The Journal of Nutrition
Community and International Nutrition

Early Life Exposure to the 1959–1961 Chinese Famine Has Long-Term Health Consequences[1,2]

Cheng Huang,[3] Zhu Li,[4] Meng Wang,[3] and Reynaldo Martorell[3]*

[3]Hubert Department of Global Health, Rollins School of Public Health, Emory University, Atlanta, GA 30322; and [4]National Center for Maternal and Infant Health, and Department of Health Care Epidemiology, Beijing University Medical Science Center, Beijing 100191, China

Abstract

The Chinese famine of 1959–1961 was the largest in human history. We used data on 35,025 women born in 1957–1963 to assess the impact of famine exposure on height, BMI, and hypertension at ~32 y of age. The data were from the China-U.S. Collaborative Project for Neural Tube Defect Prevention. The famine varied in intensity across provinces and counties and affected rural areas disproportionately. We used a measure of famine intensity at the county level based on the size of birth year cohorts in a difference-in-difference model, which compared each cohort to the unexposed 1963 cohort, after correcting for age and time trends, and estimated impact for the average level of intensity across counties. The impact was confined to rural areas, but this could be due to small sample sizes in urban areas. Height was reduced in the 1958 and 1959 cohorts by 1.7 and 1.3 cm, respectively. This corresponded to exposures during 0.5–3.5 y for the 1958 cohort and late pregnancy and 0–2.5 y for the 1959 cohort. BMI increased by 0.92 kg/m^2 in the 1957 cohort, exposed from 1.5 to 4.5 y, but decreased by 0.3 kg/m^2 in the 1960–1961 cohorts, exposed during pregnancy and infancy. Famine exposure was associated with a 3-fold increase in the odds of hypertension for the 1958 cohort. In general, postnatal exposure during the first 2–3 y of life reduced height and increased BMI and hypertension, whereas exposure during pregnancy and infancy reduced BMI. J. Nutr. 140: 1874–1878, 2010.

Introduction

Famines provide a quasi-experimental setting for research on the long-term effects of nutritional deprivation on human development. The most studied famine has been the "hungry winter" Dutch famine at the end of World War II (1). The famine was intense and had a well-defined but brief duration (~6 mo). Most of the literature about the Dutch famine has focused on the differential impact of exposure during early, middle, and late gestation on adult health outcomes.

Famines in developing countries have received comparatively little attention as settings for life-course research because of limitations of poor record keeping, attrition due to migration and mortality, and identification of appropriate controls. The 1959–1961 Chinese famine, the largest in human history, was largely unknown in the West until the 1980s when researchers examined census data released by the central government and deduced that up to 30 million people were "missing" from the famine year cohorts (2–6). The genesis of the famine was the "Great Leap Forward" campaign launched by Mao in 1958. All rural households were organized in thousands of people's communes and there was a sudden, sharp drop in grain production in 1959–1961, the immediate cause of the famine. A host of interrelated factors came into play: drought, excessive procurement by the state, delayed response to the food shortage, eroded incentives for food production due to collectivization, and resource diversion as a result of massive industrialization (2,5–7).

The famine affected all of China, but its severity varied across regions and affected rural areas disproportionately (8). Also, its duration varied geographically and is difficult to define with precision for any given area. In fact, there is debate over whether the famine began in 1958, 1 y earlier than generally accepted (8). The end of the famine is also unclear. Whereas the famine was over by the end of 1961 in many areas, it may have lasted until early 1963 in a few areas such as Sichuan (9). Lack of a distinct nonfamine area and the somewhat indeterminate duration of exposure present challenges in assessing long-term effects of the famine. On the other hand, selection due to migration is not a major concern; migration was restricted during the famine (7) and was rare before 1990 (10).

Although the Dutch famine was severe, it occurred in the context of an otherwise well-nourished population that may have been somewhat buffered. The Chinese famine was of longer duration, was superimposed on widespread chronic undernutrition, and food availability was more severely curtailed. We expected, therefore, the Chinese famine impact on adult health to be more marked than for the Dutch famine, although more difficult to measure precisely.

In the present study, we linked early life exposure to the Chinese famine of 1959–1961 to adult height, BMI, and

[1] Supported by a grant from The Global Health Institute of Emory University.
[2] Author disclosures: C. Huang, Z. Li, M. Wang, and R. Martorell, no conflicts of interest.
* To whom correspondence should be addressed. E-mail: rmart77@emory.edu.

Manuscript received February 10, 2010. Initial review completed March 20, 2010. Revision accepted July 5, 2010.
First published online August 11, 2010; doi:10.3945/jn.110.121293.

hypertension using data available for a large sample of women who participated in a folic acid supplementation trial (11,12). We assessed effects by age at exposure separately for urban and rural areas.

Methods

Data

The data came from the China-U.S. Collaborative Project for Neural Tube Defect Prevention conducted by the U.S. CDC and Beijing Medical University between 1993 and 1996. The study was designed to evaluate the efficacy of periconceptual folic acid supplementation to prevent neural tube defects and was approved by the institutional review boards of the CDC and Beijing Medical University (11,12). This program covered major regions of 3 provinces in China, Hebei, Zhejiang, and Jiangsu, and included 35 counties overall. Women preparing for marriage registered with the pregnancy-monitoring system and participated in a medical examination that provided body size and blood pressure data.

We restricted our study sample to 35,025 women born in 1957–1963 who had data for height, BMI, and hypertension and who were not pregnant at the time of measurement. The women were 31.7 ± 2.4 y old at measurement. Few women born before 1957 were available for study. The birth cohorts selected for study include cohorts born before (1957, 1958), during (1959–1961), and after (1962, 1963) the famine. The window of postnatal exposure for each birth cohort was estimated for women born mid-year, which represents the average exposure for the cohort (**Table 1**). Only the 1960 and 1961 cohorts were fully exposed during pregnancy, but there was also exposure in infancy as well for these 2 cohorts. Only the 1963 cohort was not exposed at any age to the famine.

We divided our sample into urban (n = 2,293) and rural (n = 32,732) strata according to residency status at birth.

Measures

Outcomes. Adult height, BMI, and hypertension were derived from the medical examination at registration. BMI was calculated as weight in kilograms divided by height in meters squared. Hypertension was defined as ≥140 mm Hg systolic pressure or ≥90 mm Hg diastolic pressure (13).

Famine severity. Our analytic design was patterned after the approach used by Chen and Zhou (7), who used excess mortality in 1960 at the province level to generate a measure of severity of the famine. Because we had only 3 provinces, a number too small to be useful, we used a 1% sample of China's 1990 population census to derive a measure of famine intensity at the county level based on the size of birth cohorts (the data are publicly available at https://international.ipums.org/international/) (14). The assumption was that the smaller the size of the cohorts born during the famine years relative to other years, due to reduced fertility and/or increased mortality, the greater the severity of the famine. As an illustration, cohort size in the 1% sample for 1950–1970 was aggregated by province (**Fig. 1**). The famine cohorts of 1959–1961 were significantly smaller compared with earlier and later cohorts. To derive an index of

TABLE 1 Windows of exposure to the 1959–1961 Chinese famine by birth year cohort

Cohort	Pregnancy exposure	Postnatal exposure at mid-year point, y
1957	None	1.5–4.5
1958	None	0.5–3.5
1959	Some, late	0–2.5
1960	Entire	0–1.5
1961	Entire	0–0.5
1962	Some, early	None
1963	None	None

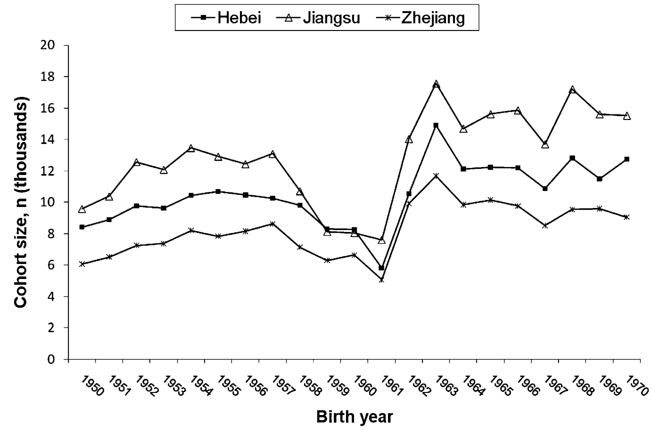

FIGURE 1 Cohort size by year of birth in the sampled provinces in a 1% sample of the 1990 Census of the Chinese Population.

famine severity for each county, we first calculated the mean cohort size of women born during the 3 y immediately before the famine (1956–1958) and the 3 y immediately after the famine (1962–1964), labeled as N_{nonfam}, as well as the mean cohort size of women born during the famine years (1959–1961), labeled as N_{famine}. We then calculated the cohort size shrinkage index $(CSSI)_r$[5] as the difference between N_{nonfam} and N_{famine} divided by N_{nonfam}. Our assumption was that the larger the $CSSI_r$, the more severe the famine was in the county. Among the 35 counties, $CSSI_r$ ranged from 0.24 to 0.64, with a mean of 0.42 and SD of 0.09. The inclusion of 3 y preceding and 3 y following the famine in the denominator provided a more reliable estimate than if we had used only the preceding years and was justified by the fact that both the drop in cohort size during the famine and the rebound afterwards were likely reflective of famine severity. The correlation between the county-level percentage drop in cohort size and the subsequent percentage rise was 0.83 (n = 35; $P < 0.01$).

Statistical model. We used an analytic model (see below) that contrasted each specific birth cohort between 1957 and 1962 to the 1963 cohort, which was not exposed to the famine. We did this by using dummy variables for each of the cohort years, leaving the 1963 cohort as the reference. Because the famine was less severe in urban compared with rural areas, we expected weaker relationships for the urban sample.

Difference-in-difference model. The ideal design for any birth cohort year exposed to famine, e.g. 1959, would first estimate the difference in the famine area, e.g. in height, between those born in 1959 and those born in the reference nonfamine year of 1963. This difference, however, may represent both a cohort effect (i.e. age effect and time trends) and a famine effect. Thus, to control for age and time trends, we would want to subtract a second difference from the first, specifically that between the same birth cohorts, 1959 and 1963, in a non famine or control area. Because the Chinese famine was nationwide, we lacked a true nonfamine or control area. Thus, we relied on the variation in famine severity across counties to construct the difference-in-difference (DID) model, similar to the approach used by Chen and Zhou (2007) (7):

$$H_{irk} = H_0 + \alpha_k Cohort_k + \sum_{k=1957}^{1962} \beta_k (CSSI_r \times Cohort_k) + \gamma CSSI_r + \varepsilon_{irk},$$

where H_{irk} is the adult height of a woman born in county r and year k (birth cohort k), α_k is the cohort fixed effect, and $CSSI_r$ is the cohort size shrinkage index in county r (an indicator of famine intensity in the county). β_k is the coefficient of the interaction between the cohort size shrinkage index and the birth cohort dummy variables (CSSI × COHORT) and is a measure of the double difference and represents

[5] Abbreviations used: $CSSI_r$, cohort size shrinkage index; DID, difference-in-difference; CHNS, China Health and Nutrition Survey.

the impact of early life famine exposure on adult height. This β_k represents the impact on height corresponding to change in CSSI of 1 unit. This unit change is hypothetical and to estimate the average effect across counties, one must multiply the interaction coefficient by the average CSSI observed across all counties (0.42). Similarly, we also estimated the impact on BMI and hypertension in adulthood. We modeled BMI as a continuous variable, because few women were overweight or obese (> 25 kg/m^2). For hypertension, the left side of the equation had a logged odds of being hypertensive. CI were adjusted for clustering. All analyses were conducted using SAS version 9.

Results

The characteristics of women born during 1957–1963 were provided by birth cohort and urban/rural residence (**Table 2**). Mean heights were between 158 and 159 cm and BMI between 21 and 22 kg/m^2. The prevalence of hypertension across groups was more variable, between 15 and 32%.

The average famine impact on adult height was represented by the coefficients for the interaction term between CSSI and the birth cohort dummy variable of interest multiplied by 0.42, the mean CSSI (**Table 3**). In the rural sample, there was significant impact of the famine on height among women in the 1958 and 1959 cohorts. On average, the 1958 birth cohort would otherwise have grown 1.66 cm taller (95% CI: 0.63, 2.69) in the absence of famine. Similarly, the 1958 cohort would have been 1.33 cm taller (95% CI: 0.46, 2.19) in the absence of famine. There was no significant famine impact on height in the urban sample.

Significant effects on BMI were found for the 1957, 1960, and 1961 rural cohorts but not for any of the urban cohorts (**Table 4**). On average, the 1957 cohort gained 0.92 kg/m^2 (95% CI: 0.32, 1.51) because of famine exposure. The direction of the relationship of famine exposure with adult BMI was reversed and the absolute magnitude was lower for the 1960 and 1961 birth cohorts compared with the 1957 cohort. The loss of BMI represented, on average, −0.32 *kg/m*^{2} (95% CI: −0.58, −0.08) and −0.30 kg/m^2 (95% CI: −0.58, −0.02), respectively, for the 1960 and 1961 birth cohorts.

We found evidence of famine impact on adult hypertension only in the rural sample (**Table 5**). The log of odds of hypertension among women born in 1958 was 1.38 (95% CI: 0.17–2.59), which suggested that the odds of hypertension for the 1958 cohort was about 3.97 [exponent (1.38) times of that for the 1963 cohort (reference group)]. A similar estimate of the log of hypertension was obtained for the 1957 cohort, 1.23, but with a wider CI (95% CI: −0.38 to –2.84).

TABLE 2 Characteristics of the study women by birth cohort and urban/rural residence[1,2]

Birth cohort	Sample size, *n*	Height,[2] *cm*	BMI,[2] *kg/m*^{2}	Hypertension, *%*
Rural sample				
1957	654	157.8 ± 5.3	22.2 ± 2.8	22.2
1958	943	157.9 ± 4.8	22.1 ± 2.7	20.5
1959	1157	157.9 ± 4.4	21.9 ± 2.7	18.6
1960	2126	158.1 ± 4.4	21.8 ± 2.6	15.1
1961	3113	158.4 ± 4.3	21.6 ± 2.5	14.7
1962	10,363	158.6 ± 4.5	21.6 ± 2.6	14.8
1963	14,376	158.8 ± 4.4	21.5 ± 2.6	16.2
Urban sample				
1957	89	158.7 ± 4.8	21.8 ± 2.3	23.6
1958	92	158.8 ± 4.3	22.3 ± 2.8	31.5
1959	121	158.4 ± 4.2	21.8 ± 2.3	25.6
1960	197	159.2 ± 5.5	21.8 ± 3.18	25.9
1961	200	158.8 ± 4.6	21.1 ± 2.3	27.5
1962	664	158.9 ± 4.5	21.2 ± 2.6	20.0
1963	930	159.1 ± 4.8	21.2 ± 2.7	24.2

[1] Values are mean ± SD or percent.
[2] Data source: the China-U.S. Collaborative Project for Neural Tube Defect Prevention (11,12).

Discussion

We found that famine exposure reduced height in rural women in the 1958 and 1959 cohorts by 1.7 and 1.3 cm, respectively. This corresponded, on average, to exposures during 0.5–3.5 y for the 1958 cohort and late pregnancy and 0–2.5 y for the 1959 cohort. Based on analyses of the China Health and Nutrition Survey (CHNS) (15), others found that exposure to the Chinese famine reduced adult height by ~3 cm for the 1959, 1960, and 1962 cohorts, with the 1957 cohort having a smaller impact (7). This corresponded to exposure during pregnancy and the first 2 y for the 1959 and 1960 cohorts; the 1962 cohort experienced famine in early pregnancy but not postnatal life. However, if the famine extended into 1962, this cohort would have been exposed in infancy. These effects were inconsistent in urban areas (7). CHNS covered 8 provinces, with only Jiangsu in common with our study (7). Among the 7 other provinces included were several (Guizhou, Henan, Hunan, and Guangxi) that were more severely affected than the provinces in our study (7,16), which may explain the differences in results.

Reports about human capital effects resulting from prenatal exposure to the Dutch famine found no effect on adult linear measures or body proportions (17) or on mental retardation or IQ at age 19 y (18). This is in contrast to our findings and those of Chen and Zhou (2007) (7), which suggest that prenatal and postnatal exposure to the Chinese famine reduced height and economic outcomes: total and home gardening hours worked; agrarian, farm, and home gardening income; and wealth, as proxied by dwelling size. These labor and earnings findings were largely confined to the 1959 and 1960 cohorts, which suggest exposure during 0–2 y as the cause (7). Effects were substantial; e.g. for the 1959 cohort, annual per capita agrarian income was reduced by 33% (7). The findings from China are consistent with those from Guatemala that report positive effects from a nutrition intervention in the first 2 y of life on height (19), intellectual functioning and schooling (20), and income (21).

We also found some famine effects on measures of adult health but only among rural women. For the 1957 cohort, our oldest cohort, exposed, on average, during ages from 1.5 to 4.5 y, we found an increase of 0.92 BMI units, whereas for the 1960 and 1961 cohorts, exposed during pregnancy and infancy, we found a loss of ~0.3 BMI units. These findings remained unchanged when we modeled BMI as a dichotomous variable using either 25 or 23.5 BMI as the cutoff point (results not shown). A report based on CHNS data concluded that BMI and overweight were significantly higher in the 1959, 1960, and 1961 cohorts than in the 1964 cohort, the nonexposed cohort, but only in women (22); however, this study should be interpreted with caution, because the findings were based entirely on direct comparisons among cohorts, which does not control for age and time trends. Another study, also based on CHNS data, used the DID approach but compared the famine

TABLE 3 Average effect of exposure in early life to the Chinese famine (1959–1961) on the height of women at early-mid 30s

Birth cohort	Rural sample		Urban sample	
	Average effect,[1] *cm*	95% CI	Average effect,[1] *cm*	95% CI
1957	−0.29	−1.41, 0.84	1.02	−2.13, 4.14
1958	−1.66**	−2.69, −0.63	−1.02	−5.26, 3.22
1959	−1.33**	−2.19, −0.46	−0.61	−3.64, 2.42
1960	0.29	−0.51, 1.08	0.05	−1.99, 2.10
1961	0.04	−0.65, 0.74	−0.05	−2.96, 2.86
1962	0.01	−0.50, 0.52	−0.39	−2.46, 1.69

[1] Average effect is estimated as coefficients for the interaction term between CSSI and the birth cohort dummy variable of interest, multiplied by 0.42, the mean CSSI. All estimates are with reference to the 1963 cohort (unexposed). ** $P < 0.01$.

years, 1959–1962, to postfamine years, 1963–1966, and concluded that overweight increased but only in women (16). These results are difficult to compare to ours because of the lumping of the famine years.

We found that the odds of being hypertensive increased by nearly 3-fold in the 1958 cohort; a similar but nonsignificant estimate was found for the 1957 cohort ($P = 0.12$). These are our oldest cohorts and the findings suggest that postnatal exposure from mid-infancy to 4 y increased hypertension. We are not aware of other reports of effects of the Chinese famine on hypertension.

Several studies of prenatal exposure to the Dutch famine and adult health are available. Whereas no association was found between famine exposure and blood pressure at age 50 (23), a significant association was observed in a similar population examined 9 y later (24). The Dutch famine studies demonstrate the complexity of the relationship of prenatal famine exposure with adult BMI (25). For example, the obesity rate was higher in men with famine exposure in the first one-half of gestation but lower in men with famine exposure in the last trimester or the immediate postnatal period than men born after the Dutch famine (25,26). In women, but not men, prenatal famine exposure, with no specific gestational window implicated, was associated with elevated total cholesterol and HDL cholesterol (27). In general, the association found between exposure to the Dutch famine and adult health is modest. Other than a few reports on BMI and our findings on hypertension, the impact of exposure to the Chinese famine on adult health has not been studied.

Our study has strengths and limitations. We used robust analytical methods that incorporate a measure of intensity of the famine at the county level based on the sizes of birth year cohort in a 1% sample of the 1990 Chinese census. This allowed us to make up for the lack of a nonfamine area in China and apply the DID approach that controls for cohort effects. We used a range of birth year cohorts that included cohorts born before, during, and after the famine and compared each individual cohort to the 1963 cohort, which was not exposed to the famine at any age. This allowed us to explore a range of age exposures and to explore differential impact by outcome. Our sample sizes for the rural areas are large, between 654 and 14,376/birth year cohort.

Among the limitations are the small sample sizes in urban areas, <200/y across all 35 counties for those born between 1957 and 1960. We had expected and found weaker relationships in urban compared with rural areas but were unable to exclude the possibility that our null findings in urban areas, particularly for our oldest cohorts, reflected small sample sizes. We had a limited set of outcomes, height, BMI, and hypertension, and only in women. The women were relatively young, 32 y on average, and were measured in the early 1990s, just as the prevalence of overweight began to rise in China (28). Greater effects on BMI and hypertension likely would be found if the study were conducted today, 50 y after the famine. Finally, the imprecise duration of the famine precluded us from specifying ages of exposure other than in broad, overlapping age ranges.

Our study adds to the literature on the effects of famine exposure in developing countries. We show that exposure in early life has important effects on height, BMI, and hypertension and that these effects vary by outcome. In general, postnatal exposure during the first 3 y of life reduces height and increases BMI and hypertension, whereas exposure during pregnancy and infancy reduces BMI. Impacts on adult health have been found for the Dutch famine, but little research has been done in regards to the Chinese famine. Future research about the effects of the Chinese

TABLE 4 Average effect of exposure in early life to the Chinese famine (1959–1961) on the BMI of women at early-mid 30s

Birth cohort	Rural sample		Urban sample	
	Average effect,[1] *kg/m²*	95% CI	Average effect,[1] *kg/m²*	95% CI
1957	0.92**	0.32, 1.51	0.03	−2.82, 2.87
1958	−0.06	−1.00, 0.87	−1.68	−3.65, 0.30
1959	−0.56	−1.42, 0.31	1.11	−1.69, 3.90
1960	−0.32*	−0.58, −0.08	0.13	−1.83, 2.09
1961	−0.30*	−0.58, −0.02	0.73	−0.92, 2.37
1962	0.00	−0.23, 0.23	−0.58	−1.63, 0.48

[1] Average effect is estimated as coefficients for the interaction term between CSSI and the birth cohort dummy variable of interest, multiplied by 0.42, the mean CSSI. All estimates are with reference to the 1963 cohort (unexposed). * $P < 0.05$, ** $P < 0.01$.

TABLE 5 Average effect of exposure in early life to the Chinese famine (1959–1961) on the log odds of hypertension in women at early-mid 30s

Birth cohort	Rural sample		Urban sample	
	Average effect,[1] *kg/m²*	95% CI	Average effect,[1] *kg/m²*	95% CI
1957	1.23	−0.38, 2.84	0.37	−2.07, 2.80
1958	1.38*	0.17, 2.59	0.07	−1.93, 2.07
1959	0.77	−0.50, 2.05	−1.00	−3.86, 1.86
1960	0.08	−0.89, 1.05	−0.66	−3.00, 1.68
1961	−0.50	−1.33, 0.33	0.19	−1.21, 1.60
1962	−0.01	−0.84, 0.82	−0.35	−1.39, 0.67

[1] Average effects are estimated as coefficients for the interaction term between CSSI and the birth cohort dummy variable of interest, multiplied by 0.42, the mean CSSI. All estimates are with reference to the 1963 cohort (unexposed). * $P < 0.05$, ** $P < 0.01$.

famine should include a comprehensive battery of outcomes, including indicators of human capital and adult health.

Acknowledgment

We thank R. J. Berry for his assistance in providing background information about the China-U.S. Collaborative Project for Neural Tube Defect Prevention conducted by the U.S. CDC and Beijing University Medical Science Center since the early 1990s. R.M. conceived the project and with C.H. designed the study; C.H. developed the analytic model and carried out the analyses; C.H. wrote the first draft and R.M. wrote subsequent drafts; and R.M. had primary responsibility for final content. All authors read and approved the final manuscript.

Literature Cited

1. Lumey LH, Stein AD, Kahn HS, van der Pal-de Bruin KM, Blauw GJ, Zybert PA, Susser ES. Cohort profile: The Dutch Hunger Winter Families Study. Int J Epidemiol. 2007;36:1196–204.
2. Ashton B, Hill K, Piazza A, Zeitz R. Famine in China, 1958–61. Popul Dev Rev. 1984;10:613–45.
3. Becker J, editor. Hungry ghosts. Mao's secret famine. New York: Henry Holt and Company, Inc.; 1998.
4. Smil V. China's great famine: 40 years later. BMJ. 1999;319:1619–21.
5. Wei L, Yang DT. The great leap forward: anatomy of a central planning disaster. J Polit Econ. 2005;113:840–77.
6. Lin JY, Yang DT. Food availability, entitlements and the Chinese famine of 1959–61. Econ J. 2000;110:136–58.
7. Chen Y, Zhou L-A. The long-term health and economic consequences of the 1959–1961 famine in China. J Health Econ. 2007;26:659–81.
8. Chang GH, Wen GJ. Communal dining and the Chinese famine of 1958–61. Econ Dev Cult Change. 1997;46:1–34.
9. Song S, Wang W, Hu P. Famine, death, and madness: schizophrenia in early adulthood after prenatal exposure to the Chinese Great Leap Forward Famine. Soc Sci Med. 2009;68:1315–21.
10. Fan C, Huang YQ. Waves of rural brides: female marriage and migration in China. Ann Assoc Am Geogr. 1998;88:227–51.
11. Berry RJ, Li Z. Folic acid alone prevents neural tube defects: evidence from the China study. Epidemiology. 2002;13:114–6.
12. Berry RJ, Li Z, Erickson JD, Li S, Moore CA, Wang H, Mulinare J, Zhao P, Wong LY, et al. Prevention of neural-tube defects with folic acid in China. China-US Collaborative Project for Neural Tube Defect Prevention. N Engl J Med. 1999;341:1485–90.
13. Chobanian AV, Bakris GL, Black HR, William CC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. 2003;42:1206–52.
14. Minnesota Population Center. Integrated Public Use Microdata Series, international: version 6.0 [Machine-readable database]. Bureau of the Census. Minneapolis: University of Minnesota; 2010.
15. Popkin BM, Du S, Zhai F, Zhang B. Cohort profile: The China Health and Nutrition Survey: monitoring and understanding socio-economic and health change in China, 1989–2011. Int J Epidemiol. Epub 2009 Nov 3.
16. Luo Z, Mu R, Zhang X. Famine and overweight in China. Rev Agr Econ. 2006;28:296–304.
17. Stein AD, Kahn HS, Rundle A, Zybert PA, van der Pal-de Bruin, Lumey LH. Anthropometric measures in middle age after exposure to famine during gestation: evidence from the Dutch famine. Am J Clin Nutr. 2007;85:869–76.
18. Stein Z, Susser M, Saenger, Marolla F. Nutrition and mental performance. Prenatal exposure to the Dutch famine of 1944–1945 seems not related to mental performance at age 19. Science. 1972;178:708–13.
19. Rivera JA, Martorell R, Ruel MT, Habicht J-P, Haas J. Nutritional supplementation during preschool years influences body size and composition of Guatemalan adolescents. J Nutr. 1995;125 (Suppl 4): S1068–77.
20. Maluccio JA, Hoddinott J, Behrman JR, Martorell R, Quisumbing AR, Stein AD. The impact of improving nutrition during early childhood on education among Guatemalan adults. Econ J. 2009;119:734–63.
21. Hoddinott J, Maluccio JA, Flores R, Martorell R. Effect of a nutrition intervention during early childhood on economic productivity in Guatemalan adults. Lancet. 2008;371:411–6.
22. Yang Z, Zhao W, Zhang X, Mu R, Zhai Y, Kong L, Chen C. Impact of famine during pregnancy and infancy on health in adulthood. Obes Rev. 2008;9:95–9.
23. Roseboom TJ, van der Meulen JH, Ravelli AC, van Montfrans GA, Osmond C, Barker DJ, Bleker OP. Blood pressure in adults after prenatal exposure to famine. J Hypertens. 1999;17:325–30.
24. Stein AD, Zybert PA, van der Pal-de Bruin K, Lumey LK. Exposure to famine during gestation, size at birth, and blood pressure at age 59 y: evidence from the Dutch famine. Eur J Epidemiol. 2006;21:759–65.
25. Ravelli AC, van Der Meulen JH, Osmond C, Barker DJ, Bleker OP. Obesity at the age of 50 y in men and women exposed to famine prenatally. Am J Clin Nutr. 1999;70:811–6.
26. Ravelli GP, Stein ZA, Susser MW. Obesity in young men after famine exposure in utero and early infancy. N Engl J Med. 1976;295:349–53.
27. Lumey LH, Stein AD, Khan HS, Romijn JA. Lipid profiles in middle-aged men and women after famine exposure during gestation: the Dutch Hunger Winter Families Study. Am J Clin Nutr. 2009;89:1737–43.
28. Zhai F, Wang H, Du S, He Y, Wang Z, Ge K, Popkin B. Prospective study on nutrition transition in China. Nutr Rev. 2009;67:S56–61.