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This Is Air: The "Nonhealth" Effects of Air Pollution

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Abstract

A robust body of evidence shows that air pollution exposure is detrimental to health outcomes, often measured as deaths and hospitalizations. This literature has focused less on subclinical channels that nonetheless impact behavior, performance, and skills. This article reviews the economic research investigating the causal effects of pollution on nonhealth end points, including labor productivity, cognitive performance, and multiple forms of decision-making. Subclinical effects of pollution can be more challenging to observe than formal health care encounters but may be more pervasive if they affect otherwise healthy people. The wide variety of possible impacts of pollution should be informed by plausible mechanisms and require appropriate hypothesis testing to limit false discovery. Finally, any detected effects of pollution, in both the short and long run, may be dampened by costly efforts to avoid exposure ex ante and remediate its impacts ex post; these costs must be considered for a full welfare analysis.

1. INTRODUCTION

> There are these two young fish swimming along and they happen to meet an older fish swimming the other way, who nods at them and says "Morning, boys. How's the water?" And the two young fish swim on for a bit, and then eventually one of them looks over at the other and goes "What the hell is water?"
>
> —David Foster Wallace, "This Is Water"

The impacts of pollution[1] on human health have been the subject of intense scrutiny for at least the past 70 years. Efforts to understand how pollution affects our lives have largely focused on easy-to-measure health outcomes such as hospitalizations or mortality. Economists have played an important role in this space, largely by improving the credibility of causal inference outside of the laboratory setting.

Yet these health encounters are likely to reflect the tip of the iceberg, as many less visible impacts of pollution also affect well-being. A burgeoning literature within economics has begun to investigate the causal effects of pollution on numerous "nonhealth" end points, such as worker productivity, school performance, decision-making, and even crime. While a distinction between the health and nonhealth harms from pollution is useful, we emphasize that the mechanisms underlying both types of harms are physiological. In the extreme, cardiovascular and respiratory impairment due to pollution exposure can lead to hospitalization or death. Yet, even those experiencing no such health harms may find their productivity diminished or their cognitive function impaired due to reduced blood flow and cell oxygenation caused by pollution exposure. Thus, the key distinction between the health and nonhealth literatures is that the former requires death or some kind of health encounter to be observed, while the latter focuses on subclinical effects that nonetheless impact behavior, performance, and skills.

Despite their shared etiological pathways, it is important to understand what is unique about the nonhealth space relative to the health one. First, the subtlety of the physiological effects that shape nonhealth end points makes behavioral responses to limit the harms from pollution more complicated. The absence of symptoms (or the manifestation of symptoms that simply feel like an off day) makes introspective causal attribution difficult. As such, ex ante avoidance behavior—preventative steps to limit pollution exposure—will be less extensive in this setting because people are imperfectly aware of the impacts of pollution. However, changes in nonhealth outcomes are often visible, and while optimizing agents may be unaware of the root causes of these changes, it is still possible to compensate for any impacts with ex post amelioration. Since this compensation is costly, it has important welfare implications.[2]

To illustrate the point, imagine that pollution exposure reduces test scores but that students and school administrators, being unaware of this link, fail to act to minimize student exposure to pollution. The realization of lower test scores, however, may lead students and the school to take remedial efforts to improve performance. The key insight here is that remediation does not require knowing that pollution lowered test scores; it only requires knowing that test scores are lower than acceptable. At the same time, repeated lectures and tutoring represent economic costs of pollution that may be hard to attribute but that matter for a comprehensive assessment of impacts.

Second, the mechanisms linking pollution to nonhealth outcomes are much less clear than the well-established biophysical pathways that link pollution to particular end points. Toxicological experiments that explore outcomes such as lung function or heart performance map clearly onto

[1] Unless otherwise specified, all references to pollution are specifically about air pollution that originates outdoors, which is the focus of this review. For an overview of the literature on the health and welfare effects of air pollution from indoor sources, readers are referred to Duflo et al. (2008).

[2] For an excellent example in the health context, readers are referred to Deschênes et al. (2017).

observational analyses that explore outcomes such as asthma and cardiovascular events. For nonhealth outcomes, the mechanism is often less clear, involving more speculative links that lean more heavily on scientific evidence from animal studies. This feature should not take away from the credibility of the findings, but the lack of an agreed-upon physiological mechanism calls for deeper study of such pathways and testing of mechanisms whenever possible. The more exploratory nature of such analyses raises concerns over potential data mining and *p*-hacking, such that prespecified analysis plans (PSAPs) are warranted when feasible. At the same time, these explorations can help shape scientific research agendas, moving beyond a paradigm in which biological science largely functions as an input to environmental economics research to one that creates a virtuous cycle in which each discipline helps illuminate and contribute to deeper insights in the other.

Third, the timing of effects for nonhealth outcomes is more varied than for health outcomes. Some consequences may be nearly immediate, where an elevated exposure leads to a physiological change that then alters a nonhealth outcome. These effects may be short lived once exposure returns to baseline, or they may endure beyond the exposure period, thereby affecting the stock of human capital. This delayed impact could be a result of latent effects, whereby no apparent impacts exist at the time of exposure, but they materialize at a future date, as is proposed in the fetal origins hypothesis (Barker 1990). Enduring effects may also arise because of dynamic complementarities in human capital accumulation (Cunha & Heckman 2007). A student whose learning is impaired in primary school due to pollution may struggle to matriculate through secondary and high school because they lack the fundamental building blocks on which knowledge accumulates. Thus, the enduring effect of pollution exposure at a young age in this case may be best represented by earnings as an adult. Moreover, these enduring impacts may lead to general equilibrium effects that have important implications for econometric and welfare analysis.

Finally, the dose-response relationship between pollution and nonhealth outcomes may be quite different than that for health outcomes. Just as subclinical impacts may arise at lower levels of pollution than more severe end points, nonhealth effects may arise at considerably lower pollution levels. Moreover, unlike the most severe health outcomes, which are largely limited to more vulnerable populations such as infants, the elderly, or those with underlying health conditions, the nonhealth outcomes that result from more subtle biophysical changes may apply to an otherwise healthy population, thereby broadening their impact.

2. SCIENTIFIC BACKGROUND

In this section, we provide a selective summary of the scientific evidence mapping air pollution exposure to physiological end points, with an eye toward mechanisms that underpin the recent nonhealth findings in the economic literature. These mechanisms include the well-known effects on respiratory and cardiovascular functioning as well as the emerging evidence documenting impacts on the central nervous system, particularly the brain, and genetic expression. Throughout this article, we focus on the impacts of short-term (as opposed to chronic) exposure to pollution from outdoor sources, even if exposure to pollutants like fine particles often takes place indoors. Much of the evidence originates in the controlled setting of the laboratory, with supporting correlational evidence from the field.

To start with the punchline, even low levels of pollution can yield cellular and organ system changes that the recipient experiences as an off day. Symptoms may include fatigue, irritability, impatience, and a lack of focus, to name a few. These symptoms, in turn, offer plausible pathways through which air pollution can affect a range of behavioral and socioeconomic outcomes. In Section 5, we review the economic literature that focuses on translating these physiological impacts into outcomes consequential for welfare analysis.

2.1. Heart and Lungs

The primary site of exposure to air pollution is the respiratory tract following inhalation. Ambient urban air pollution consists of gaseous components and particulate matter (PM). The former includes ozone (O_3), volatile organic compounds, carbon monoxide (CO), sulfur oxide, and nitrogen oxide (NO_x). PM, as the name suggests, is a measure of particles in the air, whose composition varies by location and even time of year, with size playing an important role in harm. Particles at the finer end of the spectrum are of paramount concern because fine PM, that is, particles less than 2.5 μm in aerodynamic diameter, can remain airborne for long periods, easily flow from outdoors to indoors (making exposure challenging to avoid), and lodge deep in the respiratory tract. In fact, most human exposure to PM of outdoor origin occurs indoors (Martins & Da Graca 2018, Krebs et al. 2021). The principal impact of exposure is inflammation in the lungs, which reduces the efficiency with which the body exchanges carbon dioxide for oxygen, and thus it impedes cellular function throughout the body. Repeated exposure to particle pollution aggravates the initial injury and promotes chronic inflammation (Viehmann et al. 2015).

Air pollution also impacts the cardiovascular system, partially due to the inflammatory response that has its origins in the lungs and also because some particle forms can be absorbed directly into the bloodstream (Oberdörster et al. 2004, Brook & Rajagopalan 2007). These changes caused by air pollution can affect blood pressure and heart rate variability as well as blood coagulation and atherosclerosis progression (Park et al. 2005, Giorgini et al. 2016). These physiological changes, in turn, are associated with consistent increased risk for cardiovascular events such as myocardial infarction, stroke, and heart failure (Brook et al. 2010).

While severe respiratory and cardiovascular impacts result in health system encounters, pollution exposure can also cause a range of subclinical symptoms (Novaes et al. 2010, DeMeo et al. 2004) that are insufficient to prompt a health care visit. Nonetheless, these physical manifestations can lead to fatigue, lack of focus, memory impairment, and other symptoms that can have subtle impacts important for human capital accumulation and performance (Delgado-Saborit et al. 2021).

2.2. The Brain

Mounting evidence suggests that air pollution can harm the brain (Costa et al. 2019) through associated increases in neuroinflammation and oxidative stress within the central nervous system (Calderón-Garcidueñas et al. 2008, Kraft & Harry 2011) and impaired function of receptors that regulate neuronal cell death (Ikonomidou et al. 2001). The primary route of exposure for these harms is inhalation, whereby pollutants can be translocated from the lungs to the blood and from there to the brain (Forman & Finch 2018). Fine and ultrafine PM can also enter the brain directly via the olfactory nerves and move to other regions of the brain such as the cerebral cortex and the cerebellum (Oberdörster et al. 2004).

Animal studies, mostly in mice and rats, have shown that air pollution can activate the brain's microglia in sex-dependent ways (Allen et al. 2017), causing neuroinflammation and oxidative stress that can lead to a host of neurological impairments (Win-Shwe et al. 2008, 2009, 2014; Ehsanifar et al. 2019) as well as altered motor activity (Yokota et al. 2009, Suzuki et al. 2010). Pollution can also affect brain chemistry by lowering levels of serotonin (Paz & Huitrón-Reséndiz 1996, Murphy et al. 2013), which regulates aggression and impulsivity (Coccaro et al. 2011, Siegel & Crockett 2013). Exposure can lead to changes in other emotional behaviors as well (Yokota et al. 2009), including anxiety (Ehsanifar et al. 2019) and depressive behavior (Fonken et al. 2011, Davis et al. 2013). While the timing of the exposure windows examined varies considerably, evidence indicates that impacts can arise as fast as within 24 h of exposure. All of these effects, however, appear to be more pronounced in response to pre- and/or postnatal exposure, when the central nervous system and brain are still engaged in rapid cell proliferation, migration, and differentiation (Bayer et al. 1993, Rodier 1995).

In short, the emerging evidence on the impacts of pollution on brain functioning suggests that pollution can touch almost every aspect of life by impairing cognitive function and altering emotional states. As we discuss in Section 5, this can include domains as wide ranging as decision-making, educational outcomes, and productivity, as well as criminal behavior.

2.3. Epigenetic Programming

Early pollution exposure may also have latent effects, whereby no apparent changes in human capital are evident during early childhood, but impacts manifest themselves later in life (Bale et al. 2010, Almond & Currie 2011). In some cases, it simply takes time for harms to reveal themselves, such as less severe cognitive impairments that are difficult to discern in young children who are generally not subject to formal cognitive evaluations until the later stages of elementary school. At the same time, emerging evidence suggests that these latent effects can also arise due to altered gene expression, known as epigenetics (Petronis 2010). While genetic sequences are determined by inheritance and remain unchanged, the epigenetic pattern is malleable and defines the expression of those genetic sequences. An epimutation is a change in gene activity that is associated with changes to the DNA molecule through methylation or other modifications of chromatin (Oey & Whitelaw 2014).

To date, studies suggest that polyaromatic hydrocarbons and $PM_{2.5}$ have modest effects on DNA methylation, and evidence is emerging for other air pollutants such as ozone and NO_x (Rider & Carlsten 2019). Moreover, methylation is only one of several epigenetic mechanisms that cells use to control gene expression (Phillips 2008). Indeed, recent evidence suggests that air pollution might contribute to the transmission of epimutations from gametes to zygotes by involving mitochondrial DNA, parental allele imprinting, histone withholding, and noncoding RNAs (Shukla et al. 2019).

While the evidence on both mechanisms and physiological end points in this scientific domain is still evolving, there are compelling reasons to believe that these early-life insults can manifest in later-life health and nonhealth outcomes alike. In the case of the latter, cognitive impairments appear to be especially important through impacts on synaptic plasticity, learning, and memory (Day & Sweatt 2011). As we discuss in Section 5, these impairments can in turn shape educational and labor market outcomes.

3. CONCEPTUAL MODEL

In this section, we model the behavior of an individual who is affected by pollution through both short-term and long-term channels and who pursues a mix of behaviors to minimize its ill effects.

3.1. Model Setup

Imagine a representative worker who values consumption C, dislikes labor L, and values their human capital H. Human capital is an aggregate of physical and mental attributes used in production, and the worker also enjoys it independently of its value in producing output. For instance, having poor lung function or chronic anxiety would reduce the worker's well-being even if their income were unchanged. We refer to effects that operate through the direct utility of human capital as well-being effects.

The agent lives for two periods: an initial current period, during which their human capital is exogenously given, followed by a long-term period comprising the remainder of their working life, during which their human capital will be affected by other factors. They discount their utility in this second period by a factor β:

$$U = U_1(C_1, L_1, H_1) + \beta U_2(C_2, L_2, H_2).$$

The worker decides how many labor hours to supply and how much to consume in each of two periods, subject to their budget constraint. In the first period, there is some level of ambient environmental pollution P.[3] The worker has access to an avoidance technology $f(\cdot)$ that allows them to pay to reduce the fraction of pollution that reaches them in the first period. Investing in air purifiers, limiting outdoor exercise, and wearing masks are a few examples of this kind of technology. Pollution exposure D is then a function of ambient environmental pollution P and avoidance spending A:

$$D = P[1 - f(A)].$$

Pollution exposure D has two effects on the worker. In the short run, it reduces labor productivity $F(H_1, D)$:

$$Y_1 = L_1 F(H_1, D).$$

Exposure also diminishes human capital in the second period. Second-period human capital H_2 is then determined as a function of the residual human capital H' from the first period and the worker's use of a remediation input M, which repairs some of the damage caused by pollution in the first period. Medical care is the most easily observed remediation input, but remediation can also include nonmedical interventions to improve both physical and mental functioning, such as exercise to improve overall health, or remedial instruction to compensate for reduced mental acuity:

$$H_2 = h(H', M) = H(H_1, D, M), \quad H' = H'(H_1, D).$$

The enduring effect of pollution reduces H_2 in the second period given any fixed level of remediation. This lowers labor productivity and utility from human capital in the second period:

$$Y_2 = L_2 G(H_2).$$

There are two important conceptual distinctions: First, avoidance must take place at the time of exposure, while remediation cannot take place until after sufficient time has passed for pollution to impact human capital. Second, avoidance has both an effect on current output in the first period that operates by improving the productivity of a unit of human capital F and an effect on output in the second period that operates through changes in the level of human capital H_2. In contrast, remediation impacts output only in the second period and solely through changes in the level of human capital H_2, not the marginal product of a unit of human capital. In short, avoidance reduces both short-run and enduring effects; remediation reduces only the enduring effects of pollution.

Combining the model features described above, we can write the maximized lifetime utility of the agent as[4]

$$\begin{aligned} V \equiv & \max_{C_1, L_1, A, M, C_2, L_2} U_1(C_1, H_1, L_1) + \beta U_2(C_2, H_2, L_2) \\ & + \lambda \left[L_1 F(H_1, D) + \frac{L_2 G(H_2)}{\chi} - (C_1 + A) - \frac{C_2 + M}{\chi} \right] + \mu \left[H(H_1, D, M) - H_2 \right]. \end{aligned} \tag{1}$$

Here, χ is the interest rate at which consumption can be transferred between periods.

[3]The assumption of zero pollution in the second period is for simplicity; one can interpret P as the deviation of pollution from an omitted baseline level.

[4]Throughout the following discussion, we treat λ and μ as fixed in order to discuss the impacts of small variations in pollution, which do not substantially change the marginal value of wealth or human capital.

3.2. Optimization

Our primary focus in this model is on avoidance and remediation behavior. Intuitively, optimal avoidance involves setting the marginal cost of avoidance equal to its marginal benefits. These benefits include contemporaneous productivity increases as well as improvements in future human capital that provide direct utility and increase future productivity. Optimal remediation involves setting the marginal cost of remediation equal to the marginal benefits it provides through the restoration of human capital and its resulting impacts on utility and output.

As noted above, our understanding of the relationship between human pollution exposure and nonhealth outcomes is still emerging. As such, a more realistic model of optimizing behavior would include agents who understood some but not all of the causal impacts of pollution. More formally, suppose that there are two components of human capital H, which we then write as $H_2 = Z(\Phi_2, \Psi_2)$. The component Φ represents aspects of human capital that are widely understood to be impacted by pollution, such as breathing difficulties, while the component Ψ represents other aspects of human capital that are not commonly viewed as being impacted by pollution, such as impulse control, test performance, or dementia.

The optimality condition for avoidance is

$$\frac{\partial V}{\partial A} : \lambda \left[L_1 \frac{\partial F}{\partial D}(-Pf'(A)) + \frac{L_2}{\chi} \frac{\partial G}{\partial H} \left(\frac{\partial H}{\partial \Phi} \frac{\partial \Phi}{\partial A} + \frac{\partial H}{\partial \Psi} \frac{\partial \Psi}{\partial A} \right) - 1 \right] + \mu \left(\frac{\partial Z}{\partial \Phi_2} \frac{\partial \Phi_2}{\partial A} + \frac{\partial Z}{\partial \Psi_2} \frac{\partial \Psi_2}{\partial A} \right) = 0. \qquad 2.$$

The first term represents the marginal effects on utility from increasing consumption (through an increase in productivity, plus higher output due to higher human capital in the next period, minus the cost paid), while the second represents the utility value gained through all channels by increasing later human capital.

Consider a situation in which workers incorrectly believe that $\frac{\partial \Psi}{\partial A} = 0$ when in fact $\frac{\partial \Psi}{\partial A} > 0$: In other words, they incorrectly believe that avoiding pollution has no benefits for this aspect of human capital. We can see that setting the term $\frac{\partial \Psi}{\partial A}$ in this expression to 0 would reduce the positive (benefit) terms without altering the utility cost of spending money on avoidance. The result is that the agent would choose a lower-than-optimal level of A, $A_N < A^*$, resulting in larger short-run effects of pollution than are optimal. We refer to agents who optimize while ignoring the Ψ terms in this first-order condition as partial information avoiders.

If the agent observes their health status H' coming into the second period and adjusts optimally, their choices will satisfy the following equality:

$$\frac{\partial V}{\partial M} : \lambda \left[\frac{L_2 \left(\frac{\partial G}{\partial H} \left(\frac{\partial H}{\partial \Phi} \frac{\partial \Phi}{\partial M} + \frac{\partial H}{\partial \Psi} \frac{\partial \Psi}{\partial M} \right)_{H'(A_N)} \right) - 1}{\chi} \right] + \mu \left(\frac{\partial H}{\partial \Phi} \frac{\partial \Phi}{\partial M} + \frac{\partial H}{\partial \Psi} \frac{\partial \Psi}{\partial M} \right)_{H'(A_N)} = 0. \qquad 3.$$

Here, the subscript $H'(A_N)$ indicates that these partial derivatives of the human capital evolution function depend on the suboptimal human capital $H'(A_N) < H'(A^*)$ with which a partial information avoider enters the second period. Under the reasonable assumption that remediation has higher marginal benefits for those with lower human capital, this increases remediation relative to its counterfactual level under optimal avoidance: Informational constraints lead to an increase in remediation to repair damages. Note that agents do not need to be able to accurately observe first-period pollution in order to make a choice that is optimal given their residual human capital H'.

As demonstrated above, making an optimal choice of avoidance requires observing the level of pollution P and understanding both the effectiveness of avoidance $f(\cdot)$ and the human capital evolution function $H'(H_1, \cdot)$. In contrast, when choosing remediation, it is sufficient to observe residual human capital H' and understand the effectiveness of the remediation input. In short, incomplete information about the harms from pollution will generally lead to suboptimal avoidance followed by higher levels of remediation.

3.3. Welfare Implications

For small changes in the level of pollution around the correctly anticipated level P, an envelope condition dictates that changes in the choice variables have no first-order impact on welfare. Here, we sketch out the full set of channels by which pollution affects welfare, including changes in the choice variables (consumption, labor, avoidance, and remediation).

3.3.1. First-period observables: output, consumption, leisure.

The full effects of pollution in the first period can be expressed as follows:

$$\frac{dU_1}{dP} = \frac{\partial U_1}{\partial C_1} \cdot \underbrace{\left(\underbrace{\left(L_1 \frac{\partial F}{\partial D}[1 - f(A)] + F(H_1, D)\frac{dL_1}{dP} + L_1 \frac{\partial F}{\partial D}(-Pf'(A))\frac{dA}{dP} \right)}_{\frac{dY_1}{dP}} - \frac{dA}{dP} - \frac{dS}{dP} \right)}_{\frac{dC_1}{dP}} + \frac{\partial U_1}{\partial L_1}\frac{dL_1}{dP}. \quad 4.$$

Here, S is savings from period 1, a variable previously left implicit in the budget constraint. The direct effect, dY_1/dP, depends on how avoidance and labor supply react and is distinct from the effect on consumption, dC_1/dP. The effect of changing labor supply (terms involving dL_1/dP) may contribute to output declines but also provides utility from leisure.

3.3.2. Second-period observables: now including human capital.

All the variables in Equation 4 are theoretically observable in period 1, but human capital effects do not materialize until some time after exposure. Pollution's enduring effects are reflected in second-period welfare:

$$\frac{dU_2}{dP} = \beta \left(\frac{\partial U_2}{\partial C_2} \underbrace{\left(\underbrace{L_1 \frac{\partial F}{\partial H_2}\left(\frac{\partial H_2}{\partial P} + \frac{\partial H_2}{\partial A}\frac{dA}{dP} \right) + G(H_2)\frac{dL_2}{dP}}_{\frac{dI_2}{dP}} - \frac{dM}{dP} + \chi \frac{dS}{dP} \right)}_{\frac{dC_2}{dP}} + \frac{\partial U_2}{\partial L_2}\frac{dL_2}{dP} + \frac{\partial U_2}{\partial H_2}\frac{dH_2}{dP} \right). \quad 5.$$

These enduring effects of pollution are in some ways analogous to the short-term effects, but with two key differences. The effects on productivity are mediated through changes in human capital rather than exposure. There are also direct utility effects of the human capital loss caused by pollution if remediation and avoidance are incomplete.

To recap, this simple model of consumption and production with pollution generates a few key insights:

1. Avoidance has direct effects on productivity that do not operate through its enduring effect on human capital. High-efficiency particulate air (HEPA) filters in an office do not just prevent future lung damage; they also increase today's output from workers who may not have any diagnosable health problems. Considering either impact alone understates the benefits of avoidance.
2. Agents who are unaware of some of pollution's impacts will in general pursue less avoidance than is optimal but will partly offset this with higher remediation later on. This suggests a role for public policies around providing information to improve avoidance behaviors.
3. The long-run effects of pollution on human capital, which may constitute the bulk of the impacts for some pollutants, cannot be assessed until long after the date of exposure. For other pollutants, the direct productivity effects may be substantial relative to long-run human capital harms.
4. The impact of pollution on consumption may differ significantly from its impact on income due to both consumption smoothing and changes in avoidance and remediation. Utility depends on consumption, not income; thus, an assessment of harms that studies only output effects does not fully capture harms from lost consumption.

4. EMPIRICAL METHODOLOGY

In this section, we describe several important methodological issues inherent in identifying the causal effects of air pollution on nonhealth outcomes. Many of the empirical concerns are similar to analyses on the health effects from pollution, so we eschew a complete assessment of all concerns (for a review, see Graff Zivin & Neidell 2013). Instead, we focus on a brief recap of similar issues, provide more elaboration on issues particular to nonhealth outcomes, and delve deeper into recent advances since Graff Zivin & Neidell (2013).

4.1. Defining Pollution Treatment: The Role of Avoidance and Mitigation

We begin with a framing of pollution treatment to define the distinction between pollution concentrations (or levels) and pollution exposure, as the two are often muddied in empirical work. Pollution concentrations are the ambient levels of pollution in the environment, whereas pollution exposure is an individual's exposure to pollution after any efforts to avoid it. Accordingly, studies may estimate either concentration-response or exposure-response functions depending on which treatment they observe. Importantly, any efforts to limit exposure to pollution (or mitigate any experienced harms) occur after pollution concentrations have been realized, thus representing ex post behaviors. The consequences of this distinction are twofold. First, the welfare analysis outlined above (and elsewhere—see, e.g., Cropper & Freeman 1991) defines treatment as pollution concentration and thus rests on the concentration-response function. Second, despite the challenges in measuring avoidance (and mitigation) behavior, failing to include it as a control variable does not present an econometric challenge for obtaining causal effects of the concentration-response function because it is an ex post behavior. Even if one could observe these behaviors and were interested in estimating an exposure-response function, including them in a regression model would reflect bad controls that may induce spurious correlation between the treatment and the outcome (Rosenbaum 1984, Angrist & Pischke 2010). Avoidance and mitigation behaviors must be measured for a welfare analysis—something lacking in current research—but they do not need to be included to estimate the concentration-response function properly.

4.2. Endogeneity of Pollution Concentrations

Given the focus on the concentration-response function, a major concern with identification is the endogeneity of ambient pollution levels. The issues that arise are, for the most part, similar to those that the researcher encounters when studying health outcomes. Graff Zivin & Neidell (2013) review these sources of endogeneity in detail, and we highlight two of them here. First, individuals choose residential locations based on the attributes of that area, which leads to a nonrandom assignment of pollution concentrations. Preferences over residential neighborhoods depend on factors such as employment opportunities, commuting costs, and local amenities in the area. These amenities are often bundled such that environmental quality is correlated with other attributes in a location, although the specific contents of a particular bundle vary by location. Different preferences, income levels, and susceptibility to pollution can lead to varying ambient pollution levels. The former two factors can lead to omitted-variable bias in cross-sectional studies, while the third can lead to simultaneity bias.

The second source of endogeneity is environmental confounding. Many of the factors that drive variation in pollution levels may also affect outcomes. For example, temperature can affect pollution formation, but it also has a direct impact on health, labor supply, and productivity that translates into economic costs (Dell et al. 2014, Graff Zivin & Neidell 2014, Addoum et al. 2020, Aguilar-Gomez et al. 2021). Fortunately, weather variables are readily observable; thoroughly and flexibly controlling for them is central to addressing environmental confounding.

Instrumental variables (IVs) and natural experiments have been used to overcome both sources of endogeneity mentioned above. The same instruments used in the health literature are largely valid for the nonhealth effects since the concerns listed above do not differ significantly. Recent examples include work by Deryugina et al. (2019), who instrument for air pollution using changes in local wind direction to estimate the life-years lost due to pollution exposure, and Schlenker & Walker (2016), who exploit idiosyncratic variation in daily airplane taxi time to measure the health effects of CO.

A critical caveat to any IV approach is underidentification. Researchers often possess one instrument, but there are multiple endogenous pollution variables. Further, the pollution variables are often highly correlated since they come from the same emission sources, making it difficult to attribute impacts to a specific pollutant. Estimating separate IV equations for each pollution variable does not provide unbiased estimates.[5] There are two solutions. One is to focus on the reduced-form relationship between the instrument and the outcome. This is often a relevant policy parameter because the instrument is potentially manipulable by policy. The second solution expands the number of instruments by exploiting different dimensions of an instrument. Wind speed, direction, and interactions with topography can yield a fuller set of instruments, as can the multiple dimensions of wind inversions, including speed, duration, and strength. For instance, Knittel et al. (2016) simultaneously estimate the effects of both CO and PM_{10} using changes in traffic by exploiting the fact that different weather conditions result in different pollution levels by pollutant.

4.3. Challenges to Measurement

Measurement error is a perennial concern when evaluating air pollution impacts. Ideally, air pollution monitors providing readings at high frequency would be available at the study sites (e.g.,

[5]The same is true if one uses an IV approach for one pollutant while controlling for the others.

where work is being performed or tests being taken); in practice, the pollution level assigned to an individual observation is often an inverse-distance weighted average from several monitors, which may each be several kilometers away. Hence, the measure of pollution available to the researcher likely contains noise relative to the true level of pollution at the study sites, which biases estimated impacts toward zero if the noise is random.

Recent advances that combine satellite measures with ground-based monitors using spatial mapping techniques, such as machine learning, produce high-quality reanalysis data at finer spatial scales across the entire globe. These data can yield significant improvements over the use of either fixed monitoring stations or satellite data alone (van Donkelaar et al. 2016).[6] Such data can be used at different temporal frequencies and geographical scopes depending on the users' interests. In general, the finer the temporal scale, the coarser is the spatial scale, and vice versa, with reliable measures for daily global measures (GMAO 2019) available at a 50×62.5-km grid and global annual surface $PM_{2.5}$ concentrations (van Donkelaar et al. 2016, Hammer et al. 2020) available at resolutions as fine as a 1×1-km grid cell. These trade-offs will likely become less stark as machine learning improves and longer data streams are available to train the models.

Measuring the dependent variable can also be challenging in this setting. In contrast with mortality and severe morbidity, many nonhealth outcomes are difficult to observe using typical survey data. Researchers often obtain data from proprietary sources; the digital revolution is making more of those available. Unfortunately, much of these data come from nonrepresentative samples—a single firm or a handful of schools—raising concerns about generalizability. Moreover, such data are often obtained through data use agreements and cannot be shared with other researchers to ensure reproducibility. While we are still in the early days of this literature, extending findings across settings is critical for welfare analysis and policy making going forward.

4.4. Multiple Hypothesis Testing

As discussed above, the literature on the nonhealth impacts of pollution is increasingly exploratory. A myriad of data from administrative and digital trace records allow researchers to expand the set of outcomes studied. This expansion, in turn, has the disadvantage of fostering a search for affected variables less grounded in theory. Researchers face behind-the-scenes decisions about their econometric specifications and need to be forthcoming, particularly when using proprietary data. We have two suggestions for best practices.

First, results should include adjustments for multiple hypothesis testing. Resampling methods, first proposed by Westfall & Young (1993), have become popular because they require fewer assumptions about the data-generating process, utilize data-based distributional characteristics, and can scale up reasonably well to high-dimensional settings (Westfall & Troendle 2008). The procedure proposed by Westfall & Young adjusts p-values and standard errors to account for multiple hypothesis testing and is readily available in standard software packages.

Second, we encourage the use of PSAPs when possible to limit data mining (Burlig 2018, Christensen & Miguel 2018). Although PSAPs can be limiting in certain studies (Miguel et al. 2014), the costs are likely much lower in this setting. The independent variables are often quite similar across studies (e.g., criteria pollutants), so the only differences across studies are the dependent variable, changes in context, and the temporal and spatial structure of the data. Econometric

[6]Using satellite data by themselves is problematic because satellites measure particles and the chemical composition of the entire column of air from ground to orbit (rather than surface measures) and provide poor measures on cloudy days. For a more comprehensive discussion of the trade-offs between satellite- and ground-based measures, readers are referred to Fowlie et al. (2019).

specifications may only need minimal modifications to accommodate these changes. Furthermore, PSAPs can allow sufficient flexibility to explore alternative functional forms, variations in the timing of effects, and multiple robustness checks. Deviations from the PSAPs are possible and sometimes desirable, but there are significant gains in transparency when researchers explain how they deviated from the plan and why.

5. EMPIRICAL REVIEW

Here we provide a review of the empirical literature on the nonhealth impacts of acute exposure to air pollution. While not comprehensive, it is designed to touch upon the three core and interrelated domains of influence—labor markets, cognitive performance, and decision-making—along with a discussion of latent effects within those categories. As previously discussed, we primarily focus on nonhealth outcomes with a physiological basis rather than those driven by behavioral responses to a health shock, though this distinction is sometimes unclear. We also limit our focus to studies with quasi-experimental research designs, such as the use of fixed effects and IVs, to isolate the causal effects of pollution. Since these designs are well established in the broader literature, we do not describe methods in detail; instead, a set of relevant, overarching identification issues is described in Section 4.

5.1. Labor

As discussed in the scientific background, pollution exposure can cause fatigue, irritability, impatience, and a lack of focus, among other symptoms. As highlighted in our conceptual model, these physiological sequelae potentially map onto short-run impacts on output through labor supply and productivity. In this section, we review previous findings that shed light on the type of tasks affected and the magnitude of impacts and how they add up to macro-level output changes.

5.1.1. Productivity effects. The myriad physiological impacts of pollution discussed earlier can alter task performance in a number of ways. These impacts are perhaps most intuitive in the context of physically demanding work. Graff Zivin & Neidell (2012) examine daily fluctuations in the daily exposure of piece-rate agricultural workers to ozone and find that a 10-ppb increase in ozone decreases earnings by 5.5%, despite ozone levels being below regulatory limits. As the authors note, the rapid onset and recovery from ozone exposure indicate that the observed productivity impacts are primarily due to short-term performance effects rather than declining health. Chang et al. (2016) study the effect of particulate pollution (measured as a 6-day average) on the productivity of piece-rate pear packers in a Northern California factory. They find that an increase in $PM_{2.5}$ of 10 $\mu g/m^3$ reduces productivity by approximately 6%, also at pollution levels well below current federal standards. Adhvaryu et al. (2019) study the effects of $PM_{2.5}$, measured at the hourly level at multiple locations in an Indian garment factory, on garment production. Their estimates imply a roughly 0.3% decline in productivity for every 10 $\mu g/m^3$ increase in $PM_{2.5}$, with larger effects for more complex tasks and older workers. In contrast to the above studies, He et al. (2019), who study the effects of $PM_{2.5}$ variation on piece-rate manufacturing worker output in two towns in China, fail to find a statistically significant effect of $PM_{2.5}$ during a worker's shift, even at baseline levels approximately eight times the current Environmental Protection Agency (EPA) standards. It is noteworthy, however, that they find small negative effects of prolonged exposure, with a persistent 10-$\mu g/m^3$ increase in $PM_{2.5}$ reducing daily output by roughly 1%.

Sports are highly monitored and physically demanding activities that have provided fertile ground for researchers to study the impacts of pollution. In the sports world, Lichter et al. (2017) find that higher $PM_{2.5}$ reduces the performance of professional soccer players in Germany, Guo

& Fu (2019) find that marathon runners in China run slower on days with higher pollution as measured by the Air Quality Index (AQI),[7] and Mullins (2018) finds that ozone impairs the performance of intercollegiate athletes in the United States. These studies reveal pollution effects on physically fit populations, sometimes at relatively low concentrations.

All the aforementioned studies focus on physically demanding occupations, but pollution may also affect workers' ability to perform more cognitive tasks. Chang et al. (2019) examine the performance of call center workers in Shanghai and Nantong, China. They find that a 10-unit increase in the air pollution index (API) decreases the number of daily calls by 0.35%, an effect that appears to occur through longer employee breaks.[8] Archsmith et al. (2018) study the effect of air pollution on Major League Baseball umpires, workers for whom sustained mental focus is key to job performance. They find that CO and $PM_{2.5}$ have negative effects on the accuracy of calls: a 1-ppm increase in CO reduces the fraction of accurate calls by 2%, and a 10-$\mu g/m^3$ increase in $PM_{2.5}$ reduces it by 0.4%. Kahn & Li (2020) examine the effect of $PM_{2.5}$ on the performance of trial judges during court cases in China. They find that a 1% increase in $PM_{2.5}$ leads to a 0.182% increase in case duration (an inverse measure of productivity), with effects stronger for older judges and for more complex cases. They also find that air quality alerts lower the effects of pollution, a finding consistent with avoidance behavior.

5.1.2. Labor supply. In addition to impacts on worker productivity conditional on working, evidence also points to sizeable labor supply responses to pollution when looking at populations broader than the employees of a particular firm. Aragón et al. (2017) find effects of $PM_{2.5}$ pollution on the labor supply of households in Lima, Peru, particularly for households with susceptible members: A 10-$\mu g/m^3$ increase in $PM_{2.5}$ reduces labor by 1.9 h per week. Hanna & Oliva (2015) find that for their preferred model specification, a 10-ppm decrease in SO_2 (due to the closure of a refinery in Mexico City) caused an increase of 1.3 h worked per week. Holub et al. (2021) study the impact of PM_{10} on sick days in Spain, using increases in PM_{10} caused by *calima* dust clouds from the Sahara. They find that an increase of 10 $\mu g/m^3$ raises the number of workers taking at least one sick day by 0.03 percentage points.

5.1.3. Effects at broad scales. While the previous studies find effects at particular firms or locations, an important question centers on how well the results generalize to broader scales. Using output and pollution data at the regional and national scales, Dechezleprêtre et al. (2019) examine economy-wide harms of pollution; they conclude that a 10-$\mu g/m^3$ increase in $PM_{2.5}$ reduces output by 8%. There is some indication of increasing marginal effects. Fu et al. (2018) investigate the effect of $PM_{2.5}$ on productivity for all large Chinese firms. Their estimates suggest that a 10-$\mu g/m^3$ increase in annual pollution causes an 8.2% drop in output per worker. These studies suggest that the combined productivity and labor supply effects have large impacts at national and even regional scales.

5.1.4. Labor overview. Taken together, the labor literature finds that air pollution reduces worker productivity and, in some cases, labor supply. However, productivity estimates vary considerably. There are several possible explanations for this divergence, which include, but are not limited to, differences in occupations, setting, pollutant of interest, and study design. More work

[7]The AQI is an overall index of air quality constructed by taking the maximum of rescaled measures of six criterion pollutants: SO_2, NO_2, CO, O_3, PM_{10}, and $PM_{2.5}$. It should be noted that the Chinese and American formulas for AQI differ slightly.

[8]The API is an earlier version of the Chinese AQI and is based on three pollutants; the level is driven mostly by PM_{10}.

is needed to reconcile these differences, but the ubiquity of harmful effects across pollution levels, demographics, and sectors underscores the wide reach of these impacts. The sizable macroeconomic impacts further highlight the perniciousness of these harms. Despite the measurement challenges, future work should focus on the consequences for high-skill and more creative occupations, for which the value marginal product of labor is particularly high. The role that pollution may play in sleep disruption and its knock-on effects for labor productivity is another area ripe for future exploration (Gibson & Shrader 2018). A better understanding of who bears the costs of these effects would also shed light on the incentives for private and public efforts to invest in both emissions control and exposure avoidance technologies.

5.2. Cognitive Performance

Consistent with the scientific literature on both respiratory and central nervous system effects, a growing body of evidence suggests that exposure to air pollution reduces performance on a variety of academic and cognitive tests. Ebenstein et al. (2016) examine the effects of fine PM and CO on nationwide student test performance in Israel. They find that a 1–standard deviation (SD) increase in $PM_{2.5}$ reduces scores on high-stakes tests by 1.7% of an SD, with larger effects for males. Bedi et al. (2021) investigate which types of mental processes are affected by $PM_{2.5}$ at the University of São Paulo in Brazil. They find 3% lower scores per 10 $\mu g/m^3$ on a grammatical reasoning test but no effect on other tests. Roth (2021) quantifies the effects of indoor PM_{10} on London-area university students taking high-stakes exams. He finds that a 10-$\mu g/m^3$ increase in PM_{10} reduces test scores by approximately 3% of an SD. Intriguingly, statistically significant effects are found only among males, consistent with the larger effect sizes found by Ebenstein et al. (2016). Zhang et al. (2018) use data from cognitive ability tests in a nationally representative longitudinal sample in China to evaluate the impact of pollution across a broader population at all ages. They find that API on the day of the test has a statistically significant effect for verbal tests only, with a 10-unit increase in API reducing scores by 0.4% SD. Longer lags of pollution have much larger effects on both verbal and math scores, with a 10-unit increase in average API over 3 years reducing verbal scores by 8.2% of an SD even after controlling for contemporaneous pollution. The authors find larger effects among men (particularly older men) and on verbal scores. In a related study that focuses on brain-training games, La Nauze & Severnini (2021) find that exposure to $PM_{2.5}$ significantly impairs adult cognitive function, with the largest effects found for those of prime working age. Using evidence from the same brain-training game, Krebs & Luechinger (2021) find that the effect is larger for the most experienced players.

Several additional papers provide evidence consistent with significant adverse effects of air pollution on test scores, but without detailed enough pollution data to isolate the pollutant or estimate a dose-response relationship. Graff Zivin et al. (2020) study the impact of agricultural fires in China on National College Entrance Exam scores, finding that a 1-SD increase in net upwind fires (which primarily emit PM) reduces exam scores by 1.42% of an SD,[9] with effects concentrated in high-ability students. Persico et al. (2021) find that openings of Toxic Release Inventory sites in Florida decrease standardized test scores by 2.4% of an SD and increase school absences by 0.4 percentage points for households living within a mile of the sites. These results are consistent with other evidence that shows that pollution increases school absenteeism (Currie et al. 2009, Liu & Salvo 2018).

Overall, existing research on the effects of pollution on short-run test performance leads to two tentative conclusions about heterogeneous impacts. First, effects may be larger for men (Ebenstein

[9]Reliable data on pollutant levels are not available in this setting.

et al. 2016, Zhang et al. 2018, Roth 2021); second, effects may be larger for verbal than for nonverbal tests (Zhang et al. 2018, Bedi et al. 2021). Establishing which groups of people and which mental processes are most affected by pollution may lead to insights into the pathophysiological mechanisms involved, as noted by Zhang et al. (2018). More empirical research is also needed to understand the accumulation of these effects vis-à-vis dynamic complementarities in learning (Cunha & Heckman 2007) and the intermittent feedback that enables compensatory behavior (Graff Zivin et al. 2018).

5.3. Decision-Making

Pollution can influence the decision-making process through at least three channels: (*a*) altering perceived payoffs, (*b*) altering risk perceptions, and (*c*) altering risk preferences (Bondy et al. 2020). The most direct pieces of evidence on this come from the financial sector. Huang et al. (2020) examine whether pollution negatively affects trading performance. Using account-level equity-transaction data from a large Chinese brokerage house, they find pollution exacerbates three common behavioral biases among investors: (*a*) the tendency to sell assets that have increased in value while keeping assets that have dropped in value, (*b*) excessive trading, and (*c*) the purchase of attention-grabbing stocks. Their back-of-the-envelope calculation suggests that the reduced performance due to air pollution accounts for roughly 6.8% of the average underperformance of individual investors in their sample. Dong et al. (2021) explore the effect of acute pollution exposure of investment analysts in China. They find that higher AQI during corporate site visits leads to more pessimistic projections of earnings forecasts. Meyer & Pagel (2017) find related results for individual investors in Germany, who are less likely to sit down, log in, and trade in their brokerage accounts when exposed to pollution.

Sager (2019) provides additional evidence by exploring the effect of air pollution on the number of traffic accidents in the United Kingdom between 2009 and 2014. Their findings suggest a 0.3–0.6% increase in accidents per day for each additional 1 $\mu g/m^3$ of $PM_{2.5}$. While some of these accidents may be due to the effects of diminished reaction time, as the authors speculate, they are also consistent with impaired judgment, although the precise mechanisms driving this change in decision-making remain unclear. In a completely different setting, Künn et al. (2019) find that higher levels of air pollution reduce the strategic decision-making of chess players, with a 10-$\mu g/m^3$ increase in the indoor concentration of $PM_{2.5}$ increasing a player's probability of making an erroneous move by 18.8%.[10]

Decision-making can also be affected through neuroinflammation and reduced serotonin production, which can lead to aggressive behavior. Herrnstadt et al. (2021) exploit detailed location data on over two million serious crimes reported to the Chicago Police Department over 12 years. Their estimates suggest that a 1-SD increase in PM_{10} concentrations causes a 2.9% increase in violent crime but has no impact on the commission of property crime. Burkhardt et al. (2019) examine the impact of short-term exposure to $PM_{2.5}$ and O_3 on crime and aggression by county in the United States. They find that a 10% increase in same-day exposure to $PM_{2.5}$ and O_3 is associated with increases in violent crimes of 0.14% and 0.3%, respectively, costing the country roughly $1.4 billion in crime costs per year. Bondy et al. (2020) find that pollution affects not only violent crimes but also those that are economically motivated. They employ daily administrative data for London in 2004–2005 and find that a 10-point increase in the AQI increases the crime rate by 1.2% and that experiencing an AQI above 35 (near the high end of the range classified by

[10]A related literature finds conflicting evidence on risk-taking, proxied with lottery sales (Bondy et al. 2020, Chew et al. 2021).

the EPA as good) leads to 3.7% more crimes. Importantly, all of these effects on crime manifest at pollution levels that are well below current regulatory standards, consistent with the findings of pollution effects on physical (Graff Zivin & Neidell 2012, Chang et al. 2016) and cognitive (Archsmith et al. 2018, Bishop et al. 2018) performance domains.

Despite the compelling evidence on pollution and decision-making, much remains uncertain in this space, particularly regarding the specific mechanisms driving many of these effects. A great deal of the life of *Homo economicus* is driven by time and risk preferences, and much more work is needed to understand these impacts. Cognizant of multiple hypothesis testing concerns, future research should borrow from the behavioral economics toolkit to utilize controlled experiments to assess the degree to which decision-making anomalies may be driven by ambient air pollution. Since many important decisions are made over extended periods of time, a deeper understanding of the temporal signature of the dose-response function and how that interacts within the ecology of multiple decisions is also an area rich for future exploration both inside and outside of economics.

5.4. Human Capital Effects of Early Exposure

In keeping with the fetal origins hypothesis, there is also evidence that pollution during gestation impacts nonhealth outcomes later in life.

Sanders (2012) examines the impact of prenatal exposure to total suspended particles (TSPs; particles 100 μm or smaller) on the long-term educational outcomes of students in Texas, measured as performance on a high-stakes standardized test. He finds that a 1-SD increase in TSPs in the year of birth reduces test scores by 2% of an SD. Bharadwaj et al. (2017) employ data on date of birth of children in Santiago, Chile, to assign pollution levels during gestation. They focus on CO and PM_{10}[11] and find that a 1-ppm increase in CO over the course of the pregnancy reduces math scores on a fourth-grade standardized test by 0.06 SD and language scores by 0.076 SD. Results for PM_{10} and CO separately show statistically significant negative effects on language scores but not math scores (at the 5% level), consistent with the short-run effects literature reviewed in Section 5.2.

Isen et al. (2017) examine the effects of early-life TSP exposure on both earnings and labor force participation at age 30 in the United States. They find that a 10-μg/m^3 increase in TSPs in the year of birth caused a 1.4% decline in income and a 2.8% decline in the number of quarters employed.

Voorheis (2017) brings much of this nonhealth literature together by linking the American Community Survey to Social Security and income tax data. He finds that a 10-μg/m^3 increase in TSPs in utero lowers yearly earnings by $246 and the probability of college attendance by 1.8%. Both in utero exposure and exposure during adolescence reduce high school completion rates and raise the likelihood of incarceration, though with heterogeneous effect sizes by race and parental income.

We conclude by noting that new evidence suggests that the effects of early-life pollution exposure may persist beyond the generation exposed to it. Colmer & Voorheis (2020) link cohorts of respondents in the US Census to evaluate the impact of TSPs on the educational attainment of the children of people exposed to lower TSPs. Their estimates imply that a 10-μg/m^3 increase in TSPs is associated with a reduction in college attendance of 3.8 percentage points. As effects appear to be the same for adopted and biological children, the authors theorize that differences in parental resources and investments account for most of the effects. The remarkably long reach of acute pollution exposure has important implications for welfare and thus the returns to any policies that might limit that exposure.

[11] Due to the high correlation between these two pollutants, it is not clear which one is the causal agent.

6. CONCLUSIONS

A blossoming literature has begun to link air pollution to a wide range of nonhealth outcomes. While the physiological causes of these harms are the same as those driving the better-known health impacts from pollution, their impacts are subtle, sometimes imperceptible, and in some cases may arise from impacts on brain functioning and genetic expression. Moreover, these impacts are generally not limited to vulnerable populations and manifest at quite modest levels of pollution, suggesting that even small individual impacts from air pollution exposure may have substantial economy-wide implications. However, further work is needed along several dimensions.

First, causal research designs to bridge the gap between laboratory and epidemiological evidence are essential. Evidence from the laboratory reveals impairments on a wide range of subclinical outcomes but with unclear implications for human well-being outside of the laboratory. Moreover, evidence from animal behaviors requires additional translation to the human experience. How might decreased lung functioning or increased blood pressure impact cognitive performance or decision-making? What does impaired spatial memory or increased impulsivity in a rodent imply about labor productivity or forward-looking behavior in humans? How quickly after exposure might these economic impacts manifest, and how long might they endure? In domains like cognition where dynamic complementarities are likely to effect outcomes over the long run (Cunha & Heckman 2007), the creation of suitable surrogate indices that predict the value of the long-term outcome given the short-term outcomes (Athey et al. 2019) represents a particularly fruitful area for future research. Moreover, this review has focused on the impacts of acute exposure, in part because it is more amenable to econometric techniques that rely upon quasi-experimental shocks. A causal understanding of the long-run effects of exposure on health and nonhealth end points alike remains elusive and is an area that requires far more scrutiny.[12]

Second, we need a much better understanding of the behavioral responses to pollution, including the role of avoidance behavior in limiting exposure (long understood though often poorly measured), as well as the role played by compensatory investments that ameliorate harms after exposure. Responding optimally to pollution requires weighing costs and benefits, which themselves depend on a wide range of socioeconomic factors, including mobility, school quality, and the availability of resources required to avert and compensate.[13] As we have argued throughout this review, ex post behaviors are especially important in nonhealth domains where individuals may find the relationship between pollution and outcomes opaque but the outcomes themselves relatively visible. Since many compensatory investments require the consumption of nonmarket goods (e.g., bringing work home to complete for the next day) and services that are not readily recorded in data sets (e.g., school tutors or upskilling services), they are difficult to measure.

Third, more work is needed on the design of optimal environmental policies. People employ costly behavioral responses to cope with pollution, which necessarily implies that the full welfare costs of pollution are larger than those tied to health and nonhealth outcomes alone. The relevance of behavioral responses also raises the prospect of new regulatory approaches. If

[12]The work by Bishop et al. (2018), who use a 10-year panel of Medicaid beneficiaries to estimate the impacts of $PM_{2.5}$ exposure on dementia, is a notable exception to the usual focus on acute exposure.

[13]Since exposure to poor environmental quality tends to correlate with low income, this tends to magnify the impacts on the poor, who have fewer financial resources to dedicate to avoidance and compensatory actions. While environmental justice has gained prominence within the field (for a good review, see Banzhaf et al. 2019), this aspect of the problem is largely missing from the debate.

ex post compensation is inefficient due to incomplete information, informational interventions can reduce costs. Investments that lower the costs or improve the effectiveness of avoidance and compensation technologies can yield similar dividends. Whether these objectives are best achieved through informational campaigns, behavioral nudges, tax incentives, or direct public investment is an open question, but it is clear that efforts to improve behavioral responses may also serve as an important part of the regulatory armamentarium.

Lastly, the empirical evidence that we have reviewed in this article is relatively new, spans many domains, and yields a wide range of results. Replication is critical here. Only a handful of studies produce estimates at a national level, and more could be done to estimate these relationships in other contexts. This will, in turn, help to generalize them beyond the narrow settings that have thus far been necessary to pin down empirical identification. Interdisciplinary collaborations are essential for unpacking the mechanisms driving these empirical relationships and disseminating the findings to a wider audience. Additionally, the broad etiologic basis that gives rise to so many potential impacts requires a more disciplined approach to hypothesis testing to avoid the so-called file drawer problem in which nonresults are buried on hard drives while significant ones, and sometimes only surprising ones, are published in journals.

Some level of pollution is part of the ether in which all human activity takes place. The recognition that even low levels of pollution can affect human capital accumulation and functioning and that humans generate a great deal of that pollution underscores the epibiotic relationship between humans and the environment. As David Foster Wallace noted in the quotation that began this article, the ubiquity of our environment can easily blind us to its impacts upon us. It appears that virtually no aspect of human life is unaffected by the quality of our air. While the science and economics on these impacts will continue to evolve, it should be clear that, to a significant degree, we are what we breathe.

SUMMARY POINTS

1. Air pollution causes meaningful damage to productivity in a wide range of contexts through both physical and cognitive detriments.
2. In addition to productivity impacts, air pollution has effects on human capital and decision-making.
3. The impacts from acute exposures may be larger in the long run due to latency and the dynamics of impact accumulation.
4. Care must be taken to avoid data mining given the wide range of possible impacts.

FUTURE ISSUES

1. Attempts to avoid and remediate the effects of pollution contribute to costs but are understudied.
2. Although emerging evidence suggests the generalizability of certain impacts, further investigation is needed to assess the full scope of external validity.
3. Investigating the heterogeneity of effects may cast light on pathophysiological mechanisms behind cognitive impacts.

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