

# Crisis and Human Biology<sup>\*</sup>

Prashant Bharadwaj  
UC San Diego, BREAD, and NBER

Tom Vogl  
Princeton University, BREAD, and NBER

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## Abstract

We review the literature on the effects of aggregate crises on human biological outcomes. The crises we consider are acute, severe, and unexpected negative events occurring at the population level: recessions, famines, epidemics, natural and environmental disasters, and wars. Our review of the literature suggests that the effects of aggregate crises on human biology are pervasive and long-lasting.

**Keywords:** Aggregate crises, famines, epidemics, wars, recessions, natural disasters, health, human capital, shocks

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# 1 Introduction

In modern parlance, the word “crisis” describes a pronounced shock, an adverse event, often economic or financial in nature. But, the word’s earliest appearances in English texts refer to human illness, with a “crisis” being the critical juncture in the progression of a disease that determines whether the sufferer recovers or dies.<sup>1</sup> The links between crisis and human biology extend well beyond this point, however, and they are the subject of a burgeoning literature in economics. How do the various manifestations of crisis—from deep recessions to pandemics to natural disasters—affect human health, reproduction, and child development, and how do changes in health endowments, incomes, prices, and behaviors mediate these effects?

This chapter reviews the empirical evidence on the effects of crisis on human biology, assessing the capacity of economic theory and methodology to illuminate this evidence. We define crises as acute (as opposed to chronic), severe, and unexpected negative events, helping us focus our discussion by circumscribing a subset of the vast literature linking a variety of acute shocks with human biology.<sup>2</sup> We focus on crises that affect entire populations at once, which are likely to have the gravest consequences. Crises strike at various scales, afflicting individuals, families, communities, or entire populations. But only those in the last category supersede the ability of markets and insurance systems to absorb some of the impact of their effects.

Our focus, then, is on events that most would already perceive as terrible and worth avoiding, even at considerable cost. This insight might lead some to wonder why these events merit further study at all. But government decisions regarding crisis prevention and mediation rely on information on the costs and benefits of such interventions. The evidence we review suggests that the effects of aggregate crises are both longer-lasting (felt over the lifecycle) and more extensive (involving nonobvious human biological outcomes) than many might appreciate. Thus, from a policy perspective, our review may lend additional support to arguments in favor of increased disaster preparedness or increased focus on post-disaster adaptation strategies. From a broader scientific perspective, however, the results cast in stark relief the way the human body reacts to a variety of grave insults.

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<sup>1</sup> In the Oxford English Dictionary, all quotations with the word before the year 1600 used this definition.

<sup>2</sup> See Ruhm (2006), Strauss and Thomas (2007), and Currie and Vogl (2013) for reviews.

We organize our discussion around five types of aggregate crises: recessions, famines, epidemics, natural and environmental disasters, and wars.<sup>3</sup> Taking a broad view of “human biology”—including health, child development, and reproduction—we detail the effects of each type of crisis on human biological outcomes. Whenever the literature allows, we discuss the roles of health endowments, incomes, prices, and behaviors in mediating the results, and we note how the results may vary between rich and poor settings. Notably, one type of crisis can lead to another crisis—for example, a disease epidemic can cause economic fluctuations, or a war can result in a famine. We note these possibilities as potential pathways where relevant, but due to space considerations, we focus on the impacts of the crisis that likely occurred first. In the same vein, we do not dwell on the specific pathways through which crises affect human biology. Little existing research sheds light on these mechanisms, so we point to them as a fruitful topic for future research.

## 2 Conceptual Issues

Before reviewing the evidence, we further develop our definitions of the two concepts at the center of this chapter: crisis and human biology. To begin, we set the selection criteria for the crises we study, discussing their theoretical underpinnings and implications. We describe how behavioral responses, price adjustments, and insurance arrangements may mitigate or exacerbate the effects of a crisis, with particular attention to how these mechanisms may play out at different levels of aggregation. The research we review does not necessarily shed direct light on these mechanisms, but we draw attention to them as a way to motivate our choice to focus on large-scale crises. After defining crisis, we move on to setting the parameters for the outcomes we consider.

Our definition of crisis narrows the scope of the relevant literature considerably. The *acute* criterion precludes us from focusing on serious, long-lasting problems that many label “crises,” such as what the editorial board of *The Lancet* (2013) calls the “global crisis of ... malnutrition in children.” The *severe* criterion leads us to direct our attention to studies of large shocks, rather than marginal changes in rainfall in agricultural areas, for example. And the *unexpected* criterion forces us to exclude important problems that unfold in a gradual and anticipated way, such as the

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<sup>3</sup> We omit major political crises, for example regime change (as in the Soviet Union) or political partition and reunification (as in the partition of British India or the reunification of Germany).

so-called “pension crisis.” Our restrictive definition does occasionally rule out relevant research that falls slightly outside its boundaries. In these cases, we allow ourselves to deviate. For example, we include research on the HIV/AIDS “crisis,” even if the pandemic is by now chronic and expected. We reason that it was once acute and unexpected, and that viewed through a broader lens, its history is still short.

That said, even with the three criteria, our definition includes a very wide range of negative shocks with a wide range of effects. Some crises affect individuals or families in isolation, for instance when a parent loses his job or dies. Others occur at the local level, such as when a plant closes or an agricultural community experiences a growing season with limited rain. And yet others strike entire populations or economies; these crises, which include famines and sudden epidemics, receive the most attention from policymakers and the media because they are so wide reaching. Crises at these three levels are likely to have differing effects on human biology, depending on the extent of risk-sharing, market integration, reserves, and safety nets. Aggregate crises are worse than the sum of many localized crises because they have stronger price effects and are more difficult to insure. If a single agricultural community has a bad crop year, then its members may suffer income losses. If an entire region has a bad crop year, however, these income losses may be compounded by increases in the price of food. More generally, the impact of a crisis is contingent on both the extent of *ex ante* insurance—whether through buffer-stock saving, formal insurance systems, informal risk-sharing, or government safety nets—and the extent of *ex post* behavioral responses (see Skoufias (2003) for more on coping strategies relevant to economic crises and natural disasters). For these reasons, we focus on aggregate crises. The scope of aggregation may vary—a state in one application, a country in the next—but we maintain a focus on crises broad enough to defy many risk mitigation strategies and price adjustment effects.

Apart from the characterization of crisis, our topic also requires us to define human biology. We take a broad view of the concept, including mortality, physical growth, physical morbidity, mental health, cognitive function, and fertility. These domains of human biology have considerable overlap in the literature. In some cases, the impact of crisis in one domain mediates its impact in another. In other cases, the impact in one domain complicates estimation of impacts in another. For example, impacts on mortality or fertility may introduce selection bias in estimates of impacts on morbidity or cognitive function among survivors. This concern arises in all re-

search concerning shocks with potential effects on population composition, but it is especially pertinent for the study of major health crises, such as famines or pandemics.

Much of the research we review follows how the human biology impact of a crisis spills over into social and economic outcomes. We include such analyses in our discussion only when we can confidently attribute the effects of a crisis on social and economic outcomes to a human biology pathway. For example, one can safely trace the human capital effects of disease exposure in utero to a human biology pathway, but the same link is not possible for the lifelong consequences of exposure to war in childhood.

### **3 Recessions**

Of all the crises we consider, recessions are the most natural for economists to study. Importantly, recessions typically have other root causes, some of which we consider in subsequent sections. But, a large literature considers the effects of recessions per se, perhaps because recessions with varying causes often share common features. This literature pays special attention to the interplay between income effects, owing to contractions in economic resources, and substitution effects, from reductions in the opportunity cost of time for example. Much of the literature estimates the relationship between continuous measures of aggregate economic conditions (such as growth rates or employment rates) and human biology, which bundles together booms, mild recessions, and deep recessions. Although we are most interested in the effects of deep recessions, the work on these continuous measures provides important complementary evidence, so we include it in our review.

Perhaps the most striking finding in this literature is that population health improves during economic downturns in wealthy countries. Using both aggregate time-series and state-level panel data from the United States, Ruhm (2000) finds that a percentage point increase in the unemployment rate is associated with a  $\frac{1}{2}$  percent decrease in all-age mortality. Gerdtham and Ruhm (2006) find similar evidence from across the OECD, as do Granados and Diez Roux (2009) for the historical United States, even in crises so pronounced as the Great Depression.<sup>4</sup> In seeking an explanation for these results, Ruhm (2000) emphasizes that the opportunity costs of leisure and other health inputs decrease during recessions, as do negative externalities from increased eco-

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<sup>4</sup> Despite the overall mortality decrease during the Great Depression, relief spending during this era was associated with further decreases in mortality (Fishback, Haines, and Kator 2007).

conomic activity.<sup>5</sup> Evidence for these mechanisms is mixed in contemporary data. On the one hand, he finds that sedentarism, smoking, drinking, and fat consumption are pro-cyclical, consistent with opportunity cost effects from higher wages and job stress. Additionally, mortality from vehicle accidents is strongly pro-cyclical, consistent with externalities from increased activity.

On the other hand, Stevens et al. (2011) show that pro-cyclical mortality is concentrated among young children, twenty-somethings, and especially the elderly, for whom the opportunity cost of time is small; the pattern is absent among adults of prime working age. These findings are difficult to reconcile with a theory which indicates individuals take worse care of themselves during recessions. Rather, Stevens et al. propose that mortality declines during recessions primarily reflect cyclicalities in the quality of health care, perhaps due to staffing costs. They report that staffing in skilled nursing facilities rises during recessions, and also that pro-cyclicality is more pronounced for deaths occurring in nursing homes and in states where more elderly live in nursing homes.

Pro-cyclical mortality and counter-cyclical health are also apparent among the very young, but these patterns may have other causes, most importantly because changes in fertility patterns influence the distribution of health among infants. Fertility falls during recessions in the United States (Currie and Schwandt 2014), in both national time series and state-level panel data. If this pattern varies across different types of women, then it may affect the distribution of child health. Indeed, Dehejia and Lleras-Muney (2004) present evidence that the composition of new mothers changes during recessions, in a way that increases the prevalence of maternal characteristics that promote child health. At the same time, holding these characteristics constant, maternal health behavior improves during recessions. Both margins of change—selection and behavioral adjustment—improve the average health of U.S. infants born during recessions, both across the country and within states. A percentage point increase in the unemployment rate is associated with a  $\frac{1}{4}$ - $\frac{1}{2}$  percent decline in the prevalence of low birth weight.

In poor countries, the opposite pattern generally holds. Analyzing Demographic and Health Survey data from 59 African, Asian, and Latin American countries, Baird et al. (2011) find strong counter-cyclicalities in infant mortality. Within countries over time, fluctuations in GDP per capita are negatively associated with fluctuations in infant mortality, a result robust to the inclusion of flexible country-specific time trends—thus accounting for joint secular trends in

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<sup>5</sup> For additional evidence on these mechanisms, see Ásgeirsdóttira et al. (2014).

economy and health. The result is also robust to mother fixed effects—thus controlling for changes in the composition of mothers. A 1 percent decrease in GDP per capita adds  $\frac{1}{4}$ - $\frac{1}{2}$  infant deaths per 1000 live births. Most country-specific studies in the developing world also find that mortality rises in downturns (Cutler et al. 2002; Paxson and Schady 2005; Bhalotra 2010). Where data exist, they suggest similar results for other health outcomes such as birth weight (Bozzoli and Quintana-Domeque 2014). Nevertheless, counterexamples also exist. In Colombia, sudden drops in the international price of coffee cause infant health to improve in coffee-growing regions, arguably because of falling opportunity costs of maternal time with children (Miller and Urdinola 2010). In Indonesia, child weight-for-age held steady through the financial crisis because mothers buffered children's caloric intake by eating less themselves (Block et al. 2004). Broadly speaking, however, most results point to pro-cyclical health in developing countries.

The differences in results between rich and poor countries suggest that income effects may dominate when a recession reduces resources to dire levels, as in a crisis. This theory would be compelling if the effects of booms and busts had asymmetric effects, in which busts have stronger negative effects than booms have positive effects. The literature offers some evidence to this effect. In poor countries, deep recessions elevate female infant mortality particularly strongly -- far exceeding effects proportional to the impacts of small negative shocks or positive shocks (Baird et al. 2011). But in a twist that remains open to interpretation, the effects of aggregate shocks on male infant mortality are both smaller and more symmetric. Furthermore, infant mortality is neither significantly more counter-cyclical in low-income (compared to middle-income) countries, nor in children born to less-educated (compared to more-educated) women.

Beyond these contemporaneous impacts, research suggests that survivors of early-life exposure to economic crisis may suffer life-long sequelae in developing countries. The most convincing evidence comes from the historical record. In Dutch cohorts from the 19<sup>th</sup> century, those with birth years coinciding with recessions experienced higher mortality risk through the lifecycle (van den Berg et al. 2006). Around the same time, men born during the Great French Wine Blight exhibited relative shortfalls in adult height if they were born in wine-growing regions, which underwent a deep recession (Banerjee et al. 2010). More recent evidence also suggests that exposure to economic shocks in early life can result in mental health impacts in adulthood (Adhvaryu, Fenske and Nysdhadham 2014). At least for the young, the human biology impacts of deep recessions last far longer than the direct economic impacts.

## 4 Famines

An extreme manifestation of recession is famine, a phenomenon that has received much attention in the economics literature at least since Amartya Sen's (1981) analysis of the Bengal Famine of 1944 (see also Ó Gráda 2009). The idea that famines kill is not novel; hence, the literature we summarize in this section addresses the issue of how famines affect the health and human capital of those who *survive*. This relationship is key to the notion that famines can have consequences beyond the loss of life in the short run.

Selection poses a major stumbling block to estimating the effects of famine. If mortality is concentrated among the weakest individuals, then survivors of famine will be positively selected, leading researchers to understate the impact of famine on survivors. Moreover, while the weather plays an important role in triggering famines, the intensity and duration of famines often result from failures of policy and political will. Hence, areas affected by famines might also be areas that otherwise would have received less public transfers, for example. Survivors are therefore likely to be selected in a way that resembles selective sorting into cities or neighborhoods, possibly confounding the analysis.

An influential series of papers examines the long-run impacts of the Dutch famine, which occurred in 1944-45. Reviewing these papers, Roseboom et al. (2001) conclude that prenatal exposure to the Dutch famine resulted in worse adult health along various dimensions, especially coronary heart disease (CHD). The studies focus not only on long-term health as an outcome but also on whether the *timing* of the famine in utero matters in different ways. Cohorts exposed during the first trimester were affected differently from cohorts exposed during the third trimester. For example, cohorts exposed late in gestation tended to have lower weight at birth and increased glucose concentrations (a marker for diabetes) in adulthood. Cohorts affected in the first trimester, in contrast, tended to not have any lower birth weight but had higher tendencies for CHD in adulthood. Perhaps the most interesting conclusion of the Dutch famine studies is that undernutrition during gestation can affect long-term health even if there is no indication of poorer health at birth (as measured by birth weight). Other studies on this particular famine have also found intergenerational birth weight effects, suggesting the effects of famine last across multiple generations (Stein and Lumey 2000).<sup>6</sup>

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<sup>6</sup> For more recent results on the Dutch famine, see Painter et al. (2006), Stein et al. (2007), Rooij et al. (2007), and Rooij et al. (2011).



Researchers have examined the long-run health consequences of famines in many other contexts as well. In China, Gørgens et al. (2012) study the Great Famine (1959-1961) and find that survivors of early-childhood exposure are significantly shorter than people who were not exposed. An important contribution of this study is its idea to use the *children* of survivors to control for selection into survivorship. Past research on the Chinese famine has struggled to find effects on survivors because famine mortality was apparently linked with potential height. The authors argue that while children inherit the genotype of their parents with regards to height, they do not inherit their phenotype. So they adjust for height differences in the next generation and interpret the residual height deficit in affected cohorts as the effect of famine. Notably, this strategy assumes that the scarring effects of famine do not transcend generations, which seems difficult to defend *a priori*. Nonetheless, the study makes important progress on dealing with selection effects.<sup>7</sup>

In a different context, Dercon and Porter (2010) highlight similar results stemming from one of Africa's worst famines, which occurred in Ethiopia in 1984. Using a household measure of famine intensity and comparing siblings with different famine exposure, the authors find that early-life famine exposure significantly reduces height in adulthood by at least 3 centimeters. Comparing exposed and non-exposed siblings is another way to control for selection effects, since siblings have similar height potentials.

While some studies show conflicting results of famine exposure (Luo et al. 2006, Stanner et al. 1997), the studies that explicitly account for selection and measurement error seem to consistently show a negative effect on health of famine exposure. A logical next step then is to examine how survivors fare in school, on the marriage market, and on the labor market, as recent studies have done.<sup>8</sup> Overall, these studies find that famine exposure at young ages (typically measured in utero or before the age of 2) negatively impacts long-term, non-health outcomes. Most of these studies use similar strategies of comparing exposed and non-exposed cohorts in areas with differing famine intensity. We highlight two complementary studies that examine the long-run labor market impacts of the Chinese famine.

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<sup>7</sup> Mu and Zhang (2011) find that female survivors have a higher incidence of disability, which they attribute to greater selective male mortality during the famine. Two other papers use similar strategies to find that exposed cohorts are shorter in the long run using (Chen and Zhou 2007, Meng and Qian 2009).

<sup>8</sup> See Shi (2011), Almond et al. (2006), Brandt et al. (2008), and Meng and Qian (2009) on China; Dercon and Porter (2010) on Ethiopia; Scholte et al. (2015) on the Netherlands; and Neelsen and Stratmann (2011) on Greece.

Almond et al. (2006) use multiple sources of variation to study the impacts of the famine. Choosing a narrow window of birth cohorts (1956-1964) to reduce confounding, they compare cohort-level changes across provinces with differing famine intensity. They also use residents of Hong Kong, which was under British rule and was thus unaffected by the famine, as a second control group. Among men, they find that in utero exposure to the famine increases illiteracy by 9 percent, reduces labor force participation by 6 percent, and reduces the probability of marriage by 6.5 percent. Women are similarly impacted, albeit with smaller magnitudes.

Meng and Qian (2010) build on this study by using a finer source of variation (county-level rather than province-level) and by using institutional features of the centrally planned procurement system to instrument for famine intensity. To account for positive selection into survival, they estimate the effect of exposure on the upper quantiles of the outcomes of interest. Their findings are consistent with prior studies on the famine (in terms of health and educational attainment), but accounting for measurement error and selection leads to larger magnitudes than prior studies. They find that in utero famine exposure reduces the 90<sup>th</sup> percentile of adult height by 3 centimeters, weight by 1½ kilograms, and educational attainment by half a year.

## 5 Epidemics

Like famines, epidemics have direct effects on human biology, and hence, economic research has contributed to our understanding of these crises mainly by looking for broad, long-term, or indirect impacts. Here again, changes in patterns of mortality and fertility complicate the estimation of impacts among survivors, but in this case, they may also have interesting macroeconomic implications. At least since Malthus (1798), economists have noted that widespread disease may increase the ratios of land or capital to labor, raising *per capita* living standards.<sup>9</sup> Along these lines, Voigtländer and Voth (2013) present evidence that the Black Death was a crucial turning point in the emergence of modern economic growth in Europe. A key feature of the bubonic plague in this respect is its rapid progression to death; as a result, it caused widespread mortality without sustained morbidity. More generally, the macroeconomic effects of an epidemic depend on its relative mortality and morbidity burdens. With greater morbidity, the surviving population becomes less productive, pushing back against the Malthusian benefits of epidemic mortality.

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<sup>9</sup> The same general equilibrium reasoning also applies to famine and war, but it has drawn more interest in the literature on disease.

A fitting example is HIV/AIDS, which Young (2005) argues may bring increased prosperity to Africa. Incorporating positive wage effects from AIDS mortality, negative fertility responses to HIV, and negative effects of orphanhood on the next generation's human capital, Young calibrates a positive net effect of the epidemic on future living standards. However, the calibration depends heavily on his assumption that morbidity from HIV/AIDS does not meaningfully decrease productivity and on his estimate that fertility falls with rising HIV prevalence.

Subsequent research casts doubt on both of these crucial ingredients to Young's argument. On the first, HIV has become a chronic disease, and mounting evidence suggests that it has serious productivity consequences (Thirumurthy et al. 2008; Habyarimana et al. 2010; Levinsohn et al. 2013). On the second, follow-up research has found that increases in regional and community-level HIV prevalence are not associated with falling fertility (Fortson 2009; Juhn et al. 2013). Using these revised fertility estimates, Kalemli-Ozcan and Turan (2011) recalibrate Young's model and find no macroeconomic benefit from HIV/AIDS.

A further indirect effect of the HIV pandemic on human biology is that it diverts resources and attention away from other important health care goals. Analyzing data from 14 sub-Saharan African countries from 1988 to 2005, Case and Paxson (2011) find that sub-national regions with larger increases in HIV prevalence experienced erosions in antenatal care, institutional deliveries, and immunization. In corroborating evidence, Grépin (2012) analyzes country-level panel data to show that expansions in international aid for HIV programs are associated with declines in immunization. Such crowd-out effects are not limited to HIV. In Taiwan, for example, Bennett et al. (2015) find that the onset of the SARS epidemic reduced outpatient medical visits by nearly one-third within a few weeks. Thus, epidemics affect healthcare access not only through policymakers' resource allocation decisions but also through fear of contagion in healthcare settings.

At the microeconomic level, studies show lasting effects of early-life morbidity due to epidemics. The 1918 influenza pandemic has attracted particular attention due to its sharp, unexpected, and indiscriminate nature. As with other crises involving significant mortality, positive mortality selection is likely to bias researchers toward finding no long-term effect on survivors. Nevertheless, drawing on both cohort-level variation and state-cohort panel variation in the United States, Almond (2006) estimates that in utero exposure to the flu pandemic reduced educa-

tional attainment, reduced adult income, and raised adult disability.<sup>10</sup> Cohorts that were *in utero* during the pandemic were 4 percent less likely to finish high school, 3 percent more likely to be poor, and 8 percent more likely to have a disability that prevented work in middle age.

Other research examines the lasting consequences of early-life exposure to malaria, a disease known for its widespread toll. Some of this literature studies the consequences of malaria eradication in settings where the disease is endemic (Bleakley 2010; Cutler et al. 2010; Lucas 2010), leveraging regional differences in baseline prevalence to isolate variation in the extent of malaria decline due to eradication. Comparing cohorts born before versus after eradication in high versus low prevalence areas, these studies find largely positive effects of malaria-free childhood human capital and labor market outcomes. The elimination of an endemic disease has little to do with crisis, but Lucas (2010) notes that Paraguay underwent a pronounced epidemic just before its eradication campaign. In that setting, the elimination of *epidemic* malaria at birth raised schooling by  $\frac{3}{4}$  of a year in the most infected region. Further evidence on the consequences of in utero exposure to a malaria crisis can be found in Barreca's (2010) analysis of malaria outbreaks in the early-20<sup>th</sup> century United States. Drawing on an OLS specification with state and birth cohort fixed effects, as well as an instrumental variables strategy that relies on climatic fluctuations to identify the effects of malaria outbreaks, Barreca finds that early-life exposure to epidemic malaria reduces schooling and adult labor supply, while increasing poverty. A standard deviation increase in the malaria death rate in high malaria states during gestation led to a 0.04-year decrease in educational attainment, a 0.35 percentage point decrease in full-year work, and a 0.38 percentage point increase in poverty. These analyses shed much light on how health insults in early life affect the trajectories of social and economic outcomes over the lifecycle.

## **6 Natural Disasters and Environmental Crises**

Natural disasters like earthquakes and floods often result in loss of life and property, as do environmental crises like nuclear accidents and dramatic increases in air pollution. While both are forms of "crisis," one can perhaps distinguish them on the basis of the extent of human involvement in their genesis. This differentiation is rather loose, however, because some natural disas-

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<sup>10</sup> Brown and Thomas (2011) point out that the 1918 pandemic coincided with World War I military deployments that changed the composition of new parents, which they argue can account for much of Almond's estimated flu impact. However, data from countries with less involvement in World War I indicate similar impacts of in utero exposure to the pandemic (Richter et al. 2013; Lin and Liu 2014).

ters are at least partly attributable to human action via climate change. Hence, we treat them jointly in this section.

We begin with papers that examine the impact of natural or environmental disasters on fertility. In the case of fertility, the effect of natural or environmental disaster can be positive or negative. Fertility may fall due to a loss of property or income, or it may rise if parents wish to “replace” deceased children. At least in developing countries, the evidence points toward replacement effects. Examining responses to three different earthquakes, Finlay (2009) finds that fertility rises after an earthquake. Similarly, studying the aftermath of the 2004 Indian Ocean Tsunami in Indonesia, Nobles et al. (2014) find increased fertility in harder-hit areas. The effect reflected both the behavior of women whose children died (thus replacing their *own* lost children) and the behavior of women who did not yet have children (thus replacing the *community’s* lost children). These results stand in contrast to that of Lin (2010), who finds that in Italy and Japan, short run instability due to natural disasters is associated with decreased fertility.

Many papers have documented the mortality effects of natural disasters and extreme weather events. For example, in a paper examining the effects of natural disasters in over 141 countries over two decades, Neumayer and Plumper (2007) finds that mortality effects of natural disasters tend to be concentrated among women. This is an important study as it suggests relevant inequalities in the impact of natural disasters. Since mortality effects of natural disasters are well documented in other review articles (see for example Cavallo and Noy (2010) and Bourque et al. (2007)) we now focus our attention to perhaps less widely-studied extreme weather events. For example, Deschenes and Moretti (2009) find that both extreme heat and extreme cold result in short-run mortality increases, with different causes of mortality at different ends of the temperature spectrum. Heat primarily advances the mortality of those who are already weak by a few days or weeks. In response to cold extremes, however, people who might otherwise live a few more years might die; hence, the mortality impact of extreme cold is longer-lasting. Given the general pattern of mobility in the United States, with people moving from colder to warmer climates, this finding suggests that migration could be one driving force behind increasing life expectancy. An important addition to this body of work is the recent research of Barecca et al. (2013), who find that the heat-mortality relationship in the United States has declined in recent decades. They attribute this decline to the adoption of air conditioning.

Extreme weather also has short-run consequences in the developing country context. In a broad study examining the impacts of early life weather conditions on infant mortality in 28 African countries, Kudamatsu et al. (2012) find that extreme weather fluctuations have a significant impact on infant mortality in Africa via malnutrition and malaria. Analyzing even more extreme weather variation, Anttila-Hughes and Hsiang (2012) study the aftermath of typhoons in the Philippines, finding elevated infant mortality rates that far exceed the direct effects of the storms. Burgess et al. (2013) also estimate a significant relationship between weather and mortality in India, which is mostly driven by high temperature extremes at the time of crop growth in rural areas. In a related paper that speaks to differences between aggregate crises and local shocks, Burgess and Donaldson (2012) show that the expansion of India's railroad network diminished the mortality impact of agriculturally-damaging weather shocks.

The evidence of the impact of pollution crises on health in developing countries is considerably less. Jayachandran (2009) is one of the few researchers to examine the mortality effects of forest fires. Forest fires produce atmospheric pollution that can travel large distances, with the potential of affecting the health of people far away. Using data from Indonesia, she finds that prenatal exposure to particulate matter due to forest fires in 1997 lead to around 16,400 fewer surviving infants in Indonesia. Changing focus to urban pollution, Arceo et al. (2012) use variation due to thermal inversions—which despite their frequency might qualify as crises—to find a similar result that pollution exposure is a significant contributor to infant mortality—in Mexico City.<sup>11</sup> Several other recent papers examine the effects of marginal changes in environmental damage in developing countries, which fall outside the purview of our review.

Along similar lines, a large literature considers the long-term effects of early-life exposure to pollution (Currie et al. 2014, Bharadwaj et al. 2014), but much of this literature considers marginal changes in pollution levels or environmental policies that improve pollution, neither of which qualifies as a crisis. A notable exception is Almond et al.'s (2009) analysis of the Chernobyl nuclear disaster's aftermath. Leveraging spatial and temporal variation in exposure to the radiation cloud in Sweden, Almond et al. find that prenatal exposure to radiation decreases cognitive achievement in later life, albeit without observable health impacts.<sup>12</sup>

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<sup>11</sup> A thermal inversion occurs when warm air settles over a layer of cooler air near the ground, trapping the cool air and any pollutants inside it.

<sup>12</sup> In a related paper using nuclear weapons testing rather than nuclear disaster as a source of variation, Black et al. (2014) find similar long run impacts of prenatal exposure.

## 7 Wars

Many of the crises we review in part reflect human action, but nowhere is human responsibility graver than in the case of war. Wars and other conflicts are disruptive along social, political and economic lines, with significant potential to affect human biology.<sup>13</sup> Most research on this topic has focused on the impact of conflict exposure in early childhood on subsequent physical growth. Analyses of this question must grapple with standard concerns about selective mortality, fertility, and migration, in addition to the fact that wars often accompany other undesirable phenomena: recession, disease, food shortage, and deterioration of health infrastructure, *inter alia*. While separately identifying each mechanism would be an interesting area for future research, existing research does not concern itself with isolating the underlying mechanism.

The Nigerian civil war was one of the earliest civil wars in post-independence Africa, making it a suitable context for studying long-run effects of childhood exposure. Akresh et al. (2012) examine its impact on adult stature, finding that individuals from ethnic groups most heavily exposed to the war attained significantly lower stature as adults. Exposure to conflict during adolescence was more damaging than exposure just in early childhood, which the authors attribute to possible disruptions in the normal growth spurt experienced in adolescence. Adult height is also the primary outcome in Aguero and Deolalikar's (2012) study of the Rwandan genocide of 1994. While they too find that exposure to the genocide leads to decreased height (relative to trends in neighboring countries), they find the effects to be greater when exposure occurs at a younger age. Thus, while both Akresh et al. (2012) and Aguero and Deolalikar (2012) find that childhood and adolescent exposure to wars reduces adult height, they differ on when exposure matters more.

Shifting from adult outcomes to child outcomes, Bundervoet et al. (2009) examine the consequences of exposure to civil war in Burundi on child height-for-age z-scores. The authors use variation in the timing and geographic spread of the war to estimate that an additional month of civil war exposure reduced height for age by about 0.05 z-scores. The effects are concentrated in children who were exposed to conflict between ages 0 and 2 years, consistent with the idea that the first few years of life are a critical period for physical growth.

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<sup>13</sup> While the term "war" is typically used to denote fighting across borders, and the term "conflict" often denotes within-country fighting (such as civil or ethnic conflicts), we use the terms interchangeably in this chapter.

When two countries go to war, residents of the winning country might benefit by suffering less destruction or disruptions to economic systems or public health delivery. Hence, when thinking about the impacts of inter-country conflict, a crucial question arises regarding the *net* health impacts of such conflict. Akresh et al. (2014) address this issue by examining the health impacts on *both* sides of the Eritrean-Ethiopian conflict of 1998-2000. Children on *both* sides suffered equally in terms of the effect on height-for-age z-scores, but children in the losing nation suffer more than those in the winning country.

While war-induced migration is a central concern for interpretation of the results so far, the arrival of refugees in large numbers could also pose a health risk to the locals in the areas where they arrive. Baez (2011) examines the health of *local* children as a function of the refugee influx from the genocides in Burundi and Rwanda. In 1994-95, North-Western Tanzania received hundreds of thousands of refugees; however, topographical characteristics induced geographical variation in refugee inflows. Baez uses this variation to estimate negative effects on the health (as measured by anthropometrics and child mortality) and human capital attainment of local children one year after the arrival of refugees. We hope this important study opens the door to further research on the health impacts of conflict for people not directly involved in the conflict.

## **8 Conclusion**

A burgeoning economics literature considers the effects of various shocks on human biology. Mixed into this literature are shocks large and small, positive and negative, local and aggregate. In this review, we have homed in on a subset of these shocks—aggregate crises—in the hope of highlighting commonalities in their effects. The literature suggests that these unexpected, pronounced, negative, and population-wide events affect human reproduction, mortality, and morbidity over the lifecycle. To shed further light on the roles of prices and insurance arrangements, a fruitful line of future inquiry would compare crises at different scales (Caruso 2014 and Akresh, Bundervoet and Verwimp 2011 are recent examples), or those occurring in environments with differing levels of market integration or insurance system development (Kahn 2005 is an excellent example).

More broadly, however, the literature highlights the lasting effects that social, economic, political, environmental, and pathological crises have on the human body. Children, who are never complicit in creating crisis, carry the burden of exposure for the rest of their lives. While advanc-



es in methodology and data availability have allowed researchers to uncover these nuanced but powerful effects, much work remains in improving crisis response, especially in poor countries. As the findings demonstrate, improvements in this arena would have beneficial effects long after the acute period of a crisis subsides, on outcomes far beyond its most obvious sequelae.

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