang. 2010. "Long-Term Effects of Early-Life Development: Evidence from the 1959 to 1961 China Famine." Pp. 321-345 in *The Economic Consequences of Demographic Change in East Asia*, edited by T. Ito and A. Rose. Chicago: University of Chicago Press.

Angrist, J.D.and A.B. Krueger. 2001. "Instrumental Variables and the Search for Identification: From Supply and Demand to Natural Experiments." *Journal of Economic Perspectives* 15(4):69-85.

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. 1987. *China's changing population*. Stanford, CA: Stanford University Press.

Barker, D.J.P. 1990. "The Fetal And Infant Origins Of Adult Disease: The Womb May Be More Important Than The Home." *BMJ: British Medical Journal* 301(6761):1111.

—. 1995. "Fetal Origins Of Coronary Heart Disease." *BMJ: British Medical Journal* 311(6998):171-174.

Barker, D.J.P.and C. Osmond. 1986. "Infant Mortality, Childhood Nutrition, and Ischaemic Heart Disease in England and Wales." *The Lancet* 327(8489):1077-1081.

Bateson, P., D. Barker, T. Clutton-Brock, D. Deb, B. D'Udine, R.A. Foley, P. Gluckman, K. Godfrey, T. Kirkwood, M.M. Lahr, J. McNamara, N.B. Metcalfe, P. Monaghan, H.G. Spencer, and S.E. Sultan. 2004. "Developmental plasticity and human health." *Nature* 430(6998):419-421.

Bernstein, T.P. 1984. "Stalinism, famine, and Chinese peasants." *Theory and Society* 13(3):339-377.

Burns, J.P. 1987. "Immigration from China and the Future of Hong Kong." *Asian Survey* 27(6):661-682.

Cao, S. 2005. *The Great Famine*. Hong Kong: Time International Publishing Co., Ltd.

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(4):659-681.

Coale, A.J. 1984. *Rapid Population Change in China, 1952-1982*. Washington, D.C.: National Academy Press.

Cramer, J.S.and L.H. Lumey. 2010. "Maternal Preconception Diet and the Sex Ratio." *Human Biology* 82(1):103-107.

Fung, W.and W. Ha. 2010. "Intergenerational Effects of the 1959-61 China Famine." Pp. 222-254 in *Risk, Shocks and Human Development: On the Brink*, edited by R. Fuentes-Nieva and P.A. Seck. London, UK: Palgrave-Macmillan.

Gluckman, P.D., M.A. Hanson, C. Cooper, and K.L. Thornburg. 2008. "Effect of In Utero and Early-Life Conditions on Adult Health and Disease." *New England Journal of Medicine* 359(1):61-73.

Gluckman, P.D., M.A. Hanson, H.G. Spencer, and P. Bateson. 2005. "Environmental influences during development and their later consequences for health and disease: implications for the interpretation of empirical studies." *Proceedings of the Royal Society B: Biological Sciences* 272(1564):671-677.

Huang, C., Z. Li, K.M. Venkat Narayan, D.F. Williamson, and R. Martorell. 2010a. "Bigger babies born to women survivors of the 1959–1961 Chinese famine: a puzzle due to survival selection?" *Journal of Developmental Origins of Health and Disease* 1(06):412-418.

Huang, C., Z. Li, M. Wang, and R. Martorell. 2010b. "Early Life Exposure to the 1959–1961 Chinese Famine Has Long-Term Health Consequences." *The Journal of Nutrition* 140(10):1874-1878.

Jirtle, R.L.and M.K. Skinner. 2007. "Environmental epigenomics and disease susceptibility." *Nature Reviews Genetics* 8(4):253-262.

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

Kung, J.K.-s.and S. Chen. 2011. "The Tragedy of the Nomenklatura: Career Incentives and Political Radicalism during China's Great Leap Famine." *American Political Science Review* 105(1):27-45.

Li, C.-M. 1962. *The statistical system of Communist China*. Berkeley: University of California Press.

Li, Y., V.W. Jaddoe, L. Qi, Y. He, J. Lai, J. Wang, J. Zhang, Y. Hu, E.L. Ding, X. Yang, F.B. Hu, and G. Ma. 2011. "Exposure to the Chinese famine in early life and the risk of hypertension in adulthood." *Journal of Hypertension* 29(6):1085-1092.

Luo, Z., R. Mu, and X. Zhang. 2006. "Famine and Overweight in China." *Applied Economic Perspectives and Policy* 28(3):296-304.

Meng, X.and N. Qian. 2009. "The Long Term Consequences of Famine on Survivors: Evidence from a Unique Natural Experiment using China's Great Famine." *National Bureau of Economic Research Working Paper Series* No. 14917.

Paneth, N.and M. Susser. 1995. "Early Origins of Coronary Heart Disease: The Barker Hypothesis." *BMJ* 310(2):411-412.

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., S. Du, F. Zhai, and B. Zhang. 2010. "Cohort Profile: The China Health and Nutrition Survey - monitoring and understanding socio-economic and health change in China, 1989-2011." *International Journal of Epidemiology* 39(6):1435-1440.

Roseboom, T.J., J.H.P. van der Meulen, A.C.J. Ravelli, C. Osmond, D.J.P. Barker, and O.P. Bleker. 2001. "Effects of prenatal exposure to the Dutch famine on adult disease in later life: an overview." *Molecular and Cellular Endocrinology* 185(1–2):93-98.

Song, S. 2009. "Does Famine Have a Long-term Effect on Cohort Mortality? Evidence from the 1959–1961 Great Leap Forward Famine in China." *Journal of Biosocial Science* 41(04):469-491.

—. 2010. "Mortality consequences of the 1959–1961 Great Leap Forward famine in China: Debilitation, selection, and mortality crossovers." *Social Science & Medicine* 71(3):551-558.

—. 2012. "Does famine influence sex ratio at birth? Evidence from the 1959–1961 Great Leap Forward Famine in China." *Proceedings of the Royal Society B: Biological Sciences* 279(1739):2883-2890.

—. 2013. "Identifying the intergenerational effects of the 1959–1961 Chinese Great Leap Forward Famine on infant mortality." *Economics & Human Biology* 11(4):474-487.

Song, S., W. Wang, and P. Hu. 2009. "Famine, death, and madness: Schizophrenia in early adulthood after prenatal exposure to the Chinese Great Leap Forward Famine." *Social Science & Medicine* 68(7):1315-1321.

Stanner, S.A., K. Bulmer, C. Andrès, 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." *BMJ: British Medical Journal* 315(7119):1342-1348.

Stein, A.D., P.A. Zybert, and L.H. Lumey. 2004. "Acute undernutrition is not associated with excess of females at birth in humans: the Dutch Hunger Winter." *Proceedings of the Royal Society of London. Series B: Biological Sciences* 271(Suppl 4):S138-S141.

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

Susser, E.S., H.W. Hoek, and A. Brown. 1998. "Neurodevelopmental Disorders after Prenatal Famine: The Story of the Dutch Famine Study." *American Journal of Epidemiology* 147(3):213-216.

Trivers, R.L.and D.E. Willard. 1973. "Natural Selection of Parental Ability to Vary the Sex Ratio of Offspring." *Science* 179(4068):90-92.

Yang, D.L. 1996. *Calamity and Reform in China: State, Rural Society, and Institutional Change Since the Great Leap Famine*. Stanford, CA: Stanford University Press.

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

Zhao, Y., E. Crimmins, P. Hu, Y. Hu, T. Ge, J.K. Kim, J. Strauss, G. Yang, X. Yin, and Y. Wang. 2014a. "China Health and Retirement Longitudinal Study: 2011-2012 National Baseline Blood Date Users' Guide." Beijing: China Center for Economic Research, Peking University.

Zhao, Y., Y. Hu, J.P. Smith, J. Strauss, and G. Yang. 2014b. "Cohort Profile: The China Health and Retirement Longitudinal Study (CHARLS)." *International Journal of Epidemiology* 43(1):61-68.

Table 1. Frequency distributions of high-risk biomarkers in the 2011 China Health and Retirement Longitudinal Study (CHARLS)

		Famine Cohorts (%)				Post-famine Cohorts (%)				
Biomarkers	High-risk Cut-points[a]	1959	1960	1961	Total	1962	1963	1964	Total	*P*-value[b]
Cardiovascular										
Diastolic blood pressure	>= 90 mmHg	23.0	17.7	20.2	20.3	19.6	18.9	14.0	17.8	0.181
Systolic blood pressure	>= 140 mmHg	17.6	15.6	15.4	16.2	15.4	14.6	11.9	14.1	0.222
Resting pulse (bpm)	> 90	4.2	4.9	4.3	4.5	5.4	8.6	6.7	6.9	**0.033**
Metabolic - lipids										
HDL cholesterol	< 40 mg/dL	26.4	27.2	26.4	26.7	26.5	22.8	27.1	25.3	0.524
LDL cholesterol	> 160 mg/dL	10.5	9.1	12.0	10.4	9.8	7.2	5.2	7.5	**0.033**
Total cholesterol	>= 240 mg/dL	10.0	11.5	10.1	10.6	12.7	7.9	6.4	9.2	0.331
Triglycerides	>= 200 mg/dL	16.7	18.5	16.8	17.4	15.4	14.6	17.4	15.7	0.341
Metabolic - glucose metabolism										
Glucose	>= 126 mg/dL	12.6	12.8	12.5	12.6	10.8	10.3	9.1	10.1	0.103
HbA1c	>= 6.5%	3.8	5.3	6.7	5.2	3.2	4.1	4.3	3.8	0.153
N		239	243	208	690	408	417	328	1153	

[a]Cut-points are taken from the users' guide of CHARLS (Zhao et al. 2014a).

[b]*P*-values from Pearson's chi-squared tests of prevalence of high-risk biomarkers between the famine cohorts (1959-1961) and the post-famine cohorts (1962-1964).