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ARTICLE

Intergenerational effects of early-life health shocks during the Chinese 1959–1961 famine

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Abstract
A large literature has examined early-life insult and later-life health outcomes. However, whether early-life exposure might persist into the outcomes of future generations remains unclear. Using data from the China Family Panel Studies, this study examines the intergenerational effects of early-life health shocks during the great famine in China, distinguishes the intergenerational effects of *in utero* and early-life famine exposure, and estimates whether there is a sex-specific transgenerational response. Difference-in-difference results show that first-generation male *in utero* famine exposure (1959–1961) is associated with a series of health and economic disadvantages in the second generation, compared with the unexposed post-famine-born cohort (1964–1965) in China. The effect persists in the third generation but attenuates, and there is no same-sex transgenerational response. These findings may suggest a novel source of multigenerational persistence in health and economic poverty and may point to a need to consider evidence of transgenerational mechanisms.

Keywords: intergenerational effects; great famine; China

Introduction

A large body of literature has examined early-life insult and later-life health outcomes based on the developmental origins of health and disease theory (often referred to as the Barker hypothesis). It suggests that insults *in utero* programme the foetus in ways that lead to maladaptive responses to the environment that persist throughout the life course (Barker, 1990, 1995*a*, 1995*b*). As using experimental research designs is not possible, many studies have used quasi-experimental designs by leveraging striking demographic events, such as famine and pandemics, to demonstrate the accuracy of the foetal origin hypothesis. Almond's seminal studies are among the first to exploit the 1918 flu pandemic as a natural experiment and test whether *in utero* exposure influences later-life outcomes. These studies find lower

educational attainment, higher rates of physical disability, and lower income and socio-economic status (SES) in the cohorts exposed to the flu pandemic (Almond and Mazumder, 2005; Almond, 2006). Roseboom's seminal studies also find that individuals exposed to the Dutch famine *in utero* have increased prevalence of cardiovascular disease, diabetes, obesity and breast cancer (Roseboom *et al.*, 2001, 2006, 2010; Roseboom and Watson, 2012). Subsequent studies build on these outcomes and find significant disadvantages compared with the non-exposed reference population.

With these results established, the next question is whether *in utero* effects might persist into the health outcomes of future generations. Experimental evidence (rodent studies) indicates that health traits, which have been induced by environmental insults during the *in utero* shock in one generation, may be transmitted to future generations without alterations to the genome. Feeding protein-restricted rats during gestation results in higher blood pressure and endothelial dysfunction not only in the offspring but also in the grand-offspring (Torrens *et al.*, 2008). A low-protein diet during pregnancy leads to insulin resistance in the adult male and female second-generation offspring (G2) (Zambrano *et al.*, 2005; Pinheiro *et al.*, 2008), and glucose metabolism in G3 is also affected by G0 undernutrition (Benyshek *et al.*, 2006). Transgenerational effects are also seen in humans, but studies are scarce. A historical study of three generations in Överkalix, Sweden, reports that when grandparents have a limited food supply, this influences their grandchildren's later mortality and disease risk in a sex-specific manner, in part operating exclusively through the paternal line (Pembrey *et al.*, 2014). Richter and Robling (2015) find that first-generation exposure to the 1918 flu *in utero* results in two to three fewer months of schooling for the second generation. Cook *et al.* (2019) also find multigenerational effects of the flu pandemic on educational, economic and health outcomes.

Two pathways exist for the intergenerational transmission of early-life health shocks: socio-economic channels and biological channels (phenotype-to-phenotype transmission or epigenetic inheritance) (Kuzawa and Eisenberg, 2014). Through socio-economic channels, intergenerational persistence of adverse outcomes could occur when parents experiencing a health insult while *in utero* raise children in a poor-resource environment. Biologically, in cases of shocks *in utero*, phenotype-to-phenotype transmission impacts the outcomes of the next generation through changes in parental biological systems that lead to altered gestational and/or lactation environments for offspring (Cook *et al.*, 2019). Epigenetic inheritance through gametes exists in various species and epigenetic information carriers, including DNA methylation, non-coding RNAs, and chromatin proteins in gametes, play important roles in the transmission of phenotypes from parents to offspring (Wei *et al.*, 2015). More importantly, socio-economic and biological channels can also interact with each other. That is, the effects of the adult phenotype on offspring are not limited to physiology and metabolism, but also include parental behaviour/environmental responses as potential sources of phenotypic transmission, and possibly even cumulative phenotypic changes across generations (Benyshek, 2013; Cook *et al.*, 2019).

To explore intergenerational effects, some research has focused on exogenous (*i.e.* unrelated to genetics) health shocks. Researchers have increasingly used exposure to prenatal famine or infectious diseases as a natural experiment in studies of

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long-term health effects (Lumey *et al*., 2011). Well-known famine examples include 19th-century crop failures in Sweden and Finland, the Siege of Leningrad of 1941–1944, the Dutch Hunger Winter of 1944–1945, the Chinese great famine of 1959–1961 and the Bangladesh famine of 1974. Well-known infectious diseases examples include the Black Death before the 17th century, the 1918 influenza pandemic, severe acute respiratory syndrome (SARS) in 2003 and the ongoing COVID-19 pandemic, which began in 2019. As exposure to infectious diseases or famine is beyond the control of most individuals, regardless of their genetic traits, personality or SES, the process governing an individual's prenatal exposure is arguably exogenous and resembles a random assignment.

In this study, we seek to advance the literature by examining the intergenerational or multigenerational effects of the 1959–1961 Chinese famine.[1] China adopted economic policies to transform rapidly from a predominantly agrarian society to an industrialised socialist economy supported by an agricultural collectivisation programme in the early 1950s (Chen and Zhou, 2007; Xu *et al*., 2016). All rural households were organised into thousands of People's Communes in 1958. Natural disaster, excessive state procurement and weakened production motivations due to the sweeping collectivisation programme in 1958 led to agricultural production dropping sharply, and the great famine ensued (Eckstein, 1966; Ashton *et al*., 1984; Lin and Yang, 2000; Chen and Zhou, 2007; Meng *et al*., 2015; Xu *et al*., 2018). China's grain output dropped from 200 million tons in 1958 to 170 million tons in 1959, and 140 million tons in the following two years (1960–1961) (State Statistical Bureau, 1990). The nationwide famine lasted three years (the end of 1961 in most regions, and into 1962 in some rural areas) until the government put forward the policy of 'adjusting, consolidating, enriching and improving' operation of the national economy, and sent large amounts of grain into rural areas. Our analysis conducts novel examinations of multigenerational effects of the Chinese famine and extends four aspects of prior studies: (a) we distinguish the intergenerational effects of *in utero* and early famine exposure; (b) using a difference-in-difference (DID) strategy, we identify the famine severity in different provinces by gender; (c) going further, we also estimate whether there is a sex-specific transgenerational response; and (d) we simultaneously examine the effects of grandparents' and maternal grandparents' famine exposure on the third generation.

Methods

Data

To examine intergenerational effects of early-life health shocks, we base our study on data from the China Family Panel Studies (CFPS), a nationally representative, longitudinal survey of Chinese communities, families and individuals, collected by the Institute of Social Science Survey at Peking University (Xie and Hu, 2014). The CFPS started in 2010 and gathered information on family structure and family members, health, event history and child development.

Our analysis draws on data from the fourth wave conducted in 2016, in which respondents (G1) who were *in utero* and or experienced early-life exposure to the 1959–1961 great famine might have children (G2) or grandchildren (G3), providing the structure for our intergenerational analysis. We restrict the first generation to

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cohorts born from 1956 to 1965, focusing on those born before or after the famine. We divide the first generation into four groups: (a) the pre-famine birth cohort (1956–1958), who were exposed to the famine between the ages of 0–3 years; (b) the famine birth cohort (1959–1961), who were exposed to the famine *in utero*; (c) the post-famine birth cohort (1962–1963), who experienced partial prenatal exposure to the famine; and (d) the reference cohort (1964–1965), who had no exposure to the famine. Descriptive characteristics for variables are reported in Table 1. Among the first-generation females, 30.27 per cent experienced the famine between the ages of 0–3 years and 20.75 per cent experienced the famine *in utero*. Among the first-generation males, 54.46 per cent (32.00 + 22.46 = 54.46) experienced the famine at the age of 0–3 or *in utero*.

Measurements

Following the broader economic and health focus of the Barker hypothesis, we consider two broad measures of general economic outcomes and four health outcomes in the second generation.

Education degree is assessed in the CFPS by the survey question, 'How would you describe your highest degree?' Respondents answered on a scale from 1 (illiteracy) to 8 (doctoral degree). We recode this variable so that education degree indicates the years of education. Income is another economic indicator calculated by the CFPS group based on the questionnaire. We take the logged value of income and negative income reporting is assigned a wealth value of 1 RMB before taking the natural log. Height (in centimetre) and weight (in catty) are measured by the survey questions 'How tall are you now?' and 'How much do you weigh now?', respectively; body mass index (BMI) is calculated as weight (in kilograms) divided by the square of height (in metres). Birth weight (in catty) is another primary measure of health in most analyses of health and welfare, and is measured by the question, 'How much did you weigh at birth?'

Assessing famine severity

As the place of birth of the first generation cannot be identified directly due to the CFPS data limitations, we use G2's birth province as a proxy variable for G1's birth province. The reasons are as follows. Interprovincial migration was very low from the famine year to the early 1990s. In order to ensure structural stability, the Chinese government officially adopted the family register system, the *hukou* system, to control the movement of people between urban and rural areas in 1958 (Li *et al.*, 2013; Li and An, 2015). Individuals seeking to move from rural to urban areas to take up non-agricultural work would have to apply through the relevant bureaucracies, and require six passes to work in other provinces. Therefore, the number of such workers was tightly controlled. Interprovincial migrants only accounted for 0.3–0.7 per cent of the population during the period 1959–1963 (Liang and White, 1996). In 1984, the State Council of the People's Republic of China issued a notice on the settlement of farmers in towns, showing restrictions on population movement were gradually lifted, while the spatial mobility of China's population was still very low. The 1990 census showed that the interprovincial migration was less than 0.5 per cent of the total population

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Table 1. Summary statistics

Variable	N	%	Mean	SD	Minimum	Maximum
First generation, female:						
Pre-famine cohort	732	30.27		–	–	–
Famine cohort	500	20.75		–	–	–
Post-famine cohort	641	26.60		–	–	–
Reference cohort	537	22.28		–	–	–
CSSI	2,410		0.38	0.11	0.17	0.63
Birth year	2,410		1960.7	2.98	1956	1965
First generation, male:						
Pre-famine cohort	738	32.00		–	–	–
Famine cohort	518	22.46		–	–	–
Post-famine cohort	552	23.94		–	–	–
Reference cohort	498	21.60		–	–	–
CSSI	2,306		0.39	0.11	0.18	0.63
Birth year	2,306		1960.6	2.97	1956	1965
Second generation:						
Education	3,021		9.97	4.42	0	19
Income	1,203		7.77	4.40	0	14.4
Height	3,018		166.0	7.99	112.5	196
Weight	2,803		126.9	25.4	70	260
BMI	2,800		22.9	3.65	12.7	51.4
Birth weight	1,370		6.18	1.12	2	10
Gender	3,021		0.50	0.50	0	1
Birth year	3,021		1986.2	4.19	1976	1995
Number of siblings	2,916		1.33	1.06	0	7

Notes: The table provides summary statistics for all variables used. The sample sizes of Table 1 may not be representative for all estimations. SD: standard deviation. CSSI: cohort size shrinkage index. BMI: body mass index.

(Duan *et al.*, 2008). Meanwhile, interprovincial marriage migration was also very low until the early 1990s. That is, there were 1.54 million interprovincial marriages, accounting for only 0.13 per cent of the total population in 1990 (Hu and Li, 2015).

Following Xu *et al.* (2018), we quantify the regional famine severity using the cohort size shrinkage index (CSSI) from the 1 per cent sample of the 1990 Census of Chinese Population. Specifically, due to actual incidence of famine by province and the different mortality rates by gender across lifetimes, we calculate the mean size of cohorts born three years before the famine (1956–1958) and three years after the famine (1962–1964) by province and gender, and label this

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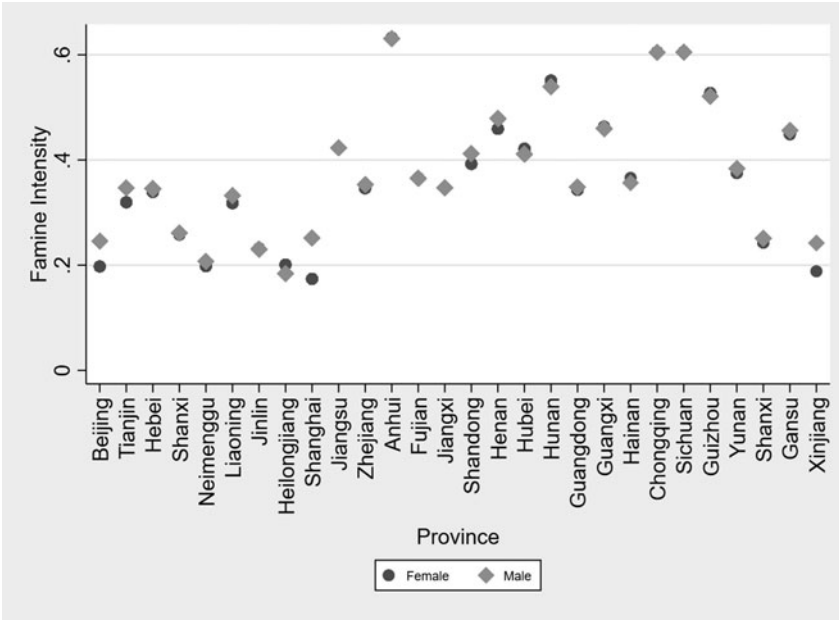

Figure 1. Geographical distribution of the cohort size shrinkage index (famine intensity) by gender.

N_{nonfam}. The mean size of cohorts born during the famine years (1959–1961) is labelled N_{fam} by province and gender.

Subsequently, we calculate the famine severity by gender in different provinces, generating the CSSI as the difference between N_{nonfam} and N_{fam}, divided by N_{nonfam}. In general, CSSI is between 0 and 1, and the severity of famine increases with the number. Measures of cohort loss can only capture the famine intensity under a series of assumptions (*e.g.* accurate census data on fertility and mortality, stable secular trends in fertility and mortality in the absence of the famine, and strictly restricted migration), however, CSSI remains the best parameterisation of that latent factor. The variations in CSSI across provinces are relatively large (Figure 1). Among the 28 provinces in our analytical sample, Anhui, Chongqing and Sichuan experience severe famine with high CSSI.

Analysis strategy

Following Cook *et al.* (2019), we examine harmful effects of being exposed *in utero* and during early-life by estimating intergenerational impacts on the second and third generations. Subsequently, we extend this analysis by using a DID method and exploit the variation of famine exposure across cohorts and regions to estimate the effects of famine:

$$y_{ig,jk} = \alpha_0 + \alpha_1 CSSI_j + \sum_{k=1}^{4} \beta_k Cohort_k + \sum_{k=1}^{4} \gamma_k (CSSI_j \times Cohort_k) + \delta X_{ig,jk} + \varepsilon_{ig,jk}$$

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where $y_{ig,jk}$ refers to the outcomes for an individual i for generation g, whose parents/grandparents are born in region j and cohort k; $X_{ig,jk}$ represents generation-specific controls; and $\varepsilon_{ig,jk}$ is representative of a random error term. Our primary focus is on the coefficient γ_k, the coefficient of the interaction between the CSSI and the cohort dummy variables, which captures the famine effect as a treatment effect of generation g in a standard DID model. A detailed discussion on estimating the treatment effect using the interaction term in non-linear DID models has been published previously (Athey and Imbens, 2006). Standard error is adjusted for clustering by family.

Results

Economic effects of the second generation

To test intergeneration effects of famine exposure, we conduct regression analysis on two primary economic outcomes of the second generation. These estimations are performed in Table 2. Columns 1 and 4 focus on an indicator for mothers' (G1 females') exposure to the great famine; columns 2 and 5 are an indicator for fathers' (G1 males') exposure; and columns 3 and 6 consider both mothers' (G1 females') and fathers' (G1 males') exposure. Controls include an indicator for sex, birth year and its square, and the number of siblings in the second generation.

To reiterate, our hypothesis is that *in utero* or early-life exposure to the 1959–1961 famine has effects that persist for multiple generations. As shown in column 1 of Table 2, mother's *in utero* exposure to the famine is followed by a decline in years of schooling in the second generation compared with the reference cohort, however, the difference is not significant. Further, we do not find a statistically significant difference in years of schooling in the second generation for those whose mother was born either before (1956–1958) or immediately after the famine (1962–1963). Column 2 shows that having a father born in 1959–1961 is associated with a statistically significant ($p < 0.001$) decline in years of schooling in the second generation. Once again, having a father born before or after the famine seems to have no effects on years of schooling in the second generation. Column 3 examines maternal and paternal influences and finds that having a father born in 1959–1961 reduces the educational attainment of offspring, net of the mother's famine experience. The findings of years of schooling are like income in the second generation, which replaces years of schooling as the dependent variable with the natural log of income for the 2016 wave. As with years of schooling, a negative association is seen throughout the specifications of fathers born in 1959–1961, which is associated with a decline in income in the second generation. The results in columns 4 and 6 show that the effect of a mother's post-famine exposure becomes statistically positively significant at the 5 per cent level ($p = 0.033$), implying that those with mothers born in 1962–1963 have much more income compared with the reference group by 2016.

Previous research has pointed to important gender differences in education and income, emphasising social structural and behavioural factors. These gender differences should merit attention insofar as they might lead to the connection between sex-linked biological variation or gender-based transgenerational responses.

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Table 2. Second-generation economic effects

	Years of schooling			Income in 2016		
	(1)	(2)	(3)	(4)	(5)	(6)
Gender	0.04	−0.01	−0.20	0.24	0.29	0.17
	(−0.27, 0.35)	(−0.33, 0.31)	(−0.56, 0.17)	(−0.34, 0.81)	(−0.27, 0.85)	(−0.50, 0.83)
Birth year	12.75	−16.66	8.85	23.27	25.54	11.85
	(−19.82, 45.33)	(−54.03, 20.70)	(−35.43, 53.13)	(−39.84, 86.38)	(−41.27, 92.36)	(−73.26, 96.96)
Birth year × birth year	−0.00	0.00	−0.00	−0.01	−0.01	−0.00
	(−0.01, 0.01)	(−0.01, 0.01)	(−0.01, 0.01)	(−0.02, 0.01)	(−0.02, 0.01)	(−0.02, 0.02)
Number of siblings	−1.29***	−1.29***	−1.25***	−0.33*	−0.38*	−0.21
	(−1.46, −1.12)	(−1.46, −1.12)	(−1.46, −1.04)	(−0.63, −0.03)	(−0.67, −0.09)	(−0.56, 0.15)
Pre-famine cohort, female	1.03		0.31	−0.99		−3.27
	(−0.50, 2.57)		(−2.01, 2.63)	(−3.82, 1.83)		(−7.98, 1.44)
Famine cohort, female	0.80		−0.24	0.93		−1.87
	(−0.79, 2.39)		(−2.32, 1.84)	(−1.83, 3.68)		(−5.08, 1.34)
Post-famine cohort, female	−1.07		−1.05	−2.61*		−3.18*
	(−2.63, 0.50)		(−2.69, 0.58)	(−5.16, −0.05)		(−6.00, −0.35)
CSSI, female	−6.09***		−22.85***	−7.79**		−10.52
	(−9.06, −3.11)		(−31.42, −14.28)	(−12.54, −3.03)		(−26.85, 5.81)
Pre-famine cohort × CSSI, female	0.02		1.31	2.71		5.45
	(−3.90, 3.94)		(−5.07, 7.68)	(−4.49, 9.92)		(−7.41, 18.32)
Famine cohort × CSSI, female	−0.10		2.64	−2.66		3.68
	(−4.31, 4.10)		(−3.02, 8.31)	(−10.24, 4.93)		(−5.59, 12.95)

Post-famine cohort × CSSI, female	3.31		3.13	8.02*		9.06*
	(−0.74, 7.35)		(−1.14, 7.39)	(1.43, 14.60)		(1.71, 16.41)
Pre-famine cohort, male		1.73*	1.48		1.22	1.53
		(0.08, 3.39)	(−0.95, 3.90)		(−1.85, 4.30)	(−3.46, 6.53)
Famine cohort, male		3.27***	2.75*		3.51*	3.49
		(1.54, 5.00)	(0.55, 4.94)		(0.49, 6.53)	(−0.72, 7.71)
Post-famine cohort, male		0.55	0.64		0.85	−2.12
		(−1.22, 2.32)	(−1.44, 2.71)		(−2.43, 4.12)	(−6.51, 2.27)
CSSI, male		−0.82	21.79***		−1.33	2.15
		(−3.77, 2.13)	(12.61, 30.96)		(−6.77, 4.12)	(−16.32, 20.62)
Pre-famine cohort × CSSI, male		−3.08	−3.76		−1.41	−1.25
		(−7.19, 1.03)	(−10.02, 2.51)		(−9.19, 6.36)	(−13.93, 11.43)
Famine cohort × CSSI, male		−8.27***	−7.82**		−9.83*	−9.67†
		(−12.73, −3.81)	(−13.46, −2.18)		(−17.68, −1.97)	(−20.46, 1.13)
Post-famine cohort × CSSI, male		−2.46	−3.28		−2.53	4.77
		(−6.75, 1.83)	(−8.33, 1.76)		(−10.56, 5.49)	(−5.61, 15.14)
N	2,326	2,234	1,644	906	907	662

Notes: Columns 1 and 4 focus on an indicator for mothers' (first generation (G1) females') exposure to the great famine; columns 2 and 5 are an indicator for fathers' (G1 males') exposure; and columns 3 and 6 consider both mothers' (G1 females') and fathers' (G1 males') exposure. The 95 per cent confidence intervals are in parentheses. The values not in parentheses represent coefficient. CSSI: cohort size shrinkage index.
Significance levels: † $p < 0.1$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Human studies have also revealed some sex differences, and sex-specific effects may represent the differential sensitivity of males and females to programming phenomenon. Stronger programming in females may further amplify the matrilineal pattern of intergenerational inheritance (Drake and Walker, 2004). Using the same model, we also identify the intergenerational effect on education and income by gender (*see* Table S1-1 in the online supplementary material). We conclude that there is no difference in education by gender for those individuals whose parents experienced the great famine. However, the interaction between being male, mother's famine and CSSI is significant for income; the effect of the mother's famine is more pronounced for males than females. That is, the role of mother's environmental exposure on income is stronger for men compared to women.

Health effects of the second generation

We consider four broad measures of general health in adulthood: height, weight, BMI and birth weight (Table 3). A similar pattern emerges across all panels. Like economic outcomes in Table 2, for adult height, G1 male *in utero* exposure to the famine is shown to have negative and statistically significant ($p < 0.05$) association with height outcomes in the second generation, reducing height. Pre-famine and post-famine exposure also have a negative association with height but not significantly. G1 female pre-famine exposure is associated with an increase in height ($p < 0.10$); however, famine and post-famine G1 female exposure do not alter the height in the second generation. Panels B and C consider the effect on second-generation weight and BMI, respectively. From columns 1–3, either parent having been born either pre- or post-famine does not affect the weight or BMI in the second generation, which means these two phenotypes are more susceptible to the nurture effect. Panel D identifies the effect on birth weight of the second generation and finds that the mother's pre-famine exposure is associated with a statistically significant increase in the offspring's birth weight.

Again, using the same model, we also identify the intergenerational effect on health outcomes by gender (*see* Table S1-2 in the online supplementary material). As shown, many of the gender differences in G2 are not statistically significant. We conclude that there is no difference in height and birth weight by gender in those individuals whose parents experience the famine. However, the interactions between being male, mother's post-famine/father's famine exposure, and CSSI are significant for weight and BMI; the effects of post-famine exposure are more pronounced for females than males. Potential reasons for this sex disparity could be tied to general trends in the differences between sons and daughters in intergenerational mobility (Chadwick and Solon, 2002). Another potential reason for the sex difference could be the larger standard deviation in G2 male outcomes.

In sum, first-generation *in utero* famine exposure consistently has a statistically significant and economically meaningful effect on second-generation health and economic outcomes. This effect appears to be driven solely by the father's exposure. Our evidence does not support the pre-famine exposure effect and homogeneous sex effect.

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Table 3. Second-generation health effects

	(1)	(2)	(3)
Panel A: Height (cm):			
Pre-famine cohort × CSSI, female	4.61†		2.47
	(−0.73, 9.94)		(−7.31, 12.25)
Famine cohort × CSSI, female	3.12		5.00
	(−2.66, 8.89)		(−2.67, 12.68)
Post-famine cohort × CSSI, female	3.90		3.53
	(−1.63, 9.43)		(−2.90, 9.96)
Pre-famine cohort × CSSI, male		−0.97	−5.58
		(−6.86, 4.92)	(−14.66, 3.49)
Famine cohort × CSSI, male		−5.30†	−8.33*
		(−11.25, 0.65)	(−16.43, −0.22)
Post-famine cohort × CSSI, male		−2.28	−5.03
		(−8.49, 3.93)	(−13.11, 3.06)
N	2,324	2,232	1,643
Panel B: Weight (catty):			
Pre-famine cohort × CSSI, female	9.11		−2.72
	(−12.90, 31.11)		(−41.19, 35.75)
Famine cohort × CSSI, female	17.52		22.14
	(−5.85, 40.89)		(−9.68, 53.97)
Post-famine cohort × CSSI, female	9.93		11.12
	(−12.80, 32.67)		(−14.85, 37.10)
Pre-famine cohort × CSSI, male		−1.35	−2.54
		(−24.73, 22.03)	(−38.87, 33.79)
Famine cohort × CSSI, male		13.63	12.83
		(−10.97, 38.22)	(−21.00, 46.66)
Post-famine cohort × CSSI, male		7.47	0.65
		(−17.37, 32.31)	(−30.90, 32.20)
N	2,171	2,075	1,537
Panel C: BMI:			
Pre-famine cohort × CSSI, female	0.53		−0.33
	(−2.87, 3.93)		(−6.08, 5.42)
Famine cohort × CSSI, female	2.01		2.64
	(−1.74, 5.77)		(−2.43, 7.72)
Post-famine cohort × CSSI, female	0.29		1.07

(*Continued*)

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Table 3. (*Continued.*)

	(1)	(2)	(3)
	(−3.45, 4.03)		(−3.11, 5.25)
Pre-famine cohort × CSSI, male		−0.31	0.34
		(−3.97, 3.36)	(−5.30, 5.99)
Famine cohort × CSSI, male		2.97	3.25
		(−1.01, 6.96)	(−1.96, 8.45)
Post-famine cohort × CSSI, male		1.46	0.75
		(−2.70, 5.62)	(−4.26, 5.76)
N	2,169	2,073	1,536
Panel D: Birth weight (catty):			
Pre-famine cohort × CSSI, female	1.33†		0.10
	(−0.15, 2.81)		(−2.19, 2.38)
Famine cohort × CSSI, female	0.13		−0.71
	(−1.49, 1.76)		(−2.83, 1.40)
Post-famine cohort × CSSI, female	−0.41		−0.95
	(−1.90, 1.08)		(−2.72, 0.81)
Pre-famine cohort × CSSI, male		−0.10	0.94
		(−1.80, 1.59)	(−1.74, 3.61)
Famine cohort × CSSI, male		−1.15	−0.43
		(−2.83, 0.53)	(−2.85, 1.99)
Post-famine cohort × CSSI, male		−1.12	−0.15
		(−2.87, 0.62)	(−2.69, 2.40)
N	1,071	1,048	779

Notes: Column 1 focuses on an indicator for mothers' (first generation (G1) females') exposure to the great famine; column 2 is an indicator for fathers' (G1 males') exposure; and column 3 consider both mothers' (G1 females') and fathers' (G1 males') exposure. Models adjust for second-generation (G2) gender, G2 birth year, G2 birth year × G2 birth year, G2 number of siblings, pre-famine cohort, famine cohort, post-famine cohort and cohort size shrinkage index (CSSI). The 95 per cent confidence intervals are in parentheses. The values not in parentheses represent coefficient. cm: centimetres. BMI: body mass index.
Significance levels: † $p < 0.1$, * $p < 0.05$.

Discussion

This study presents novel evidence of the intergenerational effect of *in utero* and early-life health insults. That is, we use the sudden and unexpected Chinese famine pandemic in 1959–1961 to trace out the effects of *in utero* and early exposure on future generations.

First, we provide evidence that environmental exposure changes experienced *in utero* can also last through subsequent generations. Individuals in our second generation – and their siblings who have fathers exposed to the 1959–1961 famine *in utero* – complete less schooling, have less income and are shorter. The result

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aligns well with those of Richter and Robling (2015), who suggest that the probability of college attendance drops by 7–11 percentage points if their fathers are potentially prenatally exposed. Cook *et al.* (2019) also find that having either parent born during the 1918 influenza pandemic is associated with a reduction of roughly 0.1 inches in height; however, this effect seems to be driven by the mother's exposure rather than the father's. Periods of nutritional restriction while *in utero* can have intergenerational consequences in human populations, and postnatal growth appears to be under patriline intergenerational influence. Kaati *et al.* (2002) study health outcomes among descendants born in 1890, 1905 and 1920. They find that food abundance during the grandfather's (but not grandmother's) slow-growth period is associated with an increase in diabetes mortality and that male descendants have a statistically increased relative risk of mortality (Pembrey *et al.*, 2006). Subsequently, we extend results to the third generation for the first time in the literature and find that individuals in the third generation still have disadvantages in health, despite reduced statistical power (*see* the online supplementary materials). Further, we find no homogeneous sex effect of transmission of the health shock.

Second, the findings do not support the intergenerational effect of early childhood health shock. We find that the pre-famine and post-famine exposure have no effect on educational attainment or related economic and health outcomes across two and three generations. As previously outlined, the intergenerational persistence in poor outcomes is due to the *in utero* channels, not the early health shock channels. Intrauterine exposure could lead to changes in gene expression via changes in methylation and be transmitted to the next generations. Intergenerational transmission may occur through epigenetic inheritance through the germline, a distinct possibility based on findings of the Överkalix studies, or phenotype-to-phenotype transmission and cumulative intergenerational phenotypic change.

Third, we conclude that evidence on the intergenerational effects hypothesis is sensitive to the choice of measurements. Compared with other health measurements, height is the indicator which is more influenced by parents. Our work is consistent with a previous study in a Gambian population. Eriksen *et al.* (2017) investigate the association of parental exposure to energy and nutrient restriction *in utero* on their children's growth in rural Gambia. They find that the timing of paternal birth in relation to nutritional stressors predicts offspring height-for-age *z* score at 24 months of age with or without controlling for possible confounders (including parental height). Further, no evidence for an association between paternal season of birth and offspring birth size, weight-for-height *z* score and weight-for-age *z* score is found.

Several study limitations are noteworthy. First, like many other famine studies, we do not have sufficient data to measure individual-level or family-level prenatal exposure accurately. The actual incidence of environmental exposure likely differs by SES and social standing. Using ecological measures of famine severity at the province level is a reasonable alternative; however, it may conceal important individual heterogeneity within the same region. This estimation strategy will underestimate the true effect of exposure, lowering the magnitude of our coefficient of interest. Second, we have difficulty demarcating the timing of *in utero* famine exposure due to the lack of reliable and accurate vital statistics for this period (Susser and Clair, 2013). Specifically, information about the importance and differential impact of famine exposure timing during gestation is still limited, which

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means the monthly temporal path of *in utero* exposure to the event based on the distinct outcomes of intrauterine growth and gestational age cannot be examined. Third, an issue that must be carefully considered in any study of *in utero* health shock is selective fertility. Using the famine pandemic as the quasi-random shock of interest, it is reasonable to assume that its unexpected nature makes selective fertility related to the famine event unlikely. Survival selection may also occur during foetal or early exposure. The foetus may be prone to miscarriage or stillbirth, and infants already born may be prone to death due to malnutrition in the famine. Infants who are healthier or have better genetic endowment are more likely to survive the famine. That is, conditions will lead to the survival of a strong foetus or baby at birth, while a weak foetus or baby at birth is less likely to survive. Therefore, survival selection has a positive effect on those who experience famine and has both a negative, intergenerational 'scarring effect' on the offspring. Finally, our analysis is unable to fully distinguish between these socio-economic and biological proposed intergenerational channels. We intend to account partially for the socio-economic mechanism by controlling for the first-generation's economic outcome (*see* the online supplementary material). However, little economic information about the first generation is found in the sample.

Despite these limitations, the results of this study offer information that may influence public policy. As the first study to examine the intergenerational and multigenerational effects of the great famine in China in 1959–1961, distinguishing the different effects of *in utero* and early-life exposure to the famine, our study adds new empirical evidence to the controversial literature on the foetal origin's hypothesis. Our findings suggest the importance of *in utero* health insults that persist across multiple generations and allow a shift in our analytical frame from the long arm of childhood circumstances to that of previous generation's circumstances – or, alternatively, the long reach of history (Kuzawa and Eisenberg, 2014). The famine of 1959–1961 in China resulted in over 23 million deaths, 30 million lost or postponed births (Song *et al*., 2009), and a large undernourished population. Besides, the unprecedented catastrophe of this famine also dramatically damaged economic as well as health outcomes for the next generation. Our results can also be interpreted in the context of assessing the benefits of making policy decisions that support environmental conditions to reduce the likelihood of *in utero* health insults, where the full benefits may unfold over multiple generations. Furthermore, our evidence may point to a need to consider evidence of multigenerational persistence in health and economic poverty through biosocial factors. Our theoretical framework and method could be expanded to study the multigenerational effects of other events, like the 1918 influenza pandemic, the Dutch famine, the unexpected terrorist attacks of 11 September 2001, the COVID-19 pandemic, *etc*. Considering the current COVID-19 global pandemic, it is particularly important that governments support their populations and prevent health effects that may last for generations.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0144686X22000113

Conflict of interest. The authors declare no conflicts of interest.

Ethical standards. Ethical approval was not required.

Note

1 It is important to distinguish the transient effects induced by the initial trigger from the truly transgenerational effects. When the G0 gestational female is exposed to environmental factors, the G1 embryo is directly exposed. Therefore, phenotypes from G0 and G1 may result from direct environmental exposure, and only the G2 and later generations can be considered as displaying truly transgenerational effects.

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