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Exposure *in utero* to Adverse Events and Health Late-in-life: Evidence from China*

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Abstract

This paper estimates the effect of *in utero* exposure to adverse events on late life diabetes, cardiovascular disease risks and cognition deficiency. We merge data on the regional violence during the Cultural Revolution and the excessive death rates during the Chinese Great Famine with data from the China Health and Retirement Longitudinal Study (CHARLS) survey. Results show that female babies who were exposed *in utero* to the famine have higher diabetes risks, while male babies who were exposed to the Cultural Revolution are shown to have lower cognitive abilities.

Keywords: Early life conditions, Chinese Great Famine, Cultural Revolution, Diabetes, Cardiovascular Disease, Cognition

JEL Codes: I10, J11, J14

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1 Introduction

A growing body of literature has shown that exposure to adverse conditions around birth can have long-term negative consequences on health. The long shadow of early-life circumstances can be explained by both biological and social mechanisms. According to the "Fetal Origin Hypothesis" by Barker (see, e.g., Barker et al. 1993, Barker 1995, Almond & Currie 2011), malnutrition during pregnancy might cause disproportional fetal growth and program later coronary heart disease and type 2 diabetes (see, e.g., Lumey, Stein & Susser 2011, Portrait et al. 2011, Xu et al. 2017, Kim et al. 2017, Hu et al. 2017, Dinkelman 2017, Doblhammer, van den Berg & Lumey 2013, Van den Berg et al. 2015). Moreover, prenatal stress might also be responsible for developing diseases such as depression and cognitive deficiency in the longer run (see, e.g., Akbulut-Yuksel 2017, Gade & Wenger 2011, Strauss et al. 2011, Bramsen et al. 2007, Grimard & Laszlo 2014, Teerawichitchainan & Korinek 2012, Islam et al. 2017). The pathway framework (Kuh & Shlomo 2004), on the other hand, rests less on the biological imprinting and states that the impact of early life conditions on health later in life depends also on the interaction of the individual with the environment. The idea is that adverse conditions around birth might set in motion lifetime trajectories of health related disadvantages. For example, children born under adverse circumstances might be exposed to poor diets, smoking, worse educational and job opportunities, and all these factors in turn will have a negative impact on health. In this paper we estimate the effect of *in utero* exposure to the Chinese Great Famine (1959-1961) and the Cultural Revolution (mainly the Red Guard Army movement 1966-1971) on a range of physical and mental health outcomes in later life. While the famine was responsible for severe malnutrition for pregnant mothers living in rural areas, the Cultural Revolution caused a sharp increase in violence and stress in urban areas. Therefore, it is interesting to look at both events. We draw data from the China Health and Retirement Longitudinal Study (CHARLS) survey, a nationally representative survey of the Chinese population aged 45 or more. The 2011 and 2015 waves of the survey include data from blood biomarkers, which allow us to construct an 8-year risk score for diabetes and a 10-year risk score for cardiovascular disease (CVD). Indeed some *in utero* effects might only manifest themselves later in life but early signals of disease might be detected at younger ages using blood-based biomarkers. For mental health, the data include standard survey scales to measure the presence of depressive symptoms and assess cognitive abilities.

Our results support the idea that adverse events around birth potentially have a long-lasting negative effect on both physical and mental health. We also find that there are gender differences in this effect: exposure *in utero* to the famine heightens the risk of type 2 diabetes for women,

while it increases depressive symptoms for men. On the other hand, the Cultural Revolution mainly harmed the cognitive abilities of men.

The rest of the paper is organized as follows: section 2 summarizes the epidemiological and biomedical evidence. Section 3 briefly explains the background of the Chinese Great Famine and the Cultural Revolution. Section 4 introduces the data the construction details of the main variables. Section 5 describes the empirical model and section 6 presents the results. Finally, section 7 concludes the paper.

2 Biological Mechanisms

In this section we summarize several commonly accepted biological mechanisms through which the *in utero* exposure to adverse events can be translated into late life diseases. Based on the literature, there are two important mechanisms: malnutrition and prenatal stress. Biomedical studies show that exposure to prenatal stress harms individuals' cortex development and it further induces depression and cognition deficiency, whereas exposure to malnutrition is mostly associated with later life cardiovascular disease or diabetes.

2.1 Stress and Cognitive Deficiency

In recent studies, prenatal stress is found to have a significant impact on the babies *in utero*. The hypothalamic-pituitary-adrenal (HPA) axis plays a crucial role in understanding the mechanism[1], especially during early fetal development because plasticity is very high (see, Braithwaite et al. 2014). Basically, the HPA axis is a neuroendocrine system that regulates the body's response to stress. Higher prenatal stress implies more responses or reactions from the HPA axis, which in turn affects the brain formation of babies[2].

The detailed processes are as follows (see, e.g., Molenaar et al. 2019, Bock et al. 2015). When pregnant mothers experience unexpected adverse events or a stressful environment, the hypothalamus (the H of HPA) in the brain area releases excessively a type of hormone called corticotropin-releasing hormone (CRH). The CRH then travels to the pituitary gland (the P of HPA) which responds by releasing more adrenocorticotropic hormone (ACTH). The ACTH then travels across human bodies and reaches the adrenal glands (the A of HPA) on the top of the kidneys. The adrenal glands are stimulated by the increase of ACTH and release more cortisol into the bloodstream. Normally the HPA axis has a feedback system that balances the hormone

[1]There are also other mediation pathways to explain the association, but the one with HPA axis is commonly accepted.

[2]Various biological literature tries to find biomarkers to quantify the activity of the HPA axis and the prenatal stress, such as hair cortisone levels (see, e.g., Molenaar et al. 2019) and dehydroepiandrosterone (DHEA) and dehydroepiandrosteronesulfate (DHEAS) (see, e.g., Schmelter et al. 2019).

levels. When the stress level becomes too high, however, the "negative feedback" hits the HPA axis and the sense receptor in the hippocampus will shut down the stress response mechanism. Finally, the cortisol level becomes out of control.

The elevated cortisol level then affects the foetus. Normally the placenta acts as a barrier between the concentrated cortisol level and the foetus. However, when the cortisol density around the placenta becomes too high, it will penetrate the placenta and the foetus becomes exposed to this excessive cortisol. Recent epigenetic literature explains the mechanisms after the foetus is exposed to excessive cortisol. It has been shown that the gene expression and the DNA-methylation processes of babies are altered to adapt to the changing intrauterine environment. The methylation process of the genes such as *Nr3c1*, *CRH*, *CRHBP*, *Crb*,and *Nr3c1* F1 promoter (see, e.g., Welberg & Seckl 2001, Hamada & Matthews 2019, McGowan & Matthews 2017)) would be altered. For instance, the *Nr3c1* gene which controls the glucocorticoids receptor production and controls the HPA axis reactivity of babies. The altered HPA axis activities then affect the shaping of the cortex and hippo-campus which reflects cognitive abilities late in life.

2.2 Malnutrition and Type-2 Diabetes and CVD Risks

Babies who experience malnutrition *in utero* are shown to have higher risks of type-2 diabetes (see, e.g., Vaiserman & Lushchak 2019) and higher risks of CVD (see, e.g., Alessie et al. 2019). The literature finds that maternal malnutrition creates intrauterine growth restriction, which then produces foetal adipose tissue and pancreatic β-cell dysfunction for the foetuses. To be more specific, the β-cell dysfunction is essential to explain the mechanisms behind the association. The β-cell is made from the stem cells when the pancreas of the embryo is developing, and it is the basis for the islets of Langerhans (the home of hormone). The decreased β-cell in reaction to the environmental cues will lead to irreversibly reduced insulin secretion. That is why babies exposed to malnutrition will develop higher diabetes and CVD risks.

The detailed processes are as follows: when pregnant mothers and their embryos experience malnutrition, the "thrifty genotype" starts to play a role (see, Hales & Barker 1992). Epigenetic evidence (mostly from animal models) shows that, with reduced nutrition supply especially amino acids, the DNA-methylation processes for genes such as *IGF2*, *GNASAS*, *IL10*, *LEP*, *ABCA1*, *INSIGF* and *MEG3* will be altered (see, e.g., Lumey, Terry, Delgado-Cruzata, Liao, Wang, Susser, McKeague & Santella 2011, Heijmans et al. 2008, Tobi et al. 2009). For example, one of the most famous epigenetic pieces of evidence by Heijmans et al. (2008) shows that, when the expression of the gene insulin-like growth factor-2 (IGF2) is reduced, the β-cell transformation from the stem cells is suppressed. Then the body becomes thriftier in keeping glucose and

releasing less insulin, and the risk of having type 2 diabetes increase once the nutrition supply after birth is improved. The babies finally have lower insulin secretion and a higher ability to store fat (see, e.g., Gluckman & Hanson 2004).

The risks of having CVD also increase because of two reasons: first, since type 2 diabetes can also cause (or caused by) CVD, the risks for having CVD also increase. Second, the lack of amino acids leads to the decrease of β-cells, while the lack of glucose and oxygen will directly lead to a reprogramming of the neuro-endocrine system (see, e.g., Almond & Mazumder 2011), similar to the HPA axis reaction due to stress. Then the CVD risks will be developed along with the type-2 diabetes risk.

The mechanisms summarized above are the most commonly accepted results. Some other research also finds that malnutrition could affect cognition (see, e.g., de Groot et al. 2011, Doblhammer, Van den Berg & Fritze 2013), *vice versa* for the literature studying prenatal stress (see, e.g., Akbulut-Yuksel 2017). These studies are mostly from the epidemiology literature and the findings might suggest that cognition deficiency is the complication of diabetes and CVD, *vice versa*.

2.3 Role of Gender

In the literature the evidence on the sex-specific health effects of prenatal exposure to adverse events is mixed (Alessie et al. 2019). For example, in terms of exposure to prenatal stress, some studies reveal that it is mostly male offspring's emotionality to be negatively affected, while others find the opposite effect, i.e. that female offspring's anxiety and depressive symptoms respond significantly to the adverse environment (see, e.g., Bock et al. 2015, for a review of both animal and human studies). A strand of research finds that the gender-specific effects in response to stress can be explained by the timing of stress exposure (see, e.g., Mueller & Bale 2008). Male babies are more responsive to prenatal stress during early gestational periods while female babies are more responsive during late gestational periods. In terms of exposure to malnutrition, there is also some evidence showing that males are affected differently than females. For example, Eriksson et al. (2010) find that boys grow faster than girls *in utero*, therefore boys need more nutrition from mothers. If mothers experience malnutrition during pregnancy, boys *in utero* will more likely be affected. Moreover, boys *in utero* tend to develop their brains first. If exposed to undernutrition, boys' visceral development will be sacrificed to sustain the nutrition supply for their brains. In the long run, boys' kidney development might be hurt, and some diseases such as hypertension and CVD will incur due to the underdevelopment of their kidney functions (see, e.g., Barker et al. 2006). In this paper, we will also empirically investigate the

gender-specific health effects of exposure to the CR and the famine to see whether males and females are affected differently.

3 The Chinese Great Famine and Cultural Revolution

3.1 The Chinese Great Famine

The Chinese Great Famine (CGF thereafter) is so far one of the largest famines in human history in terms of both the severity and the size of the affected population. The famine occurred mainly between 1959 and 1961, when grain production substantially dropped. Given the fact that grain was the major source of food, the decline in grain production caused 16.5 to 45 million deaths during the famine period (see, e.g., Meng et al. 2015). In the year 1958, some regions had already experienced some grain production drops, but it had not yet become a nationwide disaster. After the famine, grain production increased monotonically until it reached the normal level.

The famine hit primarily the rural areas. Indeed, during the famine period, China was running a centrally planned economy, and rural households were only able to eat in local communal kitchens while urban households were able to consume normal food products within quotas depending on the household size. Moreover, rural households were not allowed to store food privately. So when the famine arrived, rural households were immediately hit by the food shortage shock in the communal kitchens. What was worse, the grain produced in rural areas was over-procured by the central government so it exacerbated the starvation of the rural households.

The famine not only produced substantial deaths nationwide but also had a long-term impact on the health outcomes of survivors. With little food available for rural pregnant mothers, the foetuses in the famine cohort were more likely to suffer from malnutrition than any other cohort. In addition, mothers could have also experienced stress which could have been passed on to the babies.

The annual province-level deaths rates are provided by Lin & Yang (2000) and are available upon request from the authors. During the famine years death rates are clearly higher than in other non-famine years. Based on this, we calculate the excessive death rates and employ them to measure the famine severity across provinces. We calculate the excessive death rates in 0.1% unit by subtracting the average annual deaths rates between 1956 and 1958 from those in the famine years (1959-1961). During non-famine years, the measure of severity equals zero.

3.2 The Cultural Revolution

The Cultural Revolution (CR thereafter) was a major adverse political event that happened between 1966 and 1976 (see, e.g., Walder & Su 2003) in China. It is often called the "lost ten years" in China. In this period, there were millions of victims who suffered from various types of political movements and prosecutions, and the negative impacts persist throughout the victims' whole life course. A more detailed description of the CR can be found in Bonnin (2006). The event itself can be divided into two sub-periods: the Red Guard Army movement between 1966 and 1971, and the rustication programme between 1971 and 1976. The Red Guard Army movement was accompanied by massive conflicts and associated victims, while the rustication movement produced very few conflicts and victims. The extant studies either employ the Red Guard Army movement to study the effect of violence (see, e.g., Wang 2019) or employ the Rustication movement to study the impact of interrupted education (see, e.g., Meng & Gregory 2002).

In this paper, we focus on the Red Guard Army Movement during the CR because we would like to focus on the effect of conflicts. During this period, students were encouraged by Mao to rebel against the central/local governments. Students worshipped him, followed his extreme-left ideology and created a lot of violence in this movement. In response, the governments also repressed those student activities and produced plenty of victims and deaths. Those massive conflicts, victims, injuries and deaths were documented by local annals and gazetteers (see, e.g., Walder 2014).

Pregnant mothers who experienced or witnessed the violence were more likely to suffer from prenatal stress (perhaps also malnutrition). This stress can be passed on to the foetuses. Mothers in different regions and in different years suffered from different levels of violence, and we make use of this geographical and temporal variation to identify the health effects.

More specifically, we employ the number of collective conflicts as the measure of violence intensity during the CR (Red Guard Army movement 1966-1971) years. The measure equals 0 during the non-CR years. In a sensitivity analysis, we also use an alternative measure based on the number of deaths due to these collective conflicts. A similar strategy is also employed by Bai (2014) and Wang (2019). In Figure 1, we show the severity of total violence during the CR years by prefecture.

[Place Figure 1 about here]

4 Data on Health Outcomes and Descriptive Statistics

We draw data from the China Health and Retirement Longitudinal Study (CHARLS) 2011 and 2015 surveys. The CHARLS survey is designed to be nationally representative, and it focuses only on the population aged 45 and above. It has one national study with three regular waves (wave 2011, 2013 and 2015) and a retrospective wave (wave 2014). In this paper, we use the 2011 and 2015 waves of the national sample because they are the only waves that include a biomarker section with blood tests. Biomarkers are powerful preclinical or pre-morbid signals for diseases such as diabetes or CVD and allow us to construct risk scores.

As the CHARLS data set does not provide suitable measures for the severity of the CGF and the CR, we merge it with two additional data sources. We use the province-level excessive death rates from Lin & Yang (2000) to measure the famine severity, and we use prefecture-level conflicts and deaths counts from Walder (2014) to measure the CR severity. We merge each individual in CHARLS with these two additional data sets using the information on the prefecture/province and the year at birth. In CHARLS we also have information on the *hukou* status at birth, a household registration system used in China that identifies a person as a resident in a rural or urban area.

4.1 Sample Selection

In the original data sets, there are 17,708 observations in the 2011 wave and 21,095 observations in the 2015 wave. Only 11,847 respondents in the 2011 wave and 13,420 respondents in the 2015 wave have participated in the blood-based biomarker sections[3]. When we merge the different sections of the questionnaire, we are left with 10,138 observations in wave 2011 and 13,280 observations in wave 2013. We then construct a cross-sectional data set by pooling the two waves, and we use the latest information for each individual in the pooled data set. In other words, if a respondent has been interviewed in both 2011 and 2015, we use the information in 2015, which results in a sample size of 16,674.

We restrict our sample to those who were born between 1950 and 1971, which leaves us with 10,959 observations. We choose this period for the following reasons: first, the period not only covers both the CGF (1959-1961) and the Red Guard Army movement of the CR (1966-1971) but also includes a relatively peaceful time (1950-1959, 1962-1965). Second, we do not want to include war periods such as the Second World War (1938-1945) and the civil war (1945-1949). After merging with the CR violence data and the CGF excessive death rates

[3]Not all individuals participated in the biomarker sections. In the non-blood biomarker section, the response rate is around 78.9%, while in the blood biomarker section, the response rate is around 65%.

data, the sample size reduces to 9,794. We also drop observations with missing values in *hukou* status (144 observations), cognitive tests (595 respondents), depression symptoms (687) and the blood-based biomarkers (483).

In the final sample, there are 7,885 respondents distributed in 254 prefectures in 29 provinces[4]. In this sample, there are 4,341 female and 3,544 male respondents. Of those 7,885 respondents, 7,232 individuals have rural *hukou* at birth and 653 individuals have non-rural *hukou* at birth. There are 3,668 individuals who were born between 1950 and 1958, 799 individuals born between 1959 and 1961, 1,758 individuals born between 1961 and 1965, and 1,660 individuals born between 1966 and 1971.

4.2 Health Outcomes

4.2.1 Diabetes and CVD

Different from the extant studies which employ mostly self-reported health measures, we use biomarker data to construct risk scores for developing diabetes and CVD. For type-2 diabetes we consider the 8-year risk score, which clinically can be served as an early monitor for diabetes prevalence after 8 years. We use various sources of information to construct the risk score from the Framingham Heart Study (FHS, see, Wilson et al. 2007): age, gender, BMI, blood pressure, blood High Density Level Cholesterol (HDL Cholesterol), Triglycerides, and fasting blood glucose. The detailed calculations can be found in Appendix A2. The risk score works appropriately for undiagnosed patients but works inappropriately for those who have been diagnosed with diabetes and are taking medications. Therefore, we assign a risk score of 1 to those who were diagnosed with diabetes but have normal blood sugar levels because they are taking medications, which we can identify from the CHARLS data. The distribution of the risk score can be found in Table 2. In our sample, on average, the risk of getting diabetes in 8 years is 32.5%, and 16.2% of the respondents have already been diagnosed with diabetes. The figures are comparable with other data sources or public reports. For example, Xu et al. (2013) report that nearly half of the Chinese adult population are prediabetic, and the 2015 Report on Nutrition and Chronic Disease in Chinese Residents reveals that the prevalence rate of diabetes among Chinese adults aged 40 is 9.7 percent (see, Burns & Liu 2017, chapter 6). We also disaggregate the numbers by cohort, gender and *hukou* status, and the statistics can be found in Table **??**. From Panel A of Table **??**, we find that normally the younger cohorts have lower diabetes risks except for the famine cohort (1959-1961). The eight-year diabetes risk for the famine cohort is 35.7%, which

[4]Provinces of Xizang, Chongqing (province-level municipality) and Hainan are not included in CHARLS.

is higher than that of both the pre- and post-famine cohorts (34.1% and 31.9%)[5], indicating that the famine might play a role in increasing the risk of getting diabetes. Panel B of the table shows that females have lower diabetes risk but higher prevalence rates.

For CVD, we follow D'agostino et al. (2008) and construct a 10-year risk score from the FHS. Other studies employ the Systematic COronary Risk Evaluation (hereinafter SCORE) (see, e.g., Conroy et al. 2003, Alessie et al. 2019) but we focus on the FHS because Selvarajah et al. (2014) shows that it performs better than SCORE for the Asian population. We refer the reader to Appendix A1 for details on the construction of the risk score. As for diabetes, we assign value one to the CVD risk score for respondents with a history of CVD events such as heart failure and stroke. The descriptive statistics can be found in Table 1. On average, 8.27% of the sample has already experienced a CVD event (stroke and heart failures), and the average CVD risk in the sample is 15.4%. The figure is close to external sources. For example, the reported prevalence rate of CVD in China is about 20% (one in five adults in China has a CVD)[6].

[Place Table 1 about here]

4.2.2 Cognition and Depression

We employ various measures to describe cognitive abilities and depressive symptoms of older Chinese individuals. The first measure of cognition, *episodic memory*, uses the word recalling questions in CHARLS (see, e.g., Lei et al. 2012). Respondents were requested to read ten Chinese words, and then they were asked to recall as many words as possible at two different moments in time. The first is just after reading all the words, and the second is after answering several other questions. We measure the numbers of correct words by taking the average of the two counts. The second measure of cognition is *mental intactness*, which is generated from ten questions of the Telephone Interview of Cognitive Status (TICS hereinafter), (see, e.g., Lei et al. 2012, Huang & Zhou 2013). This measure is constructed based on whether the respondent is able to subtract 7 from 100 and keep subtracting 7 from the result for a maximum of five times, and to name the correct date (day of the week, month, day, year, and season). The maximum score is ten. The third measure considers graphical cognitive ability, which is a dummy indicating whether the respondent is able to draw a picture shown by the interviewer.

For depressive symptoms, we employ the well-known CESD-10 measure (see, e.g., Björgvinsson et al. 2013). Respondents were asked to report how often they had experienced the following situations: being bothered by small things, having difficulty in keeping their mind on what they

[5]The difference between famine and post-famine cohorts are statistically significant.
[6]The statistics are provided by World Heart Federation: https://www.world-heart-federation.org.

were doing, feeling depressed, being tired of doing things, feeling hopeful, feeling fearful, feeling restless, feeling happy, feeling lonely, and feeling hard to get going. The answers were recorded on a four-point scale from 0 to 3, corresponding to rarely, some days, occasionally, and most of the time for the negative questions, and reversed for the positive ones. The total score ranges between 0 and 30, where a cutoff score of 10 or higher indicates clinically relevant depression.

In Panel A of Table **??**, we observe that older cohorts have on average lower cognitive abilities (episodic memory, mental intactness and graphical cognition), whereas depressive symptoms are more severe among younger respondents. Panel B shows that females have lower cognitive performance than males, and they have higher levels of depressive symptoms. Rural-born residents have more depressive symptoms and less cognitive abilities than their urban counterparts.

5 Empirical Framework

The model to be estimated is of the following form:

$$y_{igpt} = \alpha + \beta_1 \cdot EDR_{pt} \cdot Rural_{igp} + \beta_2 \cdot Vio_{gt} \cdot Urban_{igp} + X_{igp}\gamma + \lambda_t + \theta_g + \epsilon_{igpt}, \qquad (1)$$

where y_{igpt} is one of the health outcomes we have mentioned in Section 4. The subscript i refers to the individual, g denotes prefecture (254), p means province (29), and t stands for the year of birth. The variable excess death rate EDR_{pt} is the excess mortality rate in province p in the year in which the respondent was born t for the CGF years (1959-1961). It is computed by subtracting the average death rate between 1956 and 1958 from the annual death rate during the famine years. During the non-famine years, EDR_{pt} equals zero. As within a province there are both rural and urban areas but the CFG only affected rural areas, we interact the provincial excess death rate with a dummy $Rural_{igp}$, which equals one if the individual was born in a rural area (having a rural *hukou* at birth) and zero if the individual was born in an urban area (having an urban *hukou* at birth). As we only consider the *hukou* status at birth, this dummy is time-invariant. The variable violence Vio_{gt} is measured at the level of the prefecture g in year t during the CR and is the number of conflicts per thousand individuals during the CR years. It is equal to zero in the non-CR years. Since also within prefectures there are both rural and urban areas but the famine mainly hit rural areas, we interact Vio_{gt} with the dummy $Urban_{igp}$.

The covariate vector X_{igp} includes variables like gender and *hukou* status. The cross-cohort correlations are captured by the cohort fixed effect λ_t, and we also introduce the prefecture fixed effects θ_g to account for unobserved cross-regional heterogeneity. The rest of the unobserved

shocks are included in the idiosyncratic error term ϵ_{igpt}. To account for the correlation within each prefecture and each cohort, the standard errors are clustered at both the prefecture and the year of birth level.

6 Results

6.1 Main Results

We estimate model (1) for the full sample, which consists of all individuals who were born between 1950 and 1971, and the results can be found in Table 2. Column (1) exhibits the regression estimates using the predicted 8-year type-2 diabetes risk as the dependent variable. Column (2) shows the estimates using the predicted 10-year CVD risk. Column (3) displays the estimates using depression as the dependent variable which is measured by the CESD 10 scale. Columns (4-6) are the estimates for three cognition measures.

[Place Table 2 about here]

The results show that mothers' exposure to a 1% (the unit of the excess deaths rates in the raw data is 0.1%) more severe famine in rural areas during pregnancy would increase the risk of type-2 diabetes of the offspring by 0.03 percentage points (Column (1)) on average, while the effect on CVD risk is insignificant. These results suggest that exposure to famine might have triggered the "thrifty genotype" mechanism which induces insulin secretion during adult life. The magnitude of the effect is non-negligible because it is around 10.9% of the sample mean and around 10.4% of a standard deviation. This increase in the risk of developing diabetes might generate substantial healthcare costs and welfare losses for society. A 1% increase in famine severity leads to a 0.6 points increase in the depression scale, while there is no significant effect on cognitive abilities. Exposure to conflicts *in utero* has no significant long-run effects on any of the health outcomes.

6.2 Heterogeneous Effects by Gender

Some studies, such as Eriksson et al. (2010), suggest that boys might be affected differently than girls (see Section 2.3). To account for potential heterogeneous effects between males and females, we interact the famine severity and regional conflicts with gender. The results can be found in Table 3.

[Place Table 3 about here]

The results show that the effect of the famine on diabetes risk matters only for females, and a 1% increase in excessive death rate would imply a 0.06 percentage points higher risk of getting type-2 diabetes. The effect is around 17.5% of the sample mean and 16.7% of a standard deviation, which is statistically and economically relevant. The effects for males are statistically insignificant. The results might also be driven by selective mortality, namely males were more likely to die earlier. In terms of the impact on CVD risk, we also find insignificant results. The pattern for depression is different: a 1% increase in famine-induced deaths increases males' depression score by 0.8, suggesting that boys' emotionality is more affected by the adverse prenatal environment, especially prenatal stress.

6.3 Robustness Checks

We conduct two robustness checks to ensure that the effects are not spurious. First, since the number of conflicts might not capture the full damage during the CR, we use an alternative violence measure: the (log) number of deaths due to the collective conflicts. Second, since the results might be sensitive to the choice of the control group, in a sensitivity analysis we restrict the control group to those who were born after 1955 (the original sample includes all respondents who were born after 1949).

In Table 4 we report the results when we replace the measure of CR violence with the log of the number of deaths. Using an alternative measure does not substantially alter the effect of the famine on diabetes and CVD risk scores. The estimated effects of violence on cognition are less robust, although both results in Columns (4-6) of Table 2 and 3 show that cognition is negatively affected by violence exposure.

[Place Table 4 about here]

The results for the second robustness check are available from request from the authors. When we restrict the sample to those who were born after 1955, the estimates do not qualitatively change from the ones in Table 3. For instance, the estimated coefficient in Column (1) for the female-EDR-rural interaction term changes from 0.00566 to 0.00558. The effect of the male-EDR-rural interaction term in Column (3) does not change at all. The coefficient on the male-conflict-urban interaction term changes from -0.167 to -0.128. Overall, the results are robust to changes in the size of the control group.

7 Conclusion

We estimate the long-run impact of exposure *in utero* to the Chinese Great Famine and the Cultural Revolution on physical and mental health later in life. We merge data on the regional violence during the Cultural Revolution and the excessive death rates during the Chinese Great Famine with data from the China Health and Retirement Longitudinal Study (CHARLS) survey. While the Chinese Great Famine was responsible for severe malnutrition and a stressful environment for pregnant mothers in rural areas, the Cultural Revolution mainly affected pregnant mothers in urban areas.

The results show that *in utero* exposure to the famine increases the risk of getting type-2 diabetes later in life, and that the effect is more pronounced for females. A 1% increase in excessive death rates increases diabetes risk by around 0.06 percentage points for girls who were exposed to famine. Boys exposed to the famine are shown to have a higher tendency to develop depressive symptoms later in life, while exposure to the Cultural Revolution has a negative effect on their cognitive abilities. As in our data we only observe those who survived into adulthood, our results are likely to underestimate the negative effect of adverse events at birth on health later in life.

Overall, our results suggest that early life intervention is crucial. Future research should focus on disentangling the mechanisms through which adverse conditions around birth translate into poor health outcomes later in life, which is crucial to identify potential targets for prevention and intervention strategies.

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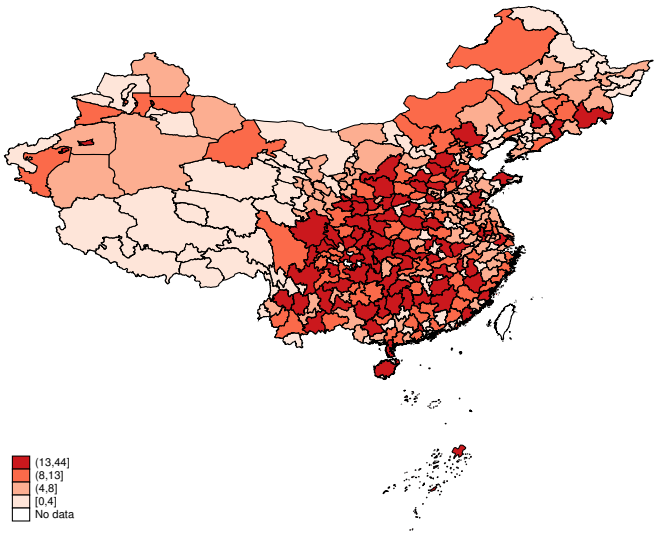
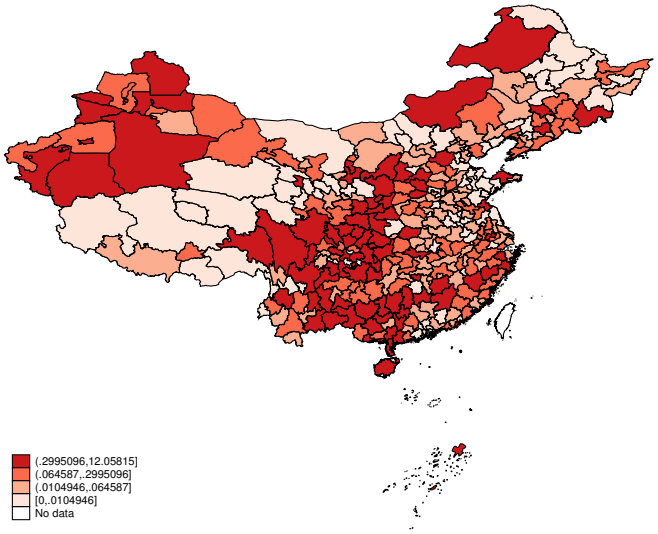
Tables and Figures in Main Texts



(a) Number of Conflicts* 1000 per capita



(b) Number of Deaths* 1000 per capita

Figure 1: **Collective Violence during the Cultural Revolution 1966-1971**

Table 1: Descriptive Statistics CHARLS 2011/15: Born during 1950-1971

VARIABLES	(1) Mean	(2) S.D.	(3) Min	(4) Max	(5) Quartile 1	(6) Median	(7) Quartile 3
			Sample size 7,885				
Female (0-1)	0.551	0.497	0	1	0	1	1
Rural Hukou (0-1)	0.917	0.276	0	1	1	1	1
Cognition & Depression							
Episodic Memory (Word Recall) (0-10)	3.699	1.765	0	10	2.500	4	5
Mental Intactness (TICS) (0-10)	6.815	2.754	0	10	5	7	9
Graphical Cognition (0-1)	0.694	0.461	0	1	0	1	1
CESD-10 (0-30)	7.792	6.295	0	30	3	6	11
CVD & Diabetes							
Diabetes	0.162	0.368	0	1	0	0	0
8-year Diabetes Risk	0.325	0.341	3.51e-05	1	0.0478	0.166	0.551
CVD	0.0827	0.275	0	1	0	0	0
10-year CVD Risk	0.154	0.266	0.00220	1	0.0249	0.0572	0.128

Note: Sample size 7,885 include all the respondents in wave 2011 and 2015, and we keep the latest information if the repondents appear twice. Rural *hukou* equals one if the repondents have the rural *hukou* at birth, zero otherwise. Episodic memory is constructed by asking the respondents recall the ten words listed on cards, ranging from 0 (no words recalled) to 10 (all words recalled). Mental intactness is measured through questions which ask the respondent to subtract 7 from 100 and keep subtracting 7 from the result for a maximum of five times and to name the correct date (day of the week, month, day, year, and season). The maximum score is 10. Graphical cognitive ability is measured by a dummy which is equal to 1 if the respondent is able to draw a picture showed by the interviewer. CESD-10 measures depressive symptoms, ranging from 0 (no depression at all) to 30 (the most serious depression). Diabetes is a dummy which equals one if the respondents have been diagnosed with diabetes and/or taking medications to control the blood sugar level. The 8-year risk score for diabetes is constructed by following Wilson et al. (2007). CVD is a dummy for actual cardiovascular disease and equals one if the respondents have been diagnosed with stroke or heart failures. The Cardiovascular Disease 10-year risk measure is from Framingham Heart Study (D'agostino et al. (2008)).

Table 2: Main Results: CHARLS 2011 and 2015

	(1)	(2)	(3)	(4)	(5)	(6)
	Diabetes	CVD	Depression	Cognition		
VARIABLES	Predicted 8-year Risk	Predicted 10-year Risk	CESD-10	Graphical Cognition	Mental Intactness (TICS)	Episodic Memory (Word Recall)
	%	%	Score 0-30(Worst)	0-1(Best)	0-10(Best)	0-10(Best)
EDR*Rural	0.00354**	0.000783	0.0597**	-0.000878	-0.0112	-0.00961
	(0.00170)	(0.00122)	(0.0291)	(0.00220)	(0.0123)	(0.00805)
Conflicts*Urban	-0.0108	-0.0358	-0.0410	-0.0644	0.369	0.0276
	(0.0372)	(0.0253)	(0.677)	(0.0437)	(0.255)	(0.210)
Rural Hukou	-0.0325	-0.0185	1.292***	-0.138***	-0.815***	-0.862***
	(0.0247)	(0.0157)	(0.353)	(0.0269)	(0.129)	(0.122)
Female	0.00872	-0.0443***	2.204***	-0.193***	-1.274***	-0.134**
	(0.0120)	(0.00781)	(0.158)	(0.0162)	(0.111)	(0.0541)
Constant	0.484**	0.216	0.808	1.053***	10.45***	5.689***
	(0.193)	(0.145)	(3.487)	(0.252)	(1.468)	(0.964)
Observations	7,885	7,885	7,885	7,885	7,885	7,885
R-squared	0.090	0.149	0.127	0.152	0.191	0.153
Interview Year Dummy	YES	YES	YES	YES	YES	YES
Year of Birth FE	YES	YES	YES	YES	YES	YES
Prefecture FE	YES	YES	YES	YES	YES	YES

Note:

1. *** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the prefecture and year of birth levels and reported in parentheses.

2. In Columns (1-2) we examine the impacts of violence (measured by log of number of collective conflicts) and famine (measured by excessive death rates) on the predicted 8-year diabetes risk and 10-year CVD risk.

3. Column (3) shows the results using CESD-10 as the measure of depression, and columns (4-6) use three measures of cognition: i) ability to draw a required picture, ii) mental intactness, and iii) memory ability using word recall questions (see the paper for details).

4. *EDR* is the province-level excessive death rates in 1959, 1960 and 1961, which is calculated by subtracting the average death rates between year 1956-1958 from the annual death rates in the three famine years. The variable *Conflicts* measures the level of violence in each of the years between 1966 and 1971. It is measured at the prefecture level per thousand population. The variable *Rural* is the indicator for rural *hukou* at birth.

Table 3: Gender Specific: CHARLS 2011 and 2015

	(1) Diabetes	(2) CVD	(3) Depression	(4)	(5) Cognition	(6)
VARIABLES	Predicted 8-year Risk	Predicted 10-year Risk	CESD-10	Graphical Cognition	Mental Intactness (TICS)	Episodic Memory (Word Recall)
	%	%	Score 0-30(Worst)	0-1(Best)	0-10(Best)	0-10(Best)
Male*EDR*Rural	0.000457	0.00138	0.0803**	0.00162	-0.0138	-0.00825
	(0.00222)	(0.00193)	(0.0402)	(0.00303)	(0.0172)	(0.0115)
Female*EDR*Rural	0.00566***	0.000376	0.0454	-0.00258	-0.00937	-0.0105
	(0.00210)	(0.00170)	(0.0348)	(0.00260)	(0.0147)	(0.00962)
Male*Conflicts*Urban	-0.00619	-0.0622	0.869	-0.167**	-0.421	-0.444
	(0.0531)	(0.0391)	(0.937)	(0.0764)	(0.395)	(0.283)
Female*Conflicts*Urban	-0.0138	-0.0189	-0.621	0.00115	0.871***	0.329
	(0.0438)	(0.0316)	(0.799)	(0.0547)	(0.319)	(0.272)
Rural Hukou	-0.0324	-0.0188	1.302***	-0.139***	-0.823***	-0.867***
	(0.0248)	(0.0158)	(0.356)	(0.0271)	(0.129)	(0.121)
Female	0.00638	-0.0442***	2.231***	-0.192***	-1.286***	-0.139**
	(0.0118)	(0.00810)	(0.163)	(0.0161)	(0.112)	(0.0542)
Constant	0.486**	0.216	0.783	1.052***	10.46***	5.692***
	(0.193)	(0.145)	(3.487)	(0.251)	(1.468)	(0.963)
Observations	7,885	7,885	7,885	7,885	7,885	7,885
R-squared	0.091	0.149	0.127	0.153	0.192	0.153
Interview Year Dummy	YES	YES	YES	YES	YES	YES
Year of Birth FE	YES	YES	YES	YES	YES	YES
Prefecture FE	YES	YES	YES	YES	YES	YES

Note:

1. *** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the prefecture and year of birth levels and reported in parentheses.

2. This table show the gender-specific estimates with the full sample.

3. In Columns (1-2) we examine the impacts of violence (measured by log of number of collective conflicts) and famine (measured by excessive death rates) on the predicted 8-year diabetes risk and 10-year CVD risk.

3. Column (3) shows the results using CESD-10 as the measure of depression, and columns (4-6) use three measures of cognition: i) ability to draw a required picture, ii) mental intactness, and iii) memory ability using word recall questions (see the paper for details).

4. *EDR* is the province-level excessive death rates in 1959, 1960 and 1961, which is calculated by subtracting the average death rates between year 1956-1958 from the annual death rates in the three famine years. The variable *Conflicts* measures the level of violence in each of the years between 1966 and 1971. It is measured at the prefecture level per thousand population. The variable *Rural* is the indicator for rural *hukou* at birth.

Table 4: Gender Specific: CHARLS 2011 and 2015 deaths

	(1) Diabetes	(2) CVD	(3) Depression	(4)	(5) Cognition	(6)
VARIABLES	Predicted 8-year Risk	Predicted 10-year Risk	CESD-10	Graphical Cognition	Mental Intactness (TICS)	Episodic Memory (Word Recall)
	%	%	Score 0-30(Worst)	0-1(Best)	0-10(Best)	0-10(Best)
Male*EDR*Rural	0.000443	0.00133	0.0807**	0.00163	-0.0135	-0.00790
	(0.00222)	(0.00194)	(0.0402)	(0.00303)	(0.0172)	(0.0115)
Female*EDR*Rural	0.00566***	0.000327	0.0458	-0.00262	-0.00934	-0.0104
	(0.00209)	(0.00169)	(0.0348)	(0.00260)	(0.0147)	(0.00962)
Male*Deaths*Urban	-0.891	-0.691	15.00	-2.562	-7.533	-7.455*
	(0.883)	(0.667)	(15.98)	(1.753)	(6.729)	(4.416)
Female*Deaths*Urban	0.390	0.880	-30.04*	-0.318	16.24**	-1.765
	(1.106)	(0.737)	(16.78)	(1.210)	(7.067)	(5.082)
Female	0.00601	-0.0442***	2.230***	-0.191***	-1.282***	-0.134**
	(0.0119)	(0.00808)	(0.163)	(0.0162)	(0.112)	(0.0544)
Rural Hukou	-0.0321	-0.0142	1.266***	-0.136***	-0.837***	-0.881***
	(0.0247)	(0.0157)	(0.352)	(0.0269)	(0.127)	(0.115)
Constant	0.486**	0.216	0.790	1.051***	10.45***	5.686***
	(0.193)	(0.145)	(3.486)	(0.251)	(1.468)	(0.963)
Observations	7,885	7,885	7,885	7,885	7,885	7,885
R-squared	0.091	0.149	0.128	0.153	0.192	0.153
Interview Year Dummy	YES	YES	YES	YES	YES	YES
Year of Birth FE	YES	YES	YES	YES	YES	YES
Prefecture FE	YES	YES	YES	YES	YES	YES

Note:

1. *** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the prefecture and year of birth levels and reported in parentheses.

2. This table shows the results using the log of number of deaths due to Cultural Revolution as the measure of violence.

3. In Columns (1-2) we examine the impacts of violence (measured by log of number of deaths due to collective conflicts) and famine (measured by excessive death rates) on the predicted 8-year diabetes risk and 10-year CVD risk.

4. Column (3) shows the results using CESD-10 as the measure of depression, and columns (4-6) use three measures of cognition: i) ability to draw a required picture, ii) mental intactness, and iii) memory ability using word recall questions (see the paper for details).

5. *EDR* is the province-level excessive death rates in 1959, 1960 and 1961, which is calculated by subtracting the average death rates between year 1956-1958 from the annual death rates in the three famine years. The variable *Deaths* measures the number of deaths due to CR violence in each year between 1966 and 1971. It is measured at the prefecture level per thousand population. The variable *Rural* is the indicator for rural *hukou* at birth.

A Appendices not intended for publication

A.1 Ten-year CVD Risk Calculation

The procedures for calculating the 10-year CVD risk can be found in the appendix of D'agostino et al. (2008). The study estimates a simple CVD risk prediction model and provides the regression coefficients of the model using data from the Framingham Heart Study. We reproduce the estimates in Table A1. The general formula for calculating the CVD risk $\hat{p}_{CVD}$ is

$$\hat{p}_{CVD} = 1 - S_0(10)^{\exp(\sum_{i=1}^{k} \hat{\beta}_i X_i - \sum_{i=1}^{k} \hat{\beta}_i \bar{X}_i)}, \tag{2}$$

where $S_0(10)$ is the baseline survival rate in 10 years. In this paper, we use the same baseline gender-specific survival rates as D'agostino et al. (2008), which is 0.95012 for females and 0.88936 for males. The covariate vector X_i includes the log of the i_{th} risk factor where i ranges from 1 to k. Vector $\bar{X}_i$ stands for the sample means of the covariates. The risk factors include age, total cholesterol, high density level cholesterol, a dummy for whether being undertreated for hypertension, a dummy for whether smoking or not, and a dummy for whether being diagnosed with diabetes or not. The regression coefficients $\hat{\beta}$ are provided in the Table 2 of D'agostino et al. (2008). For those who were diagnosed with CVD, we assign value 1 to the risk score.

Table A1: CVD Risk Factors, Table 2 of D'agostino et al. (2008)

Variable	$\hat{\beta}$	P Value
Women ($S_0(10) = 0.95012$)		
Log of age	2.32888	<.0001
Log of total cholesterol	1.20904	<.0001
Log of HDL cholesterol	-0.70833	<.0001
Log of SBP if not treated	2.76157	<.0001
Log of SBP if treated	2.82263	<.0001
Smoking	0.52873	<.0001
Diabetes	0.69154	<.0001
Men ($S_0(10) = 0.88936$)		
Log of age	3.06117	<.0001
Log of total cholesterol	1.12370	<.0001
Log of HDL cholesterol	-0.93263	<.0001
Log of SBP if not treated	1.93303	<.0001
Log of SBP if treated	1.99881	<.0001
Smoking	0.65451	<.0001
Diabetes	0.57367	<.0001

A.2 Eight-year Diabetes Risk Calculation

For the eight-year diabetes risk calculation, we use the prediction model in Table 4 of Wilson et al. (2007) which is reproduced in Table A2 . We adopt the prediction model using continuous variables. The general prediction model for the 8-year risk $\hat{p}_{Diabetes}$ is

$$\hat{p}_{Diabetes} = \frac{1}{1 + \exp(-\sum_{i=1}^{m} \hat{\gamma}_i X_i)}, \tag{3}$$

where the m risk factors include age, gender, parental history of diabetes mellitus, BMI, systolic blood pressure, HDL cholesterol level per mg/dL, triglyceride level per mg/dL, waist circumference in cm, and fasting glucose per ng/dL. Notably, because in the data set we do not have information on parental history of diabetes, we use the default value 0.17 suggested by Wilson et al. (2007) for all respondents. Similar to the calculation of CVD, we assign value 1 to the risk score if the respondents have been diagnosed with diabetes or are now taking the medications to control the blood sugar levels.

Table A2: Diabetes Risk Factors, Table 4 of Wilson et al. (2007)

Variable	OR (95% CI)	P Value
Age, y	0.99 (0.97-1.01)	.42
Male	0.65 (0.41-1.02)	.06
Parental history of diabetes mellitus	1.55 (1.01-2.38)	.04
BMI	1.04 (0.97-1.11)	.24
Systolic blood pressure, mm Hg	1.01 (1.00-1.02)	.11
HDL-C level per mg/dL	0.96 (0.95-0.98)	<.001
Triglyceride level per mg/dL	1.00 (1.00-1.00)	.16
Waist circumference, cm	1.05 (0.97-1.12)	.22
Fasting glucose level, mg/dL	1.15 (1.12-1.17)	<.001

Table A3: Provincial Excessive Death Rates (unit 0.1%) between 1955 and 1964

Province / year	1955	1956	1957	1958	1959	1960	1961	1962	1963	1964
Anhui	11.80	14.25	9.10	12.36	16.72	68.58	8.11	8.51	7.92	8.59
Fujian	8.26	8.43	9.02	7.50	7.95	15.61	12.18	8.27	7.51	8.68
Gansu	11.98	10.78	11.33	21.11	17.38	41.32	11.48	8.25	10.38	15.55
Guangdong	10.70	11.19	8.42	9.15	11.76	15.09	10.67	9.32	11.78	8.32
Guangxi	14.80	12.48	12.42	11.98	17.32	29.20	20.37	10.15	10.34	10.55
Guizhou	16.23	13.01	12.35	15.26	20.28	52.33	23.27	11.64	17.14	20.66
Hebei	11.86	11.26	11.59	10.92	12.31	12.19	13.34	8.97	10.66	10.48
Heilongjiang	11.33	10.08	10.45	9.17	12.76	10.52	11.12	8.61	8.56	11.47
Henan	11.75	14.00	11.80	12.69	14.10	39.65	10.10	8.03	9.39	10.65
Hubei	11.60	10.81	9.61	9.60	14.49	21.22	9.08	8.77	9.83	10.94
Hunan	16.41	11.50	10.35	11.58	12.92	29.26	17.48	10.23	10.26	12.88
Inner Mongolia	11.40	7.90	10.50	7.90	11.00	9.40	8.80	9.00	8.50	11.80
Jiangsu	11.65	12.81	10.03	9.33	14.55	18.41	13.35	10.36	9.03	10.13
Jiangxi	16.23	12.49	11.47	11.33	13.01	16.05	11.54	11.00	9.76	10.87
Jilin	9.91	7.53	9.05	9.11	13.43	10.13	11.12	9.96	9.44	12.62
Liaoning	9.40	6.60	9.40	8.80	11.80	11.50	17.50	8.50	7.90	9.30
Ningxia	N.A.	N.A.	N.A.	14.10	15.81	13.88	10.71	8.49	10.22	13.44
Qinghai	13.76	9.34	10.40	12.64	16.29	40.72	11.68	5.35	8.36	15.53
Shaanxi	10.55	9.85	10.31	11.04	12.76	12.27	8.76	9.35	10.55	15.60
Shandong	13.73	12.16	12.05	12.77	18.14	23.51	18.48	12.35	11.78	12.06
Shanxi	12.93	11.60	12.68	11.73	12.84	14.21	12.20	11.34	11.44	13.98
Sichuan	13.26	11.79	11.82	17.37	19.22	47.78	28.01	14.61	12.82	13.87
Yunnan	13.76	15.21	16.29	21.62	17.96	26.26	11.84	10.86	14.14	15.23
Zhejiang	12.58	9.46	9.32	9.15	10.81	11.88	9.84	8.61	7.89	9.21

Note: Date source comes from State Statistical Bureau and is summarised by Lin & Yang (2000).

Table A4: Robustness Check: Born after 1955

	(1)	(2)	(3)	(4)	(5)	(6)
	Diabetes	CVD	Depression	Cognition		
VARIABLES	Predicted 8-year Risk	Predicted 10-year Risk	CESD-10	Graphical Cognition	Mental Intactness (TICS)	Episodic Memory (Word Recall)
	%	%	Score 0-30(Worst)	0-1(Best)	0-10(Best)	0-10(Best)
Male*EDR*Rural	-0.000245	0.000773	0.0803**	0.000921	-0.0182	-0.00691
	(0.00223)	(0.00202)	(0.0401)	(0.00293)	(0.0168)	(0.0115)
Female*EDR*Rural	0.00558**	-0.00135	0.0429	-0.00414	-0.0204	-0.0136
	(0.00218)	(0.00147)	(0.0348)	(0.00271)	(0.0144)	(0.00976)
Male*Conflicts*Urban	-0.00514	-0.0385	0.815	-0.128*	-0.212	-0.367
	(0.0549)	(0.0365)	(0.932)	(0.0750)	(0.387)	(0.285)
Female*Conflicts*Urban	-0.0137	-0.00579	-0.705	0.0311	0.942***	0.435*
	(0.0449)	(0.0296)	(0.831)	(0.0541)	(0.314)	(0.253)
Female	-0.0133	-0.0270***	2.221***	-0.166***	-1.118***	-0.0744
	(0.0149)	(0.00696)	(0.170)	(0.0170)	(0.105)	(0.0604)
Rural Hukou	-0.0246	-0.00868	1.362***	-0.102***	-0.675***	-0.724***
	(0.0258)	(0.0173)	(0.425)	(0.0266)	(0.154)	(0.154)
Constant	0.290	0.119	-0.510	1.042***	10.23***	5.723***
	(0.233)	(0.164)	(4.183)	(0.299)	(1.736)	(1.173)
Observations	5,355	5,355	5,355	5,355	5,355	5,355
R-squared	0.100	0.144	0.136	0.134	0.188	0.143
Interview Year Dummy	YES	YES	YES	YES	YES	YES
Year of Birth FE	YES	YES	YES	YES	YES	YES
Prefecture FE	YES	YES	YES	YES	YES	YES

Note:

1. *** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the prefecture and year of birth levels and reported in parentheses.

2. This table shows the gender-specific effects using the subsample of people who were born after 1955.

3. In Columns (1-2) we examine the impact of violence (measured by log of number of collective conflicts) and famine (measured by excessive death rates) on the predicted 8-year diabetes risk and 10-year CVD risk.

4. Column (3) shows the results using CESD-10 as the measure of depression, and columns (4-6) use three measures of cognition: i) ability to draw a required picture, ii) mental intactness, and iii) memory ability using word recall questions (see the paper for details).

5. *EDR* is the province-level excessive death rates in 1959, 1960 and 1961, which is calculated by subtracting the average death rates between year 1956-1958 from the annual death rates in the three famine years. The variable *Conflicts* measure the level of violence in each of the years between 1966 and 1971. It is measured at the prefecture level per thousand population. The variable *Rural* is the indicator for rural *hukou* at birth.