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THIS IS AIR: THE "NON-HEALTH" EFFECTS OF AIR POLLUTION

Sandra Aguilar-Gomez
Holt Dwyer
Joshua S. Graff Zivin
Matthew J. Neidell

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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 "non-health" endpoints, 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, both in 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

Sandra Aguilar-Gomez
University of California, San Diego
saguilargomez@ucsd.edu

Holt Dwyer
Department of Economics
University of California, San Diego
La Jolla, CA 92093
bdwyer@ucsd.edu

Joshua S. Graff Zivin
University of California, San Diego
9500 Gilman Drive, MC 0519
La Jolla, CA 92093-0519
and NBER
jgraffzivin@ucsd.edu

Matthew J. Neidell
Department of Health Policy and Management
Columbia University
722 W 168th Street, 6th Floor
New York, NY 10032
and NBER
mn2191@columbia.edu

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"

1 Introduction

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 "non-health" endpoints, such as worker productivity, school performance, decision making, and even crime. While a distinction between the health and non-health 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 non-health 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 non-health space relative to the health one. First, the very subtlety of the physiological effects that shape non-health endpoints 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) make 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

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

pollution. On the other hand, changes in non-health 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 minimise 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 which matter for a comprehensive assessment of impacts.

Second, the mechanisms linking pollution to non-health outcomes are much less clear than the well-established biophysical pathways that link pollution to particular endpoints. Toxicological experiments that explore outcomes such as lung function or heart performance map clearly onto observational analyses that explore outcomes such as asthma and cardiovascular events. For non-health outcomes, the channel 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 pre-specified analysis plans 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 non-health outcomes is more varied than for health outcomes. Some consequences may be nearly immediate, where an elevated exposure leads to a physiological change, which alters a non-health outcome. These effects may be short-lived once exposure returns to baseline or they may endure beyond the exposure period, 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

[2]See Deschênes et al. (2017) for an excellent example in the health context.

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 non-health outcomes may be quite different than for health outcomes. Just as subclinical impacts may arise at lower levels of pollution than more severe endpoints, non-health 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 non-health 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 into physiological endpoints, with an eye toward mechanisms that underpin the recent "non-health" findings in the economic literature. These 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, in turn, offer plausible pathways through which air pollution can affect a range of behavioral and socioeconomic outcomes. In Section 5, we will 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 (VOCs), carbon monoxide (CO), and sulfur and nitrogen oxides (SO_x and NO_x). Particulate matter, 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 a paramount concern because fine PM, particles less than 2.5 microns 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 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, in part due to the inflammatory response that has its origins in the lungs but 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 (Giorgini et al., 2016; Park et al., 2005). These, 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 healthcare 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

In recent years, mounting evidence suggests that air pollution can harm the brain (Costa et al., 2019) through associated increases in neuro-inflammation 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, where pollutants can be translocated from the lungs to the blood and from there to the brain (Forman & Finch, 2018). Fine and ultrafine particulate matter can also enter the brain directly via the olfactory nerves and onward 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 neuro-inflammation 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 exposure windows examined varies considerably, evidence indicates that impacts can arise as fast as within 24 hours of exposure. All of these effects, however, appear to be more pronounced in response to pre- and/or post-natal 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 will see 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 (PAHs) and $PM_{2.5}$ have modest effects on DNA methylation, with emerging evidence for other criteria air pollutants such as ozone and nitrogen oxides (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 transmission of epimutations from gametes to zygotes by involving mitochondrial DNA, parental allele imprinting, histone withholding and non-coding RNAs (Shukla et al., 2019).

While the evidence on both mechanisms and physiological endpoints 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 non-health 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 will see in Section 5, these 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.

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, but which the worker also enjoys 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 will 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 $F(H_1, D)$.

[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.

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

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 non-medical 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]

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

[4]Throughout the following discussion, we will 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.

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

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 earlier, our understanding of the relationship between human pollution exposure and non-health 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 which are not commonly viewed as being impacted by pollution, such as impulse control, test performance, or dementia.

The optimality condition for avoidance is:

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

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 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 "partial information avoiders."

If the agent observes their health status H' coming into the second period and adjusts optimally, they will solve:

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

Here the subscript $H'(A_N)$ indicates that these partial derivatives of the human capital evo-

lution function depend on the sub-optimal 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.

3.1 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).

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 left implicit in the budget constraint previously. 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 provide utility from leisure.

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. 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 which 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 non-health 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; see Graff Zivin & Neidell (2013) for a review. Instead, we focus on a brief recap of similar issues, provide more elaboration on issues particular to non-health 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 consequence of this distinction is 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 challenge in measuring avoidance (and mitigating) 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 translate into economic costs (Dell et al., 2014; Graf 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 for addressing environmental confounding.

Instrumental variables (IV) 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 non-health effects since the concerns listed above do not differ significantly. Recent examples include (Deryugina & Hsiang, 2014), 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 instrumental variables approach is under-identification. 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

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

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., 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 the spatial scale and vice versa, with reliable measures for daily global measures (GMAO, 2019) available at a 50 km $\times$ 62.5 km grid and global annual surface $PM_{2.5}$ concentrations (Hammer et al., 2020), (van Donkelaar et al., 2016) available at resolutions as fine as a 1km x 1km 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 non-health 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 this data comes from non-representative samples – a single firm or a handful of schools – raising concerns about generalizability. Moreover, such data are often obtained through DUAs 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.

[6] Using satellite data by itself is problematic because satellites measures 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 tradeoffs between satellite and ground-based measures, see Fowlie et al. (2019)

4.4 Multiple hypothesis testing

As discussed above, the literature on the non-health impacts of pollution is increasingly exploratory. A myriad of data from administrative and digital trace records allows 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 in 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 pre-specified analysis plans (PSAPs) when possible to limit data mining (Christensen & Miguel, 2018; Burlig, 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 that the only differences across studies are the dependent variable, changes in context, and the temporal and spatial structure of the data. Econometric 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 PSAP 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 non-health impacts of acute exposure to air pollution. While not comprehensive, it is designed to touch upon the three core, and inter-related 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 non-health 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 instrumental variables, 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 are described in Section 4.

5.1 Labor

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 indicates 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 six-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 μg/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 μg/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 8$\times$ current EPA standards. It is noteworthy, however, that they find small negative effects of prolonged exposure, with a persistent 10μg/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 focused on physically-demanding occupations, but pollution may also affect workers' ability to perform more cognitive tasks. Chang et al. (2019)

[7]The AQI is an overall index of air quality, constructed by taking the maximum of re-scaled 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.

examine the performance of call center workers in Shanghai and Nantong, China. They find that a 10-unit increase in the air pollution index (AQI)[8] decreases the number of daily calls by 0.35%, an effect that appears to occur through longer employee breaks. 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 μg/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.

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 labor supply of households in Lima, Peru, particularly for households with susceptible members: a 10 μg/m^3 increase in $PM_{2.5}$ reduces labor by 1.9 hours 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 hours 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 μg/m^3 raises the number of workers taking at least one sick day by 0.03 percentage points.

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 scale, Dechezleprêtre et al. (2019) examine economy-wide harms of pollution; they conclude that a 10 μg/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 μg/m^3 increase in annual pollution causes an 8.2% drop in output per worker. These studies suggest the combined productivity and labor supply effects have large effects at national and even regional scale.

[8] In this sample, the maximum is almost always PM_{10}, which therefore determines the AQI.

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 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 particulate matter and carbon monoxide on nationwide student test performance in Israel. They find that a 1 s.d. increase in $PM_{2.5}$ reduces scores on high-stakes tests by 1.7% of a standard deviation, with larger effects for males. Bedi et al. (2021) investigate which types of mental processes are affected by $PM_{2.5}$ at the University 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 a standard deviation. Intriguingly, statistically significant effects are found only among males, consistent with the larger effect sizes found in 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% s.d. Longer lags of pollution have much larger effects on both verbal and math scores, with a 10-unit increase in average API over three years reducing verbal scores by 8.2% of a s.d. 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.

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 one s.d. increase in net upwind fires (which primarily emit PM) reduces exam scores by 1.42% of a s.d.[9], with effects concentrated in high-ability students. Persico et al. (2021) find that openings of Toxic Release Inventory (TRI) sites in Florida decreases standardized test scores by 2.4% of a s.d. and increases school absences by 0.4 p.p. for households living within a mile of the sites.

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 et al., 2016; Roth, 2021; Zhang et al., 2018); second, effects may be larger for verbal than for nonverbal tests (Bedi et al., 2021; Zhang et al., 2018). 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-a-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: (i) altering perceived payoffs, (ii) altering risk perceptions, or (iii) 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 under-performance 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

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

exposed to pollution.

Sager (2019) provides additional evidence by exploring the effect of air pollution on the number of traffic accidents in the U.K. 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 μg/m^3 increase in the indoor concentration of $PM_{2.5}$ increasing a player's probability of making an erroneous move by 18.8%.

Decision making can also be affected through neuro-inflammation 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 twelve years. Their estimates suggest that a 1 s.d. 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 U.S. 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 ten-point increase in the AQI increases the crime rate by 1.2%, while experiencing an AQI of above 35 (near the high end of the "Good" range) 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 Chang et al. (2016); Graff Zivin & Neidell (2012) 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 with regards to 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 non-health outcomes later in life.

Sanders (2012) examines the impact of prenatal exposure to total suspended particles (TSP, 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 s.d. increase in TSP in the year of birth reduces test scores by 2% of a s.d.. 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}[10] 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 s.d. and language scores by 0.076 s.d. 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 U.S. They find that a 10 μg/m^3 increase in TSP 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 non-health 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 TSP 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 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) links cohorts of respondents in the Census to evaluate the impact of TSP on the educational attainment of the children of people exposed to lower TSP. Their estimates imply that a 10 μg/m^3 increase in TSP is associated with a reduction in college attendance of 3.8 p.p. 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.

[10]The high correlation between these two pollutants means that which one is the causal agent is not clear.

6 Conclusions

A blossoming literature has begun to link air pollution to a wide range of 'non-health' 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 paper 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 non-health endpoints alike remains elusive and is an area that requires far more scrutiny.[11]

Second, we need a much better understanding of the behavioral responses to pollution. This includes 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.[12] As we have argued throughout this paper, *ex-post* behaviors are especially important in non-

[11] 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.

[12] 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 (see Banzhaf et al. (2019) for a good review) this aspect of the problem is largely missing from the debate.

health 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 non-market 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 non-health outcomes alone. The relevance of behavioral responses also raises the prospect of new regulatory approaches. If *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 paper 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 which gives rise to so many potential impacts requires a more disciplined approach to hypothesis testing to avoid the 'file drawer' problem in which non-results 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 quote which 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.

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