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Early-Life Exposures and Social Stratification

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Abstract

Adverse environmental exposures—war and violence, natural disasters, escalating heat, worsening air quality—experienced in pregnancy are consequential for multiple domains of well-being over the life course, including health, cognitive development, schooling, and earnings. Though these environmental exposures become embodied via biological processes, they are fundamentally sociological phenomena: Their emergence, allocation, and impact are structured by institutions and power. As a result, consequential early-life environmental exposures are a critical part of the sociological understanding of social stratification, intergenerational mobility, and individual and cohort life course trajectories. We review theory and evidence on prenatal exposures, describe enduring methodological issues and potential solutions for elucidating these effects, and discuss the importance of this evidence for the stratification of opportunity and outcomes in contemporary societies.

1. INTRODUCTION

The past two decades have seen burgeoning attention to the early period of life as a determinant of people's health, cognition, and economic well-being. Biomedical scientists have established physiological mechanisms through which prenatal exposures get under the skin and shape individual trajectories. Social scientists have credibly identified the causal impact of specific environmental insults experienced early in life—weather events, economic downturns, pollution, violence, income-support policies, among others—and have shown their consequences across the life course. While warnings that "the womb might be more important than the home" (Barker 1990, p. 1111) or that "a child who falls behind might never catch up" (Heckman 2006, p. 1900) might be exaggerated, they reflect evidence that the early-life period—from conception to early childhood—is both vulnerable to the environment and consequential for individual development, attainment, and well-being.

These insights have been slow to permeate sociological research in general, and stratification research in particular. We argue that considering the early-life period—and, specifically, exposure to environmental insults before birth—is important to understand stratification processes and the persistence of disadvantage across generations. In this review, we focus on the prenatal period because it is the earliest stage of the life course that can be observed, and because it is both exceptionally sensitive to the environment and consequential for later development, thus influencing outcomes from birth to older ages.[1]

We suggest that prenatal exposures are an important sociological concern for several reasons. First, these exposures are common in contemporary societies, and in the case of many of them, including heat, pollution, disasters, and accompanying physical and psychosocial stress, their prevalence will likely increase in the future. Second, even if the mechanisms accounting for their impact are biological, the experience and consequences of early-life exposures are social phenomena rooted in stratification dynamics (Muller et al. 2018). As Kai Erikson [2012 (1976), p. vi]) wrote, "Earthquakes do not seek out the ill-housed; they strike evenly at all of the structures in their way, but do the most damage to the frailest and most shoddily built of them, the ones in which the needy have been invited to live." In similar fashion, zoning regulations allocate exposure to pollution, racial discrimination influences recovery after neighborhood disruption, and policy decisions regulate access to insurance against risks. The claim that there is not such a thing as a (purely) natural disaster (Squires & Hartman 2006) applies to virtually all environmental exposures experienced in pregnancy: Their emergence, allocation, and impact depend on decisions regulated by institutions and power. A third reason these exposures fall under the purview of sociologists is that when they occur early in life, the causal impact of environmental exposures would go undetected and could easily be mistaken for genetic inheritance or other innate factors.

These features render prenatal environmental exposures an important part of the understanding of social stratification, intergenerational mobility, and life course trajectories. This review examines the impact of adverse prenatal exposures on individual development and well-being, and explores mechanisms and socioeconomic heterogeneity in these effects. The relevant literature is interdisciplinary; we therefore draw on work in sociology, demography, epidemiology, economics, psychology, and the biomedical sciences. Section 2 integrates arguments from multiple fields that theorize how and why the prenatal period has enduring consequences. Section 3 reviews evidence on the impact of four specific exposures that are of concern for sociologists, and that are widespread and unequally distributed: violence, natural disasters and weather events,

[1] An emergent literature considers preconception exposures and circumstances of both mother and father (e.g., Blanc et al. 2023, Provençal & Binder 2015, Selevan et al. 2000).

extreme temperatures, and air quality and pollution. Recent empirical work strives to identify the causal effects of these exposures, a critical endeavor to advance knowledge and to inform policy intervention. Section 4 examines a nascent literature on the socioeconomic heterogeneity in the impact of adverse prenatal exposures and the mechanisms driving heterogeneity. Section 5 discusses analytical and methodological challenges and potential solutions, and Section 6 discusses implications of this work for contemporary stratification research.

2. THE RELEVANCE OF THE PRENATAL PERIOD

The intuition that early-life circumstances shape people's health, behavior, and well-being has been present for centuries, permeating conventional wisdom and cultural representations. By the early twentieth century, this intuition had found empirical support in population-level analyses, when researchers concluded that mortality patterns of cohorts born in the late nineteenth and early twentieth century behaved "as if the expectation of life was determined by the conditions which existed during the child's early years" (Kermack et al. 1934, p. 700).

In the 1970s, physician Anders Forsdahl (1977) demonstrated an association between infant mortality rates of Norwegian cohorts and metabolic disease in middle age. Forsdahl hypothesized that this effect emerged from permanent biological damage caused by nutritional deprivation in utero, combined with high fat consumption during adulthood in the context of newfound affluence. A decade later, epidemiologist David Barker observed a geographic area–level association between cohort birthweight (a proxy for nutritional deprivation in utero) and rates of coronary disease in late adulthood in England (Barker & Osmond 1986). Barker also documented individual-level correlations between birthweight and coronary heart disease, hypertension, and type 2 diabetes in adulthood (Barker et al. 1993).

These correlations were replicated in many national contexts (Godfrey & Barker 2001) and gave rise to the influential "fetal programming hypothesis." This hypothesis suggests that developments that enable the fetus to adapt to an adverse uterine environment result in programming via permanent changes to physiology and metabolism, leading to illness and early death (Barker 1998). A contribution of the fetal programming hypothesis is the notion of latency: the idea that the effect of programming might remain latent for decades, emerging in adulthood, somewhat irrespective of intervening experience and proximate risk factors (Almond & Currie 2011, Palloni & Beltrán-Sánchez 2017).

The fetal programming hypothesis has been criticized on multiple fronts: that it relies on rough associations vulnerable to confounding, that it piles up evidence without any effort to falsify, and that the notion of latency is extremely difficult to test empirically (Almond & Currie 2011, Paneth & Susser 1995, Rasmussen 2001). Despite reasonable critique, the hypothesis provided enormous impetus in the social sciences to elucidate the impact of the months spent in utero—until then nearly invisible for social scientists—on a diverse set of outcomes, including health, education, and socioeconomic attainment.

Complementary research in developmental psychology and neurobiology offers a conceptual apparatus to explain why early exposures have long-term, and even permanent, consequences. Sensitive and critical developmental periods act as "windows of opportunity" for essential developmental processes (Brown 2005). These windows open during gestation, peak in early childhood, and decline thereafter (Hertzman & Boyce 2010). This approach explains why, for example, lead exposure causes neurodevelopmental deficits if experienced in utero but is less harmful when experienced later in life (Lynch & Smith 2005). There is now consensus that early life is highly susceptible to the environment and ripe with developmental opportunity given plasticity of immune, neurological, and other biological systems (Gluckman et al. 2008).

The biological embedding perspective (Hertzman & Boyce 2010, Russ et al. 2014) directly links the developmental approach to the social environment, suggesting that early life provides a roughly ordered sequence of windows of opportunity that allow early-life experiences to "get under the skin" by altering neural, endocrine, and immunologic systems and even genetic expression, affecting the course of human development. The processes underlying biological embedding involve several pathways, including fetal adaptive responses, the hypothalamic-pituitary-adrenal axis and its regulation of cortisol, and social epigenetics.

Social epigenetics is the most novel and expansive pathway linking the environment to durable changes in health and well-being (Landecker & Panofsky 2013). The field describes how environmental factors alter gene expression and activity in ways that are persistent but which do not involve changes in DNA sequence (Martin & Fry 2018). Prenatal exposures as diverse as nutritional deprivation, pollutants, and maternal psychosocial stress have been linked to epigenetic dysregulation (Bock et al. 2015, Perera & Herbstman 2011, Thayer & Kuzawa 2011), which in turn could alter outcomes such as preterm birth and neurobehavioral development (Lapehn & Paquette 2022). While the biological workings of epigenetic processes are not the domain of sociologists, their consequences for individual well-being and for the transmission of inequality across generations very much are.

Overlaying the biological mechanisms described by programming and embedding are socioeconomic mechanisms that link early-life circumstances to later well-being. The social sciences describe the life course as a dynamic, hierarchical process in which prior experience shapes outcomes over time, and therefore shapes individual trajectories from birth to late adulthood (Ben-Shlomo & Kuh 2002, Cunha & Heckman 2007, Hertzman & Boyce 2010, Lynch & Smith 2005). These pathways involve cumulative effects—for instance, the toll that chronic poverty or persistent discrimination takes on individual trajectories—and mediating processes—for instance, when poor health in childhood hinders learning, which in turn reduces educational attainment. These fields also provide support for circumstances in which early adverse effects can be mitigated (Grossman 1972, Lynch & Smith 2005) partly by compensatory behaviors of parents (Bernardi 2014) and medical institutions (Bharadwaj et al. 2013).

Finally, selective survival helps understand the population-level links between prenatal exposures and later-life well-being (Bruckner & Catalano 2018, Nobles & Hamoudi 2019). Exposure to environmental insults could increase the risk of fetal loss, particularly among the frailest members of a cohort—a mortality pattern often called "culling" in demography (Catalano & Bruckner 2006, Elo & Preston 1992, Vaupel et al. 1979). If only the hardiest members of the cohort survive an environmental insult, then researchers will obtain a downwardly biased assessment of its impact: We could observe no effect—or, in the extreme, a positive impact—driven by the positive selectivity of the survivors (Bozzoli et al. 2009, Triyana & Xia 2023). Selective survival in utero is difficult to observe, so researchers rely on indirect measures such as changes in fertility rates and in the sex ratio at birth (James & Grech 2018).

Combined, these approaches suggest that harmful prenatal exposures could lead to adverse outcomes over the entire life course, starting at birth (e.g., low weight or preterm birth), during childhood (e.g., reduced cognitive function), and in adulthood (e.g., reduced earnings and early mortality). These outcomes could express themselves later in life or can ensue as chains or risks that cumulate over time. These theoretical approaches suggest that while the impact of early-life shocks can fade out or be moderated by compensatory responses, they also have the potential to persist and even magnify over the life course. Finally, the outcomes that researchers get to observe depend on the selectivity of those surviving the early-life insult, which is usually positive and unobserved.

3. ENVIRONMENTAL EXPOSURES AND OUTCOMES OVER THE LIFE COURSE

Insights from the fetal programming hypothesis permeated social science in the 1990s. In sociology and demography, an initial literature incorporated the early life to the understanding of adult health trajectories, with a primary focus on longevity (Blackwell et al. 2001, Elo & Preston 1992, Hayward & Gorman 2004, Palloni 2006, Palloni et al. 2023, Preston et al. 1998). Related work in economics documented associations between early circumstances and later health, education, and economic attainment (Case et al. 2005, Case & Paxson 2009, Costa 2000). An interdisciplinary body of work has since ballooned, broadening the scope of exposures studied: Natural disasters, extreme temperatures, pollution, community violence, famine, family death, and safety net transfers, among others, were all explored as prenatal exposures that shape developmental trajectories. Instead of summarizing this vast, rapidly growing body of work, we focus on research in four areas that are relevant to population well-being and inequality in contemporary societies: natural disasters and weather events, extreme temperatures, air quality and pollution, and contextual violence.[2]

In each of these areas, early studies explored correlations between adverse exposures and health and development. Many of the early studies faced multiple threats to inference, but they provide rich background on which the field has built (see reviews of the descriptive scholarship focused on each of these exposures by Chersich et al. 2020, Nyadanu et al. 2022, Saulnier & Brolin 2015, Syed et al. 2022, Wright et al. 2017). More recent studies address inference challenges by combining creative use of natural experiments and causal identification for observational data. We restrict our discussion below to studies that exploit plausibly exogenous exposures or other quasi-experimental designs, include a control group representing counterfactual outcomes absent the exposure, and which use population-representative data.

Much of this scholarship focuses on short-term outcomes observed at birth: gestational age and birthweight, deemed important because they provide rough indicators of endowments at the "starting gate" of life (Conley et al. 2003). Indeed, birth outcomes are the main predictors of infant morbidity and mortality (Behrman & Butler 2007, Lau et al. 2013). Among surviving infants, they increase the risk of illness and neurodevelopmental delay, and they shape long-term socioeconomic outcomes (Luu et al. 2017, Moster et al. 2008). In each of the four domains described below, a smaller set of well-designed studies directly evaluates the effects of adverse exposures on later outcomes including cognitive development, educational attainment, income, and later-life health. We discuss a few exemplary studies in each section.

3.1. Natural Disasters and Weather Events

Floods, earthquakes, hurricanes, tornadoes, and winter storms are increasingly relevant due to evidence linking climate change with extreme weather event severity (Arcaya et al. 2020). In the United States alone, the annual number of federally designated disasters surged from about 200 in the early 1980s to about 2,000 in the 2010s (Boustan et al. 2020); these events will almost certainly continue to increase in frequency and devastation in the future.

[2]We exclude exposures abundantly studied in the literature, in particular nutritional deprivation in utero. Much research uses the Dutch Hunger Winter of 1944–1945 in the Netherlands as a natural experiment to examine the impact of prenatal nutritional deprivation on health outcomes and later-life mortality (Painter et al. 2005, Roseboom et al. 2001), neurodevelopment (Susser et al. 1998), and reproductive outcomes (Lumey 1998). Other research exploits the 1941–1942 Greek famine (Neelsen & Stratmann 2011) and the 1959–1961 Chinese famine (Chen & Zhou 2007, Li & Lumey 2017, St Clair et al. 2005). Researchers have also used Ramadan fasting among Muslims (Almond & Mazumder 2011, Greve et al. 2017) and twins (Conley et al. 2003, Royer 2009) as natural experiments to examine the impact of nutritional deprivation.

3.1.1. Health at birth. Simeonova (2011) finds that exposure to natural disasters in the United States between 1968 and 1988, including storms, winter weather, and floods, had a small impact on infant health. Using year and county fixed effects, she reports a decline in gestational age by less than one-tenth of a day and of birthweight by 1.5 g when the disaster is experienced in mid-pregnancy (see **Figure 1** for a depiction of effect estimates, confidence intervals, and timing of exposure across studies reviewed in this article). Currie & Rossin-Slater (2013) find a harmful impact of late-pregnancy hurricane exposure between 1996 and 2008 in Texas on abnormal conditions of the newborn and complications of labor and delivery, but no impact on standard measures of infant health such as birthweight and gestational age. This study is exemplary in its careful assessment of identification bias. In addition to providing a comparison between exposed and unexposed siblings, the authors account for the mechanical correlation between longer gestational length and increased probability of exposure, and for migration in response to the storms; they also implement a placebo test examining the (implausible) impact of hurricanes 6 months after birth.

A small literature examines the impact of earthquakes on birth outcomes. Earthquakes provide a unique basis for identification because they come unannounced and typically last only a few minutes, reducing the scope for selective exposure. These studies consistently find negative impacts, of varying magnitudes, of prenatal earthquake exposure. Torche (2011) examines the impact of prenatal exposure to an earthquake in 2005 in Chile using a difference-in-differences approach. She finds that infants who experienced the earthquake during early pregnancy had a 1.7 percentage point higher risk of low birthweight (from a baseline of 5.4%). Kim et al. (2017) find that first- and third-trimester exposure to the 1994 Northridge earthquake in California resulted in a much smaller 0.2 percentage point increase in the probability of low birthweight using a similar methodology, and Menclova & Stillman (2020) report decreased gestational age associated with first- and third-trimester exposure to an earthquake in 2010 in New Zealand.

3.1.2. Outcomes over the life course. A few studies have documented impacts of prenatal disaster exposure on cognitive, educational, economic, and health outcomes. Torche (2018) finds a markedly stratified impact of prenatal earthquake exposure on children's cognitive ability, with a strong negative effect on kids' cognition among socioeconomically disadvantaged families and no effect among advantaged ones. Several studies have found that in-utero earthquake exposure reduces educational attainment (Caruso & Miller 2015, Paudel & Ryu 2018, Tian et al. 2022). Frankenberg et al. (2017) use longitudinal survey data to demonstrate that 1–2 years after the 2004 Indonesian earthquake and tsunami, children in utero at the time of the disaster were one-third of a standard deviation shorter than children not similarly exposed. Four years later, exposed children had largely caught up in growth, except for those born to mothers exhibiting symptoms of extreme post-traumatic stress. Karbownik & Wray (2019) reported a remarkable 5% decline in adult income in the United States as a consequence of prenatal exposure to tropical cyclones. In terms of health outcomes, Noghanibehambari (2022) finds a 1.8-month reduction in longevity associated with exposure to earthquakes during the early twentieth century in the United States. Interestingly, Hong et al. (2021) found children exposed to a 1974 tornado outbreak in the United States experienced physical and cognitive delays but limited longer-term economic consequences, suggesting parental compensatory responses.

3.2. Extreme Temperatures

In complement to scholarship on acute disaster events, scholars have also studied the effects of enduring changes in weather. The most consistent evidence on harmful health effects emerges from exposure to extreme heat during pregnancy (Kim et al. 2021, Randell et al. 2020), which

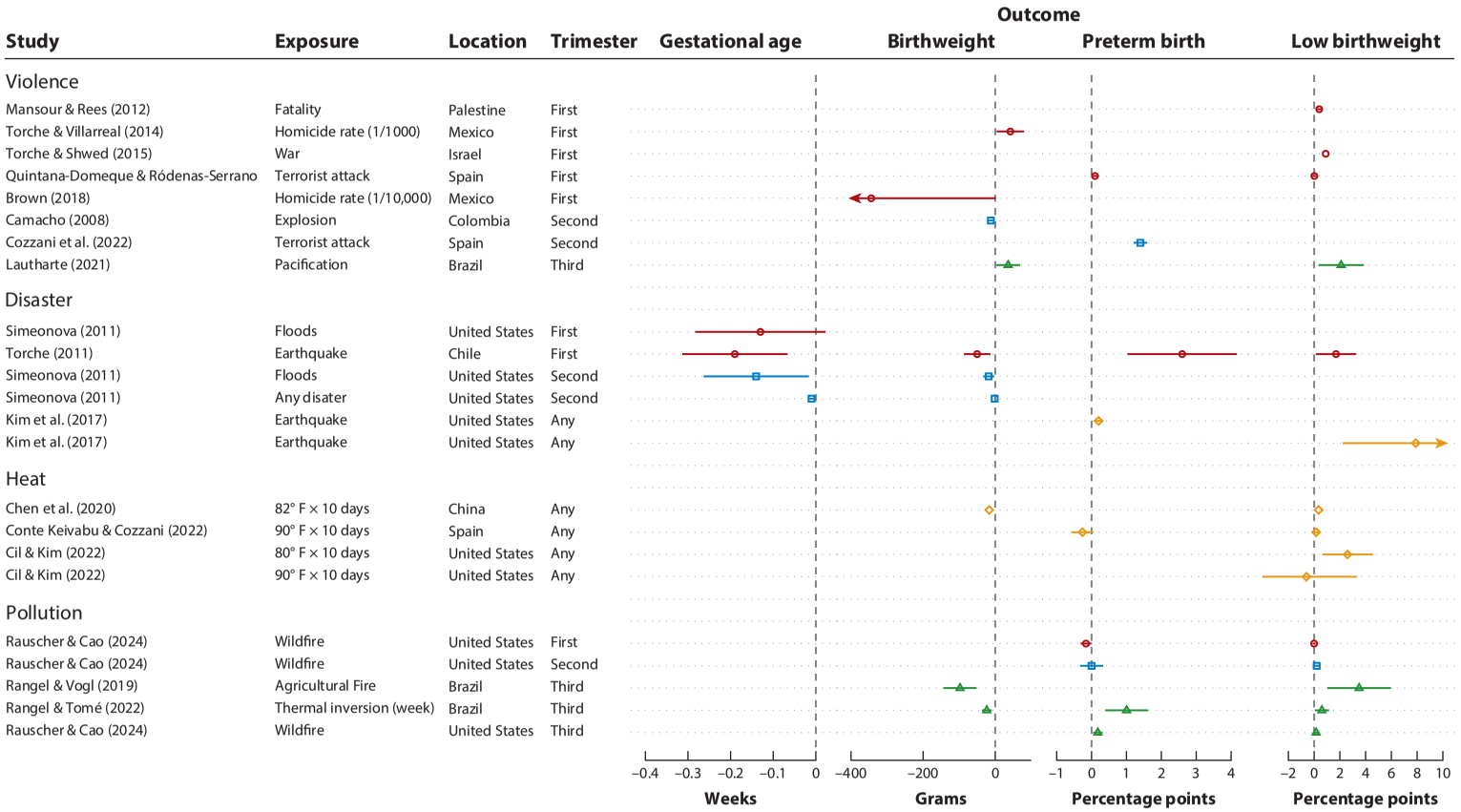

Figure 1

Selected estimates of effects of prenatal exposures on birth outcomes. Effect estimates and 95% confidence intervals are as reported in published studies. Color differentiates pregnancy trimester of exposure (*red* indicates the first trimester, *blue* the second trimester, *green* the third trimester, and *yellow* any trimester). Arrows indicate truncated confidence intervals. Truncation is used to improve legibility of effect sizes. Note that Rangel & Vogl (2019) and Rangel & Tomé (2022) measure exposures in weeks prior to birth—effects reported here reflect exposures 0–12 weeks before delivery. Most of the exposures occur in the third trimester of gestation, including some exposures for preterm births. A small number of exposures for preterm births occur in the second trimester.

operates through multiple mechanisms, including maternal heat stress and, in lower-income contexts, nutritional deprivation and infectious disease exposure (Grace et al. 2021).

3.2.1. Health at birth. To study heat exposure in pregnancy, many researchers link vital records to detailed climate data and address sources of confounding with place and time fixed effects. In an early study, Deschênes et al. (2009) examined 1972–1988 US births and demonstrated a 0.003–0.009% reduction in birthweight for every day above 85°F during gestation. Cil & Kim (2022) study 2009–2018 US births and show that exposure each day to temperatures between 80 and 90°F increases the risk of low birthweight. They also find that each day of exposure above 90°F increases very preterm birth risk by 0.24% and neonatal intensive care unit admissions by 0.2%. Conte Keivabu & Cozzani (2022) find that each day of exposure to ≥90°F temperatures among pregnancies in Spain is associated with a 0.016 percentage point increase in the probability of preterm birth; the authors do not find an association with low birthweight. Barreca & Schaller (2020) document an effect of daily temperature on birth rates in the United States, including evidence of a spike in births on or after ≥90°F days. They show how this effect contributes to a distributional shift in gestational timing: Each day above 90°F accounts for 9 lost days of gestation per 100,000 reproductive-age women.

In lower-income settings, Chen et al. (2020) find that each day of exposure to ≥82.4°F temperatures in rural China is associated with 1.66-g reduction in birthweight and a 0.035 percentage point increase in the probability of low birthweight. Le & Nguyen (2021) find that extreme heat exposure in Vietnam during in the first trimester reduces birthweight by 67 g, or 2.2%.

3.2.2. Outcomes over the life course. Isen et al. (2017) study how these exposures translate into long-run effects on socioeconomic status in the United States. They demonstrate that an additional day of exposure to temperatures above 90°F in utero or in infancy reduces annual earnings in the United States at age 30 by 0.1%. Fishman et al. (2019) find a relationship between in-utero heat exposure and reductions in completed education and earnings in Ecuador. Wilde et al. (2017) find a surprising positive effect of first-trimester heat exposure on educational attainment in sub-Saharan Africa, suggesting that it might be driven by in-utero selection.

Several scholars offer excellent discussions of best practices when studying the relationship between climate and health (Dorélien & Grace 2023, Randell 2022). Innovations in the study of prenatal heat exposure include attention to (*a*) measurement, e.g., using information on humidity and wet-bulb temperature, which may better capture maternal heat stress (Geruso & Spears 2018); (*b*) adaptive processes, e.g., measuring temperature deviations from expected values (Cil & Kim 2022), or forms of adaptation, such as air conditioning access (Isen et al. 2017); and (*c*) the likely nonlinear and nonuniform climate changes in the future (Barreca & Schaller 2020, Verdin et al. 2021). Barreca & Schaller (2020), for example, use output from climate models to project the effects on gestational age alongside expected temperature increases into the late twenty-first century.

3.3. Air Quality and Pollution

Socioeconomic development, urbanization, and changes in transportation have increased global population exposure to air pollution. Climate change and the growth in the frequency and scope of wildfires have amplified exposure to reduced air quality. A large body of research now suggests that exposure to air pollution in utero—particularly carbonized pollution, such as that created by fire and automobiles—is consequential for later health and socioeconomic status (Bekkar et al. 2020, Currie et al. 2014, Goyal et al. 2019). Studies use a range of methodological approaches, including leveraging changes in policies that regulate burning and pollution levels; variation in

wind direction; and the legacy of acute events, such as the 1952 Great Smog event in London or the 1997 wildfires in Indonesia.

3.3.1. Health at birth. A consistent finding is that late-pregnancy exposure to harmful levels of particulate matter increases the risk of preterm birth and reduces average birthweight. Researchers have merged vital records to area-level climate conditions and employed specifications with place by time fixed effects. Rauscher & Cao (2024) demonstrate that third-trimester wildfire exposure during gestation in the United States predicts a 0.18 percentage point increase in the probability of preterm birth and a 0.2 percentage point increase in the probability of low birthweight. They also find a 0.16 percentage point reduction in preterm birth associated with first-trimester wildfire exposure, possibly because of fetal selection.

Rangel & Vogl (2019) examine the effects of exposure to controlled agricultural fires in Brazil. They use shifts in wind direction as a source of exogenous variation in air quality and find that a one-standard-deviation increase in fire exposure in the 12 weeks before delivery reduces birthweight by 97 g and increases low birthweight by 3.5 percentage points. Jayachandran (2009) linked smoke exposure to census records to demonstrate that third-trimester exposure to the large 1997 forest fires in Indonesia resulted in the reduction in the size of child cohorts by 1.2%, amounting to over 15,000 fetal, infant, and child deaths.

3.3.2. Outcomes over the life course. Turning to measures of socioeconomic status, studies have found that particulate matter exposure during pregnancy in India and Indonesia predicts shorter height and reduced lung function in childhood (Rosales-Rueda & Triyana 2019, Spears et al. 2019). Using a sibling comparison, Bharadwaj et al. (2017) demonstrate that in-utero exposure to particulate matter reduces fourth grade test scores in Chile. The authors control for the frequency of public health alerts about air quality to adjust for potential variation in pollution avoidance behaviors. In the United States, Isen et al. (2017) compare cohorts born before and after improvements in air quality driven by the 1970 Clean Air Act; they demonstrate that the reduction in pollution exposure in pregnancy contributed to modest increases in labor force participation and earnings at age 30. O'Brien et al. (2018) link measures of particulate matter to area-level intergenerational income mobility. They find that a one-standard-deviation increase in suspended particles during the birth year is associated with a 0.31 percentage point reduction in the mean income percentile ranking at age 30—a small effect, roughly akin to $140 less annual income.

Researchers developing this line of scholarship have argued the importance of addressing the correlation between economic activity and pollution (Rangel & Tomé 2022) and addressing selective migration behavior away from fire activity or polluted communities (Currie 2013). This latter issue is particularly important when place of residence is not measured until birth or, in some cases, until later in life.

3.4. Contextual Violence

A growing literature examines the impact of prenatal exposure to violence emerging from terrorist attacks, homicides, war, and civil unrest. While fatal violence has declined dramatically since the 1990s in the United States and other wealthy nations (Sharkey 2018), violence associated with crime, terrorism, and political strife is on the rise around the world, particularly in low-income countries and disadvantaged communities in wealthier nations (United Nations 2020).

Several studies consider singular violent historical events. Cozzani et al. (2022) find that exposure to a 2004 train bombing terrorist attack in Madrid during the second trimester of gestation increased the risk of preterm birth by 1.4 percentage points using a difference-in-differences design. Torche & Shwed (2015) examine the impact of the 2006 Israel–Hezbollah war, a 33-day

conflict, on infant health. By comparing siblings exposed to missiles in northern Israel with their unexposed siblings, the authors demonstrate that exposure to war in early and mid-pregnancy increased the probability of low birthweight about 0.7–0.9 percentage points. Lautharte (2021) offers an unique analysis of a beneficial treatment: the decline in violence resulting from a "favela pacification" program implemented between 2008 and 2014 in Brazil. He finds that women exposed to a pacification intervention in the third trimester of pregnancy experienced increased birthweight by 36 g and a reduced risk of low birthweight by 2.1 percentage points, compared with pregnancies residing on the same street but occurring before the pacification intervention.

A related body of work examines impacts of changes in violence over time rather than single historical events. In a pioneering study, Camacho (2008) examined the impact of land mine explosions in the municipality maternal residence in Colombia and found that second-trimester exposure reduced birthweight by 12 g, based on time and municipality fixed effects models. Several studies examine the impact of local homicides. Torche & Villarreal (2014) found that exposure to each drug-trafficking-related homicide at the local level in Mexico 2008–2010 increased average birthweight by approximately 42 g when experienced early in the pregnancy. This unexpected positive finding is observed only among disadvantaged women and is at least partially accounted for by increased prenatal care use, plausibly as a protective strategy by pregnant women (but for divergent findings, see Brown 2018).

Mansour & Rees (2012) find that each fatality in the context of the 2000–2005 al-Aqsa Intifada in the West Bank and Gaza results in a modest increase—about 0.3 percentage points—in the probability of low birthweight. Foureaux Koppensteiner & Manacorda (2016) find that early-pregnancy exposure to within-municipality homicide in Brazil increases risk of low birthweight by around 0.6 percentage points, but only in small municipalities (probably because homicides are rare and residents are more aware of their occurrence than in large municipalities), and Quintana-Domeque & Ródenas-Serrano (2017) report a minimal increase in the risks of low birthweight, by 0.02 percentage points, and preterm births, by 0.1 percentage points, associated with each bomb casualty from terrorism by the group ETA (Basque Homeland and Liberty) in Spain between 1980 and 2003.

Most studies using plausibly exogenous sources of variation suggest detrimental effects on health and birth as well as developmental, educational, and economic outcomes across broad domains of natural disasters, extreme temperatures, air pollution, and contextual violence. A few exceptions document positive effects, potentially driven by in-utero selection, behavioral responses to exposure, or other mechanisms not yet well understood by which biological systems adapt to exposures in ways that increase infant survival (Kuzawa & Kim 2022). In what follows we discuss the socioeconomic stratification of these consequences.

4. SOCIOECONOMIC STRATIFICATION IN THE IMPACT OF PRENATAL EXPOSURES

Population-level effects could mask substantial socioeconomic heterogeneity. The same event may have profound negative consequences for some groups, minor or no impact for others, and even provide a positive turning point for others. Researchers are starting to examine sources of socioeconomic variation and theorize its sources (Torche et al. 2024). Based on this emerging research, we distinguish four sources of socioeconomic variation: exposure, sensitivity, behavioral responses, and early-life survival.[3]

[3] Other relevant sources of heterogeneity in the impact of prenatal exposures, such as by trimester of exposure and infant sex, are not reviewed here as they largely emerge from biological mechanisms.

4.1. Stratified Exposure

Harmful prenatal exposures are patterned by socioeconomic advantage, ethnoracial privilege, and place of residence. The theory of social conditions as fundamental causes of health suggests that socioeconomic status provides multiple resources such as money, power, knowledge, and social connections that can be mobilized to avoid harmful exposures (Clouston & Link 2021, Link & Phelan 1995). Observed stratification of risk during early life is consistent with the fundamental cause approach: Advantaged families are less exposed to environmental insults such as pollution and environmental toxicants (Currie 2011, Hajat 2015, Muller et al. 2018), violence (Buggs et al. 2022, United Nations 2020), natural disasters (Tierney 2019) and extreme heat (Gronlund 2014, Hoffman et al. 2020). They are more likely to benefit not only from financial resources but also from political influence (Elsässer & Schäfer 2023, Erikson 2015), social networks, and technological innovations (Timmermans & Kaufman 2020) to secure safe and nurturing environments.

Socioeconomic gradients in exposure invite scholars to at least consider the specific pathways stratifying prenatal exposures across the settings studied and, ideally, to advance common and comparable measures of stratification across settings (for example, short batteries of survey questions that would capture differential exposure). If stratification of exposure results in differential dosages of the disruptive event, then assuming a homogeneous treatment across the population will conflate treatment stratification with effect stratification, resulting in biased inference and missed research opportunities.

4.2. Stratified Sensitivity

Disparities in early-life exposures may be compounded by disparate vulnerability to their consequences: The effect of exposures could be stronger among disadvantaged populations if persistent economic deprivation or discrimination acts as a predisposing factor for the influence of a novel exposure. That is, a new stressor will cause more damage to individuals already debilitated by chronic disadvantage. Several theoretical approaches, including allostatic load (McEwen & McEwen 2017), weathering (Geronimus 1992), and cumulative risk factors (King et al. 2011) suggest that the wear and tear emerging from disadvantage depletes coping mechanisms and increases vulnerability to a novel exposure. It is important to note, however, that the opposite stratification pattern is also possible: Given prior experiences of disruption, disadvantaged populations might be less affected by novel environmental insults, a protective mechanism variedly termed habituation, inoculation, and adaptation (Feder et al. 2009, Gump & Matthews 1999). It is also possible that the harm caused by prenatal exposures is harder to detect among the most disadvantaged because of multiple redundant adverse factors (Muller et al. 2018).

4.3. Stratified Parental Responses

Parental responses to early-life shocks could exacerbate or compensate for their harmful consequences on children, contributing to socioeconomic gaps over the life course. Wealthier parents are more likely to invest in children's development and enrichment (Schneider et al. 2018), to engage in health-increasing behaviors during pregnancy such as using prenatal care and abstaining from smoking (Pampel et al. 2010), and to negotiate with educational and other institutions to secure benefits for their children (Calarco 2018, Lareau 2011)—all of which may mitigate the effect of adverse exposures. Evidence about intrafamily dynamics provides indirect support for this claim, showing that advantaged parents are more likely to compensate for the negative consequences of early-life shocks, while disadvantaged parents tend to reinforce differences between siblings (Bernardi 2014, Hsin 2012).

4.4. Stratified Early-Life Survival

Selective attrition in the form of fetal loss or early-life mortality may further contribute to the stratification of observed effects. Attrition from prenatal and postnatal mortality will typically narrow (and in some cases reverse) stratification effects, because socioeconomically disadvantaged families are at higher risk both of adverse exposures and of fetal loss and infant mortality (Bozzoli et al. 2009, Nobles & Hamoudi 2019).

These four sources of stratification—exposure, sensitivity, parental responses, and early-life survival—could co-occur and even offset each other, for example, if both the sensitivity to prenatal stressors and the ability to compensate are more pronounced among advantaged populations, leading researchers to miss important socioeconomic heterogeneity that is hidden by these offsetting patterns.

4.5. Stratification in the Impact of Early-Life Exposures: Empirical Assessments

Multiple studies have considered variation in the impact of early-life exposures, generally using maternal education to measure socioeconomic advantage (occasionally, researchers have used racial minoritized status, occupation, marital status, and wealth). Most of these studies report a stronger, or exclusive, impact among disadvantaged groups (Bozzoli & Quintana-Domeque 2014, Brown 2018, Cil & Kim 2022, Conte Keivabu & Cozzani 2022, Cozzani et al. 2022, Foureaux Koppensteiner & Manacorda 2016, Jayachandran 2009, Le & Nguyen 2021, Mark & Torrats-Espinosa 2022, O'Brien et al. 2018, Torche 2018, Torche & Villarreal 2014), although some report no significant socioeconomic variation (Currie & Rossin-Slater 2013, Guantai & Kijima 2020, Mansour & Rees 2012, Persson & Rossin-Slater 2018, Vyas 2023), and at least in one case, a positive socioeconomic gradient is found, in which the effect of pollution is stronger among advantaged mothers (Rauscher & Cao 2024).

This nascent body of work does not rise yet to a systematic assessment. Measures of socioeconomic advantage are usually ad-hoc and rely on convention and data availability. In some studies, it is not clear whether researchers did not report socioeconomic heterogeneity because they did not consider it or because they did not find evidence for it. Furthermore, sample size constraints might reduce the ability to detect variation. Largely, the literature documenting heterogeneity in the impact of early-life exposures has not attempted to identify the mechanisms at play. When it does, researchers tend to provide indirect evidence on a single mechanism at a time. For example, Conte Keivabu & Cozzani (2022) rely on evidence of wide socioeconomic gaps in access to air conditioning to suggest that the stronger impact of prenatal exposure to extreme heat on disadvantaged children is driven by stratified exposure. Other research has found evidence of stratified parental responses to prenatal stressors in the form of smoking (Quintana-Domeque & Ródenas-Serrano 2017) and prenatal care use (Torche & Villarreal 2014), suggesting that a socioeconomic gradient in parental responses drives stratification in effects. These are plausible hypotheses, but adjudicating across different mechanisms requires simultaneous testing of all of them.

Torche (2018) explicitly attempts to adjudicate between different mechanisms for stratification in the impact of prenatal exposure to an earthquake on children's cognitive ability. She empirically rules out differential in-utero survival and directly measures exposure to the earthquake (using multiple survey indicators, such as whether the dwelling suffered any damage, electric power was lost, or the household suffered a drop in income) and sensitivity to the earthquake (using questions such as "everything brought back memories of the earthquake," "I had difficulty sleeping," or "I felt irritable and edgy"). She documented minimal socioeconomic variation in these mechanisms, arguing that the most likely stratification pathway was differential parental responses. This

strategy is challenging, as it relies on primary data collection among the affected populations to capture the experience and corollaries of prenatal exposures.

As a discipline, sociology has developed expertise in stratification processes, their embeddedness in institutions and power, and the social and cultural mechanisms driving them. Given this expertise, sociologists are well positioned to examine the stratification in the impact of early-life exposures, its mechanisms, and its contribution to the persistence of inequality across generations.

5. METHODOLOGICAL CONSIDERATIONS AND STRATEGIES

Providing high-quality evidence of the impact of early-life exposures is a challenging task. We discuss several common issues of design and analysis and highlight how they have been navigated by researchers advancing the field.

5.1. Mechanisms

A key challenge is understanding how exposures shape well-being across the life course. Most scholarship studies environmental disruptions that are reasonably well-defined in time and place. These events can be large in scale; they capture multiple, simultaneous "treatments." Identifying which factor(s) drive effects—for example, infrastructure damage, economic shocks, maternal stress, decline in prenatal care access, or compositional changes via migration, mortality, and fertility—is difficult. Maternal stress is arguably overused in the field as both a physiological response in the pregnant person and a catch-all explanation for adverse effects. Consideration of mechanisms is also often data driven rather than theory driven. For example, it is unlikely that maternal smoking and access to prenatal care are primary explanations for most of the US exposures discussed here, but they are often used because they are available in vital statistics.

Attention to the bundled nature of treatments is critical given the use of specific prenatal exposures to identify the effect of generalizable mechanisms. For example, some studies use natural disasters and violent events as instruments to capture the effect of maternal stress. The goal is worthwhile, but is it possible? Exposures such as natural disasters and violent crime are indubitably stressful events (Schneiderman et al. 2005, Villarreal & Yu 2017), and there are plausible physiological, endocrine, and immune pathways linking maternal stress with adverse outcomes at birth and during childhood (Beijers et al. 2014, Dunkel Schetter 2011). In most cases, however, it is unlikely that stress is the only, or primary, pathway linking exposure to outcomes—i.e., the exclusion restriction that supports instrumental variable identification is unwarranted.

What is the applied researcher to do? Scholars have examined environmental exposures characterized by minimal mortality, displacement, economic disruption, or changes to health care provision, arguing that stress is the only plausible pathway of influence (Kim et al. 2017, Torche 2011). Others control away alternative pathways, such as changes in medical care (Currie & Rossin-Slater 2013) or declines in economic activity (Tian et al. 2022). These strategies are presumed to leave stress as the only "residual explanation that remains when we have ruled out other possibilities" (Currie & Rossin-Slater 2013, p. 499). This is a strong claim. Setting aside estimation bias emerging from controlling for post-treatment factors such prenatal care use (Elwert & Winship 2014), it is difficult to establish that nothing other than maternal stress changed after events such as natural disasters or terrorist attacks.

This limitation probes our ability to identify mechanisms such as stress. If the events studied have corollaries other than maternal stress, then the estimated effects are an upper bound. In the extreme, maternal stress could have a null or substantively irrelevant impact, and it is difficult to even assess effect overestimation, given the widely different characteristics of the events studied. Researchers could abandon the hope of using environmental exposures as instruments for stress,

restricting estimation to the impact of the exposures themselves. But if they did so, variation in the magnitude and characteristics of each event would matter tremendously. We do not have a conclusive answer to these questions, but they are relevant concerns as the causal literature on prenatal exposures matures and attention shifts from celebrating clever identification strategies to discussing the substantive and policy implications of the findings.

5.2. Effect Magnitude and Sources of Bias

The combination of natural experiments and causal inference with population-representative data alleviates confounding in the association between the exposure and adverse outcomes. However, other sources of bias remain, as suggested by the enormous variation in effect magnitude across studies with putatively similar exposures (see **Figure 1**). Bias arises in part because similar exposures bundle diverse treatments of varying geographic scope and duration. Additionally, intent-to-treat designs, which are common in this research, capture population-level effects including individuals that are minimally or not exposed at all and will usually provide an underestimation of the effect of the treatment on the treated. Spillover effects—when the exposure has implications for the control population, too—can bias effects in either direction, depending on the nature of the spillover. Effect estimates will be downwardly biased when social networks extend the reach of effects into control populations, for example, if resources are relocated to affected areas from unaffected adjacent areas. By contrast, effect estimates will be upwardly biased when adverse exposures incur benefits for the untreated population.

5.3. Population Composition Bias: Fertility, Migration, Survival

Environmental exposures often happen in the context of demographic change that alters population composition. Ignoring composition changes can introduce significant estimate bias. For example, Beach et al. (2022) revisit the effects of in-utero exposure to the 1918 flu on later-life socioeconomic status as described by Almond (2006), and they demonstrate that the exposed cohort came from lower-socioeconomic-status families due to wartime deployment and potential selective fertility; this population composition change was sufficiently large to drive much of the long-term effect previously attributed to prenatal influenza exposure. Understanding and characterizing concurrent demographic change is an important and often achievable study design element.

In some cases, demographic processes mediate the effects of exposures—e.g., if conflict both displaces people and subjects them to worse living conditions. In these cases, bias is introduced if the processes selectively remove observations from study—e.g., via fetal death or outmigration—and the attrition is ignored (see Sections 1 and 4). Approaches to detect and remedy this include (*a*) tests for discontinuities in cohort composition on observed covariates with high-temporal-resolution data (Isen et al. 2017, appendix table S8), (*b*) explicit measurement of attrition in panel studies, (*c*) ancillary tests for migration or fetal death (Cozzani et al. 2022, Torche & Villarreal 2014), and (*d*) modeling of fetal loss or early mortality with simulations and/or distributional assumptions (Bozzoli et al. 2009, Nobles & Hamoudi 2019).

5.4. Taking Insights from Different Disciplines Seriously

Currently, scholarship on early-life exposures proceeds largely in silos, with scholars in different fields taking insights from their fields only. This leads to avoidable mistakes. A salient example is outcome selection: Social scientists are usually unaware that some variables widely used as infant health outcomes are measured poorly in vital records. While gestational age and birthweight are measured well in US birth certificates, severe maternal morbidity and complications of pregnancy and delivery are not (Gemmill & Leonard 2022). The same applies to maternal smoking and

prenatal care, which are widely used to capture mechanisms (Jurek & Greenland 2013, Penrod & Lantz 2000). Furthermore, data quality varies depending on class, race, and place (Thoma et al. 2019), compromising findings about stratification (Corman et al. 2019). Including facility or enumerator fixed effects can alleviate some location-specific recording errors. A better approach is to pursue linkages between administrative data and data sources with better measurement of a wider variety of outcomes, such as electronic health records. At very least, outcome selection should be informed by research on measurement validity in epidemiology.

Another common example is the use of birthweight as an outcome measure, given its ready availability in vital statistics. It is now well established that birthweight results from two processes with distinct etiologies and implications for child development: intrauterine growth and gestational age (Hobel et al. 2008). Given this, biomedical research now actively discourages the use of birthweight as a population health measure and encourages the separate study of distinct outcomes such as preterm birth and intrauterine growth restriction. The social sciences would benefit from closer (or any!) reading of expert literature on the variables used, be it the biomedical, psychological, or other social sciences.

5.5. Tests of Symmetry

The most common approach to uncover effects of prenatal exposures is to leverage spatial-temporal variation in the exposure and employ place-time fixed effects (or difference-in-differences) in a regression framework. Methodological concerns of two-way fixed effect designs are reviewed elsewhere (de Chaisemartin & D'Haultfoeuille 2023). Here, we note that these designs assume symmetric effects of increases and decreases in the exposure—e.g., that the negative effect of an increase in local homicides is the exact inverse of the positive effect of a decrease in homicides. This assumption is never tested, despite strong theoretical support for effect asymmetry. For example, to the extent that maternal stress is a function of the perception of worsening conditions, social psychology demonstrates that the emergence of an adverse exposure is often more salient than its decline (Montgomery 2000).

5.6. Publication Bias and (Pre-)Registration of Research

Publication bias is the failure to submit and publish results based on the findings—especially, the preference for statistically significant effects over substantively relevant questions (Christensen et al. 2019). As in all fields, this source of bias distorts our understanding of prenatal exposure impacts, leading to many null findings going unreported (Franco et al. 2014, Nobles & Hamoudi 2019). Techniques to adjust for publication bias are widely available but are still ignored in the field (Andrews & Kasy 2019). The slow trend toward preregistering research may reduce some of these problems (Christensen et al. 2019); so far, sociology has been slower than other disciplines to adopt this approach. Incentives from journals requiring preregistration and supporting publication of null findings may be a way forward to improve the quality of research and reduce the risk of selective publication (Christensen et al. 2019). The novel idea of registration takes preregistration one step further, with studies being reviewed based on the scientific value of the question and analysis rather than the results (Manago 2023). Because learning that a plausible adverse exposure does not have an effect is as important as detecting effects (insofar as the null effect is precisely identified), the field would benefit from a registered report approach.

6. CONCLUSIONS

Theoretical approaches and early descriptive research have suggested that environmental exposures during pregnancy are consequential for multiple domains of well-being, starting at birth. A

robust literature emerging over the past two decades supports this hypothesis. **Figure 1** compiles some evidence discussed in this review and shows statistically significant, and plausibly causal, effects of prenatal exposure to violence, natural disasters and weather events, extreme temperatures, and pollution on measures of infant health such as preterm birth or low birthweight. **Figure 1** also shows that many of these effects appear modest in size. Two factors underscore the relevance of these exposures, however. First, even a modest increase in preterm birth by, say, 1 percentage point, will have vast implications if it affects large populations. If an exposure such as rising temperatures, for example, affected the entire United States in 2022, it would result in more than 36,000 additional preterm births (3.66 million births × 1%) that would have not occurred in the absence of the harmful exposure, imposing enormous costs for families and society. Second, any impact of prenatal exposures on early-life health could persist over the affected cohorts' life courses, shaping later outcomes such as cognitive development, educational attainment, and earnings, with substantial consequences over time.

Our argument does not diminish the relevance of exposures at later ages, e.g., in childhood or adolescence. Though the impact of some environmental exposures may be particularly acute in pregnancy, other exposures could be buffered by maternal biological systems (e.g., transient nutrition shocks; Thayer et al. 2020) and may be more consequential when suffered in childhood. Comparisons of the relative magnitude of prenatal and childhood effects have not yet been pursued with the kind of rigorous study design that characterizes the studies referenced above. Current evidence unambiguously identifies the prenatal period as a sensitive developmental stage. Ignoring this stage misses a moment in which intergenerational transfers occur and in which life trajectories are patterned in ways that undergird all subsequent individual exposures. This pattern has relevant policy implications, including the systematic underestimation of returns to investments in the health of reproductive-age populations (Hoynes et al. 2016).

We have argued that the prenatal exposures we consider are fundamentally social phenomena: their emergence, allocation, and impact are structured by institutions and power. As a result, early-life environmental exposures are a critical part of the sociological understanding of stratification and intergenerational mobility. We discuss several implications of the findings reviewed here.

The most striking implication is that life course trajectories are indeed initiated very early. The processes of stratification that we study—even those that occur as early as young childhood—are already undergirded by inequalities in exposures to toxicants, neurological development, and epigenetic change, likely compounded by unequal behavioral responses to environmental insults. This pattern has two immediate implications. First, early differences in endowments of infants are in part the result of social phenomena, even when they manifest through biological mechanisms, and should not be confused with genetic resources. Second, prenatal exposure effects documented here underscore the potentially large (and previously underestimated) value of policies and interventions that improve the welfare of reproductive-age populations, particularly among the most disadvantaged and marginalized groups. Note that the precise mechanisms to best support the well-being of the next generation still require further clarification—i.e., how do these adverse exposures render insults on developing pregnancies, and why are these impacts patterned by socioeconomic advantage (Section 5)? This is an ongoing task for the field.

Critically, because the prenatal period provides the most basic of links across generations—driven by the physiological dependence of the fetus on the pregnant person—any exposure and its consequences are certain to have intergenerational effects. Prenatal exposures explicitly concern the circumstances of parents, and therefore they provide another arm of scientific support for a core premise in stratification research: that the life course has an inherent intergenerational component (Duncan 1969). That a child born today is biologically endowed with unequal resources connected to the adverse environments that parents experienced during pregnancy further

supports forms of progressive social policy whose consequences might materialize only decades later.

The legacy of prenatal exposures has general implications for scholars studying population welfare. First, the study of cohorts, starting at birth and tracking them over the life course, has much to contribute to the study of change in population well-being. This is an old idea (Elder 1994, Palloni & Beltrán-Sánchez 2017, Palloni et al. 2023, Ryder 1985). It bears repeating in the contemporary data environment in which cross-sectional information is voluminous and in which tracking cohorts over time is expensive and administratively challenging. Second, if effects of early exposures are at least partially latent, it is easy to miss their origin in experiences occurring decades earlier. The findings reviewed here suggest potential long temporal lags between exposures and markers of individual and population welfare—e.g., the structure of wages among US adults in the 2000s is in part a function of extreme temperature conditions in the 1970s (Isen et al. 2017). These temporal lags are often left unexplored in traditional assessments of population welfare, in favor of a focus on more contemporaneous and proximate determinants of socioeconomic outcomes.

We have reviewed the literature examining prenatal exposures to natural disasters and weather events, extreme temperatures, pollution, and environmental violence. These exposures are, unfortunately, prevalent and stratified, and likely to become more so in the near future. This review did not, however, consider other pregnancy exposures known to be harmful—especially exposures to poverty and racism—because rigorous studies identifying the effects of these exposures during pregnancy are still in development. For key exceptions, we point the reader to a small but growing, well-designed scholarship on the legacy of antipoverty programs in the United States (East et al. 2023, Hoynes et al. 2016, Miller & Wherry 2019) and to studies that capture exposures to overt periods of racism and xenophobia against specific groups in the United States (Gemmill et al. 2019, Lauderdale 2006, Samari et al. 2020, Vu et al. 2023).

An essential element of research that uncovers the impact of exposures in pregnancy is attention to how the work is used. We now have ample evidence of the ways that research findings have been used to motivate the surveillance and criminalization of pregnant people, their bodies, and their behaviors (Roberts 1998). This is not a fringe concern—framing and clarity around the implications of research that uncovers effects of prenatal exposures are critical (Flavin 2008, Hammer & Burton-Jeangros 2013). This issue is more relevant given evidence on differential effects of adverse exposures on low-income and racialized populations (Section 4), whose reproduction is also differentially monitored and punished (Roberts 1998, Ross & Solinger 2017).

Finally, we have argued that the prenatal period is an important moment in the life course in which social stratification manifests—a moment in which the parent and child's generation are linked and a moment in which the social patterning of environmental conditions becomes biologically embedded, with consequences that may proliferate over the life course. In an era of rising economic and natural resource inequality, increasing climate hardship, and persistent discrimination, adverse exposures in utero may well increase in frequency and severity, contributing to socioeconomic inequality. By combining theoretical understanding of stratification processes with diverse methodological approaches, sociologists can make these processes visible, elucidating how early-life exposures shape individual biographies, cohort trajectories, and population well-being.

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LITERATURE CITED

Almond D. 2006. Is the 1918 influenza pandemic over? Long-term effects of *in utero* influenza exposure in the post-1940 U.S. population. *J. Political Econ.* 114(4):672–712

Almond D, Currie J. 2011. Killing me softly: the fetal origins hypothesis. *J. Econ. Perspect.* 25(3):153–72

Almond D, Mazumder B. 2011. Health capital and the prenatal environment: the effect of Ramadan observance during pregnancy. *Am. Econ. J. Appl. Econ.* 3(4):56–85

Andrews I, Kasy M. 2019. Identification of and correction for publication bias. *Am. Econ. Rev.* 109(8):2766–94

Arcaya M, Raker EJ, Waters MC. 2020. The social consequences of disasters: individual and community change. *Annu. Rev. Sociol.* 46:671–91

Barker D. 1990. The fetal and infant origins of adult disease. *BMJ* 301(156):1111

Barker D. 1998. In utero programming of chronic disease. *Clin. Sci.* 95:115–28

Barker D, Godfrey KM, Gluckman PD, Harding JE, Owens JA, Robinson JS. 1993. Fetal nutrition and cardiovascular disease in adult life. *Lancet* 341(8850):938–41

Barker D, Osmond C. 1986. Infant mortality, childhood nutrition, and ischaemic hearth disease in England and Wales. *Lancet* 1(8489):1077–81

Barreca A, Schaller J. 2020. The impact of high ambient temperatures on delivery timing and gestational lengths. *Nat. Clim. Change* 10(1):77–82

Beach B, Brown R, Ferrie J, Saavedra M, Thomas D. 2022. Reevaluating the long-term impact of in utero exposure to the 1918 influenza pandemic. *J. Political Econ.* 130(7):1963–90

Behrman R, Butler AS. 2007. *Preterm Birth: Causes, Consequences, and Prevention*, Vol. II. Washington, DC: Natl. Acad. Press

Beijers R, Buitelaar JK, de Weerth C. 2014. Mechanisms underlying the effects of prenatal psychosocial stress on child outcomes: beyond the HPA axis. *Eur. Child Adolesc. Psychiatry* 23(10):943–56

Bekkar B, Pacheco S, Basu R, DeNicola N. 2020. Association of air pollution and heat exposure with preterm birth, low birth weight, and stillbirth in the US: a systematic review. *JAMA Netw. Open* 3(6):e208243

Ben-Shlomo Y, Kuh D. 2002. A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. *Int. J. Epidemiol.* 31:285–93

Bernardi F. 2014. Compensatory advantage as a mechanism of educational inequality: a regression discontinuity based on month of birth. *Sociol. Educ.* 87(2):74–88

Bharadwaj P, Gibson M, Zivin JG, Neilson C. 2017. Gray matters: fetal pollution exposure and human capital formation. *J. Assoc. Environ. Resour. Econ.* 4(2):505–42

Bharadwaj P, Løken KV, Neilson C. 2013. Early life health interventions and academic achievement. *Am. Econ. Rev.* 103(5):1862–91

Blackwell DL, Hayward MD, Crimmins EM. 2001. Does childhood health affect chronic morbidity in later life? *Soc. Sci. Med.* 52(8):1269–84

Blanc N, Liao J, Gilliland F, Zhang J, Berhane K, et al. 2023. A systematic review of evidence for maternal preconception exposure to outdoor air pollution on children's health. *Environ. Pollut.* 318:120850

Bock J, Wainstock T, Braun K, Segal M. 2015. Stress in utero: prenatal programming of brain plasticity and cognition. *Biol. Psychiatry* 78(5):315–26

Boustan LP, Kahn ME, Rhode PW, Yanguas ML. 2020. The effect of natural disasters on economic activity in US counties: A century of data. *J. Urban Econ.* 118:103257

Bozzoli C, Deaton A, Quintana-Domeque C. 2009. Adult height and childhood disease. *Demography* 46(4):647–69

Bozzoli C, Quintana-Domeque C. 2014. The weight of the crisis: evidence from newborns in Argentina. *Rev. Econ. Stat.* 96(3):550–62

Brown R. 2005. Critical period. In *Encyclopedia of Human Development*, ed. NJ Salkind, pp. 325–27. Thousand Oaks, CA: SAGE

Brown R. 2018. The Mexican drug war and early-life health: the impact of violent crime on birth outcomes. *Demography* 55(1):319–40

Bruckner TA, Catalano R. 2018. Selection in utero and population health: theory and typology of research. *SSM Popul. Health* 5:101–13

Buggs SAL, Kravitz-Wirtz ND, Lund JJ. 2022. Social and structural determinants of community firearm violence and community trauma. *Ann. Am. Acad. Political Soc. Sci.* 704(1):224–41

Calarco J. 2018. *Negotiating Opportunities: How the Middle Class Secures Advantages in School*. Oxford, UK: Oxford Univ. Press

Camacho A. 2008. Stress and birth weight: evidence from terrorist attacks. *Am. Econ. Rev.* 98(2):511–15

Caruso G, Miller S. 2015. Long run effects and intergenerational transmission of natural disasters: a case study on the 1970 Ancash earthquake. *J. Dev. Econ.* 117:134–50

Case A, Fertig A, Paxson C. 2005. The lasting impact of childhood health and circumstance. *J. Health Econ.* 24(2):365–89

Case A, Paxson C. 2009. Early life health and cognitive function in old age. *Am. Econ. Rev.* 99(2):104–9

Catalano R, Bruckner T. 2006. Secondary sex ratios and male lifespan: damaged or culled cohorts. *PNAS* 103(5):1639–43

Chen X, Tan CM, Zhang X, Zhang X. 2020. The effects of prenatal exposure to temperature extremes on birth outcomes: the case of China. *J. Popul. Econ.* 33:1263–302

Chen Y, Zhou L-A. 2007. The long-term health and economic consequences of the 1959–1961 famine in China. *J. Health Econ.* 26(4):659–81

Chersich MF, Pham MD, Areal A, Haghighi MM, Manyuchi A, et al. 2020. Associations between high temperatures in pregnancy and risk of preterm birth, low birth weight, and stillbirths: systematic review and meta-analysis. *BMJ* 371:m3811

Christensen G, Freese J, Miguel E. 2019. *Transparent and Reproducible Social Science Research: How to Do Open Science*. Berkeley: Univ. Calif. Press

Cil G, Kim J. 2022. Extreme temperatures during pregnancy and adverse birth outcomes: evidence from 2009 to 2018 U.S. national birth data. *Health Econ*. 31(9):1993–2024

Clouston SAP, Link BG. 2021. A retrospective on fundamental cause theory: state of the literature and goals for the future. *Annu. Rev. Sociol.* 47:131–56

Conley D, Strully K, Bennett NG. 2003. *The Starting Gate: Birth Weight and Life Chances*. Berkeley: Univ. Calif. Press

Conte Keivabu R, Cozzani M. 2022. Extreme heat, birth outcomes, and socioeconomic heterogeneity. *Demography* 59(5):1631–54

Corman H, Dave D, Reichman NE, Corman H, Dave D, Reichman NE. 2019. The effects of prenatal care on birth outcomes: reconciling a messy literature. In *Oxford Research Encyclopedia of Economics and Finance*. **https://doi.org/10.1093/acrefore/9780190625979.013.375**

Costa DL. 2000. Understanding the twentieth-century decline in chronic conditions among older men. 37(1):53–72

Cozzani M, Triventi M, Bernardi F. 2022. Maternal stress and pregnancy outcomes evidence from a natural experiment: the 2004 Madrid train bombings. *Eur. Sociol. Rev.* 38(3):390–407

Cunha F, Heckman J. 2007. The technology of skill formation. *Am. Econ. Rev. Pap. Proc.* 97(2):31–47

Currie J. 2011. Inequality at birth: some causes and consequences. *Am. Econ. Rev.* 101(3):1–22

Currie J. 2013. Pollution and infant health. *Child Dev. Perspect.* 7(4):237–42

Currie J, Rossin-Slater M. 2013. Weathering the storm: hurricanes and birth outcomes. *J. Health Econ.* 32(3):487–503

Currie J, Zivin JG, Mullins J, Neidell M. 2014. What do we know about short- and long-term effects of early-life exposure to pollution? *Annu. Rev. Resour. Econ.* 6:217–47

de Chaisemartin C, D'Haultfoeuille X. 2023. Two-way fixed effects and differences-in-differences with heterogeneous treatment effects: a survey. *Econom. J.* 26(3):C1–30

Deschênes O, Greenstone M, Guryan J. 2009. Climate change and birth weight. *Am. Econ. Rev.* 99(2):211–17

Dorélien A, Grace K. 2023. Climate change-related demographic and health research: data and approaches. In *Environmental Impacts on Families: Change, Challenge, and Adaptation*, ed. SE Ortiz, SM McHale, V King, JE Glick pp. 43–66. New York: Springer
Duncan OD. 1969. Inequality and opportunity. *Popul. Index* 35:361–66
Dunkel Schetter C. 2011. Psychological science on pregnancy: stress processes, biopsychosocial models, and emerging research issues. *Annu. Rev. Psychol.* 62:531–58
East CN, Miller S, Page M, Wherry LR. 2023. Multigenerational impacts of childhood access to the safety net: early life exposure to Medicaid and the next generation's health. *Am. Econ. Rev.* 113(1):98–135
Elder GH. 1994. Time, human agency, and social change: perspectives on the life course. *Soc. Psychol. Q.* 57(1):4–15
Elo IT, Preston SH. 1992. Effects of early-life conditions on adult mortality: a review. *Popul. Index* 58(2):186–212
Elsässer L, Schäfer A. 2023. Political inequality in rich democracies. *Annu. Rev. Political Sci.* 26:469–87
Elwert F, Winship C. 2014. Endogenous selection bias: the problem of conditioning on a collider variable. *Annu. Rev. Sociol.* 40:31–53
Erikson K. 2012 (1976). *Everything in Its Path: Destruction of Community in the Buffalo Creek Flood*. New York: Simon and Schuster
Erikson RS. 2015. Income inequality and policy responsiveness. *Annu. Rev. Political Sci.* 18:11–29
Feder A, Nestler EJ, Charney DS. 2009. Psychobiology and molecular genetics of resilience. *Nat. Rev. Neurosci.* 10(6):446–57
Fishman R, Carrillo P, Russ J. 2019. Long-term impacts of exposure to high temperatures on human capital and economic productivity. *J. Environ. Econ. Manag.* 93:221–38
Flavin J. 2008. *Our Bodies, Our Crimes: The Policing of Women's Reproduction in America*. New York: NYU Press
Forsdahl A. 1977. Are poor living conditions in childhood and adolescence an important risk factor for arteriosclerotic heart disease? *Br. J. Prev. Soc. Med.* 31:91–95
Foureaux Koppensteiner M, Manacorda M. 2016. Violence and birth outcomes: evidence from homicides in Brazil. *J. Dev. Econ.* 119:16–33
Franco A, Malhotra N, Simonovits G. 2014. Publication bias in the social sciences: unlocking the file drawer. *Science* 345(6203):1502–5
Frankenberg E, Friedman J, Ingwersen N, Thomas D. 2017. Linear child growth after a natural disaster: a longitudinal study of the effects of the 2004 Indian Ocean tsunami. *Lancet* 389:S21
Gemmill A, Catalano R, Casey JA, Karasek D, Alcalá HE, et al. 2019. Association of preterm births among US Latina women with the 2016 presidential election. *JAMA Netw. Open.* 2(7):1–10
Gemmill A, Leonard S. 2022. Comment: Risk of adverse pregnancy outcomes among US individuals with gestational diabetes by race and ethnicity. *JAMA* 328(4):397
Geronimus AT. 1992. The weathering hypothesis and the health of African-American women and infants: evidence and speculations. *Ethn. Dis.* 2:207–21
Geruso M, Spears D. 2018. *Heat, humidity, and infant mortality in the developing world*. NBER Work. Pap. 24870
Gluckman P, Hanson M, Cooper C, Thornburg K. 2008. Effects of in-utero and early-life conditions on adult health and disease. *New Engl. J. Med.* 359(1):61–73
Godfrey KM, Barker DJP. 2001. Fetal programming and adult health. *Public Health Nutr.* 4(2B):611–24
Goyal N, Karra M, Canning D. 2019. Early-life exposure to ambient fine particulate air pollution and infant mortality: pooled evidence from 43 low-and middle-income countries. *Int. J. Epidemiol.* 48(4):1125–41
Grace K, Verdin A, Dorélien A, Davenport F, Funk C, Husak G. 2021. Exploring strategies for investigating the mechanisms linking climate and individual-level child health outcomes: an analysis of birth weight in Mali. *Demography* 58(2):499–526
Greve J, Schultz-Nielsen ML, Tekin E. 2017. Fetal malnutrition and academic success: evidence from Muslim immigrants in Denmark. *Econ. Educ. Rev.* 60:20–35
Gronlund C. 2014. Racial and socioeconomic disparities in heat-related health effects and their mechanisms: a review. *Curr. Epidemiol. Rep.* 1(3):165–73
Grossman M. 1972. On the concept of health capital and the demand for health. *J. Political Econ.* 80(2):223–55
Guantai F, Kijima Y. 2020. Ethnic violence and birth outcomes: evidence from exposure to the 1992 conflict in Kenya. *Demography* 57(2):423–44

Gump BB, Matthews KA. 1999. Do background stressors influence reactivity to and recovery from acute stressors? *J. Appl. Soc. Pyschol.* 29(3):469–94

Hajat A. 2015. Socioeconomic disparities and air pollution exposure: a global review. *Curr. Environ. Health Rep.* 2(4):440–50

Hammer RP, Burton-Jeangros C. 2013. Tensions around risks in pregnancy: a typology of women's experiences of surveillance medicine. *Soc. Sci. Med.* 93:55–63

Hayward MD, Gorman BK. 2004. The long arm of childhood: the influence of early-life social conditions on men's mortality. *Demography* 41(1):87–107

Heckman JJ. 2006. Skill formation and the economics of investing in disadvantaged children. *Science* 312(5782):1900–2

Hertzman C, Boyce T. 2010. How experience gets under the skin to create gradients in developmental health. *Annu. Rev. Public Health* 31:329–47

Hobel CJ, Goldstein A, Barrett ES. 2008. Psychosocial stress and pregnancy outcome. *Clin. Obstet. Gynecol.* 51(2):333–48

Hoffman JS, Shandas V, Pendleton N. 2020. The effects of historical housing policies on resident exposure to intra-urban heat: a study of 108 US urban areas. *Climate* 8(1):12

Hong SC, Kim D, Lee E. 2021. The long-term human toll of natural disasters: a study of fetal exposure to the 1974 tornado super outbreak. *Appl. Econ.* 53(4):469–81

Hoynes H, Schanzenbach DW, Almond D. 2016. Long-run impacts of childhood access to the safety net. *Am. Econ. Rev.* 106(4):903–34

Hsin A. 2012. Is biology destiny? Birth weight and differential parental treatment. *Demography* 49(4):1385–405

Isen A, Rossin-Slater M, Walker R. 2017. Relationship between season of birth, temperature exposure, and later life wellbeing. *PNAS* 114(51):13447–52

James WH, Grech V. 2018. Can sex ratios at birth be used in the assessment of public health, and in the identification of causes of selected pathologies? *Early Hum. Dev.* 118:15–21

Jayachandran S. 2009. Air quality and early-life mortality: evidence from Indonesia's wildfires. *J. Hum. Resour.* 44(4):916–54

Jurek AM, Greenland S. 2013. Adjusting for multiple-misclassified variables in a study using birth certificates. *Ann. Epidemiol.* 23(8):515–20

Karbownik K, Wray A. 2019. Long-run consequences of exposure to natural disasters. *J. Labor Econ.* 37(3):949–1007

Kermack WO, McKendrick AG, McKinlay PL. 1934. Death-rates in Great Britain and Sweden. Some general regularities and their significance. *Lancet* 226:698–703

Kim B, Carruthers CK, Harris MC. 2017. Maternal stress and birth outcomes: evidence from the 1994 Northridge earthquake. *J. Econ. Behav. Organ.* 140:354–73

Kim J, Lee A, Rossin-Slater M. 2021. What to expect when it gets hotter: the impacts of prenatal exposure to extreme temperature on maternal health. *Am. J. Health Econ.* 7(3):281–305

King KE, Morenoff JD, House JS. 2011. Neighborhood context and social disparities in cumulative biological risk factors. *Psychosomatic Med.* 73(7):572–79

Kuzawa C, Kim A. 2022. Early environments, developmental plasticity, and chronic degenerative disease. In *Human Growth and Development*, ed. N Cameron, B Bogin, pp. 325–41. Amsterdam: Elsevier. 2nd ed.

Landecker H, Panofsky A. 2013. From social structure to gene regulation, and back: a critical introduction to environmental epigenetics for sociology. *Annu. Rev. Sociol.* 39:333–57

Lapehn S, Paquette AG. 2022. The placental epigenome as a molecular link between prenatal exposures and fetal health outcomes through the DOHaD hypothesis. *Curr. Envir. Health Rep.* 9(3):490–501

Lareau A. 2011. *Unequal Childhoods: Class, Race, and Family Life*. Berkeley: Univ. Calif. Press. 2nd ed.

Lau C, Ambalavanan N, Chakraborty H, Wingate MS, Carlo WA. 2013. Extremely low birth weight and infant mortality rates in the United States. *Pediatrics* 131(5):855–60

Lauderdale D. 2006. Birth outcomes for Arabic-named women in California before and after September 11. *Demography* 43(1):185–201

Lautharte I. 2021. Babies and bandidos: birth outcomes in pacified favelas of Rio de Janeiro. *J. Health Econ.* 77:102457

Le K, Nguyen M. 2021. The impacts of temperature shocks on birth weight in Vietnam. *Popul. Dev. Rev.* 47(4):1025–47

Li C, Lumey L. 2017. Exposure to the Chinese famine of 1959–61 in early life and long-term health conditions: a systematic review and meta-analysis. *Int. J. Epidemiol.* 46(4):1157–70

Link BG, Phelan J. 1995. Social conditions as fundamental causes of disease. *J. Health Soc. Behav.* 35:80–94

Lumey LH. 1998. Reproductive outcomes in women prenatally exposed to undernutrition: a review of findings from the Dutch famine birth cohort. *Proc. Nutr. Soc.* 57(1):129–35

Luu TM, Rehman Mian MO, Nuyt AM. 2017. Long-term impact of preterm birth. *Clin. Perinatol.* 44(2):305–14

Lynch J, Smith GD. 2005. A life course approach to chronic disease epidemiology. *Annu. Rev. Public Health* 26:1–35

Manago B. 2023. Preregistration and registered reports in sociology: strengths, weaknesses, and other considerations. *Am. Sociol.* 54(1):193–210

Mansour H, Rees DI. 2012. Armed conflict and birth weight: evidence from the al-Aqsa Intifada. *J. Dev. Econ.* 99(1):190–99

Mark NDE, Torrats-Espinosa G. 2022. Declining violence and improving birth outcomes in the US: Evidence from birth certificate data. *Soc. Sci. Med.* 294:114595

Martin EM, Fry RC. 2018. Environmental influences on the epigenome: exposure-associated DNA methylation in human populations. *Annu. Rev. Public Health* 39:309–33

McEwen CA, McEwen BS. 2017. Social structure, adversity, toxic stress, and intergenerational poverty: an early childhood model. *Annu. Rev. Sociol.* 43:445–72

Menclova AK, Stillman S. 2020. Maternal stress and birth outcomes: evidence from an unexpected earthquake swarm. *Health Econ.* 29(12):1705–20

Miller S, Wherry LR. 2019. The long-term effects of early life Medicaid coverage. *J. Hum. Resour.* 54(3):785–824

Montgomery MR. 2000. Perceiving mortality decline. *Popul. Dev. Rev.* 26(4):795–819

Moster D, Lie RT, Markestad T. 2008. Long-term medical and social consequences of preterm birth. *New Engl. J. Med.* 359(3):262–73

Muller C, Sampson RJ, Winter AS. 2018. Environmental inequality: the social causes and consequences of lead exposure. *Annu. Rev. Sociol.* 44:263–82

Neelsen S, Stratmann T. 2011. Effects of prenatal and early life malnutrition: evidence from the Greek famine. *J. Health Econ.* 30(3):479–88

Nobles J, Hamoudi A. 2019. Detecting the effects of early-life exposures: why fecundity matters. *Popul. Res. Policy Rev.* 38(6):783–809

Noghanibehambari H. 2022. In utero exposure to natural disasters and later-life mortality: evidence from earthquakes in the early twentieth century. *Soc. Sci. Med.* 307:115189

Nyadanu SD, Dunne J, Tessema GA, Mullins B, Kumi-Boateng B, et al. 2022. Prenatal exposure to ambient air pollution and adverse birth outcomes: an umbrella review of 36 systematic reviews and meta-analyses. *Environ. Pollut.* 306:119465

O'Brien RL, Neman T, Rudolph K, Casey J, Venkataramani A. 2018. Prenatal exposure to air pollution and intergenerational economic mobility: evidence from U.S. county birth cohorts. *Soc. Sci. Med.* 217:92–96

Painter RC, Roseboom TJ, Bleker OP. 2005. Prenatal exposure to the Dutch famine and disease in later life: an overview. *Reprod. Toxicol.* 20(3):345–52

Palloni A. 2006. Reproducing inequalities: luck, wallets, and the enduring effects of childhood health. *Demography* 43(4):587–615

Palloni A, Beltrán-Sánchez H. 2017. Discrete Barker frailty and warped mortality dynamics at older ages. *Demography* 54(2):655–71

Palloni A, Ramirez D, Daza S. 2023. Early childhood origins of modern social class health disparities. In *Handbook of Health Inequalities Across the Life Course*, ed. R Hoffmann, pp. 232–50. Cheltenham, UK: Edward Elgar

Pampel FC, Krueger PM, Denney JT. 2010. Socioeconomic disparities in health behaviors. *Annu. Rev. Sociol.* 36:349–70

Paneth N, Susser M. 1995. Early origin of coronary heart disease (the "Barker hypothesis"). *BMJ* 310(6977):411–12

Paudel J, Ryu H. 2018. Natural disasters and human capital: the case of Nepal's earthquake. *World Dev*. 111:1–12

Penrod JR, Lantz PM. 2000. Measurement error in prenatal care utilization: evidence of attenuation bias in the estimation of impact on birth weight. *Matern. Child Health J*. 4:39–52

Perera F, Herbstman J. 2011. Prenatal environmental exposures, epigenetics, and disease. *Reprod. Toxicol.* 31(3):363–73

Persson P, Rossin-Slater M. 2018. Family ruptures, stress, and the mental health of the next generation. *Am. Econ. Rev.* 108(4–5):1214–52

Preston SH, Hill ME, Drevenstedt GL. 1998. Childhood conditions that predict survival to advanced ages among African-Americans. *Soc. Sci. Med.* 47(9):1231–46

Provençal N, Binder EB. 2015. The effects of early life stress on the epigenome: from the womb to adulthood and even before. *Exp. Neurol.* 268:10–20

Quintana-Domeque C, Ródenas-Serrano P. 2017. The hidden costs of terrorism: the effects on health at birth. *J. Health Econ.* 56:47–60

Randell H. 2022. Heat, mortality, and health. In *International Handbook of Population and Environment*, ed. LM Hunter, C Gray, J Véron, pp. 283–99. New York: Springer

Randell H, Gray C, Grace K. 2020. Stunted from the start: early life weather conditions and child undernutrition in Ethiopia. *Soc. Sci. Med.* 261:113234

Rangel MA, Tomé R. 2022. Health and the megacity: urban congestion, air pollution, and birth outcomes in Brazil. *Int. J. Environ. Res. Public Health* 19(3):1151

Rangel MA, Vogl TS. 2019. Agricultural fires and health at birth. *Rev. Econ. Stat.* 101(4):616–30

Rasmussen KM. 2001. The "fetal origins" hypothesis: challenges and opportunities for maternal and child nutrition. *Annu. Rev. Nutr.* 21:73–95

Rauscher E, Cao X. 2024. Unequal effects of wildfire exposure on infant health by maternal education, 1995–2020. *RSF* 10(1):255–74

Roberts D. 1998. *Killing the Black Body: Race, Reproduction, and the Meaning of Liberty*. New York: Vintage

Rosales-Rueda M, Triyana M. 2019. The persistent effects of early-life exposure to air pollution evidence from the Indonesian forest fires. *J. Hum. Resour.* 54(4):1037–80

Roseboom TJ, Barker DJP, Bleker OP. 2001. Effects of prenatal exposure to the Dutch famine on adult disease in later life: an overview. *Mol. Cell. Endocrinol.* 185(1–2):93–98

Ross L, Solinger R. 2017. *Reproductive Justice: An Introduction*, Vol. 1. Berkeley: Univ. Calif. Press

Royer H. 2009. Separated at girth: US twin estimates of the effects of birth weight. *Am. Econ. J. Appl. Econ.* 1(1):49–85

Russ SA, Larson K, Tullis E, Halfon N. 2014. A lifecourse approach to health development: implications for the maternal and child health research agenda. *Matern. Child Health J*. 18(2):497–510

Ryder NB. 1985. The cohort as a concept in the study of social change. In *Cohort Analysis in Social Research: Beyond the Identification Problem*, ed. WM Mason, SE Fienberg, pp. 9–44. New York: Springer

Samari G, Catalano R, Alcalá HE, Gemmill A. 2020. The Muslim ban and preterm birth: analysis of US vital statistics data from 2009 to 2018. *Soc. Sci. Med.* 265:113544

Saulnier DD, Brolin K. 2015. A systematic review of the health effects of prenatal exposure to disaster. *Int. J. Public Health* 60(7):781–87

Schneider D, Hastings OP, LaBriola J. 2018. Income inequality and class divides in parental investments. *Am. Sociol. Rev.* 83(3):475–507

Schneiderman N, Ironson G, Siegel SD. 2005. Stress and health: psychological, behavior, and biological determinants. *Annu. Rev. Clin. Psychol.* 1:607–28

Selevan SG, Kimmel CA, Mendola P. 2000. Identifying critical windows of exposure for children's health. *Environ. Health Perspect*. 108(suppl. 3):451–55

Sharkey P. 2018. *Uneasy Peace: The Great Crime Decline, the Renewal of City Life, and the Next War on Violence*. New York: W.W. Norton

Simeonova E. 2011. Out of sight, out of mind? Natural disasters and pregnancy outcomes in the USA. *CESifo Econ. Stud.* 57(3):403–31

Spears D, Dey S, Chowdhury S, Scovronick N, Vyas S, Apte J. 2019. The association of early-life exposure to ambient PM2. 5 and later-childhood height-for-age in India: an observational study. *Environ. Health* 18(1):1–10

Squires G, Hartman C. 2006. *There Is No Such a Thing as a Natural Disaster: Race, Class, and Hurricane Katrina*. London: Routledge

St Clair D, Xu M, Wang P, Yu Y, Fang Y, et al. 2005. Rates of adult schizophrenia following prenatal exposure to the Chinese famine of 1959–1961. *JAMA* 294(5):557–62

Susser E, Hoek HW, Brown A. 1998. Neurodevelopmental disorders after prenatal famine: the story of the Dutch Famine Study. *Am. J. Epidemiol.* 147(3):213–16

Syed S, O'Sullivan TL, Phillips KP. 2022. Extreme heat and pregnancy outcomes: a scoping review of the epidemiological evidence. *Int. J. Environ. Res. Public Health* 19(4):2412

Thayer ZM, Kuzawa CW. 2011. Biological memories of past environments: epigenetic pathways to health disparities. *Epigenetics* 6(7):798–803

Thayer ZM, Rutherford J, Kuzawa CW. 2020. The maternal nutritional buffering model: an evolutionary framework for pregnancy nutritional intervention. *Evol. Med. Public Health* 2020(1):14–27

Thoma ME, De Silva DA, MacDorman MF. 2019. Examining interpregnancy intervals and maternal and perinatal health outcomes using US vital records: important considerations for analysis and interpretation. *Paediatr. Perinat. Epidemiol.* 33(1):O60–72

Tian X, Gong J, Zhai Z. 2022. Natural disasters and human capital accumulation: evidence from the 1976 Tangshan earthquake. *Econ. Educ. Rev.* 90:102304

Tierney K. 2019. *Disasters: A Sociological Approach*. Cambridge, UK: Polity

Timmermans S, Kaufman R. 2020. Technologies and health inequities. *Annu. Rev. Sociol.* 46:583–602

Torche F. 2011. The effect of maternal stress on birth outcomes: exploiting a natural experiment. *Demography* 48(4):1473–91

Torche F. 2018. Prenatal exposure to an acute stressor and children's cognitive outcomes. *Demography* 55:1611–39

Torche F, Brand J, Fletcher J. 2024. Disparate effects of disruptive events in children. *RSF* 10(1):1–30

Torche F, Shwed U. 2015. The hidden costs of war: exposure to armed conflict and birth outcomes. *Sociol. Sci.* 2:558–81

Torche F, Villarreal A. 2014. Prenatal exposure to violence and birth weight in Mexico: selectivity, exposure, and behavioral responses. *Am. Sociol. Rev.* 79(5):966–92

Triyana M, Xia X. 2023. Selective mortality and the long-term effects of early-life exposure to natural disasters. *Oxf. Bull. Econ. Stat.* 85(4):773–804

United Nations. 2020. *A new era of conflict and violence*. Fact Sheet, United Nations, Geneva

Vaupel JW, Manton KG, Stallard E. 1979. The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography* 16(3):439–54

Verdin A, Grace K, Davenport F, Funk C, Husak G. 2021. Can we advance individual-level heat-health research through the application of stochastic weather generators? *Clim. Change* 164:1–13

Villarreal A, Yu W-H. 2017. Crime, fear, and mental health in Mexico. *Criminology* 55(4):779–805

Vu H, Noghanibehambari H, Fletcher J, Green T. 2023. *Prenatal exposure to racial violence and later life mortality among males: evidence from lynching*. Work. Pap., Univ. Wis.–Madison, Madison, Wis. **https://hoa-vu.github.io/research/lynching_mortality.pdf**

Vyas S. 2023. The child health impacts of coal: evidence from India's coal expansion. *J. Hum. Resour.* 58(6):0320-10784R2

Wilde J, Apouey BH, Jung T. 2017. The effect of ambient temperature shocks during conception and early pregnancy on later life outcomes. *Eur. Econ. Rev.* 97:87–107

Wright AW, Austin M, Booth C, Kliewer W. 2017. Exposure to community violence and physical health outcomes in youth: a systematic review. *J. Pediatr. Psychol.* 42(4):364–78