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Identifying the intergenerational effects of the 1959–1961 Chinese Great Leap Forward Famine on infant mortality

Shige Song *

Queens College & CUNY Institute for Demographic Research, City University of New York, 65-30 Kissena Blvd., Queens, NY 11367, USA

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ABSTRACT

Using the 1959–1961 Chinese Great Leap Forward Famine as a natural experiment, this study examines the relationship between mothers' prenatal exposure to acute malnutrition and their children's infant mortality risk. According to the results, the effect of mothers' prenatal famine exposure status on children's infant mortality risk depends on the level of famine severity. In regions of low famine severity, mothers' prenatal famine exposure significantly reduces children's infant mortality, whereas in regions of high famine severity, such prenatal exposure increases children's infant mortality although the effect is not statistically significant. Such a curvilinear relationship between mothers' prenatal malnutrition status and their children's infant mortality risk is more complicated than the linear relationship predicted by the original fetal origins hypothesis but is consistent with the more recent developmental origins of health and disease theory.

1. Introduction

The 1959–1961 Great Leap Forward Famine (GLFF) in China was one of the most tragic events in modern human history. Based on commonly accepted estimates, the famine caused 30 million deaths and 33 million fertility losses during the three-year period (Ashton et al., 1984; Peng, 1987; Yao, 1999). Recent research in the fields of demography, economics, public health, and medical sciences suggests that the impact of the GLFF has extended beyond the three famine years. People born during the famine suffered from increased risk of obesity (Luo et al., 2006), schizophrenia (Song et al., 2009), metabolic syndrome (Li et al., 2011b), and hypertension (Li et al., 2011a; Huang et al., 2010b) as well as impaired fecundity (Song, 2013), reduced adult height (Chen and Zhou, 2007; Huang et al., 2010b), and poor labor market outcomes (Chen and Zhou, 2007), among others (Song, 2010; Mu and Zhang, 2011; Almond et al., 2010). A number of studies show that prenatal exposure to the GLFF may even have an "intergenerational" effect and influence the health and well-being of children of the famine cohort (Huang et al., 2010a; Kim et al., 2012; Fung and Ha, 2010).

The current study aims to contribute to this rapidly expanding literature by focusing on the relationship between mothers' prenatal exposure to the GLFF and the infant mortality risk of their children. Using data from the 2001 National Family Planning and Reproductive Health Survey, a large, nationally representative sample survey conducted in China, and data from the 1982 Chinese Population Census, I conducted simple cohort difference (SCD) analysis, difference-in-differences (DID) analysis, and conditional cohort difference (CCD) analysis to isolate the effects of developmental plasticity, development disruption, and selection simultaneously. To the best of my knowledge, no such effort has been made before.

The remainder of this article proceeds as follows. First, I briefly review the relevant literature that connects women's prenatal famine exposure to the infant mortality risk of their children, from the "fetal origins" hypothesis to life history regulation theory and the developmental origins of health and disease (DOHaD) framework. I then

* Tel.: +1 718 997 2822; fax: +1 718 997 2820.
E-mail addresses: shige.song@qc.cuny.edu, songsg@gmail.com.

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introduce the empirical context – the 1959–1961 GLFF in China – and describe the data, variables, and analytic strategy. Finally, I present the key statistical results and discuss their demographic significance and implications.

2. Conceptual framework

The following three distinctive types of long-term effects of prenatal famine exposure have been proposed in the literature: a developmental disruption effect, developmental plasticity effect, and selection effect. The disruption effect, also known as the "fetal origins" effect, "scarring effect", or "debilitation effect", states that prenatal famine exposure leads to an increase in disease risk (and thus a decrease in health status and life expectancy) (Razzaque et al., 1990; Chen and Zhou, 2007; Huang et al., 2010b). The possibility that prenatal famine exposure has a positive, or plasticity, effect on health has been mentioned only recently (Gluckman and Hanson, 2006; Gluckman et al., 2005; Painter et al., 2008), based on biological theory of developmental plasticity and life history regulation (Bateson et al., 2004; Stearns, 1992). A selection effect caused by differential population attrition may also "reduce" the disease risk of the famine cohort. Unlike the individual-level biological effects discussed above, however, the selection effect operates at the cohort-level and must be controlled for when estimating the individual-level biological effects.

2.1. Fetal origins, life history regulation, and developmental origins: a critical review

Barker and colleagues played a pivotal role in promoting the idea that prenatal conditions may have a long-lasting health consequences by articulating the "fetal origins" hypothesis and providing some early epidemiological evidence of an inverse relationship between birth weight and the risk of chronic disease later in life (Barker and Osmond, 1986; Barker, 1995, 1992). The notion that prenatal exposure to adverse conditions may have a long-lasting and nontrivial effect on adult health has been gradually accepted by health researchers as well as social scientists, especially economists (Almond and Currie, 2011; Doyle et al., 2009; Osmani and Sen, 2003). Critics of the fetal origins hypothesis claimed that the argument was too broad and vague to be rigorously tested and refuted. In addition, the lack of experimental results and the inherent weaknesses of the observational study design used in these studies made it difficult to make causal inference (Joseph et al., 1996; Paneth and Susser, 1995; Rasmussen, 2001; Tu et al., 2005; Lucas et al., 1999; Huxley et al., 2002). As a response to these criticisms, researchers have recently employed a famine-based natural experimental approach to isolate the causal effect of prenatal exposure to acute malnutrition on adult disease risk and to test the fetal origins hypothesis (Roseboom et al., 2001; Ravelli et al., 1998; Painter et al., 2005; Song et al., 2009; Chen and Zhou, 2007; Sotomayor, 2013; Almond and Mazumder, 2011).

Lumey and Stein (1997) tested the potential long-term effect of prenatal malnutrition on reproductive outcomes using the 1944–1945 Dutch famine as a natural experiment. Their results showed that prenatal famine exposure did not significantly affect women's age at menarche, the proportion of women without children, women's age at first delivery, or the number of children born. However, they found an excess of stillbirth and perinatal death among children of the famine-born women, providing some support to the fetal origins hypothesis. In a more recent study, Barker and colleagues reported that prenatal exposure to the 1944–1945 Dutch famine led to an *enhanced* female reproductive function, as evidenced by more children born, more twin births, earlier onset of childbirth, and lower likelihood of childlessness (Painter et al., 2008). The authors utilized developmental plasticity and life history regulation theories to explain these results: fertility and body maintenance are mutually balanced; if prenatal famine exposure led to a reduced survival opportunity (as predicted by the fetal origins hypothesis), an enhanced reproductive success was likely to be observed.

The DOHaD framework provides an opportunity to explain these inconsistent empirical findings. Gluckman et al. (2005) presented a schematic map of the nonlinear relationship between prenatal nutrition and adult fitness. They acknowledged the critical importance of the prenatal nutritional condition in determining health and reproductive success later in life and argued that both overly poor and overly rich nutritional conditions can negatively influence prenatal development. In contrast, it is the moderately low prenatal nutritional condition that is more likely to achieve optimal fitness. The key to this process is the predictive adaptive response mechanism of developmental plasticity during the prenatal stage, which predicts the future (postnatal) environment based on the current (prenatal) environment and then regulates the life history strategy of the unborn child to rebalance between survival and reproductive efforts based on such predictions (Gluckman et al., 2005; Gluckman and Hanson, 2006; Bateson et al., 2004). In the case of famine, prenatal malnutrition signals the unborn child that it is about to enter a resource-poor world in which a compromised survival (e.g., poor health, reduced longevity) may be unavoidable. In this situation, adopting a life history strategy that optimizes reproductive success, as Painter et al. (2008) suggested, may be evolutionarily advantageous. However, when the famine-induced malnutrition becomes too severe for the fetus to respond adaptively, normal prenatal development will be disrupted, which leads to pathological developmental outcomes that do not have adaptive values. This leads to the hypothesis that prenatal exposure to low level of famine-induced malnutrition may lead to improved reproductive outcomes, whereas exposure to severe malnutrition may lead to deteriorated outcomes.

2.2. Famine, natural experiment, and selection

From a study design point of view, famine-based natural experimental studies have important advantages over observational studies that use birth weight as a proxy for prenatal conditions. Birth weight is an imperfect measure of prenatal nutritional condition. It may be