21. Environmental inequality and health outcomes over the life course

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INTRODUCTION

Environmental inequalities – persistent social inequalities in exposure to environmental bads such as air pollution or noise and to environmental goods such as green spaces – are increasingly recognized as potentially important sources of social inequalities in health and other major life outcomes. The main goals of the present chapter are to review conceptual and empirical work on the health impacts of environmental quality and on the social processes that lead to environmental inequality as defined above.

We begin by embedding our review in a life-course framework (Ben-Shlomo and Kuh, 2002; Kuh et al., 2003) that allows for interdependent and dynamic linkages between environmental quality, health, and other life domains (e.g., educational attainment). Life-course approaches suggest that the effects of early-life insults, including exposure *in utero*, may not be confined to their often severe short-run consequences (Barker and Osmond, 1986). Early-life experiences might additionally trigger processes that lead to manifest disease (much) later in life, after long periods of latency – possibly reinforced by processes of cumulative disadvantage (DiPrete and Eirich, 2006).

The main part of the chapter reviews empirical evidence for a (causal) effect of environmental quality on health. We begin with the health impacts of air pollution, the most widely studied aspect of environmental quality. We discuss some of the most obvious threats to causal identification and review a by now large body of literature that has exploited quasi-experimental variation to provide compelling evidence for substantial causal effects of air pollution on health. Our extensive discussion of research on the effects of air pollution is complemented by shorter sections on the health impacts of extreme heat, green space presence, and noise.

While our review covers a sizable literature, we do have to be somewhat selective. We encourage readers to consult excellent earlier reviews on the health effects of air pollution (Pope and Dockery, 2006; Currie, 2013; Graff Zivin and Neidell, 2013; Currie et al., 2014; Kelly and Fussell, 2020) and green spaces (Kondo et al., 2018) that we build upon and extend. We also note that we focus on evidence from high-income countries where environmental regulation is rather strict and where levels of pollution tend to be low by international standards. Our review demonstrates that the health impacts of air pollution and environmental influences more broadly can be substantial, even under these comparably favorable conditions.

The final sections of the chapter turn towards the extent and sources of social inequalities in residential environmental quality. Research on the health effects of environmental quality treats residential sorting as a nuisance that complicates causal identification: it represents a form of selection into treatment. From a social stratification and health equity perspective, sorting constitutes a major social problem and a phenomenon of substantive interest. Why are some social groups more likely to move into or continue living in unhealthy environments than

others? We briefly review the most prominent answers to this question, including financial constraints, housing and credit market discrimination, as well as "legacy effects" due to the historical clustering of (minority) population groups in certain areas. We also present German data that highlight the importance of fine-grained spatial data for capturing the full extent of environmental inequality. The chapter concludes with a summary and some open questions.

A LIFE-COURSE PERSPECTIVE ON ENVIRONMENTAL AND HEALTH INEQUALITIES

Life-course perspectives (Ben-Shlomo and Kuh, 2002; Kuh et al., 2003) highlight a number of general explanations and explanatory mechanisms for health inequalities, including critical developmental periods and long-term effects of early-life experiences, the dynamic interplay of biological and social factors, as well as processes of cumulation and cumulative disadvantage (see Figure 21.1 for a stylized graphical representation).

An influential example of an explanation emphasizing long-term effects is the *fetal origins hypothesis* (Barker, 1995), the idea that insults to health experienced *in utero* can lead to a fundamental "reprogramming" that puts the organism on a path towards later-life disease. The related literature on *adverse childhood experiences* (Hughes et al., 2017) emphasizes the lasting impact of childhood adversity (e.g., poverty, abuse, or violence) on health in adulthood. While the strong version of the fetal origins hypothesis is distinct from explanations emphasizing childhood experiences in that it implies a discontinuity at the time of birth (Almond and Currie, 2011), both rest on a *critical period model* assuming that early insults to health can have lasting – sometimes irreversible – effects on health and other life outcomes through a variety of both biological (e.g., physiological or epigenetic) and social pathways (Ben-Shlomo and Kuh, 2002). The characterization of early (prenatal and postnatal) life as "critical" further suggests that adverse exposures during these periods tend to have stronger effects on later outcomes than comparable experiences during later stages of the life course (Ben-Shlomo and Kuh, 2002). A similar idea is that developmental capacities may be confined to certain "developmental windows" and that delayed and/or impaired growth during these windows is difficult to compensate for or reverse after a certain point (Andersen, 2003).

A related concept is that of *latency*. While some health effects of early-life insults may become apparent more or less immediately, others may remain latent for long periods, as with cardiovascular disease and other health conditions that typically do not manifest themselves until middle age (Almond and Currie, 2011). Long latency does not imply that early-life insults have no effects in the short term. It could even be argued that *some* changes (e.g., on a physiological or epigenetic level) have to occur immediately in order to pave the way for measurable impacts on health later in life. The notion of latency – represented by the dashed arrows in Figure 21.1 – rather emphasizes the idea that these alterations do not result in manifest disease right away but only after a certain (possibly long) period of time.

Whether initial insults eventually result in pathology may depend on the presence of further stressors over the subsequent life course, that is, on whether life courses follow a pattern of *cumulative disadvantage* (DiPrete and Eirich, 2006). A specific form that cumulative disadvantage may take is that of spillover and feedback effects between different life domains. Environmental insults early in life may cause direct short- and long-term harm to health, but they may also do so indirectly by affecting cognitive development, education, employment,

or income, which subsequently influence health (Zhang, Chen and Zhang, 2018).[1] Cumulative disadvantage in this sense can be conceptualized as a twofold process consisting of latent health but also human capital effects that further affect later-life health. Crucially, both mechanisms can be assumed to cluster in socially patterned ways (Ben-Shlomo and Kuh, 2002), that is, to vary according to socio-economic status (SES).[2]

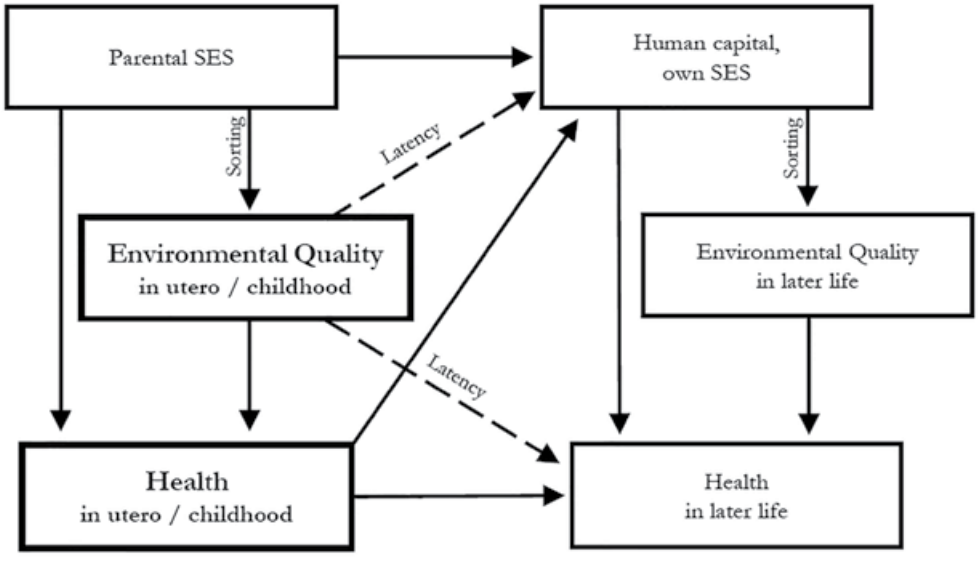


Figure 21.1 Environmental inequalities and health over the life course

Moving from these general considerations to possible impacts of environmental quality, there are many reasons why children might be particularly susceptible to (positive and negative) environmental influences from air pollution, green space presence, or heat. First, children's bodily systems are not yet fully developed making them particularly sensitive to environmental conditions. For example, the respiratory system grows and develops rapidly during early childhood, which may render it more vulnerable to assaults, with limited ability to fully repair after disruption of the morphogenesis (Bateson and Schwartz, 2007). Children's immune systems are immature, which does play an important role in the development of diseases such as asthma (Schwartz, 2004; Bateson and Schwartz, 2007). Similarly, children's thermoregulatory systems have not yet fully developed, resulting in lower capacity to adapt to extreme temperatures (Xu et al., 2014). Second, children physiologically differ from adults in other ways relevant to susceptibility. Their baseline ventilation rates are higher, thus exposing their lungs to more air pollution even under similar conditions. They also have a greater body surface area-to-mass ratio compared to adults, allowing greater heat and cold transfer between the environment and the body (Xu et al., 2014). Finally, children may be disproportionately affected by outdoor conditions as they spend more time outside and are physically more active than adults.

ADVERSE HEALTH EFFECTS OF AIR POLLUTION

We now review empirical work on the health effects of air pollution. After discussing associational epidemiological work and selected toxicological studies, we turn to key problems of causal identification and to research that tries to overcome these challenges with quasi-experimental designs. We show that empirical evidence is broadly consistent with critical period models in that the most severe short-term effects of pollution exposure are found for young children (including those exposed *in utero*) and for older people with pre-existing conditions. Evidence for long-term effects of early pre- and/or postnatal exposure seems to be growing as well, although compelling quasi-experimental studies remain scarce, partly due to demanding data requirements.

Epidemiological and Toxicological Evidence

Much of our understanding about the relationship between exposure to air pollution and health stems from epidemiological and toxicological studies. The list of air pollutants is long and includes compounds such as carbon monoxide (CO), ground-level ozone (O_3), lead (Pb), and nitrogen (NO_x) or sulphur oxides (SO_x). Perhaps the most widely studied aspect of air quality is particulate matter (PM) concentration, usually measured in microgram per cubic meter (μg/m³) and typically classified into fine particulate matter with particle diameters below 2.5 μm ($PM_{2.5}$) and coarse particulate matter with diameters below 10 μm (PM_{10}).

Epidemiological studies have established strong and robust associations between ambient air pollution and various health outcomes. Particularly strong links have been found for respiratory and cardiovascular morbidity, including reduced lung function and severity of symptoms in individuals with asthma and chronic obstructive pulmonary disease (COPD) as well as ischemic heart disease (Brook et al., 2010; Kelly and Fussell, 2011; WHO, 2013).

Temporary spikes in air pollution have been linked to increased hospital admissions. Using data from Chicago area hospitals between 1988 and 1993, Schwartz (2001) found an increase in PM_{10} by 10 μg/m³ to be associated with 2.00 percent, 1.45 percent, and 1.27 percent increases in admissions for pneumonia, chronic obstructive pulmonary disease, and heart disease, respectively (see also Zanobetti, Schwartz and Dockery, 2000).

With regard to mortality, Ostro, Hurley, and Lipsett (1999) found that an increase in PM_{10} concentration by 10 μg/m³ in Coachella Valley, California, was associated with a 1 percent increase in daily all-cause mortality. An analysis of ten large US cities similarly indicated that heightened PM_{10} concentration (10 μg/m³) was associated with a 0.67 percent increase in total mortality and a 0.89 percent increase in out-of-hospital mortality (Schwartz, 2000). For the European context, Katsouyanni et al. (2001) demonstrated that PM_{10} and black smoke predicted daily mortality in 29 countries.

In addition to these short-term associations, prospective cohort studies with long-term follow-up periods have documented robust links between levels of air pollution and lung cancer, cardiopulmonary, and all-cause mortality. One of the most comprehensive early studies – the American Cancer Society study – linked individual health risks of residents from approximately 150 US cities with data on ambient air pollution and found a risk ratio of 1.17 for all-cause mortality between the areas most and least affected by fine particulates (Pope et al., 1995; see also Dockery et al., 1993). Results of a subsequent follow-up study, observing death or survival of participants over more than 16 years (1982–1998), indicated that the risk

of dying from lung cancer and cardiopulmonary diseases increased by 8 percent and 6 percent, respectively, for each 10 μg/m³ increase in $PM_{2.5}$ (Pope, 2002).

Several groups have been found to be at higher risk for air-pollution-induced cardiovascular morbidity and mortality, including individuals with pre-existing cardiovascular conditions, people with diabetes, and elderly individuals (Zanobetti and Schwartz, 2002; Devlin et al., 2003). As many relevant conditions are more prevalent among low-SES populations – due in part to long-term exposure to pollution itself – these differential vulnerabilities tend to amplify social inequalities in health and can be interpreted as examples of cumulative disadvantage (McNamara et al., 2017).

Epidemiological evidence is bolstered by research on biological and physiological responses to air pollution exposure in humans and non-human species (Salvi et al., 1999). Bové et al. (2019) show that black carbon particles stemming from combustion-derived PM pass the human placental barrier, elucidating a potential mechanism for fetal origins of later-life disease. They further find the black carbon load from placentae to be positively associated with residential black carbon exposure of mothers throughout pregnancy (see Luyten et al., 2018 for a review of placental markers associated with prenatal air pollution exposure). A number of studies on adults have examined immediate effects of pollution exposure on humans, for example, by exposing human volunteers to diesel exhaust for 1–2h (Salvi et al., 1999, 2000; Stenfors et al., 2004; Behndig et al., 2006). These studies provide compelling evidence of systemic and pulmonary inflammatory responses following even short-term exposure to toxic pollutants. Toxicological examinations have shown that exposure to air pollution leads to changes in many cardiovascular indicators, with some (e.g., in heart rate, heart rate variability, blood pressure, vascular tone, and blood coagulability) occurring more or less immediately, and others, such as accelerated progression of arthrosclerosis, after more prolonged exposure (Simkhovich, Kleinman and Kloner, 2008). While such toxicological approaches provide valuable evidence on biological and physical pathways, they also have clear limitations. Studies involving long-term or high levels of exposure of humans are ethically indefensible, as are studies of high-risk populations. Such concerns do not loom quite as large in studies of nonhuman animal species (e.g., Sun et al., 2005), but their results cannot necessarily be extrapolated to humans.

Analysis of observational data on real-world exposures, as in the epidemiological literature discussed above, is therefore indispensable for gauging the full impact of air pollution on human health and health inequalities. Such analysis is complicated by well-known threats to causal identification in observational studies, and this is where the social sciences have made significant contributions through the application of quasi-experimental approaches (Currie et al., 2014).

Challenges to Identification

As in practically any observational study, a general concern in estimating the health impacts of air pollution is *selection on unobservables*. Greater exposure to air pollution might go hand in hand with other individual and environmental risk factors, including socio-economic deprivation, unhealthy behaviors, work-related health hazards, or lack of green spaces. Failure to adequately control for such confounding factors will lead to biased estimates of pollution exposure effects.

In the context of environmental inequality, selection on unobservables is often linked to *residential sorting*. A growing body of literature indicates that sorting based on environmental quality is a key factor shaping residential segregation. For example, Banzhaf and Walsh (2008) show that high-income families tend to move away from polluted areas (see also Best and Rüttenauer, 2018). In addition, research highlights the tendency of potentially health-relevant neighborhood characteristics such as crime, school quality, and public infrastructure more broadly to cluster in space (Alba and Logan, 1993), suggesting possible confounding at the local contextual level.

From the viewpoint of identifying the health effects of air pollution and other dimensions of environmental inequality, residential sorting is a nuisance that complicates causal identification. In the remainder of this chapter, we will initially adopt this perspective and focus on studies that seek to address the resulting selectivities through clever (quasi-experimental) designs. Yet residential sorting clearly is a social process that is of utmost substantive interest, a point that we emphasize when turning towards the mechanisms underlying environmental inequality in the later parts of the chapter.

Avoidance behavior is another potential source of bias. People may try to protect themselves and their children– temporarily or persistently – against poor environmental conditions, for example, by spending more time indoors (Neidell, 2009; Currie et al., 2014). In the presence of avoidance behavior, measures of ambient air pollution will overstate actual differences in exposure. Avoidance behavior thus results in the underestimation of health effects, in the sense that the latter would be even larger under unmitigated exposure to measured differentials in outdoor pollution (Neidell, 2009). Another interpretation is that a narrow focus on health outcomes misses some of the welfare costs of air pollution by ignoring the individual and social costs of avoidance behavior (Moretti and Neidell, 2009; Currie et al., 2014).

Quasi-Experimental Approaches

Given the ethical and practical unviability of randomized experiments, identification of plausibly causal effects of air pollution on health mostly comes from various types of quasi-experimental designs (see Morgan and Winship, 2007 for a thorough discussion; see also Chapters 7 and 9 by Gebel and by Hoffmann and Doblhammer in this volume). Plausibly exogenous variation in pollution concentration often stems from so-called *natural experiments* that lead to sudden, unexpected, and sometimes unobserved changes in environmental conditions. Important types of events include policy changes, changes in weather conditions (e.g., changes in wind directions or temperature inversions), and unexpected changes in industrial production or traffic intensity. In the absence of anticipation effects, such "shocks" can be argued to affect the same population just before and after their implementation, making distortions due to changes in population composition unlikely, at least in the short run. Whenever an event and its implications for pollution exposure are difficult to observe, avoidance and other changes in health-relevant behaviors do not pose major threats to identification either.

Fixed-effects designs that exploit (short-term) variation in measured pollution within geo-spatial and administrative units follow a similar basic logic. The primary goal of such designs again is to rule out confounding from unobserved factors that might bias estimates based on between-unit comparisons (e.g., population composition or healthcare infrastructure). While this assumption is often plausible over short periods of time, it becomes more difficult to defend when the observation period spans several months or even years. Another limitation

of designs that take measured pollution as given, even when focusing on short-term variability, is that the sources of this variation are unclear and can include factors (e.g. fireworks or traffic congestion due to major sport events) that may be associated with other (health-relevant) behavioral changes. This is a potentially crucial difference to natural-experiment approaches that can render such changes unlikely by focusing on specific (weather) events.

Another kind of fixed-effects design focuses on *within-family variation*, usually comparing the health outcomes of siblings who have experienced different environmental conditions *in utero* and in childhood, induced by families moving to another area or by changes in environmental quality over time. The strength of this approach is to control for time-invariant unobserved characteristics shared by children from the same family, including parental health behaviors or genetic predispositions. At the same time, there are obvious threats to a causal interpretation of within-family associations. For example, residential moves might go hand in hand with changes in numerous other health-relevant domains, not all of which may be adequately controlled in a given application (for a useful critical discussion of within-family designs, see Engzell and Hällsten, 2022).

The designs discussed in this section address key limitations of simple associational and conditioning-on-observables approaches, yet they certainly are no panacea. Generally, the more time elapses after the onset of a (plausibly exogenous) event, the greater the scope for residential sorting to complicate the picture (Currie et al., 2014). When natural experiments are based on unexpected shocks or policy changes, these events can also directly affect health through other mechanisms. Shocks to industrial productivity due to recessions, for example, have been used to investigate the effects of decreased industrial emissions of air pollutants (Sanders, 2012). However, economic recessions may affect health through other channels (e.g. loss of employment and/or health insurance) that, if not accounted for, can result in biased estimates. Finally, approaches exploiting temporary weather events or short-term spatiotemporal variability more generally are not well-suited for identifying health effects that have long latency, nor for studying the impact of long-term (accumulated) exposure. With these caveats in mind, we now turn to a review of quasi-experimental evidence on the health impacts of air pollution.

Short-Term Variability in Exposure and Immediate Health Consequences

Deryugina et al. (2019) exploit variation in local wind direction at the US county level to investigate adverse health effects of air pollution on US *Medicare* beneficiaries. They find that a 1 μg/m³ increase in $PM_{2.5}$ leads to 2.7 additional Emergency Room (ER) visits and 0.69 additional deaths per million beneficiaries over the three-day window that spans the day of the increase and the following two days. Jans, Johansson and Nilsson (2018) examine whether poor air quality affects Swedish children's respiratory health, exploiting variation in air quality induced by night-time temperature inversions.[3] They find that temperature inversions temporarily raise PM_{10} and NO_2 concentrations by 25 and 16 percent, respectively, and that they lead to increases in health care visits due to respiratory illness of around 5.5 percent. Low-income children are more strongly affected, which seems to be primarily due to differences in baseline health status and preexisting conditions. These effects are unlikely to be confounded by avoidance behavior, as (night-time) temperature inversions usually cannot be observed with the naked eye and are rarely reported in the media.

Dominici et al. (2006) use a design with local area fixed effects to investigate short-term health effects of air pollution using a database comprising daily information on hospital admissions of Medicare enrollees (aged > 65 years), ambient $PM_{2.5}$ levels, and weather controls for 204 US urban counties. They find short-term increases in hospital admission for all health outcomes under study except for injuries. Effects are largest for heart failure, respiratory tract infections, and COPD with statistically significant estimated increases of 1.28, 0.92, and 0.91 percent per 10 μg/m³ increase in $PM_{2.5}$.

Ground-level ozone (O_3) concentration is another aspect of air quality that has received quite a bit of attention. Neidell (2009) shows that accounting for avoidance behavior drastically increases estimated health effects of ozone on vulnerable groups – by roughly 160 percent for children and 40 percent for the elderly – while having no effect on impact estimates for other adults. Moretti and Neidell (2009) use boat traffic at the port of Los Angeles – a major source of nitrogen dioxide (NO_2), which forms O_3 when reacting with sunlight – as an instrumental variable (IV) in estimating effects of O_3 on respiratory-related hospitalizations. Simple OLS yields a statistically significant but relatively small estimate of $11.1 million ozone-related hospital costs per year. The IV estimate is roughly four times as large ($44.5 million per year).

Medium- to Long-Term Health Consequences of Air Pollution

Policy changes and other events that lead to sudden and persistent changes in environmental conditions can be exploited to investigate longer-term consequences of air pollution exposure. However, this goes hand in hand with challenges to causal identification such as changes in the composition of the affected populations.

Currie and Walker (2011) use the introduction of an electronic toll collection scheme (called E-ZPass) across the northeastern United States around the year 2000. E-ZPass reduced delays and traffic congestion at toll plazas, thereby leading to local declines in pollution caused by decelerating, idling, and accelerating. Currie and Walker (2011) compare health outcomes of infants born to mothers living near toll plazas (0–2 km) to those born to mothers living farther away (2–10 km) but close to major highways (0–3 km), starting three to six years prior to the implementation of the E-ZPass until one to two years thereafter. Difference-in-difference models suggest that the incidence of low birth weight fell by 8.5–11.3 percent among infants born to mothers within 2 km of a toll plaza, while prematurity fell by 6.7–9.2 percent (Currie and Walker, 2011). Housing prices and mothers' observable characteristics remained largely unchanged around the adoption of the E-ZPass, assuaging concerns about residential sorting.

Following a very similar logic, several studies have investigated health effects of newly established low emission zones[4] (LEZs) in urban areas. Gehrsitz (2017) studies the impact of the staggered introduction of LEZs in multiple German cities and finds modest average reductions in air pollution of approximately 2.5 percent for PM_{10}, with larger effects in cities that implemented stricter policies. Gehrsitz reports a moderate negative and statistically significant effect on stillbirths but none for average birthweight or the prevalence of low-weight births. Another German study by Margaryan (2021) finds similar reductions of PM_{10} levels and a 2–3 percent reduction in the number of patients with cardiovascular disease based on outpatient care data, with stronger effects for cerebrovascular disease (e.g., stroke) and for older people aged 65 and above. Studies looking at the introduction of an LEZ in London, United Kingdom, in February 2008 have found that the initial introduction had little to no effect on air pollution and health outcomes (Wood et al., 2015; Mudway et al., 2019).

Another widely studied policy change is the 1970 Clean Air Act Amendments (CAAA) which required so-called "nonattainment counties" with total suspended particulate (TSP) levels above threshold values to reduce air pollution, while "attainment counties" below the threshold were not required to do so. Chay and Greenstone (2003) use the nonattainment status of counties to instrument for changes in TSP between 1971 and 1972. They find that nonattainment status was indeed associated with subsequent reductions in air pollution and that a 1 percent reduction in TSP results in a 0.5 percent decline in overall infant mortality rate at the county level. Using a similar empirical approach, Chay, Dobkin and Greenstone (2003) find no systematic effect of reduced pollution on adult and elderly mortality rates, however. In a more recent paper focusing on labor market outcomes, Isen, Rossin-Slater, and Walker (2017) study the long-term consequences of pollution exposure *in utero* by comparing individuals born just before and just after the mandated improvements in air quality in nonattainment counties, using individuals born in attainment counties as a control group (difference-in-differences approach). They find that regulation-induced improvements in air quality are associated with a 0.7 percent increase in the number of quarters worked and a 1 percent increase in mean annual earnings at age 30. Their study is a so far rare case of a quasi-experimental study that provides evidence for long-term effects of *in utero* exposure.

Applying a sibling design, Currie, Neidell and Schmieder (2009) investigate health effects of O_3, CO, and PM_{10} on birth outcomes and infant mortality in New Jersey, USA, in the 1990s. Their models include pollution monitor and maternal fixed effects to account for time-invariant characteristic of neighborhoods and mothers. Results indicate that a one-unit rise in average CO parts per million (ca. 1.27 standard deviations) during the last trimester of gestation increases the risk of low birth weight by 8 percent. Additionally, a one-unit increase in mean CO during the first two weeks after birth increases the risk of infant mortality by 2.5 percent, conditional on health at birth.

In a recent study, Alexander and Schwandt (2022) exploit spatial variation in the share of emissions-cheating Volkswagen "clean" diesel cars over the 2008–2015 period as a natural experiment. They estimate that an additional cheating diesel car per 1,000 cars increases $PM_{2.5}$ concentrations by 2 percent, and low birth weight and infant mortality rates by 1.9 and 1.7 percent, respectively.

The research reviewed in this section provides compelling evidence that air pollution has substantial adverse effects on health even in high-income countries where levels of pollution tend to be low by international standards. Short-term effects of pollution exposure are strongest among the old, partly as a result of preexisting conditions, for young children and *in utero* (as indicated by effects on birth weight and stillbirths). Extant research also points towards long-term effects of *early-life* exposure, although compelling quasi-experimental evidence remains rare, partly due to formidable data requirements (e.g., information on parental and childhood residential location). Nevertheless, empirical support for the fetal origins hypothesis seems to be growing, also in terms of mechanistic biological evidence.

In the remaining sections, we extend our discussion in two main ways: The next section discusses selected dimensions of environmental quality – access to green space and exposure to noise or extreme heat – that have so far received less attention than air pollution. The subsequent section then focuses on environmental *inequality* as an explanation for health inequalities and on underlying processes of residential sorting and segregation.

GOING BEYOND AIR POLLUTION: OTHER DIMENSIONS OF ENVIRONMENTAL QUALITY

Green Space

Proximity and access to green spaces, particularly in urban areas, is another aspect of environmental quality whose potential health-promoting effects have received increasing attention in recent years. While proximity to green spaces, an environmental "good", is likely to correlate negatively with exposure to environmental "bads" such as pollution and noise, there are also plausible channels through which green spaces might independently affect both physical and mental health. For example, Markevych et al. (2017) argue that green space can affect health by restoring (e.g. stress recovery) and building capacities (e.g. encouraging physical exercise). In addition, negative correlations with environmental bads could partly be viewed as mediation (rather than confounding), for example, if the creation of a new park at the cost of road space leads to a reduction in traffic.

So far, evidence on the health effects of green spaces mostly comes from conditioning-on-observables approaches that try to account for potential confounders with multiple regression and related approaches. This may also be because natural experiments involving clear temporal and/or spatial discontinuities in green space "exposure" are more difficult to find than in the case of ambient pollution.

Extant studies report associations between exposure to natural environments and a wide range of physical and mental health outcomes, such as preterm birth (Abelt and McLafferty, 2017), childhood asthma (Donovan et al., 2018), student performance (Sivarajah, Smith and Thomas, 2018), anxiety and depression (Cohen-Cline, Turkheimer and Duncan, 2015), more aggregated indices of psychiatric disorders and mental health (Alcock et al., 2014), and respiratory as well as cancer mortality (James et al., 2016). In an exemplary and widely cited recent study, Engemann et al. (2019) combine satellite images with Danish population register data to investigate the association between green space presence in childhood (before age ten) and mental health in adolescence and young adulthood, controlling for multiple alternative explanations. High levels of green space presence in childhood are negatively associated with several psychiatric disorders.

With regard to the underlying mechanisms, several studies have investigated the role of natural environments in recovering from psychological stress, relying on salivary and hair cortisol concentration as short- and long-term biomarkers (Ward Thompson et al., 2012; Roe et al., 2013; Gidlow et al., 2016). Results suggest that exposure to natural environments is indeed negatively correlated with short-term cortisol levels (Roe et al., 2013), although associations are not always robust to controls for confounding factors such as neighborhood deprivation (Gidlow et al., 2016). In an analysis of a small-scale intervention in the UK, Chalmin-Pui et al. (2021) find that adding plants to formerly bare front gardens led to lower perceived stress levels and improvements in diurnal cortisol patterns. Results concerning physical activity as another potential mediator between residential green space and health are mixed (Triguero-Mas et al., 2015; James et al., 2016).

As noted above, clean natural experiments are more difficult to find for green space than for pollution exposure. At the same time, concerns about residential sorting and other threats to causal interpretation loom just as large as in the pollution case. Several studies have therefore tried to address concerns about unobserved confounding, mostly using (longitudinal) designs

with individual fixed effects. Using such an approach, both Alcock et al. (2014) and White et al. (2013) find positive effects of green space presence on mental health in the UK, while Noordzij et al. (2020) find no evidence for such a relationship using Dutch data. Even these longitudinal results have to be interpreted cautiously, however, as other neighborhood characteristics that co-vary with green space presence may not be fully accounted for. Cohen-Cline, Turkheimer and Duncan (2015) investigate the link between access to green space and self-reported depression, stress, and anxiety in a sample of same-sex monozygotic twin pairs. Pooled models show negative relationships between green space presence and all three mental health outcomes, but only the association with depression remains statistically significant in within-pair models, suggesting partial genetic confounding.

Extreme Heat

Extreme heat is another environmental factor with potentially severe consequences. Studies of high-income countries have so far mostly focused on countries and regions with a warm climate such as California, USA, or Australia. Climate change, however, will likely result in heat waves occurring more often, including in regions where they have been rare in the past (Koppe et al., 2004). We will not be able to review the sizable literature on the health effects of extreme heat in detail, but robust short-term association between heat waves and health have been documented for a variety of specific outcomes – including sunburn, heat stress, heat exhaustion, kidney failure, and heart attacks (Koppe et al., 2004; Guirguis et al., 2014) – as well as for broader measures of morbidity and mortality (Nitschke et al., 2011).

Urban areas are generally more likely to be affected due to "heat islands" resulting from high levels of soil sealing, high-rise buildings, and the absence of green and open spaces. Importantly, both the risks of exposure and vulnerability to extreme heat are likely to be shaped by social inequalities, an aspect that has received relatively little attention so far (for initial evidence, see Vandentorren et al., 2006; Gronlund et al., 2015). Disadvantaged populations are likely to be at greater risk of experiencing extreme heat (e.g., due to lower levels of green space presence, lower building quality, or inability to afford air conditioning and other protective measures) and tend to be more vulnerable because of pre-existing conditions. Extreme heat thus seems likely to become an increasingly important source of health inequalities in the coming years.

Noise

Associational evidence suggests a link between environmental noise exposure and a range of health and well-being outcomes such as noise annoyance, sleep quality, cognitive impairment, birth outcomes, cardiovascular disease, and metabolic disorders. Aircraft and road traffic are the most important sources of noise, suggesting that it is highly correlated with other measures of environmental quality, most importantly air pollution, but also green space presence (or the absence thereof). Establishing causality therefore requires adjustment for confounders not only on the individual (e.g. age, SES, BMI) but also on the residential neighborhood level. While this also applies to other aspects of environmental quality, the link between noise and air pollution is particularly obvious.[5]

Munzel et al. (2014) review the cardiovascular effects of noise exposure and highlight that night-time noise exposure in particular can cause sleep disturbance and increased blood pres-

sure, heart rate, and stress hormone levels, which in turn may result in arterial hypertension. Kälsch et al. (2014) jointly investigate the cardiovascular effects of PM and noise and find that both air pollution and night-time noise exposure are associated with subclinical atherosclerosis after mutual adjustment. In a study by Liu et al. (2014), the association between blood pressure in children and air pollution even becomes insignificant once controlling for noise but not vice versa. Gehring et al. (2014) show that noise exposure during pregnancy is negatively associated with birth weight even after controlling for air pollution. Exploiting plausibly exogenous variation in night-time noise introduced by a night flight ban at Frankfurt Airport, Germany, Müller et al. (2016) find that the number of awakenings per night declined from 2.0 to 0.8 after the implementation of the ban.

While the list of environmental factors considered in this chapter inevitably remains incomplete, we have discussed four of the most important and widely studied dimensions of environmental quality: air pollution, green space presence, heat, and noise. We have reviewed multidisciplinary evidence that collectively provides compelling evidence for both short- and longer-term effects of environmental quality on health and mortality. Environmental quality is not distributed equally. Disadvantaged and vulnerable populations live in less healthy environments, that is, there is environmental inequality. From a causal identification standpoint, such inequalities are a nuisance, a source of confounding that may bias estimates of the health effects of environmental quality. But of course, environmental inequality and its contribution to health inequalities and inequities more broadly are important matters in their own right. In the following sections, we therefore turn to the processes shaping environmental inequality and to some aspects of its measurement.

Mechanisms Underlying Environmental Inequality

Motivated by the profound effects of environmental quality on health and life chances reviewed in the previous section, research on *environmental inequality* seeks to understand, first, which population subgroups face low levels of residential environmental quality and, second, which mechanisms explain the socially unequal distributions of environmental goods and bads.

A key finding of the international empirical literature on social inequalities in pollution exposure is that individuals with low income (Ash and Fetter, 2004), and even more so racial or ethnic minorities (Pais, Crowder and Downey, 2013; Ash and Boyce, 2018), carry a disproportionate burden of exposure. With regard to urban green space, studies similarly show clear associations between neighborhood- or community-level greenspace presence and socio-economic (Chen et al., 2020) as well as ethnic composition (Byrne, Wolch and Zhang, 2009; Matthew McConnachie and Shackleton, 2010; Kabisch and Haase, 2014).

The processes that underlie these empirical regularities are not yet well understood. Theoretically, environmental inequality has often been attributed to two broad classes of causal mechanisms. The *selective siting hypothesis* states that environmental hazards such as polluting facilities or airports are disproportionately sited in or close to areas characterized by economic deprivation and/or high minority shares. Regarding green space, this would suggest that investments in public green spaces or urban renewal would primarily target residential areas with affluent and/or majority (white) residents. The *selective migration hypothesis*, in contrast, assumes that environmental inequality is the result of (post-siting) *residential sorting*,

with advantaged groups leaving areas with low environmental quality and moving into environmentally attractive areas at higher rates.

Residential sorting along environmental lines could occur for several reasons that are not mutually exclusive (for a detailed overview, see Banzhaf, Ma and Timmins, 2019). *Housing prices*, which have been shown to be sensitive to air pollution (Chay and Greenstone, 2005) as well as green space presence (Franco and Macdonald, 2018; Panduro et al., 2018), likely play an important role (Mohai and Saha, 2015): disadvantaged groups may be priced out of environmentally attractive neighborhoods. The racial income inequality hypothesis (Crowder and Downey, 2010) accordingly states that ethnic and racial minorities move to and live in lower-quality neighborhoods because they have, on average, fewer economic resources.

This seems to be only part of the explanation for ethno-racial disparities in environmental quality, however, as many studies show that ethnic minorities face lower environmental quality even after controlling for income and other resources (Downey et al., 2008; Rüttenauer, 2018). *Housing and credit market discrimination* are further structural explanations, in terms of both institutional rules and regulations (e.g. restrictive zoning, steering, credit scoring) and individual discrimination by landlords and other actors (Morello-Frosch and Jesdale, 2006; Small and Pager, 2020).

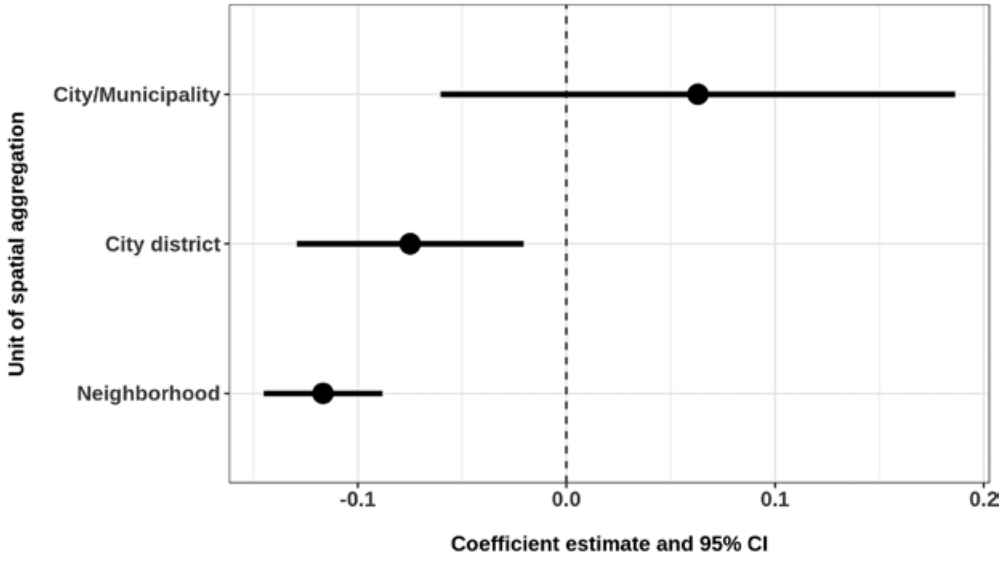
Finally, historical "*legacy effects*" could explain the persistence of environmental inequality. If (for whatever reasons) racial and ethnic minorities settled in areas with low environmental quality in the past, current members of these groups may continue to be drawn towards these areas. Research based on observational (Mossaad et al., 2020) and experimental data (Ibraimovic and Masiero, 2014) suggests that living with co-ethnics plays an important role in the internal location and migration decisions of immigrants and ethnic minorities. Studies of residential mobility show that the presence of close relatives and friends reduces the probability of moving (Clark, Duque-Calvache and Palomares-Linares, 2017; Ermisch and Mulder, 2019; Hünteler and Mulder, 2020). Such "local social capital" is assumed to deter residential mobility because the resources stemming from it are location-specific and will be less valuable if a household moves (Kan, 2007). Qualitative research from the *Moving to Opportunity* (MTO) experiment corroborates the importance of residential proximity to close ties, showing that many households that initially moved out of high-poverty neighborhoods either keep spending much of their time at their former communities or even move back (Popkin et al., 2002). The bottom line is that, in the presence of legacy effects, ethno-racial minorities may face a trade-off between living with co-ethnics and enjoying better environmental conditions that does not exist for members of majority groups.

ENVIRONMENTAL INEQUALITY AT DIFFERENT LEVELS OF SPATIAL RESOLUTION

Investigating patterns of environmental inequality and testing underlying causal mechanisms with observational data are complex tasks that require spatially fine-grained and longitudinal data on different dimensions of environmental quality as well as individual- or neighborhood-level SES. Longitudinal data is required to shed light on the mechanisms of selective siting and sorting. While large industrial plants may affect the health of residents that live even some kilometers away, other dimensions of environmental quality such as air pollution and noise from road traffic primarily affect those who live close by. Similarly, some

of the health-promoting effects of green space (e.g. stress restoration) have been argued to be concentrated among those who live in sight. High spatial granularity can also be important for exploiting quasi-experimental (spatial) variation. Conversely, measuring environmental quality and/or ethnic and socio-economic composition at overly coarse levels of spatial aggregation – e.g. counties, districts, or municipalities – can conceal the true extent of environmental inequality.

We illustrate the importance of fine-grained spatial data with a concrete example in Figure 21.2, which shows the relationship between neighborhood ethnic composition and green space presence at different levels of spatial resolution for German municipalities with at least 100,000 inhabitants. Combining data on the proportion of non-nationals at the block level[6] with high-resolution satellite data,[7] we calculated the surface share of natural environments (urban green space, sport and leisure facilities, and forests) for three levels of spatial aggregation (both the share of non-nationals and the green space surface share are expressed in percentage terms). The first two levels of aggregation are the administrative units of city/municipality and of districts within cities and municipalities. The third and most fine-grained level of aggregation, the neighborhood level, was constructed by calculating the natural environment share in a 250 m circular buffer around each block. We then ran bivariate OLS models regressing the share of non-nationals, that is, residents without German citizenship, on our measure of



Note: The figure shows the association between the population share of non-nationals and urban green space presence (measured as the surface share covered by natural environments) at different levels of spatial aggregation, with measures expressed in percentage terms. Depicted are the coefficients from three separate bivariate linear regressions for the three different levels of aggregation (OLS estimates, all standard errors clustered at the municipality level). For example, the neighborhood-level coefficient estimate of –0.12 implies that the (neighborhood-level) population share of non-nationals declines by 0.12 percentage points for each percentage point increase in green space surface share.

Figure 21.2 Association between urban green space availability and share of non-nationals at different levels of spatial aggregation

green space presence. Figure 21.2 shows the resulting regression coefficient estimates and 95 percent confidence intervals for the three different levels. At the city level, we do not observe a statistically significant association between green space availability and demographic composition. The city-level point estimate is positive, suggesting that – if anything – higher green space presence goes hand in hand with higher population share of non-nationals. The picture changes completely once we look at the association at the level of city districts: districts with higher shares of non-national residents, on average, tend to have lower green space presence. Importantly, this association becomes even stronger when we turn to the neighborhood level. The neighborhood-level coefficient estimate implies that the population share of non-nationals declines by 0.12 percentage points per percentage-point increase in the green space surface share. The fully standardized coefficient estimate is –.13, that is, a standard deviation increase in the surrounding green space surface share is associated with a decline in the block-level share of non-nationals of approximately .13 standard deviations on average.[8] These results are a simple, yet powerful illustration of how spatial aggregation can understate or even conceal the magnitude of environmental inequalities.

CONCLUSION

We have reviewed multidisciplinary evidence on the health effects of various dimensions of residential environmental quality. In so doing, we have paid particular attention to issues of causal identification and to the by now sizable body of quasi-experimental research indicating substantial causal effects of environmental quality on health, particularly for air pollution. As for the life course patterning of these effects, research supports the notion that *in utero* and childhood exposures are "critical". This is certainly true in the sense that children, in addition to the elderly and adults with preexisting health problems, are often found to suffer the most from adverse environmental influences in the short run, in terms of both mortality (stillbirths) and morbidity (e.g., asthma) as well as with respect to indicators of overall development (e.g., birthweight). There is some empirical support for long-term cumulative effects of environmental exposures *in utero* and in childhood, although evidence – and especially quasi-experimental evidence – remains much more limited on this front, partly due to formidable data requirements and the potentially long latency of effects.

In the later parts of this chapter, we have discussed the social mechanisms that generate and reproduce patterns of environmental inequality. We have emphasized and illustrated the importance of granular geo-spatial data for mapping the full extent of environmental inequality and for implementing compelling quasi-experimental designs. On a theoretical level, we have discussed several explanations for environmental inequality. In addition to resource constraints and housing or credit market discrimination, we have pointed to "legacy effects" as one potentially important reason why ethno-racial minorities may face a disproportionate environmental burden. If co-ethnics tend to cluster in areas with low environmental quality, minority individuals will face a trade-off between environmental quality and living close to co-ethnic friends and infrastructures – even if the reasons for residential clustering are purely historical. This trade-off may simply not exist for non-minority individuals, at least not to the same extent.

While we have mostly focused on the health effects of environmental quality, a growing body of literature investigates effects on human capital development (Nilsson et al., 2009;

Sanders, 2012; Persico, 2020) that are likely to mediate a portion of the total long-term effect of (early-life) environmental conditions on health (cf. Figure 21.1 above). The literature reviewed in this chapter is, thus, also linked to the broader literature on trends in morbidity and mortality. For example, Case and Deaton (2017) show that college education protects against a range of undesirable outcomes, including (involuntary) singlehood, social isolation, and detachment from the labor market, all of which are linked to rising mortality from alcohol and drug abuse as well as suicide ("deaths of despair").

We have reviewed a substantial literature from a fairly wide range of disciplines, but many open questions remain. We highlight two of them here. First, as noted at the beginning of the chapter, the evidence discussed above stems from high-income countries. This is partly due to space limitations but also a matter of data availability: estimating (long-term) causal health effects of environmental quality requires spatially fine-grained and longitudinal data that are more readily available for high-income countries (e.g. Sweden, Denmark, USA). Such countries tend to be characterized by rather moderate levels of, for example, air pollution. Adverse health effects of environmental pollution are likely to be more severe in the high-pollution contexts found in many low- and middle-income countries. Second, we have focused on *outdoor residential* environmental quality, ignoring environmental influences experienced in non-residential settings and environments, for example, in and around schools or workplaces. Approximating an individual's level of exposure solely by measures of outdoor environmental quality does not account for aspects of indoor environments that may be both conducive (e.g., protection from pollution and heat through insulation or air conditioning) or detrimental to health (e.g., indoor smoking). Restricting measures of environmental quality to the place of residence may introduce considerable measurement error, as individuals spend considerable amounts of time elsewhere. Moreover, different social groups may do so to different extents and face very different exposures in non-residential environments, as illustrated by recent work on social inequalities in infection risks during the first wave of the Covid-19 pandemic (Chang et al., 2021). Longitudinal and spatio-temporally fine-grained data at the contextual-environmental, household, and individual levels is needed to answer these and other important questions about the environment-health nexus – and to foster a richer evidence base that helps build healthy environments for all members of society.

NOTES

1. Of course, it is equally possible that health partly or largely mediates the effects of environmental insults on these outcomes, e.g., when children miss school because of health issues.
2. Ben-Shlomo and Kuh (2002) provide a more fine-grained conceptualization of four different pathways: social, biological, socio-biological, and bio-social. The fact that infants born to households with low socio-economic status, on average, show less favorable birth outcomes (e.g. due to differential *in utero* environmental quality) qualifies as a socio-biological pathway, whereas early life illnesses affecting educational participation and performance as well as subsequent labor market outcomes constitute a bio-social pathway.
3. Under normal conditions, temperature decreases with altitude, leading air pollutants emitted close to the ground to rise and disperse. During inversion periods, a warmer layer of air traps air pollutants close to the ground, thereby slowing down or preventing vertical exchange of air and increasing pollution concentration close to the surface.
4. Low Emission Zones are (mostly inner-city) areas where polluting vehicles are regulated. This usually implies that vehicles have to meet certain emission standards to be allowed to enter the area.

5. The same problem, intuitively, also applies for many of the studies investigating adverse health effects of traffic-induced air pollution. Natural experiments exploiting variation in traffic-related pollution do not necessarily isolate the health effects of air pollution from the health effects of noise. Policy changes affecting traffic, such as the introduction of LEZs, could also affect noise. For studies based on weather events, such as inversion periods, this should be less of a problem.
6. Socio-demographic data at the neighborhood level were obtained from infas 360 GmbH (https://www .infas360 .de/). There are 186,423 neighborhood blocks containing around 13.92 million households across all German municipalities with at least 100,000 inhabitants, so the average block comprises approximately 75 households.
7. High-resolution land use maps for urban areas based on satellite imagery were obtained from the EU Copernicus Urban Atlas (https://land.copernicus.eu/local/urban-atlas).
8. The population-weighted average share of non-nationals is equal to 17.36 for all three levels of spatial disaggregation. The city/district/block-level standard deviations are 5.3, 9.3, and 10.7, respectively. The population-weighted average city/district/block-level green space surface shares are 27.0/22.6/12.6, with standard deviations of 9.7/15.2/11.7.

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