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Association of exposure to Chinese famine in early life with the incidence of hypertension in adulthood: A 22-year cohort study

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Abstract *Background and aims:* Undernutrition in early life may have a lifelong effect on adult health. The conclusions on the association of exposure to famine with the risk of hypertension were inconsistent. The aim of this study was to examine the association of exposure to the Chinese famine with incident hypertension.
Methods and results: Data were obtained from the China Health and Nutrition Survey. All included participants were divided into five birth cohorts: no exposure, born in or after 1962 (N = 2 088); fetal exposure, between 1959 and 1961 (N = 880); early childhood exposure, between 1956 and 1958 (N = 1 214); mid-childhood exposure, between 1953 and 1955 (N = 1 287); and late childhood exposure, between 1949 and 1952 (N = 1 445). Hypertension was defined as SBP/DBP ≥140/90 mmHg, use of hypertensive medications, or a self-reported diagnosis. A total of 6 914 participants were included. The exposure to famine decreased the incidence of hypertension ($P = 0.0018$, 0.0001, <0.0001, and <0.0001; *HR*: 0.715, 0.686, 0.622, and 0.527, respectively) in males. Similarly, the exposure to famine might also decrease incident hypertension in the rural areas ($P = 0.0013$, <0.0001, <0.0001, and <0.0001; *HR*: 0.735, 0.706, 0.679, and 0.539, respectively). There were interaction effects between famine severity and exposure to famine in early ($P = 0.024$) and late childhood ($P = 0.009$).
Conclusion: Exposure to the Chinese famine decreased the incidence of hypertension, especially in males and in the rural areas. Furthermore, the exposure postponed the age at the onset of hypertension.

Introduction

Hypertension, a common chronic disease, is a major public health problem worldwide and the greatest attributable risk factor for death [1]. It is considered as a major modifiable risk factor for cardiovascular disease (CVD) and accounts for approximately 45% of global morbidity and mortality due to CVD [2,3]. The World Health Organization (WHO) reported that the worldwide prevalence of hypertension was approximately 22% among the adult

Acronyms: cardiovascular disease, CVD; World Health Organization, WHO; China Health and Nutrition Survey, CHNS; Systolic blood pressure, SBP; Diastolic blood pressure, DBP; Excess death rate, EDR; Standard deviations, SDs; Hazard ratios, *HRs*.

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population in 2014 [4], and this figure is expected to increase to 29% by 2025, which equals to approximately 1.55 billion people [5,6]. Therefore, taking active measurements is warranted to prevent and control hypertension.

According to the fetal origins hypothesis, undernutrition in early life is a major determinant of health in adulthood [7,8]. Previous studies on famine have provided a number of evidence to examine the developmental origins hypothesis, including the Dutch and Ukraine famine studies [9, 10]. Compared to other famines, the Chinese famine of 1959–1961 had a longer duration and affected most areas in China [11]. This famine resulted in 15–43 million excess deaths and was considered as the worst famine in the human history [12]. Most Chinese aged ≥57 years suffered from the famine at some point in their early life. The associations of exposure to the Chinese famine in early life with the risks of overweight, schizophrenia, labor supply and income, hyperglycemia, and metabolic syndrome in adult life have been well documented [13–15]. However, the association of famine with the incidence of hypertension has remained controversial [16–18], the main reason for this being the fact that most findings from the published Chinese famine studies might be confounded by age [19]. Participants in the exposed groups were older than those in the non-exposed group; therefore, the effect of age on the incidence of hypertension could not be excluded in these studies. Furthermore, previous studies were limited to cross-sectional design, which was poor to examine the association of famine with the incidence of hypertension.

Given the limitations of the previous studies, the present study analyzed data from the China Health and Nutrition Survey (CHNS), a 22-year national cohort study, and aimed to examine the association of exposure to Chinese famine in early life with the incidence of hypertension in adulthood which would allow the identification of individuals who may benefit more from targeted management and screening of hypertension among the famine survivors.

Methods

Study design

This study was based on the CHNS, which is an ongoing, open-cohort project. A multistage random cluster process was employed to create samples in nine provinces that vary substantially in geography, economic development, public resources, and health indicators. The first round of the CHNS was conducted in 1989, and subsequent rounds were conducted in 1991, 1993, 1997, 2000, 2004, 2006, 2009, and 2011. A detailed description of the survey design and procedures has been published elsewhere [20]. This study was approved by the IRB of the National Institute for Nutrition and Food Safety, China Center for Disease Control and Prevention, and University of North Carolina at Chapel Hill. All subjects provided informed consent for participation in the study.

Study population

Data covered all nine waves of the CHNS conducted from 1989 to 2011. All subjects aged ≥18 years at baseline and with available data on sex and detailed physical examination (e.g., weight and height) were eligible for inclusion in the study. Participants who were pregnant or lactating at the time of survey and those with missing or implausible outlying data (e.g., weight >300 kg or <20 kg) were excluded.

Weight and height were measured by trained healthcare workers following standardized protocols. Height was measured to the nearest 0.1 cm without wearing shoes using a portable stadiometer. Weight was measured to the nearest 0.1 kg using a calibrated beam scale while wearing lightweight clothing. BMI was calculated as weight (in kg) divided by the square of height (in m). Three measurements were taken for all indicators, and the average of the three measurements was for analysis.

Smoking and drinking status, as well as physical activity, were recorded for each subject using a questionnaire. Participants' current smoking status was defined as the use of any form of smokeless products, such as at least 100 cigarettes, 20 cigars, or 20 tobacco pipes in their lifetime in the last 30 days preceding the survey [21, 22]. Participants' nonsmoking status was defined as not smoking at the time of survey or never smoked in their lifetime. Participants' current drinking status was defined as having consumed at least 50 g alcohol daily in the last 30 days [23].

Blood pressure (BP) measurements were taken after rest for 10 min in the seated position, with 30-s intervals between the cuff inflations, using standard mercury sphygmomanometers [24]. Cuff size was carefully selected according to the participant's arm circumference. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded as the points at which the first and fifth Korotkoff sounds appeared, respectively. Both SBP and DBP were measured three times, and the average of the three measurements was used. Hypertension was defined as SBP ≥140 mmHg, DBP ≥90 mmHg, use of hypertensive medications, or a self-reported diagnosis [24, 25].

Famine cohorts and region categories

As the Chinese famine happened from 1959 to 1961, birthdate was taken as the proxy variable of exposure to famine in this study. According to a previous study [26], after transforming all lunar birthdates into western birthdates, all participants were divided into five birth cohorts: no exposure, defined as born between 1962 and 1965; fetal exposure, defined as born between 1959 and 1961; early childhood exposure, defined as born between 1956 and 1958; mid-childhood exposure, defined as born between 1953 and 1955; and late childhood exposure, defined as born between 1949 and 1952.

Because 1960 was the worst year of the Chinese famine, the excess death rate (EDR) was varied across regions and used as the proxy variable of famine severity. The EDR was calculated as the change in mortality rate from the average level in 1956–1958 to the highest level over the period of 1959–1961 [13]. The EDR of 100% was used as the threshold. Regions with EDR ≥100% were categorized as severely affected famine areas, whereas the other aears were categorized as less severely affected famine areas. The gross family income was standardized using 2011 as the reference.

Statistical analysis

Data were presented as means ± standard deviations (SDs) for continuous variables and frequencies (percentages) for categorical variables. Baseline characteristics were compared between the non-hypertension and hypertension groups using *t*-test for continuous variables, *chi-square* tests for categorical variables, and *Wilcoxon rank sum* test for ordinal variables. *Cox* regressions were conducted to obtain hazard ratios (*HRs*), with hypertension as the end-event and the time interval from the birth year to the occurrence of hypertension, death, loss to follow-up, or the end of this study, whichever came first, as the time variable. The censored outcomes were from two groups of patients: i. those who were not diagnosed with hypertension until either drop-out from the cohort or the end of the study (2011) and ii. those who were not diagnosed with hypertension until death prior to the end of the study. In order to correct the competing risks of death for hypertension, all models were adjusted for death. All *Cox* regression models met the proportional hazards assumption. In the adjusted models, age, sex, smoking, drinking, physical activity, BMI, gross family income, famine severity, regions, and ethnicity at baseline were adjusted. The effect of exposure to famine in different periods was analyzed, with no exposure as the reference. Linear regressions were employed to analyze the effect of famine on the age at onset of hypertension. Given the cluster of data due to the sample method, family unit was corrected as the random effect term. All analyses were conducted using SAS 9.4 (SAS Institute Inc., Cary, NC, USA.), with two-tailed $P \leq 0.05$ considered to indicate statistical significance.

Results

In this study, 6 914 subjects were included, comprising 4 152 non-hypertensive and 2 762 hypertensive subjects. The median follow-up duration was 47 and 45 years in the non-hypertension and hypertension groups, respectively. The mean age was 36.94 years, and BMI was 22.33 kg/m^2. Baseline characteristics were compared between the non-hypertension and hypertension groups. There were significant differences in all characteristics except physical activity ($P = 0.268$) and gross family income ($P = 0.581$) (Table 1).

Table 2 shows the associations of famine with the incidence of hypertension from the *Cox* regressions in the total population as well as in the male and female subgroups. In total, exposure to famine decreased the incidence of hypertension ($P_{trend} < 0.001$) (Fig. 1). When stratified by sex, the results of crude models in both males and females were similar to those in the total population. After adjusting for covariates, exposure to famine in fetal, early, mid, and late childhood was found to decrease the incidence of hypertension (adjusted $P_{trend} < 0.001$, $P = 0.003$, <0.001, <0.001, and <0.001; *HR*: 0.728, 0.652, 0.620, and 0.522; and *HR 95% CI*: 0.591–0.896, 0.537–0.792, 0.505–0.760, and 0.419–0.650, respectively) in males, whereas in females, the exposure in any period was not associated with hypertension, except in late childhood (adjusted $P_{trend} = 0.010$, $P = 0.010$; *HR*: 0.733; and *HR 95% CI*: 0.579–0.929).

When stratified by regions, significant associations of exposure to famine in mid- (adjusted $P = 0.029$; *HR*: 0.752; and *HR 95% CI*: 0.582–0.971) and late (adjusted $P = 0.007$; *HR*: 0.689; and *HR 95% CI*: 0.525–0.902) childhood with the incidence of hypertension were observed in the urban regions after adjusting for covariates, whereas in the rural regions, the exposure to famine in fetal, early, mid, and late childhood was shown to decrease the incidence of hypertension (adjusted all $P < 0.001$; *HR*: 0.698, 0.706, 0.682, and 0.569; and *HR 95% CI*: 0.578–0.842, 0.594–0.840, 0.570–0.816, and 0.467–0.693, respectively) (Fig. 2).

The associations of exposure to famine with age at the onset of hypertension are displayed in Table 3. In total, compared to no exposure, exposure to famine in any period of childhood could postpone the age at the onset of hypertension ($P_{trend} < 0.001$). These results were comparable before and after adjustment for covariates (all $P < 0.001$). When stratified by sex or region, the results of all subgroups were consistent with those of the total population, except in fetal childhood and in the urban regions ($P = 0.585$).

Considering that exposure to famine could postpone the age at the onset of hypertension, the interaction effect between exposure to famine and famine severity on incident hypertension is presented in Table 4. Whether adjusting for covariates or not, there were interaction effects between early childhood exposure and famine severity ($P_{crude} = 0.020$ and $P_{adjusted} = 0.024$) as well as between late childhood exposure and famine severity ($P_{crude} = 0.016$ and $P_{adjusted} = 0.009$). Regarding severe famine, there were stronger effects of early and late childhood exposure on the incidence of hypertension ($\beta = -0.281$ and -0.300, respectively).

Discussion

Because the associations of exposure to famine with the risk of hypertension were controversial, this study attempted to examine this association based on a 22-year cohort study from the CHNS. The results suggested that exposure to Chinese famine in early life was associated with the incidence of hypertension and could decrease the risk of hypertension. The results of subgroup analysis indicated that exposure to Chinese famine in early life

Table 1 The characteristics of all participants at baseline.

Variables	All subjects (N = 6914)	Non-hypertension (N = 4152)	Hypertension (N = 2762)	*P*
Age (years)[a]	36.94 ± 8.17	36.22 ± 8.19	38.02 ± 8.01	<0.001
BMI (kg/m^2)[a]	22.33 ± 2.90	21.81 ± 2.64	23.12 ± 3.10	<0.001
Sex				<0.001
Male	3390 (49.03)	1874 (45.13)	1516 (54.89)	
Female	3524 (50.97)	2278 (54.87)	1246 (45.11)	
Smoking				<0.001
No	3975 (59.73)	2501 (64.00)	1474 (53.66)	
Yes	2680 (40.27)	1407 (36.00)	1273 (46.34)	
Drinking				<0.001
No	3329 (50.05)	2104 (53.88)	1225 (44.61)	
Yes	3322 (49.95)	1801 (46.12)	1521 (55.39)	
Ethnicity				<0.001
Han	5812 (84.06)	3383 (81.48)	2429 (87.94)	
Other	1102 (15.94)	769 (18.52)	333 (12.06)	
Physical activity				0.268
No	6525 (94.37)	3908 (94.12)	2617 (94.75)	
Yes	389 (5.63)	244 (5.88)	145 (5.25)	
Famine[b]				<0.001
No exposure	2088 (30.20)	1453 (35.00)	635 (22.99)	
Fetal childhood	880 (12.73)	570 (13.73)	310 (11.22)	
Early childhood	1214 (17.56)	707 (17.03)	507 (18.36)	
Mid- childhood	1287 (18.61)	702 (16.91)	585 (21.18)	
Late childhood	1445 (20.90)	720 (17.34)	725 (26.25)	
Gross family income				0.581
Low	3549 (51.33)	2120 (51.06)	1429 (51.74)	
High	3365 (48.67)	2032 (48.94)	1333 (48.26)	
Famine severity				<0.001
Less severe famine	2205 (31.89)	1180 (28.42)	1025 (37.11)	
Severe famine	4709 (68.11)	2972 (71.58)	1737 (62.89)	
Region				<0.001
Urban	2704 (39.11)	1755 (42.27)	949 (34.36)	
Rural	4210 (60.89)	2397 (57.73)	1813 (65.64)	

[a] These variables were analyzed by *t-test*.
[b] The variable was analyzed by *Wilcoxon rank sum* test.

Table 2 The associations of famine with the incidence of hypertension from *Cox* regressions.

Famine	Crude				Adjusted※			
	β	*P*	HR	HR 95% CI	β	*P*	HR	HR 95% CI
Total (N = 6 914)								
No exposure	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Fetal childhood exposure	−0.316	<0.001	0.729	0.630–0.845	−0.235	0.002	0.791	0.680–0.920
Early childhood exposure	−0.502	<0.001	0.605	0.531–0.690	−0.275	0.001	0.760	0.661–0.873
Mid-childhood exposure	−0.712	<0.001	0.491	0.431–0.559	−0.343	<0.001	0.710	0.613–0.822
Late childhood exposure	−0.948	<0.001	0.387	0.340–0.441	−0.490	<0.001	0.613	0.523–0.718
P_{trend}	–	<0.001	–	–	–	<0.001	–	–
Males (N = 3 390)								
No exposure	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Fetal childhood exposure	−0.365	<0.001	0.694	0.573–0.841	−0.318	0.003	0.728	0.591–0.896
Early childhood exposure	−0.625	<0.001	0.535	0.451–0.635	−0.427	<0.001	0.652	0.537–0.792
Mid-childhood exposure	−0.793	<0.001	0.452	0.382–0.536	−0.479	<0.001	0.620	0.505–0.760
Late childhood exposure	−1.090	<0.001	0.336	0.284–0.398	−0.650	<0.001	0.522	0.419–0.650
P_{trend}	–	<0.001	–	–	–	<0.001	–	–
Females (N = 3 524)								
No exposure	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Fetal childhood exposure	−0.251	0.029	0.778	0.621–0.975	−0.157	0.190	0.855	0.676–1.081
Early childhood exposure	−0.341	0.001	0.711	0.585–0.865	−0.094	0.384	0.910	0.737–1.125
Mid-childhood exposure	−0.580	<0.001	0.560	0.462–0.679	−0.172	0.126	0.842	0.676–1.050
Late childhood exposure	−0.781	<0.001	0.458	0.378–0.556	−0.311	0.010	0.733	0.579–0.929
P_{trend}	–	<0.001	–	–	–	0.010	–	–

※In adjusted models, age, sex, BMI, smoking, drinking, physical activity, ethnicity, income, famine severity, region, and death were adjusted in total population; and age, BMI, smoking, drinking, physical activity, ethnicity, income, famine severity, region, and death were adjusted in male and female population.

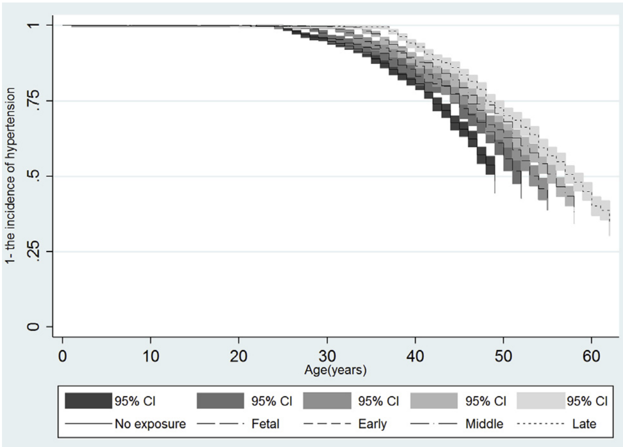

Figure 1 The survival curve of exposure to famine on the incidence of hypertension.

would lower the risk of hypertension in males and in the rural regions. Although several previous studies have reported that exposure to famine in early life increases the risk of hypertension [16, 21, 27], these studies were limited to cross-sectional study, which was poor in examining the association of famine with hypertension. Furthermore, some studies have also mentioned the potential age bias [19, 28]. While famine cohorts were defined according to the birth year, age increased from fetal to late childhood and all participants included in the exposed groups were older than those included in the non-exposed groups [29]. If age differences were not corrected, the effect of famine on hypertension would be affected by age.

As the worst famine in the human history, the Chinese famine resulted in over 30 million excess deaths in most areas of China. The famine survivors might be healthier and stronger than the frail members kicked out by the famine, a finding that is in line with Darwin's theory of survival of the fittest [30]. In this case, the surviving famine cohort might have better performance and should show a lower risk of hypertension [16]. Therefore, exposure to famine could decrease the risk of hypertension in adulthood. However, there might be selection bias due to the competing risk. As those died due to famine were frailer and would have been more likely to develop hypertension in adulthood, no data were available for individuals who died from the famine and the competing risk could not be corrected. This should be examined in combination with the death register data in further research.

In this study, participants born between 1962 and 1965 were considered as having been not exposed to famine. However, their parents must have inevitably experienced the famine. A previous study reported that exposure to extreme malnutrition might lead to a low birth-weight offspring, which has been shown to be associated with a reduction in the number of nephrons to raise BP during adulthood of these offspring [29]. Therefore, individuals with no exposure to famine might be more likely to become hypertensive than those with exposure to famine.

Sex and region differences could modify the association of famine with hypertension in stratified analyses. Because of the male preference in China [31], women might be more adaptable to famine than men. Therefore, exposure to famine rarely affected the incident hypertension in women. Although the Chinese famine covered most regions, famine severity was varied across different regions, especially between the urban and rural regions. As a result, significant associations were observed only in mid- and late childhood exposure in the urban regions. In the present study, only year of birth rather than the exact date of birth was used to define exposure, which might have led to misclassification. However, another study has confirmed that no impact of the variation on the reported outcomes [19].

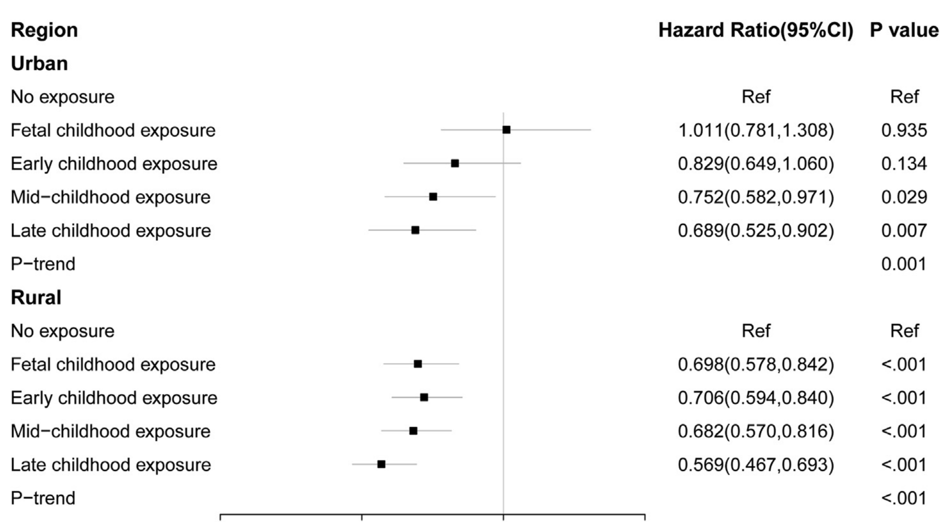

Figure 2 The associations of exposure to famine with the incidence of hypertension in both urban and rural areas.

Table 3 The associations of famine with the onset age of hypertension in hypertension population.

Famine	Crude			Adjusted※		
	β	t	*P*	β	t	*P*
Total						
No exposure	Ref	Ref	Ref	Ref	Ref	Ref
Fetal childhood exposure	3.128	6.28	<0.001	1.966	4.22	<0.001
Early childhood exposure	5.826	13.60	<0.001	3.284	7.83	<0.001
Mid-childhood exposure	8.077	19.60	<0.001	4.269	9.76	<0.001
Late childhood exposure	10.768	27.55	<0.001	5.814	12.69	<0.001
P_{trend}	–	–	<0.001	–	–	<0.001
Males						
No exposure	Ref	Ref	Ref	Ref	Ref	Ref
Fetal childhood exposure	3.343	5.01	<0.001	1.870	2.99	0.003
Early childhood exposure	6.425	11.02	<0.001	3.320	5.76	<0.001
Mid-childhood exposure	7.588	13.47	<0.001	3.327	5.52	<0.001
Late childhood exposure	11.093	20.83	<0.001	5.282	8.26	<0.001
P_{trend}	–	–	<0.001	–	–	<0.001
Females						
No exposure	Ref	Ref	Ref	Ref	Ref	Ref
Fetal childhood exposure	2.690	3.62	<0.001	1.820	2.60	0.010
Early childhood exposure	4.861	7.74	<0.001	2.980	4.84	<0.001
Mid-childhood exposure	8.293	13.77	<0.001	5.135	8.05	<0.001
Late childhood exposure	10.094	17.59	<0.001	6.180	9.37	<0.001
P_{trend}	–	–	<0.001	–	–	<0.001
Urban						
No exposure	Ref	Ref	Ref	Ref	Ref	Ref
Fetal childhood exposure	1.852	2.31	0.021	0.401	0.55	0.585
Early childhood exposure	6.134	8.59	<0.001	3.581	5.28	<0.001
Mid-childhood exposure	8.174	11.86	<0.001	3.923	5.48	<0.001
Late childhood exposure	11.152	17.29	<0.001	5.568	7.63	<0.001
P_{trend}	–	–	<0.001	–	–	<0.001
Rural						
No exposure	Ref	Ref	Ref	Ref	Ref	Ref
Fetal childhood exposure	3.893	6.14	<0.001	2.833	4.73	<0.001
Early childhood exposure	5.667	10.60	<0.001	3.131	5.89	<0.001
Mid-childhood exposure	8.028	15.64	<0.001	4.422	8.03	<0.001
Late childhood exposure	10.550	21.49	<0.001	5.903	10.08	<0.001
P_{trend}	–	–	<0.001	–	–	<0.001

※In adjusted models, age, sex, BMI, smoking, drinking, physical activity, ethnicity, income, famine severity, region, and death were adjusted in total population; and age, BMI, smoking, drinking, physical activity, ethnicity, income, famine severity, region, and death were adjusted in male and female population; and age, sex, BMI, smoking, drinking, physical activity, ethnicity, income, famine severity, and death were adjusted in urban and rural regions.

Table 4 The interaction effect between famine exposure and famine severity on the incident hypertension.

Variables	Crude model			Adjusted model※		
	β	χ^2	*P*	β	χ^2	*P*
No exposure	Ref	Ref	Ref	Ref	Ref	Ref
Fetal childhood exposure	−0.319	7.482	0.006	−0.238	4.072	0.044
Early childhood exposure	−0.302	9.222	0.002	−0.098	0.927	0.336
Mid-childhood exposure	−0.602	36.889	<0.001	−0.270	6.697	0.010
Late childhood exposure	−0.731	59.172	<0.001	−0.283	7.307	0.007
Less severe famine	Ref	Ref	Ref	Ref	Ref	Ref
Severe famine	−0.132	2.444	0.118	−0.148	2.977	0.085
No exposure× Less severe famine	Ref	Ref	Ref	Ref	Ref	Ref
No exposure× Severe famine	Ref	Ref	Ref	Ref	Ref	Ref
Fetal childhood exposure× Less severe famine	Ref	Ref	Ref	Ref	Ref	Ref
Fetal childhood exposure× Severe famine	0.007	0.003	0.960	0.015	0.011	0.917
Early childhood exposure× Less severe famine	Ref	Ref	Ref	Ref	Ref	Ref
Early childhood exposure× Severe famine	−0.289	5.458	0.020	−0.281	5.109	0.024
Mid-childhood exposure× Less severe famine	Ref	Ref	Ref	Ref	Ref	Ref
Mid-childhood exposure× Severe famine	−0.124	1.070	0.301	−0.107	0.783	0.376
Late childhood exposure× Less severe famine	Ref	Ref	Ref	Ref	Ref	Ref
Late childhood exposure× Severe famine	−0.273	5.774	0.016	−0.300	6.850	0.009

※In adjusted models, age, sex, BMI, smoking, drinking, physical activity, ethnicity, income, region, and death were adjusted.

Strengths and limitations

The strengths of our study were that this study was based on a national and representative cohort study. This was a prospective study that provided more power and better understanding regarding the association of famine with incident hypertension. In multivariate analyses, age, socioeconomics, famine severity, and regions were adjusted, which could correct the effects of confounding factors and provide a more accurate association of famine with hypertension. Moreover, the relationship between famine and the age at the onset of hypertension would further confirm the protective effects of exposure to famine. Nevertheless, the limitations of this study should also be stated. Due to the exact start and end dates of the Chinese famine being unknown, the year of famine alone was used to determine the Chinese famine. As data related to the specific diet intake were unavailable in the CHNS, nutrients related to hypertension such as salt intake could not be adjusted. Further, because of the large proportion of missing data related to the history of diseases such as diabetes and cardiovascular diseases and lack of family history of hypertension and birth weight, disease and family history and birth weight were not adjusted.

In conclusion, exposure to Chinese famine in fetal, early, mid, and late childhood was associated with the incidence of hypertension and would decrease the risk of hypertension. The protective effects could be fully observed in the male population and in the rural regions. Furthermore, exposure to famine postponed the age at the onset of hypertension.

Conflicts of interest

The authors have nothing to disclose.

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