Three Essays on Health and the Environment

By

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A dissertation submitted in partial fulfillment of

the requirements for the degree of

Doctor of Philosophy

(Agricultural and Applied Economics)

at the

UNIVERSITY OF WISCONSIN-MADISON

2017

Date of final oral examination: 04/24/2017

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Acknowledgements

The completion of a PhD is not possible without a community of support. I am indebted to my advisor, Dan Phaneuf, for his guidance throughout my graduate career. His constant enthusiasm in my work was motivating and much appreciated. I am also grateful to the rest of my dissertation committee, John Mullahy, Nick Parker, Corbett Grainger, and Jason Fletcher, for always having an open door to discuss research or job market intricacies. I have also greatly benefited from the greater Taylor Hall community, through administrative support, presentation feedback, and countless coffee hours.

Grants from the Robert Wood Johnson Foundation and the UW-Madison Global Health Institute acknowledged the broad appeal of my work and kept me motivated. Research funding through the departments of Agricultural and Applied Economics and Population Health Sciences allowed me to focus on my dissertation and become a well-rounded researcher.

The friendships I have developed here made my time in Madison truly memorable. They helped me grow personally and professionally, while providing necessary support and stress relief. I am grateful to my parents, who have continued to support me morally, and often financially, throughout my undergraduate and graduate careers. I know that without this safety net, many of the things I have accomplished would not have been possible. Lastly, I am thankful to Bryton, who has endured my occasional grumpiness throughout the past few years and has shared and celebrated my achievements along the way.

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Introduction

This dissertation explores relationships between environmental factors and human health outcomes in various settings. A primary reason for valuing environmental quality is for its associated health benefits, but the activities that generate pollution often also have economic value. Optimal environmental regulation must take both costs and benefits into consideration, so understanding the full extent of these values is essential. In my essays, I focus on measuring the costs of pollution, which comes with a number of empirical challenges. Exposure to environmental factors is not randomly assigned because environmental quality is in part determined by human activities and choices in complex economic systems. Each chapter in this dissertation yields new information on the health benefits of environmental quality and accounts for confounding factors that inhibit causal inference.

The first chapter estimates how medical expenditures respond to changes in air quality. The health-related costs of poor air quality come in a variety of forms, including defensive expenditures, medical expenditures, lower worker productivity, suffering from illness, and premature mortality. As medical technology improves and demographics shift in the United States, more people are living longer with chronic respiratory conditions, which are exacerbated by pollution exposure. In this chapter, I find that increases in fine particulate matter significantly increase medical expenditures for these types of conditions. To estimate this effect, I first connect household level medical expenditure panel data with measures of ambient pollution. The data allows me to estimate the impact of pollution on a variety of different types of medical expenditures, such as medication purchases and hospital visits, and for various disease types, such as respiratory or cardiovascular conditions. Identification first comes from including household fixed effects, which control for unobserved household characteristics and some types of long-term avoidance behaviors. Next, I instrument for pollution measures using variation in emissions from distant sources. These pollution sources are less likely to be related to unobserved local sources of economic activity that may confound estimated effects. The instrumental variables strategy also accounts for potential measurement error in the pollution monitoring data. With this estimation strategy, I find that a one standard deviation decrease in fine particulate matter decreases medical expenditures for chronic respiratory diseases by 8.3 percent. Considering total spending on these conditions in the United States, this corresponds to $6.3 billion in savings annually. The costs measured in this chapter are only one piece of the overall economic costs of air pollution. Still, reductions in spending on chronic respiratory disease may alone be sufficient to justify annual environmental compliance costs for electricity generating units, which are the source of most of the emissions reductions during the study period.

The second chapter of this dissertation examines relationships between built-environment features and human health outcomes. Recent research in public health finds associations between the proximity of an individual to greenspace and various health outcomes, including obesity, cardiovascular disease rates, depression, and anxiety. Based on these associations, it is sometimes asserted that better access to parks will lead to improved health outcomes. This chapter tests this assertion by focusing on an issue sometimes ignored in this literature: people sort themselves into neighborhoods based on the characteristics of those neighborhoods and their personal preferences. Using observed neighborhood location decisions by young adults from the National Longitudinal Study of Adolescent to Adult Health (Add Health), I find that living near a park decreases obesity. My estimation strategy reveals that accounting for time varying unobserved variables is crucial when estimating the relationship between greenspace and health. To identify the effect, I first exploit the panel design of the data and include individual fixed effects to control for unobserved heterogeneity. I then utilize a novel instrumental variables strategy that instruments for built environment features using other neighborhood characteristics that are unrelated to health outcomes. This approach reveals that one additional park within one kilometer of an individual's residence decreases body mass index (BMI) by 1.25 percent. My study complements the current literature by yielding new evidence on how greenspace amenities impact health outcomes, and how heterogeneous amenity valuations may account for observed greenspace/health associations.

The last chapter investigates the defensive actions people take to avoid exposure to air and water pollution. These avoidance behaviors are generally not observed by researchers and obfuscate the true relationship between ambient pollution levels and health outcomes. Avoidance behaviors themselves are costly, so understanding their prevalence gives a clearer idea of the magnitude of this piece of pollution costs and the subgroups that are disproportionately bearing these costs. The answers to these questions also enhance our understanding of the motivations behind avoidance behaviors. Using survey response data that asks individuals if they have engaged in a number of defensive behaviors, I examine which types of behaviors are most common and which demographic and economic characteristics are the strongest predictors of defensive behavior decisions. I access confidential behavioral data that is geographically matched to data on weather and air quality outcomes, which are important determinants of some types of avoidance behavior. Further, I control for a variety of proxies for unobserved health and risk preferences and implement instrumental variables estimation to address endogeneity issues. I find that leisure time preference and labor market variables have significant impacts on the decision to engage in defensive behaviors, and the determinants of defensive behavior vary substantially over behavior type.

Chapter 1: The Morbidity Costs of Air Pollution: Evidence from Spending on Chronic Respiratory Conditions

1.1 Introduction

People living in urban areas are frequently exposed to unhealthy levels of air pollution, and the biological consequences of this exposure include damage to respiratory and cardiovascular health. The Clean Air Act (CAA) introduced policies to reduce ambient levels of pollution in order to minimize these negative health consequences. In accordance with the CAA, the Environmental Protection Agency (EPA) sets National Ambient Air Quality Standards (NAAQS) that limit allowed concentrations of six criteria pollutants, including sulfur dioxide and particulate matter. Evaluating the efficiency of the criteria pollutant standards requires estimates for pollution abatement costs, and the demand for pollution reductions. In 2010, economy-wide compliance costs for the CAA were estimated to be over $50 billion (US EPA, 2011). The economic benefits from pollution reduction arise mainly from reductions in mortality and morbidity rates, along with improved environmental quality. For the health impacts, measuring demand requires an empirical link between health-related outcomes and ambient pollution, and theory linking health outcomes to welfare measures. The empirical task is complicated by the inability to observe individual-level exposure to ambient pollution. Furthermore, even if a proxy pollution measure is available, theory predicts that people will engage in defensive actions aimed at reducing their exposure to pollution, or alleviating the ill effects of exposure once it has occurred. These behaviors decrease mortality and morbidity rates and, if not addressed, will compromise efforts to estimate the causal impact of pollution on health outcomes.

Defensive behaviors also provide an avenue for understanding the demand for air pollution reductions, since the actions undertaken have an opportunity cost. Medical services consumption is an important example of a costly action that alleviates damage from pollution exposure, so understanding the link between medical services and ambient pollution is critical for measuring the demand for air quality improvements. In this paper, we focus on how changes in ambient air pollution induce changes in medical expenditures related to asthma and chronic obstructive pulmonary disease (COPD) – two highly prevalent chronic respiratory diseases that together accounted for over $75 billion in US healthcare spending in 2012 (Cohen, 2014).

There are several motivations for studying the relationship between pollution and healthcare spending. First, although early-mortality costs from pollution are thought to be much larger than the costs of morbidity, understanding the impact of pollution on morbidity is increasingly recognized as important for evaluating policy (Cameron, 2014). Direct estimates of morbidity costs may be more politically palatable than mortality costs that rely on estimates of the value of a statistical life, and the costs of morbidity may be more salient to individuals who have personal experience with chronic disease. Morbidity costs operate through several channels, including lower worker productivity, decreased earnings, and costly medical conditions. Accounting for medical utilization is therefore an important aspect of the relationship between pollution and health outcomes, yet few studies have measured the contribution of pollution exposure to these morbidity-related costs. Furthermore, as health technologies improve, mortality rates are decreasing, while the prevalence rates of people surviving and living with chronic diseases such as asthma and COPD are increasing (American Lung Association, 2013). This increase in prevalence implies an increase in expenditures needed to manage the conditions, all else equal. Asthma and COPD consistently rank among the top five most costly conditions in terms of health care expenditures, and so if ambient pollution has even a modest impact on this spending, a reduction could have large welfare benefits. Also, the use of medication and medical services is likely one of the more common ways in which individuals with respiratory conditions respond to poor air quality, so understanding the nature and magnitude of this response is necessary for developing a comprehensive understanding of defensive behaviors. Finally, the relationship between CAA criteria air pollutants such as fine particulates, and newly regulated pollutants such as carbon and mercury, is increasingly important for environmental policy. Initiatives such as the Clean Power Plan (for carbon) and the Mercury and Air Toxics Standards will simultaneously decrease particulate concentrations, and the resulting decrease in morbidity and mortality will be important for determining if the standards provide positive net economic benefits. This study fills in some of the current gaps in our knowledge of the morbidity costs of fine particulates and can help inform future cost-benefit analyses for environmental regulations.

We show how defensive expenditures, or medical services consumption in the current case, can be used to construct welfare measures for changes in ambient pollution. We apply the defensive expenditures model in a new and important setting, using data on over 10,000 households living in 26 US metropolitan areas between 1996 and 2003. This time period is particularly interesting because it overlaps with the first and second phases of Title IV of the 1990 Clean Air Act amendments, which established a sulfur dioxide (SO_2) allowance market in the US. Though the program was initially intended to protect water and forest ecosystems from acid rain, the vast majority of benefits are thought to have arisen from decreases in mortality, due to the large reduction in SO_2 and the fine particulate matter associated with it (Schmalensee and Stavins, 2013). The pollution reductions induced by this policy provide significant variation in ambient air quality levels over time and across US metropolitan areas, allowing identification of the relationship between pollution and medical expenditures.

We rely on the Medical Expenditure Panel Survey (MEPS) data, collected by the Agency for Healthcare Research and Quality (AHRQ), in our analysis. This, when combined with ambient air pollution data from the EPA's Air Quality System database, allows us to track household medical expenditures and local pollution levels over two-year periods. We aggregate this spending by quarter and use fixed effects and instrumental variable models to measure the response of spending to changes in ambient pollution at the Metropolitan Statistical Area (MSA) level. We find that a one standard deviation increase in fine particulate matter ($PM_{2.5}$) increases asthma/COPD spending by over 8 percent. A one standard deviation change in mean pollution levels is large, representing 56 and 27 percent changes in average SO_2 and $PM_{2.5}$, respectively. However, these magnitudes are generally consistent with the decreases in ambient levels of pollution occurring during this time period. According to the EPA, the annual 99th percentile of daily maximum SO_2 concentrations decreased by 76 percent between 1990 and 2014. In addition, annual averages of $PM_{2.5}$ decreased by 37% between 2000 and 2015. Since spending on these conditions is so high, an 8 percent change represents over $6 billion in annual expenditures (relative to 2012 expenditure levels). We show that our estimates can be used to derive partial welfare measures for changes in pollution, which suggest that this figure is a lower bound on the annual willingness to pay for a standard deviation reduction in ambient pollution.

This paper adds to the literature on defensive actions and air quality valuation in several ways. First, we quantify the contribution of SO_2 and $PM_{2.5}$ to healthcare spending. In this sense, we are directly estimating one aspect of pollution-related morbidity costs, as a complement to the large volume of research that has focused on pollution and mortality/morbidity risk. A more complete understanding of this aspect of the welfare costs of pollution exposure is necessary for evaluating and updating air quality standards. Second, we use household level data, while the one similar study of which we are aware uses aggregate measures of medical expenditures. Household fixed effects allow us to control for unobserved characteristics, which includes the propensity of individuals to engage in avoidance behavior or other non-medical defensive actions. Household level data also provides the opportunity to investigate how household attributes, such as insurance status and income, influence behavioral responses to pollution. Next, we break down expenditures by disease type to identify the main drivers of medical expenditure changes, rather than focusing on broad categories such as ‘respiratory conditions’. Importantly, we find no effect when using a broad respiratory categorization, but strongly significant results for asthma and COPD spending. This provides a more nuanced explanation for how households alter their consumption behavior in response to pollution changes. Finally, we employ a distant-source instrumental variable (IV) strategy, similar to Bayer et al. (2009) and Hamilton and Phaneuf (2015), to account for local unobserved macroeconomic factors that might be related to both pollution levels and healthcare markets, and to address measurement error concerns. As in previous research, we find that implementing this type of strategy increases the magnitude of our estimates, which highlights the importance of properly dealing with pollution endogeneity and measurement concerns.

1.2 Background

Understanding the impact of pollution exposure on human health has been a central public health concern for the past few decades, and imposing appropriate pollution standards remains a contentious policy issue. A wealth of literature in economics, epidemiology, and other health sciences has described the relationship between air pollution and health outcomes and given insight into how these pollutants should be regulated. The EPA currently regulates several six criteria air pollutants: SO_2, particulate matter, nitrogen dioxide (NO_2), carbon monoxide (CO), ground-level ozone (O_3), and lead. Particulate matter is further categorized by particulates smaller than 2.5 micrometers and particulates smaller than 10 micrometers, denoted $PM_{2.5}$ and PM_{10}, respectively. The EPA's Integrated Science Assessments provide detailed summaries of the sources, distribution, and known health impacts of each pollutant type. All are thought to harm human health, but may impact different conditions. Epidemiological studies that find statistical associations between pollution and morbidity, combined with results from human clinical studies that demonstrate causal pathways between exposure and symptoms, such as airway inflammation and limited lung function, provide strong evidence of a causal negative impact of air pollution on health. Careful consideration of this evidence suggests that all of the criteria pollutants increase respiratory morbidity, and that $PM_{2.5}$, O_3, and CO exposure increase cardiovascular morbidity and overall mortality (US EPA 2008a, 2008b, 2009, 2010, 2013).

Though work in epidemiology and other fields has established a clear link between pollution and health outcomes, economists have noted problems with casual interpretation in these studies, since pollution exposure is not randomly assigned, and is likely correlated with unobserved drivers of health and other outcomes. Chay and Greenstone (2003) use exogenous variation in pollution reductions resulting from the 1980-82 recession to address this issue. They find that the estimated impact of total suspended particulates (TSPs) on health is consistently larger, when accounting for these potential sources of bias. Similarly, Schlenker and Walker (2015) use exogenous variation in airport congestion to estimate the impact of CO on health outcomes for individuals living near airports. They find a one standard deviation increase in CO pollution increases daily asthma attacks by 21 percent, relative to the baseline average. These estimates are an order of magnitude larger than estimates that ignore the potential endogeneity. These papers demonstrate the need for careful vetting of the sources of pollution variation, when examining the causal relationship between health outcomes and ambient air quality.

Even when variation in pollution is exogenous, ignoring the defensive actions people take to protect themselves against pollution can bias estimates. For instance, if a person wears a mask (which filters out particulate matter) on poor air quality days, she will not experience respiratory damage as severe as a person who does not take this precaution. If a large proportion of the population engages in defensive behavior, then observed health outcomes on a poor air quality day will be only a fraction as severe as the counterfactual, in which no person engages in defensive behaviors. Morretti and Neidell (2011) provide evidence of this using ship arrivals in Los Angeles ports as an exogenous source of variation in ozone. Their objective is to estimate the causal relationship between hospitalizations (a proxy for health outcomes) and ozone concentrations. Port activity is a large source of ozone in the area but generally unobserved by individuals, so defensive behavior is unlikely to directly respond to it. Using ship traffic as an instrument for ozone, Morretti and Neidell find IV estimates that are four times larger than similar estimates using OLS. Specifically, they show that hospital costs increase by more than 4.5 percent in response to a 0.01 part per million (20 percent of mean) increase in average ozone levels.

Other studies focus directly on the defensive actions. Behavioral changes induced by a decrease in environmental quality are often to as defensive expenditures. Graff Zivin et al. (2011) find that consumers respond to notices of public drinking water violations by increasing purchases of bottled water between 17 and 22 percent. For microorganism violations, they find a higher response rate in communities with a larger elderly (over 65) population, corroborating the theory that vulnerable groups will have more incentive to respond to violation notices. In other examples, Mu and Zhang (2014) and Zheng et al. (2015) find that purchases of anti-pollution facemasks and air filters increase with ambient pollution levels.

Our paper examines defensive expenditures related to medication purchases, and so contributes to the literature using this approach to measure the morbidity-related welfare effects from changes in air pollution. In contrast to studies with a regional or niche product emphasis, we focus on a nationally-relevant category of spending that totals over $75 billion per year. In addition, previous evidence suggests this spending may be sensitive to ambient air quality. For example, an EPA report on the costs and benefits associated with the 1990 Clean Air Act Amendments suggests that particulate matter accounted for $11.6 billion in medical expenditures in 2010 (EPA, 2011). This figure is derived from estimated pollution-induced changes in cardiovascular and respiratory disease incidence, multiplied by an average cost of illness measure. While the EPA estimate provides a useful benchmark, its methodology likely suffers from the critiques described above. Our paper builds on the existing literature's focus on plausibly exogenous pollution measures and careful accounting for avoidance behavior, to provide a valid estimate of the impact of air pollution on respiratory disease spending.

The paper closest to our study is Deschenes et al. (2016), who use county level insurance claims data to investigate how reductions in ozone attributed to the implementation of the NOx Budget Program – a cap and trade policy established in the northeastern US – impact healthcare expenditures. Using policy implementation differences across states as an exogenous treatment, they find that the program reduced respiratory and cardiovascular spending by 2 percent, an effect that primarily operates through the reduction in ambient ozone concentrations. Our research builds on and complements this by exploring the connection between spending and air pollution in a wider geographic context, using household level panel data. While Deschenes et al. exploit variation in ozone, we use variation in SO_2 and particulate matter to investigate analogous effects from pollutants that have thus far not been examined in this context. Furthermore, we apply an identification strategy that is new to the pollution-health relationships literature.

1.3 Theoretical model

Exposure to pollution has biological consequences that can result in degraded respiratory health. Defensive actions occur after pollution levels have been realized, and people can utilize healthcare to alleviate damage caused by the resulting pollution exposure. Here we present a static version of Grossman's (1972) health production framework to frame this problem more precisely, and to motivate our empirical analysis. We assume the person receives utility from health *H* along with spending on a composite good *x* based on the direct utility function $U(H,x)$. Health is produced according to the production function $H=h(m,a)$, where *m* is spending on healthcare (e.g. medication), and *a* is the ambient level of air pollution. We assume that *m* has positive marginal product and *a* negative marginal product, so that higher air pollution reduces health. The person's optimization problem is given by

$$V(a,y) = \max_m U\left(y - \Pi - pm, h(m,a)\right), \tag{1}$$

where *p* is the out of pocket cost of medication, *y* is income, and Π is an insurance premium paid by the consumer. The solutions to this problem are given by $m(p,a,y)$ and $x(p,a,y)$, where $x = y - \Pi - pm.$ This allows us to express the health outcome as

$$H = h(m(p,a,y),a). \tag{2}$$

Taking the total derivative of (2) respect to *a* while holding *H* fixed we obtain

$$0 = \frac{\partial h}{\partial m}\frac{\partial m(p,a,y)}{\partial a} + \frac{\partial h}{\partial a}, \tag{3}$$

which, after rearranging and multiplying both sides by *p*, we have

$$p\frac{\partial m(p,a,y)}{\partial a} = -p\frac{\partial h/\partial a}{\partial h/\partial m}. \tag{4}$$

Note that the left hand side of (4) is the marginal change in out of pocket expenditure on *m* following a marginal change in *a*, and the right hand side is the value of the units of *m* needed to offset a change in *a*, holding *H* constant. To relate (4) to marginal willingness to pay, we differentiate (1) with respect to *a* to obtain

$$\begin{aligned}\frac{\partial V}{\partial a} &= \frac{\partial U}{\partial x}\left[-\frac{\partial \Pi}{\partial a} - p\frac{\partial m}{\partial a}\right] + \frac{\partial U}{\partial H}\left[\frac{\partial h}{\partial m}\frac{\partial m}{\partial a} + \frac{\partial h}{\partial a}\right] \\ &= \frac{\partial U}{\partial x}\left[-\frac{\partial \Pi}{\partial a} - p\frac{\partial m}{\partial a}\right],\end{aligned} \tag{5}$$

where the second equality follows from equation (3). Dividing through by the marginal utility of income, we obtain an expression for marginal willingness to pay as

$$MWTP(a) = -\frac{\partial V/\partial a}{\partial V/\partial y} = \frac{\partial \Pi}{\partial a} + p\frac{\partial m(p,a,y)}{\partial a}. \tag{6}$$

Thus knowledge of the insurance premium structure and household demand for *m* allows calculation of marginal willingness to pay for a small change in *a*.

For insight on the structure of the insurance premium, we consider a highly stylized model of a competitive insurance provider. We abstract from adverse selection and assume that the insurance company knows the demand for health services, but is uncertain over ambient pollution levels. Specifically, the insurance company sets its premium and out of pocket copay before the level of ambient pollution is realized, so that expected profit is

$$E(\Omega) = \Pi - \int (c-p)\cdot m(p,a,y)\cdot f(a)da, \tag{7}$$

where c is the marginal cost of providing a unit of m and $f(a)$ is the probability distribution for a. At zero expected profit, for a given p the equilibrium premium level is characterized by[1]

$$\begin{aligned}\Pi &= \int (c-p)\cdot m(p,a,y)\cdot f(a)da \\ &= (c-p)\int m(p,a,y)\cdot f(a)da \\ &= (c-p)\cdot E\left(m(p,a,y)\right).\end{aligned} \tag{8}$$

Equation (8) shows that, holding p fixed, a influences the actuarially fair premium level through its influence on health care demand. Specifically, after differentiating we have

$$\frac{\partial \Pi}{\partial a} = (c-p)E\left(\frac{\partial m(p,a,y)}{\partial a}\right). \tag{9}$$

Substituting this expression into equation (6), we can write the household's marginal willingness to pay for a change in a as

$$\begin{aligned} MWTP(a) &= \frac{\partial \Pi}{\partial a} + p\frac{\partial m(p,a,y)}{\partial a} \\ &= (c-p)E\left(\frac{\partial m(p,a,y)}{\partial a}\right) + p\frac{\partial m(p,a,y)}{\partial a},\end{aligned} \tag{10}$$

and the average marginal willingness to pay is the expected change in the full cost of health care:

$$\begin{aligned} E\left(MWTP(a)\right) &= (c-p)\cdot E\left(\frac{\partial m(p,a,y)}{\partial a}\right) + p\cdot E\left(\frac{\partial m(p,a,y)}{\partial a}\right) \\ &= c\cdot E\left(\frac{\partial m(p,a,y)}{\partial a}\right).\end{aligned} \tag{11}$$

Equation (11) is our key result. It shows that an estimate of the change in *total expenditures* from a change in a can be linked to marginal willingness to pay, even when households' out of pocket expenditures are less than total expenditures. This result motivates of our use of total expenditures in our main empirical analyses and welfare predictions.

[1] Cutler and Zeckhauser (2000) present a more detailed exposition of an optimal insurance policy, including selection of the optimal coinsurance rate.

Appendix A extends this theory to include cases with additional defensive actions. If both medication use and behavioral adjustments are available, the household simultaneously equates the marginal benefit to the marginal cost of producing health through each channel. In our empirical section we focus on estimating the demand for medication, while controlling for an individual's propensity to engage in other defensive activities. Bartik (1988) and Phaneuf and Requate (2016) extend this analysis to the case of non-marginal changes in pollution. They show that the change in defensive expenditures following a change in pollution provides a lower bound on compensating variation (CV). We use this result in our empirical analysis to argue that our predicted changes in expenditures provide a lower bound estimate of the willingness to pay for the pollution reduction magnitudes observed during our study period.

Our model shows how welfare measures can be derived from observed consumption behavior in a static context. However, a more complex dynamic model may be necessary to fully describe some types of consumption behavior over time. A dynamic model would need to vary by the type of medical spending being analyzed. For example, for chronic respiratory ailments, such as asthma and chronic obstructive COPD, there are two broad types of medications. The first group consists of 'rescue' inhalers that are used in the event of an acute asthma attack. Purchase of these types of medicines can be thought of as self-insurance, aimed at reducing the severity of an asthma attack if it occurs. It is intuitive that use of this type of medication will be immediately responsive to environmental irritants, but not impact future use. It is therefore consistent with a static model of behavior.

The second group includes long-acting medications, such as inhaled corticosteroids, that reduce the probability of a future asthma attack. Purchase of this type of medication can be thought of as self-protection. Long-acting inhaler purchases, and possibly office visits, may come before or in expectation of a forthcoming pollution season, in contrast to rescue inhaler use or emergency room visits, which come after exposure has occurred. Furthermore, long-acting medications are often recommended for daily use, suggesting they would not be responsive to short term environmental changes. Despite these considerations, evidence from the health literature shows that utilization of these medications is reactive in nature, and exhibits seasonal variation similar to rescue medications (Sloan et al. 2013). In addition, long-acting inhalers are refilled, on average, two to four times annually (Stempel et al. 2005). This frequency allows observation of changes in use within quarterly data, even for medications that are more long-term in nature. In our empirical analysis, we focus on asthma and COPD spending, which this reasoning suggests may be responsive at a quarterly time span, and so provides a suitable setting for framing under the static health-substitutes model described here.

1.4 Data

1.4.1 Medical Expenditure Panel Survey

Our analysis relies on data describing health care expenditures, ambient air quality, and several additional controls. The health expenditure data come from the Medical Expenditure Panel Survey (MEPS) from 1996 to 2003. These data consist of seven overlapping panels. Households in each panel are drawn from the previous year's National Health Interview Survey (NHIS), and are interviewed five times over a two year period. The NHIS selects a nationally representative sample of households and then collects data for every member in a chosen household. This protocol allows us to include personal and household characteristics, account for some pre-existing conditions, and identify a household's location at the Metropolitan Statistical Area (MSA) level. Linking the MEPS and NHIS files requires a confidential data use agreement through the Agency for Healthcare Research and Quality (AHRQ). Although we can match spending to individuals, we aggregate the data to the household level, and use this as our cross sectional unit of observation, for several reasons. First, some characteristics that influence healthcare decisions, such as income, insurance status, and genetic risk factors for illness, are typically shared by all members of a household. Also, consumption decisions may be made jointly or primarily by a head of household figure. Finally, some drugs, such as allergy and cold medications, are likely shared among members of a household.

Interview rounds in the MEPS cover a five month period on average, but some variables are available on a monthly or even daily level. For example, a prescription medication refill date is only known at the interview-round level, but ambulatory care or emergency room visit dates are known exactly. We specify expenditures at the quarterly (three month) level and use quarters as our time unit of observation. For medication purchases only observed at the interview-round level, and interview-rounds that span more than one quarter, expenditures are divided proportionally across the quarters. This results in eight observations per household over their two year interview period.

Categories of spending that we consider include prescription medications, office-based physician visits, and emergency room visits. Medical conditions related to these spending categories can be identified using Internal Classification of Diseases (ICD-9) codes. Expenditure data is cross checked with insurance providers and pharmacies during data collection, so there is little worry about measurement error for these variables. Our main analysis uses spending on conditions for chronic obstructive pulmonary disease (COPD) and allied conditions (ICD-9 codes 490-496). The ‘allied conditions’ in this category include bronchitis, emphysema, and asthma. We further separate asthma from the other conditions under the hypothesis that the management of asthma symptoms may differ from other chronic respiratory conditions, due to the nature of the disease and/or differences in the populations with each condition. Additional categories we examine include diseases of the cardiovascular (ICD-9 410-414) and digestive (ICD-9 520-579) systems, where the latter serves as a placebo test. Expenditures on all categories are converted to 2003 dollars, and deflated using the appropriate Personal Health Care (PHCE) or Component Price Indices (CPI), as recommended by the AHRQ (AHRQ, 2015). Summaries of household spending by type are presented in Table 1. Over 13 percent of households have positive spending on asthma medications during at least one quarter, while 11.4 and 11.9 percent of households have positive spending on rescue inhalers and office based asthma/COPD visits, respectively. On the other hand, COPD medication purchases and emergency room visits are more rare, but also more costly per event. The MEPS data also provide individual level characteristics, such as insurance status and type, age, income, and diagnosed health conditions. Table 2 summarizes some of these variables. The indicator *Asthma* equals one if the individual has ever been diagnosed with asthma or emphysema, has had a recent asthma attack, or has used asthma medications in the past. Among sampled individuals, 7.2 percent belong to the asthma category.

1.4.2 Pollution Data

Data on ambient pollution concentrations come from EPA's Air Quality System database, which we use to characterize pollution at the metropolitan statistical area (MSA) level, since this is the finest geographical resolution available in the MEPS data. For reasons we discuss below, we focus primarily on sulfur dioxide (SO_2) and particulate matter, though we consider other criteria air pollutants as controls. The combustion of fossil fuels for electricity production or industrial use generates the vast majority of SO_2 emissions in the United States. Transportation-related sources also emit SO_2, but contribute only five percent of total emissions. Particulate matter finer than 2.5 micrometers ($PM_{2.5}$) comes from natural sources, such as forest fires, as well as from reactions to chemicals emitted during the combustion of fossil fuels for electricity production (US EPA, 2009). The largest anthropogenic source of particulate matter smaller than 10 micrometers (PM_{10}), however, is from transportation. In terms of other criteria pollutants, nitrogen dioxide (NO_2) emissions result primarily from on-road mobile sources, but electricity production is also a significant contributor. Ground level ozone (O_3) occurs when sunlight interacts with nitrogen oxides (NOx), which include NO_2, and volatile organic compounds (VOCs). Carbon monoxide (CO) is also a precursor to ambient levels of O_3. Transportation is the largest contributor to these ozone precursors.

The Air Quality System database provides an inventory of pollution concentration measures taken from individual monitors across the country. To match medical expenditures to ambient pollution levels, we aggregate pollution data from monitors at the MSA level for three month periods. Pollution measures are calculated by first averaging the daily mean values across all of the monitoring stations within an MSA for a given day. Then, these daily means are averaged within a three month quarter. Similar MSA/quarter measures are created using daily maximums and the 98^{th} or 99^{th} percentiles of daily maximums, depending on the form of the EPA's ambient air pollution standards for each particular pollutant. Since pollution monitors may come into service or retire during the study period, and because some monitors only operate seasonally, we restrict attention to monitors that operate continuously, to minimize any potential bias from selective monitoring. Continuous operation in our context means there is at least one weekly observation for 47 weeks per year, for both years of an individual's survey period. This cut-off point is admittedly arbitrary, but it retains the majority of the monitor data and mirrors the strategy used in Deschenes et al. (2016). Pollution summary statistics, including means, within-person standard deviations, and the number of cities sampled with consistent monitors for the pollutions we consider are included in Table 3. Relative to its mean, SO_2 has the highest overall and within-panel variation, which is consistent with the contemporaneous policy environment. A full list of MSAs represented in our data, by monitoring type, is included in Appendix B as Table B1.

Though our expenditure data requires us to characterize pollution at the MSA level, there may be significant variation in pollution levels within an MSA, causing deviation from the MSA average in actual exposure for people in different areas of the city. The bias caused by this measurement error will attenuate our estimates, leading to conservative estimates of the true effect. However, this not be a large concern in our setting. Fine particulate matter concentrations tend to be more homogenously distributed within a city than pollutants such as NO_2 and CO (US EPA 2008b, 2009, 2010). For SO_2, cities with high readings from monitors across the city are more highly correlated than in cities with low ambient levels (US EPA 2008a). In addition, the source of a pollutant determines its intra-urban homogeneity: $PM_{2.5}$ on road mobile sources will be more heterogeneously distributed than $PM_{2.5}$ originating from distant regional electricity production (US EPA 2009). As we discuss in detail below, our instrumental variables strategy exploits variation in the latter source of pollution. Table B2 in Appendix B shows within-MSA correlations for monitors included in the EPA data inventory. Average pairwise correlations within a city range from 0.46 for CO to 0.76 for $PM_{2.5}$. More generally, $PM_{2.5}$, O_3, and SO_2 have the highest correlations, while CO and NO_2 have the lowest. This is one motivation for our focus on $PM_{2.5}$ and SO_2.

The timing of the available MEPS data and implementation of the 1990 Clean Air Act Amendments provide a second motivation. Our 1996-2003 study period overlaps the first and second phases of the SO_2 allowance trading program in the United States. This policy led to large reductions in SO_2 in some regions of the country, offering more year to year variation than other pollutants during this period. Importantly, sulfur dioxide is a precursor for $PM_{2.5}$, which also exhibited large decreases over the study period. Most of the mortality-centered health benefits from the cap and trade program are attributed to this reduction, making particulate matter of central interest. One limitation is that $PM_{2.5}$ measurements are not widely available until 1999, while PM_{10} readings are available throughout the study period. This presents a tradeoff between sample size and our ability to use our preferred pollutant. For this reason, we estimate models on samples including either $PM_{2.5}$ or PM_{10} and compare the results, which we find to be remarkably similar.

According to the EPA, national averages of $PM_{2.5}$ decreased by 37 percent between 2000 and 2015. In our estimates, we report the effects of a one standard deviation change in $PM_{2.5}$, which equate to approximately 27 percent of our sample average concentration. While this is a large change, it corresponds with the large reductions in $PM_{2.5}$ that occurred over the past two decades. To assess the contribution of the 1990 CAA Amendments to this reduction, the EPA developed counterfactual estimates of $PM_{2.5}$ concentrations in the absence of the policy changes (EPA 2011). They find that concentrations in some large population centers to be dramatically smaller; for example, they estimate $PM_{2.5}$ concentrations to be 27 percent lower in Los Angeles and Pittsburgh, 37 percent lower in Manhattan, and 67 percent lower in Chicago than they would have been by the year 2000 in the absence of the CAA Amendments.

Figure 1 displays pollution trends over the study period. Sulfur dioxide shows a clear decrease from year to year, with peaks over the winter months. Ozone concentrations have a clear seasonal pattern that peaks during the summer, but there are no evident aggregate year to year trends during the sample period, and so quarter fixed effects should remove most of the natural O_3 variation in the data. Fine particulate matter ($PM_{2.5}$) levels are more volatile, but still exhibit seasonal patterns, with a slight downward trend over time. The existence of significant year to year variation in SO_2 and particulate matter provides quantitative evidence in support of our decision to focus primarily on these two pollutants.

1.4.3 Additional Controls

Additional controls in our regressions include weather data, which comes from the National Climatic Data Center (NCDC). Weather variables include mean precipitation, tenth percentiles of daily maximum temperatures and relative humidity, and the standard deviation of barometric pressure. The tenth percentile measures are used since cold and dry air are known to agitate respiratory conditions (Hyrkas et al. 2014, Makinen et al. 2009). There is some evidence that changes in atmospheric pressure may also irritate asthma, so a measure of variability is used for the pressure variable (Hashimoto et al. 2004).

1.5 Empirical Approach

Our objective is to estimate the effect of contemporaneous air pollution on household medical expenditures. Our baseline model is

$$Y_{it} = \beta_1 pollution_{jt} + \beta_2 weather_{jt} + \beta_3 X_{it} + \theta_i + \mu_{q(t)} + \gamma_{cr(j)}^{y(t)} + \varepsilon_{it}, \tag{12}$$

where Y_{it} is one of the (log-transformed) expenditure categories listed in Table 1, for household i during quarter t. Medical expenditure data from a general population often contains many zero observations with some rare high-cost events. A logarithmic transformation is a simple method commonly used in health economics to address the right-skewed nature of the data (Manning and Mullahy 2001). Due to the existence of zeros in our data, the exact transformation of the dependent variable takes the form $\log(x+1)$. Other transformations, including inverse hyperbolic sine, were tested but led to qualitatively similar results. Further estimation strategies that address the zeros and skewness in our data are included in the robustness section below. Insurance premium payments are not included in the publicly available MEPS data, so we primarily use total, rather than out of pocket, expenditure measures as dependent variables. Our theoretical model implies that total expenditures more comprehensively account for the medical costs of air pollution. The main coefficient of interest is β_1, which measures the proportionate change in expenditures from a unit change in pollution, expressed in our models as a standard deviation change in concentration. For controls we include household (θ_i) and quarter of year ($\mu_{q(t)}$) fixed effects, so that β_1 is identified off within-household variation purged of seasonal trends. The variables $weather_{jt}$ and X_{ijt} account for time-varying weather shocks at the MSA (j index) level and co-pollutants; the latter also includes household controls. Finally, $\gamma_{cr(j)}^{y(t)}$ is a year-by-census-region dummy variable that flexibly accommodates geographic variation in aggregate annual time trends. These trends include regional macroeconomic cycles that may impact both emissions and healthcare expenditures.

Consistent estimation of β_1 in equation (12) requires that, conditional on the fixed effects and observables, $pollution_{jt}$ and ε_{it} are uncorrelated. Unobserved determinants of medical expenditures may pose a threat to this condition. For example, people may engage in avoidance behaviors, such as spending more time indoors on poor air quality days, so as to reduce exposure to outdoor ambient pollution. Insofar as people are engaging in these types of behavior during more-polluted periods, estimates of the impact of pollution on respiratory disease and spending will be biased downwards, providing a conservative estimate of the true costs. However, the household fixed effects in the model account for propensities to engage in avoidance behaviors generally, as well as other unobserved household characteristics that do not change over time, and so this downward bias may be attenuated. In addition, household fixed effects can improve estimation when measurement error in right hand side variables is constant over time (Bound and Krueger, 1991). In our case, MSA level ambient pollution measures may be poor proxies for actual ambient levels where a household is located. However, if pollution in a household's neighborhood is consistently (say) higher than the MSA average, due to fixed geographic influences, then the household fixed effect will absorb that difference.

An additional threat to identification is due to correlation between pollution concentrations and local macroeconomic conditions, which may also impact our outcome variable. To address this and other endogeneity concerns, we implement an instrumental variable (IV) strategy similar to the one used by Bayer et al. (2009) and Hamilton and Phaneuf (2015). Their approach uses an atmospheric source-receptor matrix, developed by US EPA contractors, that predicts how emissions of SO_2 and nitrous oxides (NO_X) originating from a source s (s=1,…,S) affect concentrations of particulate matter at a receptor r (r=1,…,R).[2]

[2] An example of research using this source receptor matrix to predict the impacts on particulate matter concentrations of place-specific emission reductions is Shadbegian et al. (2007). These authors interacted with EPA staff and Abt Associates analysts to document how to use the matrix, which was originally described in Latimer (1996). The latter reference seems to no longer exist, though technical information on the source-receptor matrix is also provided in Abt Associates (2000, pp. E1-E5). Bayer et al. (2009) and Hamilton and Phaneuf (2015) relied on personal communication with Wayne Gray to obtain the necessary files and documentation, which we have drawn

Sources in the matrix we use include specific point sources (generally electricity generating units) with stacks over 500 meters tall, as well as county-level aggregates for shorter stacks and other emission sources. Receptors are at the level of US counties, and the elements of the matrix are pollutant-specific transfer coefficients that are calibrated based on prevailing winds, stack heights, and other physical features. Given data on SO_2 and/or NO_X emissions from a set of sources at a given time, it is possible to predict the county-level concentrations of particulate matter derived from that pollutant using

$$SR^k \times E_t^k = A_t^k, \tag{13}$$

where E_t^k is an S×1 vector of emissions of pollutant k (SO_2 or NO_X) at time t from each of the S sources, SR^k is the R×S source-receptor matrix specific to pollutant k, and A_t^k is a vector of particulate matter concentrations predicted from pollutant k, for the R receptor counties.

Since the sources and receptors included in SR^k are spatially explicit, it is possible to determine the distance between each source-receptor pair. With this, we define the R×S matrix D such that d_{rs}=1 if receptor r is located more than some cutoff distance from source s, and zero otherwise. We then define an alternative prediction of concentrations in the R counties according to

$$\left(D.\times SR^k\right)\times E_t^k = \tilde{A}_t^k, \tag{14}$$

where the operator '.×' denotes element by element multiplication. Note that $\tilde{A}_t^k$ is a prediction of concentrations based on pollutant k, which *excludes* all emission sources located less than the cutoff distance from the receptor county. In our main models we use a cutoff distance of 50km

on for this paper. Documents on using the matrix, including emails sent to Shadbegian et al. from developer Douglas Latimer, are available upon request.

and present robustness checks for 120km. This makes it useful as an instrument. Specifically, the design of the source-receptor matrix (and variation in geography and prevailing wind patterns) imply that $\tilde{A}_t^k$ will be correlated with observed concentrations at a particular location, but its reliance on only distant pollution sources in calculation suggests it is plausibly uncorrelated with local macroeconomic conditions, and any time-varying propensity individuals may have to engage in avoidance behavior.[3] Additional details on how we construct $\tilde{A}_t^k$ using the source-receptor matrix are available in Appendix C.

Though previous papers have developed this instrument at the annual level, we use quarterly emissions data from the EPA's Air Market Program Data (AMPD), which provides comprehensive emissions data for SO_2 and NO_X for thousands of facilities. Using these emissions data, we construct two separate instruments using distant emissions of SO_2 and NO_X individually, which vary quarterly across our study years. These instruments are correlated with our focus pollutants (MSA level concentrations of sulfur dioxide and fine particulate matter), but plausibly uncorrelated with locally unobserved drivers of medical expenditures via the distance exclusion.

We implement our IV strategy using the following generalization of equation (12). The first stage is given by

$$pollution_{jt} = \delta_1 \tilde{A}_{jt}^k + \delta_2 weather_{jt} + \delta_3 X_{it} + \alpha_i + \tau_{q(t)} + \phi_{cr(j)}^{y(t)} + u_{it}, \tag{15}$$

which we then use to construct an instrument for $pollution_{jt}$ in

[3] That is, though most local ambient pollution comes from local sources, some emissions are carried long distances due to weather and geographical patterns. As such, local pollution concentrations depend on both 'endogenous' locally produced emissions, and 'exogenous' emissions produced by distant sources.

$$Y_{it} = \beta_1 pollution_{jt} + \beta_2 weather_{jt} + \beta_3 X_{it} + \theta_i + \mu_{q(t)} + \gamma_{cr(j)}^{y(t)} + \varepsilon_{it}. \tag{16}$$

If distant emissions are uncorrelated with local unobserved factors serving as correlated omitted variables, this IV strategy will yield improved estimates of β_1.

1.6 Results

1.6.1 Main Findings

Table 4 presents baseline OLS fixed effects results for specifications that include and exclude co-pollutants, and use the $PM_{2.5}$ and PM_{10} samples. For each column the dependent variable is log-transformed total spending on asthma and COPD, which includes medication purchases, office-based visits, and emergency room visits. The pollutant measures are quarterly averages of the observed daily mean, normalized to have zero mean and a standard deviation of one. Standard errors are clustered at the MSA/survey cohort level.[4]

Our OLS results show a strong and robust positive marginal effect of SO_2 on expenditures. Specifically, a one standard deviation increase in average SO_2 is associated with an increase in asthma and COPD spending of between 2.4 and 4.2 percent. All other pollutants, including both measures of particulate matter, have insignificant coefficients across all models. This provides initial evidence that air pollution affects spending on respiratory medications, though the potential endogeneity of our pollution measures requires additional analysis to establish causality.

Table 5 presents our main IV results using distant source emissions as an instrument for

[4] That is, respondents included in the same cohort timeframe and living in the same MSA are included in the same cluster. For our $PM_{2.5}$ sample this produces 68 clusters, and for the PM_{10} sample there are 133 clusters.

local pollution concentrations. Here we focus on the $PM_{2.5}$ sample and again examine total spending on asthma and COPD conditions. In addition to the household and time fixed effects, these estimates control for time-varying unobservable drivers of healthcare consumption that may be correlated with local pollution. As described above and in Appendix C, we construct instruments for SO_2 and $PM_{2.5}$ concentrations using the source-receptor matrix and distant emissions of SO_2 and NO_X, though similar endogeneity concerns may exist for the co-pollutant controls we include in the model. Without instruments to estimate all endogenous pollutants jointly, we face a tradeoff between omitted variable bias from excluding co-pollutants, and bias that comes from including non-instrumented endogenous variables. Table 5 presents a systematic exploration of this tradeoff. Each column displays models containing a different subset of (non-instrumented) co-pollutants, and the rows show estimates of the two coefficients of interest for different IV strategies. The bottom row is our ex ante preferred specification, in that it jointly instruments for both SO_2 and $PM_{2.5}$.

Our first stage F-statistics show that our instruments based on distant source emissions are reasonably strong predictors of local concentrations.[5] In our second stage results, we find that instrumenting for $PM_{2.5}$ produces statistically and economically significant effects for this pollutant across the range of co-pollutant specifications. Point estimates from the bottom two rows of Table 5 suggest a one standard deviation increase in $PM_{2.5}$ concentration increases spending on medication between 3.6 and 8.3 percent. Importantly, in our preferred bottom-row specification that jointly instruments for both pollutants of interest, only $PM_{2.5}$ retains its statistical and economic significance. Here we find that a one standard deviation increase in $PM_{2.5}$ increases expenditures for asthma and COPD by 4.7 to 8.3 percent. Omitting O_3 as a

[5] Table R1 in the reviewer's appendix shows representative first stage results.

control, as in columns (2) and (4), decreases the coefficients on $PM_{2.5}$. This makes sense, since O_3 is negatively correlated with $PM_{2.5}$ in general, while CO and NO_2 are positively correlated with $PM_{2.5}$. Consistent with findings in previous studies, our IV estimates are larger than their OLS counterparts, perhaps due to improved accounting for time-varying avoidance behavior. In addition, the switch in significance from the SO_2 to $PM_{2.5}$ variables could be indicative of measurement error in our $PM_{2.5}$ variable. In particular, the alternative $PM_{2.5}$ measures from our instruments may help isolate the true $PM_{2.5}$ effect, whereas in the OLS regressions, SO_2 acted as the proxy for particulate matter.

1.6.2 Disaggregate Findings

Our main findings show that particulate matter concentrations have an economically important influence on asthma and COPD spending. In this section we further examine the relationship by presenting regressions using disaggregated categories of spending. Table 6 shows results from several models that use our preferred IV strategy of instrumenting for both SO_2 and $PM_{2.5}$. Panel A focuses on the total expenditures (out of pocket costs plus insurance contributions) that have been our main emphasis, while Panel B uses out of pocket household expenditures as the dependent variables.

In Panel A, the estimates on $PM_{2.5}$ are positive and significant for most of the categories, while the estimate on SO_2 generally remains insignificant. More specifically, the $PM_{2.5}$ estimate is qualitatively consistent across asthma-related spending on medications, office visits, and emergency room visits (columns 2, 5, and 6). From these regressions we see that asthma-specific spending has an economically significant relationship to pollution, even when examined separately from COPD. Given the quite different populations suffering from these two conditions, this is a useful insight. Columns 3 and 4 break spending on asthma medications into long-acting and rescue inhalers. The former is found to be more responsive to air pollution – a result consistent with findings in Deschenes et al. (2016). This gives new insight into how people adapt to air pollution patterns. In particular, long-acting medications are more responsive over quarterly periods than rescue medications. Individuals with chronic respiratory conditions may purchase rescue inhalers proactively, which would lead to the insignificant effects in column 4. Columns 7 and 8 illustrate the importance of specificity in relating pollution to health care expenditures. Column 7 presents estimates when the dependent variable is broadly defined to include spending on all types of respiratory-related ailments, while column 8 does so for cardiovascular ailments. In contrast to our robust findings for asthma and COPD, these broad categories produce null and/or counterintuitive results. We speculate that the broad spending categories nest too much heterogeneity in physiological responses to different air pollutants to allow clean identification of a single-pollutant effect. Finally, column 9 in Panel A provides a placebo test relating spending on digestive ailments, which should not respond to air pollution, to our preferred measures of air pollution. Consistent with intuition, there is no significant relationship between this category of spending and $PM_{2.5}$ or SO_2.

The estimates focusing on out of pocket expenditures (Panel B) mirror their total expenditure counterparts in sign, but they are consistently smaller in magnitude. This is intuitive from a practical perspective, but also has a potential moral hazard explanation. For example, an annual cap on total out of pocket spending could produce the observed differences in pollution response effects, even if the quantity consumed at a given level of pollution remains constant. More speculatively, if moral hazard causes inefficiently high use of medical services, the marginal willingness to pay for a pollution reduction based on out of pocket costs may be smaller than the marginal effect on total expenditures. Further research could investigate this link more completely. For our analysis here, we rely on our main theoretical predictions as motivation for focusing on total expenditures for our welfare interpretation.

1.6.3 Robustness

We completed several additional analyses to check the robust of our main findings to alternative assumptions and data configurations. In this section we describe a subset of these efforts that serve to illustrate the stability of our main findings. To test the dependence of our findings on how MSA-level air pollution is represented, we examined alternative pollution measures in our main regressions. Table 7 replicates our preferred IV specification for total asthma and COPD spending using the quarterly average of daily maximum and percentiles of daily maximums that mirror the form of EPA's NAAQS. The daily maximum and percentile measures in columns 1 and 2, respectively, result in estimates similar to those from our baseline joint IV estimates. This is encouraging, in that it is not entirely clear ex ante which pollution measure should be preferred.

Our main regression analysis focused on medical service expenditures, which combines both quantity demanded and pricing aspects. To check that our findings are driven by consumption responses and not a spurious relationship between pollution and medical services pricing, perhaps due to drug company pricing policies, we sought out models that would hold price effects fixed. Since there are hundreds of differentiated products included in each spending type, and because we only have expenditure and quantity information for those who purchased medication, directly including price effects in this setting is infeasible. It is possible, however, to investigate if the number of prescriptions or their quantity, such as number of doses per prescription, increase in response to increases in pollution. Table 8 presents two models using our preferred IV strategy that investigate these notions. In column 1, the number of asthma medications purchased per survey round is used as the dependent variable. Similarly, the dose quantity per asthma medication is the dependent variable in column 2. There is clear evidence that both number of prescriptions and doses per prescription increase in response to increases in particulate matter, which should alleviate concerns about endogenous pricing by drug companies, or other spurious correlations between pollution and prices.

Our IV strategy relies on the exclusion restriction that emissions generated more than 50km from a receptor county are uncorrelated with time-varying unobservable drivers of health care spending. This decision followed from the Bayer et al. (2009) and Hamilton and Phaneuf (2015), but is nonetheless an arbitrary cutoff point. Table 9 replicates the results in Table 6, but does so using a cutoff distance of 120km to construct the instruments. Comparison of estimates in the two tables confirms that our findings are not sensitive to our choice of cutoff distance.

Another concern relates to our use of continuous linear models for our limited dependent variable outcomes. We check the robustness of our non-IV results in Table 4 by presenting estimates from correlated random effects (CRE) probit and CRE tobit models. The CRE versions of these estimators model unobserved heterogeneity as functions of the observed covariates. The CRE tobit model is appealing in our context due to the large number of zeros in medical expenditure data. In addition, the average partial effects are estimated, which are the objects of interest because our data are corner-solution in nature rather, than censored (Wooldridge 2010). Table 10 displays results from models that use both the $PM_{2.5}$ (Panel A) and PM_{10} (Panel B) samples. In column 1, the CRE tobit model produces results similar to those presented in Table 4. Columns 2 and 3 use a binary indicator of positive spending or no spending as the dependent variable in a linear probability model and CRE probit, respectively. These models are qualitatively consistent with Table 4, in that they show a positive association between SO_2 and the likelihood of a household having positive spending. Comparison of Panels A and B implies similar associational findings for the two different samples, across the tobit, probit, and linear probability models. Though these estimates do not support the robustness of our causal findings, they do suggest that our findings are robust to a fuller accounting of the limited dependent variable nature of our data.

The final columns in Panel A of Table 10 does contribute to the robustness of our causal inference. Column 4 our preferred IV, fixed effects analysis of the binary outcome describing spending or no spending by a household. Consistent with our main findings, we show that a higher concentration of particular matter leads to a higher probability that the household will have positive spending on asthma or COPD medial conditions. In column 5 of Panel A, we use an inverse hyperbolic sine function to transform the continuous spending variable. This transformation is appealing as it does not require the addition of an arbitrary constant to data with zeros, and coefficient estimates can be interpreted in a similar way to those produced by a log transformation. With this alternative specification, an increase in $PM_{2.5}$ is found to increase expenditures by over 9 percent. The magnitude of this estimate is consistent with those found using our preferred log transformation.

As a final robustness check, we depart from our household-level analysis and consider individual level spending. Table 11 presents our results. Column 1 is comparable to Table 6, Column 1, Panel A. The magnitude of the $PM_{2.5}$ estimate is now smaller, but still positive and significant. Columns 2 and 3 divide the sample into two age groups. The first groups consists of adults between the ages of 18 and 64, and the second group includes what are generally considered more vulnerable groups: children under the age of 18 and seniors 65 and older. We find no significant effect for the vulnerable age groups, but a strong effect for the 18-64 group. A possible explanation for this result is that vulnerable individuals with diagnosed respiratory conditions utilize high levels of medications and medical services regardless of pollution level, and they therefore are less likely to respond to quarterly variation in pollution.

1.7 Conclusions

Our empirical work in this paper demonstrates an economically significant and robust relationship between air pollution and asthma and COPD related medical expenditures. In our preferred specifications, we find that a one standard deviation increase in $PM_{2.5}$ increases spending on asthma and COPD by as much as 8.3 percent. This estimate implies that a substantial amount of spending is attributable to air pollution, given that total spending on these conditions exceeded $75 billion in 2012. We identify this effect using high quality data on household medical expenditures, combined with ambient air quality and emissions data, which we analyze using a fixed effects panel IV estimator. By controlling for time-invariant household characteristics and instrumenting for local ambient pollution using emissions from distance sources, we provide a convincing causal estimate at a large spatial scale, for a pollutant and medical expenditure category that have until now not been carefully examined.

We also frame our empirical analysis in the context of a static health production framework, which allows us to link changes in medical expenditures to welfare effects. Specifically, by integrating a model of household behavior with a stylized competitive insurance provider, we show that the marginal willingness to pay for a change in pollution is at least as large as the marginal change in total expenditures (out of pocket plus insurance) resulting from the change. Using this and the Bartik (1988) logic for bounding the willingness to pay for discrete environmental changes, we conclude that the changes in fine particulate matter concentrations stemming from the 1990 Clean Air Act Amendments and contemporaneous events – which correspond approximately to a standard deviation reduction across our data years – generate over $6.3 billion in in annual economic benefits via lower expenditures on asthma and COPD medical services. Since the economic benefits from this policy have mainly focused on the reduced mortality stemming from the decreases in $PM_{2.5}$, this is a heretofore unmeasured benefit of the program. As people live longer with accurate respiratory and cardiovascular ailments, morbidity-focused benefits of this type are likely to become increasingly important for evaluating air pollution policy.

Our findings complement the small number of other studies that have examined how the demand for medical services responds to air quality. For example, Deschenes et al. (2016) find evidence that a 6 percent reduction in ambient ozone concentrations led to 2 percent reduction in respiratory and cardiovascular medication expenditures.[6] Our results add to this work by showing a convincing pathway between particulate matter and asthma and COPD expenditures, using household level data representative of the US urban population.

Our paper also highlights possibilities for additional study. In our empirical models we have controlled for concentrations of co-pollutants – i.e. pollutants beyond $PM_{2.5}$ and SO_2 that may also affect health outcomes. The estimates for these were generally insignificant, which may suggest that respiratory medical expenditures are not responsive to these pollutants over the relatively long seasonal time periods that we consider. Alternatively, it may be that the large

[6] For completeness and comparison, Reviewer's Table R2 shows estimates when we use our instrument strategy to identify ozone's effect on asthma and COPD expenditures.

variation in SO_2 and $PM_{2.5}$ induced by acid rain program allows statistically precise identification of their effects, while leaving the other pollutants relatively invariant at our timescale. It would be useful to know if a limited focus on SO_2 and $PM_{2.5}$ translates to other respiratory ailment contexts. Also, our analysis identifies differences in the responsiveness to particulate matter of total spending and out of pocket spending. We have focused on the former for our welfare analysis, but suggest that there may be implications for the welfare effects of air pollution changes – both conceptually and empirically – of moral hazard and other institutional features of medical services markets in the US.

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1.9 Tables

Table 1: Expenditure Categories and Descriptions

Spending Category	Average Positive Total Expenditure (2003 $)	Average Positive Out of Pocket (2003 $)	Inferred Coinsurance Rate	# Households with Positive Spending	% Households with Positive Spending
Asthma Medication	144.88	51.09	0.35	1411	13.1
COPD Medication	241.01	105.17	0.44	125	1.2
Long-Acting Inhalers	167.00	56.33	0.34	533	4.9
Rescue Inhalers	61.95	22.22	0.36	1231	11.4
Office Based Visits for Asthma/COPD	152.40	19.39	0.13	1280	11.9
Emergency Room for Asthma/COPD	729.43	287.76	0.39	250	2.3
Digestive Medication	149.65	53.57	0.36	2625	24.3

Average expenditure values are conditional on positive spending by a household within a quarter. Coinsurance rate indicates the percentage of a health care service paid out of pocket. We observe 10,187 unique households over 8 quarters. Percentages are based on pooled sample.

Table 2: Individual Summary Statistics

Variable	Observations	Mean	Std. Dev.	Min	Max
Insurance	27051	0.8106	-	0	1
Age	27051	34	22	1	90
Asthma	27051	0.072	-	0	1
Income	27051	$19,635	25,308	-$47,387	$186,145

We observe 27,051 individuals in 10,187 unique households. Income is reported here at the individual level. Negative income indicates an individual is taking on debt in that period. Values for individuals in the same household are averaged for household level analysis.

Table 3: Pollution summaries

Pollutant	SO_2	$PM_{2.5}$	PM_{10}	O_3	NO_2	CO
Average Daily Mean	4.45	13.90	25.81	25.09	18.27	.718
Overall Standard Dev.	2.52	3.78	7.43	7.92	6.15	.279
Within Standard Dev.	1.37	2.39	4.71	7.31	3.43	.198
Minimum	.35	6.25	9.20	7.88	2.99	.300
Maximum	13.61	28.55	62.34	49.82	48.11	2.392
99^{th} Percentile	73.54	na	na	na	na	na
98^{th} Percentile	na	34.50	60.84	na	65.24	na
Observations	1026	598	1030	726	946	1074
MSAs	39	34	40	29	37	41

Unit of observation is an MSA-quarter pair. SO_2, O_3, CO, and NO_2 are measured in parts per billion (ppb), and PM_{10} and $PM_{2.5}$ units are in micrograms per cubic meter (μg/m^3).

Table 4: Impact of SO_2 and PM on Asthma & COPD Spending, with and without Co-Pollutant Controls

	(1)	(2)	(3)	(4)
SO_2	0.0411***	0.0419***	0.0242***	0.0272***
	(0.00889)	(0.00844)	(0.00838)	(0.00806)
$PM_{2.5}$	0.00609	0.000818		
	(0.00929)	(0.00713)		
PM_{10}			-0.00628	-0.00920
			(0.00659)	(0.00641)
O_3	-0.00848		-0.0113	
	(0.00908)		(0.00743)	
NO_2	-0.0178		-0.00583	
	(0.0161)		(0.0135)	
CO	-0.000102		0.00293	
	(0.0117)		(0.00974)	
Insurance	0.0428	0.0427	0.0208	0.0208
	(0.0278)	(0.0279)	(0.0196)	(0.0196)
Income	1.35e-07	1.33e-07	1.14e-07	1.13e-07
	(6.32e-07)	(6.31e-07)	(4.72e-07)	(4.72e-07)
Temperature	0.000906	0.000469	0.00148*	0.00114
	(0.000727)	(0.000644)	(0.000890)	(0.000753)
Air Pressure	-0.000225	-0.000268	5.33e-05	3.38e-07
	(0.000200)	(0.000195)	(0.000156)	(0.000152)
Precipitation	-0.000226	-0.000102	3.73e-05	0.000243
	(0.000599)	(0.000579)	(0.000540)	(0.000547)
Humidity	0.00108	0.00128	0.000255	0.000290
	(0.000813)	(0.000790)	(0.000382)	(0.000369)
Household FE	X	X	X	X
Region by Year FE	X	X	X	X
Quarter FE	X	X	X	X
Observations	48,626	48,626	76,870	76,870
Households	6,137	6,137	10,187	10,187

OLS estimator with log-transformed total spending on asthma and COPD as dependent variable. Pollution units are standard deviations of mean concentrations. Household level probability weights are used. Standard errors, clustered at the MSA/survey cohort level, are included in parentheses. *** p<0.01, ** p<0.05, * p<0.1

Table 5: IV Estimation, Impact of SO_2 and $PM_{2.5}$ on Total Asthma and COPD Spending

	Pollutant	(1) All Co-Pollutants	(2) No Co-pollutants	(3) O3 Only	(4) NO_2, CO Only	First Stage F-Stat
SO_2 instrumented	SO_2	0.0350**	0.0298**	0.0338**	0.0312**	42.79
		(0.0154)	(0.0148)	(0.0154)	(0.0146)	
	$PM_{2.5}$	0.00681	0.00164	0.0022	0.00437	
		(0.00921)	(0.00710)	(0.00721)	(0.00867)	
$PM_{2.5}$ instrumented	SO_2	0.0333***	0.0375***	0.0320***	0.0400***	
		(0.00958)	(0.00772)	(0.00943)	(0.00780)	
	$PM_{2.5}$	0.0636**	0.0404**	0.0568**	0.0394**	23.53
		(0.0247)	(0.0174)	(0.0227)	(0.0174)	
Jointly instrumented	SO_2	0.00594	0.0161	0.00923	0.0175	54.18
		(0.0193)	(0.0153)	(0.0188)	(0.0144)	
	$PM_{2.5}$	0.0834***	0.0471**	0.0682***	0.0480**	35.67
		(0.0282)	(0.0192)	(0.0256)	(0.0187)	
Observations		48,626	48,626	48,626	48,626	
Households		6,137	6,137	6,137	6,137	

Contributions from distant SO_2 and NOx emissions to local predicted particulate matter concentration are used as instruments for all regressions. All regressions include household fixed effects, household, co-pollutant and weather controls, quarter of year fixed effects, and region-year fixed effects. The dependent variables are log-transformed expenditures on Asthma and COPD. Pollution units are standard deviations of mean concentrations. Household level probability weights are used. Standard errors, clustered at the MSA/survey cohort level, are included in parentheses. *** p<0.01, ** p<0.05, * p<0

Table 6: Effect by Category, Out of Pocket vs Total Expenditures, IV Estimation

	(1) Asthma & COPD	(2) Asthma Meds	(3) Long-Acting Meds	(4) Rescue Meds	(5) Office Asthma	(6) Emergency Asthma	(7) Respiratory Meds	(8) Cardio Meds	(9) Digestive Meds
Panel A: Total Expenditures									
SO_2	0.00594	-0.00466	-0.00474	-0.0209	0.0173	-0.0271**	-0.0708	-0.0496**	-0.00261
	(0.0193)	(0.0118)	(0.0124)	(0.0148)	(0.0203)	(0.0133)	(0.0439)	(0.0208)	(0.0295)
$PM_{2.5}$	0.0834***	0.0473**	0.0333**	0.0166	0.0627***	0.0345**	0.0857	0.0355	-0.0296
	(0.0282)	(0.0208)	(0.0167)	(0.0227)	(0.0239)	(0.0163)	(0.0674)	(0.0435)	(0.0388)
Panel B: Out of Pocket Expenditures									
SO_2	-0.00725	-0.00884	0.00535	-0.0146	0.00437	-0.00244	-0.0675**	-0.0177	-0.00201
	(0.0174)	(0.0126)	(0.0101)	(0.0125)	(0.0118)	(0.00293)	(0.0326)	(0.0196)	(0.0227)
$PM_{2.5}$	0.0581***	0.0405**	0.0161	0.0202	0.0310**	0.00666	0.0778	0.00957	-0.0208
	(0.0218)	(0.0183)	(0.0132)	(0.0146)	(0.0132)	(0.00409)	(0.0477)	(0.0366)	(0.0320)
Observations	48,626	48,628	48,628	48,628	48,622	48,628	48,621	48,628	48,628
Households	6,137	6,137	6,137	6,137	6,137	6,137	6,137	6,137	6,137

Ambient particulate matter contributions from distant SO_2 and NOx emissions are used as to jointly instrument for SO_2 and $PM_{2.5}$ levels. All regressions include household fixed effects, household, co-pollutant and weather controls, quarter of year fixed effects, and region-year fixed effects. The dependent variables are log-transformed expenditures on the categories listed in column headings. Pollution units are standard deviations of mean concentrations. Household level probability weights are used. Standard errors, clustered at the MSA/survey cohort level, are included in parentheses. *** p<0.01, ** p<0.05, * p<0.1

Table 7: Alternative Pollution Measures

	(1)	(2)
Max SO_2	0.0105	
	(0.0353)	
Max $PM_{2.5}$	0.0798***	
	(0.0298)	
99th Percentile SO_2		0.0574
		(0.0441)
98th Percentile $PM_{2.5}$		0.0618**
		(0.0266)
Observations	48,626	48,626
Households	6,137	6,137

Contributions from distant SO_2 and NOx emissions to local predicted particulate matter concentration are used as instruments. Both regressions include household fixed effects, household, co-pollutant and weather controls, quarter of year fixed effects, and region-year fixed effects. The dependent variables are log-transformed expenditures on Asthma and COPD. Pollution units are standard deviations of mean concentrations. Household level probability weights are used. Standard errors, clustered at the MSA/survey cohort level, are included in parentheses. *** $p<0.01$, ** $p<0.05$, * $p<0.1$

Table 8: Impact on Number and Size of Prescriptions for Asthma

	(1)	(2)
	# RX	Quantity
SO_2	-0.0143	-0.877
	(0.0207)	(1.549)
$PM_{2.5}$	0.0796**	4.339**
	(0.0315)	(1.732)
Observations	48,628	48,628
Households	6,137	6,137

#RX indicates number of prescriptions used as dependent variable. Quantity indicates number of doses per prescription. Contributions from distant SO_2 and NOx emissions to local predicted particulate matter concentration are used as instruments. Both regressions include household fixed effects, household, co-pollutant and weather controls, quarter of year fixed effects, and region-year fixed effects. Pollution units are standard deviations of mean concentrations. Household level probability weights are used. Standard errors, clustered at the MSA/survey cohort level, are included in parentheses. *** $p<0.01$, ** $p<0.05$, * $p<0.1$

Table 9: Effect by Category using 120km Cutoff Distance for IV Estimation

	(1) Asthma & COPD	(2) Asthma Meds	(3) Long-Acting Meds	(4) Rescue Meds	(5) Office Asthma	(6) Emergency Asthma	(7) Respiratory Meds	(8) Cardio Meds	(9) Digestive Meds
Panel A: Total Expenditures									
SO_2	0.0201	-0.000894	-0.00830	-0.0157	0.0361**	-0.0205*	-0.0522	-0.0418*	0.00696
	(0.0168)	(0.00998)	(0.0107)	(0.0126)	(0.0177)	(0.0123)	(0.0393)	(0.0222)	(0.0254)
$PM_{2.5}$	0.0589**	0.0386*	0.0276	0.0107	0.0389	0.0254*	0.0628	0.000133	-0.0448
	(0.0280)	(0.0215)	(0.0173)	(0.0210)	(0.0246)	(0.0150)	(0.0647)	(0.0492)	(0.0378)
Panel B: Out of Pocket Expenditures									
SO_2	0.000984	-0.00593	0.00473	-0.0110	0.0150	-0.00240	-0.0546*	-0.0132	0.00558
	(0.0142)	(0.0102)	(0.00828)	(0.0107)	(0.0102)	(0.00241)	(0.0283)	(0.0179)	(0.0202)
$PM_{2.5}$	0.0428**	0.0337*	0.0117	0.0171	0.0189	0.00664	0.0556	-0.00772	-0.0313
	(0.0213)	(0.0183)	(0.0130)	(0.0120)	(0.0126)	(0.00407)	(0.0442)	(0.0406)	(0.0303)
Observations	48,626	48,628	48,628	48,628	48,622	48,628	48,621	48,628	48,628
Households	6,137	6,137	6,137	6,137	6,137	6,137	6,137	6,137	6,137

Ambient particulate matter contributions from distant SO_2 and NOx emissions are used as to jointly instrument for SO_2 and $PM_{2.5}$ levels. The F statistic is on the excluded instruments is 45.55 for $PM_{2.5}$ and 50.25 for SO_2. All regressions include household fixed effects, household, co-pollutant and weather controls, quarter of year fixed effects, and region-year fixed effects. The dependent variables are log-transformed expenditures on the categories listed in column headings. Pollution units are standard deviations of mean concentrations. Household level probability weights are used. Standard errors, clustered at the MSA/survey cohort level, are included in parentheses. *** p<0.01, ** p<0.05, * p<0.1

Table 10: Alternative Specifications

Panel A: $PM_{2.5}$ Sample

	(1) CRE Tobit	(2) LPM	(3) CRE Probit	(4) LPM IV	(5) IHS IV
SO_2	0.0257***	.01055***	0.0054***	0.00408	0.00934
	(0.0088)	(0.00231)	(0.00196)	(0.00519)	(0.0223)
$PM_{2.5}$	0.0061	0.00049	0.0011	0.0140**	0.0932***
	(0.0082)	(0.00214)	(0.00191)	(0.00678)	(0.0323)
Observations	48,628	48,628	48,628	48,628	48,628
Households	6137	6137	6137	6137	6137

Panel B: PM_{10} Sample

	(1) CRE Tobit	(2) LPM	(3) CRE Probit
SO_2	0.0229***	0.0065***	0.0047***
	(0.0065)	(0.0021)	(0.0015)
PM_{10}	-0.0092	-0.0017	-0.0019
	(0.0066)	(0.0014)	(0.0016)
Observations	77,579	77,579	77,579
Households	10,187	10,187	10,187

The dependent variable is log-transformed total spending on asthma and COPD in column (1), a binary indicator of positive spending for columns (2), (3), and (4), and inverse hyperbolic sine transformed total spending in column (5). All regressions include household, co-pollutant and weather controls, quarter of year fixed effects, and region-year fixed effects. Columns 2 and 4 include household fixed effects. Ambient particulate matter contributions from distant SO_2 and NOx emissions are used as to jointly instrument for SO_2 and $PM_{2.5}$ levels in columns (4) and (5). Pollution units are standard deviations of mean concentrations. Household level probability weights are used for (2) and (4). Standard errors, clustered by MSA-panel, are included in parentheses. *** $p<0.01$, ** $p<0.05$, * $p<0.1$

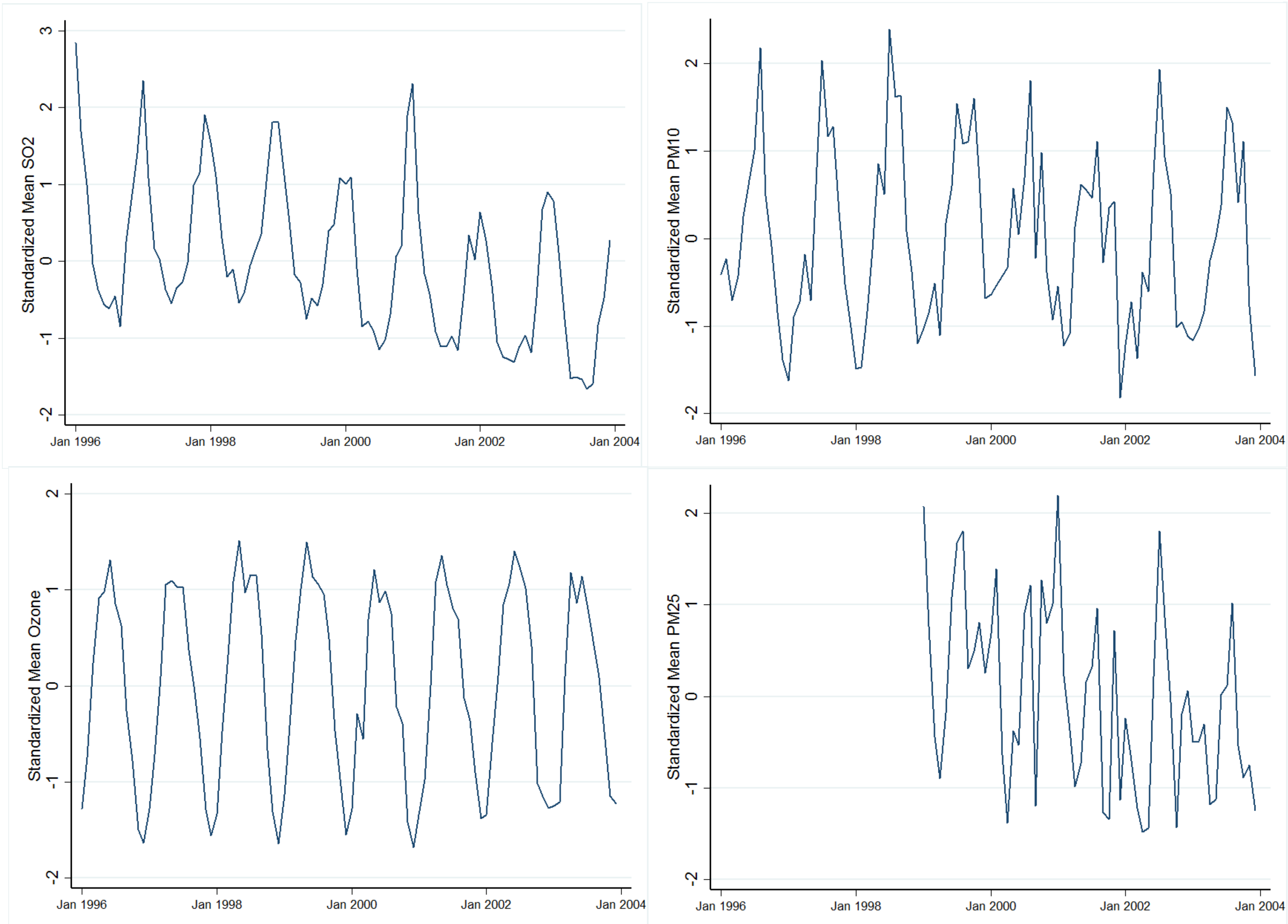
Table 11: Individual Level Estimates

	(1) Full Sample	(2) 18-64	(3) <18 & ≥65
SO_2	0.0124	0.00946	0.0152
	(0.00909)	(0.0135)	(0.0205)
$PM_{2.5}$	0.0314**	0.0541***	-0.00302
	(0.0138)	(0.0196)	(0.0254)
Observations	125,668	77,237	48,256
Individuals	15,896	9,957	6,339

Contributions from distant SO_2 and NOx emissions to local predicted particulate matter concentration are used as instruments for all regressions. All regressions include household fixed effects, household, co-pollutant and weather controls, quarter of year fixed effects, and region-year fixed effects. The dependent variables are log-transformed expenditures on Asthma and COPD. Pollution units are standard deviations of mean concentrations. Individual level probability weights are used. Standard errors, clustered at the MSA/survey cohort level, are included in parentheses. *** p<0.01, ** p<0.05, * p<0.1

1.10 Figures

Figure 1: Pollution Trends



1.11 Appendices

1.11.1 Detailed Exposition of Health-Substitutes Model in the Context of Medical Expenditures

In this this generalization we introduce an additional health input *d* that contributes to health by mitigating the impact of pollution. In contrast to the purchased input with out of pocket price *p*, this can be thought of as avoidance behavior that uses time, which has a shadow cost of *w*. With this, the health production function is *H*=*h*(*m*,*d*,*a*) and the individual's optimization problem is

$$V(p,w,a,y)=\max_{m,d} U\left(y-\Pi-pm-wd,h(m,d,a)\right). \tag{A.1}$$

The solutions to this problem are given by *m*(*p*,*w*,*a*,*y*) and *d*(*p*,*w*,*a*,*y*), which allows us to express the health outcome as

$$H=h\left(m(p,w,a,y),d(p,w,a,y),a\right). \tag{A.2}$$

Taking the total derivative of (A.2) respect to *a* while holding *H* fixed we obtain

$$0=\frac{\partial h}{\partial m}\frac{\partial m(p,w,a,y)}{\partial a}+\frac{\partial h}{\partial d}\frac{\partial d(p,w,a,y)}{\partial a}+\frac{\partial h}{\partial a}. \tag{A.3}$$

To develop a measure of marginal willingness to pay, we differentiate (A.1) with respect to *a* to obtain

$$\begin{aligned}\frac{\partial V}{\partial a}&=\frac{\partial U}{\partial x}\left[-\frac{\partial \Pi}{\partial a}-p\frac{\partial m(\cdot)}{\partial a}-w\frac{\partial d(\cdot)}{\partial a}\right]+\frac{\partial U}{\partial H}\left[\frac{\partial h}{\partial m}\frac{\partial m(\cdot)}{\partial a}+\frac{\partial h}{\partial d}\frac{\partial d(\cdot)}{\partial a}+\frac{\partial h}{\partial a}\right]\\&=\frac{\partial U}{\partial x}\left[-\frac{\partial \Pi}{\partial a}-p\frac{\partial m(\cdot)}{\partial a}-w\frac{\partial d(\cdot)}{\partial a}\right],\end{aligned} \tag{A.4}$$

where the second equality follows from equation (A.3). Dividing through by the marginal utility of income, we obtain an expression for marginal willingness to pay as

$$MWTP(a)=-\frac{\partial V/\partial a}{\partial V/\partial y}=\frac{\partial \Pi}{\partial a}+p\frac{\partial m(p,w,a,y)}{\partial a}+w\frac{\partial d(p,w,a,y)}{\partial a}, \tag{A.5}$$

which is analogous to equation (6). This result illustrates that estimates of the MWTP for air quality that only account for medication use will be lower bounds on the true MWTP, because additional value accrues from the marginal decrease in costly avoidance behaviors (the last term in the expression). It also shows that the demand for medical services depends on the shadow price of avoidance behavior *w*, which can manifest as an omitted variable in a regression of medical expenditures on air pollution.

1.11.2 Additional Pollution Information

Table B1: Pollutant Monitoring by MSA

Included MSAs	SO_2 PM_{10}	$PM_{2.5}$	O_3	NO_2	CO
Akron, OH	X	X			X
Atlanta-Sandy Springs-Marietta, GA	X	X		X	X
Baltimore-Towson, MD	X	X	X	X	X
Boston-Cambridge-Quincy, MA-NH	X	X	X	X	X
Bridgeport-Stamford-Norwalk, CT	X			X	X
Buffalo-Niagara Falls, NY	X		X	X	X
Charlotte-Gastonia-Concord, NC-SC	X	X		X	X
Chicago-Naperville-Joliet, IL-IN-WI	X	X	X	X	X
Cincinnati-Middletown, OH-KY-IN	X	X		X	X
Columbus, OH	X	X			X
Dallas-Fort Worth-Arlington, TX	X	X	X	X	X
Dayton, OH	X				X
Detroit-Warren-Livonia, MI	X	X		X	X
Hartford-West Hartford-East Hartford, CT	X	X	X	X	X
Houston-Sugar Land-Baytown, TX	X	X	X	X	X
Jacksonville, FL	X	X	X	X	X
Kansas City, MO-KS	X	X	X	X	X
Los Angeles-Long Beach-Santa Ana, CA	X	X	X	X	X
Louisville/Jefferson County, KY-IN	X	X		X	X
Memphis, TN-MS-AR	X	X	X	X	X
Miami-Fort Lauderdale-Pompano Beach, FL	X	X	X	X	X
Milwaukee-Waukesha-West Allis, WI	X	X		X	X
Minneapolis-St. Paul-Bloomington, MN-WI	X	X	X	X	X
New Haven-Milford, CT	X			X	X
New York-Northern New Jersey-Long Island, NY-NJ-PA	X	X	X	X	X
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	X	X	X	X	X
Phoenix-Mesa-Scottsdale, AZ	X	X	X	X	X
Pittsburgh, PA	X	X	X	X	X
Providence-New Bedford-Fall River, RI-MA	X	X	X	X	X
Richmond, VA	X			X	X
Riverside-San Bernardino-Ontario, CA	X	X	X	X	X
Rochester, NY	X	X	X		X
Sacramento--Arden-Arcade--Roseville, CA	X	X	X	X	X
San Diego-Carlsbad-San Marcos, CA	X	X	X	X	X
San Francisco-Oakland-Fremont, CA	X	X	X	X	X
Seattle-Tacoma-Bellevue, WA	X	X	X	X	X
St. Louis, MO-IL	X	X	X	X	X

Tampa-St. Petersburg-Clearwater, FL	X	X	X	X	X
Washington-Arlington-Alexandria, DC-VA-MD-WV	X	X	X	X	X

$PM_{2.5}$ monitoring begins in 1999. MSAs without monitoring for SO_2, weather covariates, or household expenditure data are excluded from this table.

Table B2: Spatial Correlation of Monitor Readings within an MSA

Pollutant	Correlation	# of MSAs	Monitors per MSA
SO_2	0.652	212	2.75
$PM_{2.5}$	0.760	383	2.37
PM_{10}	0.497	350	3.26
O_3	0.614	332	3.09
NO_2	0.469	142	2.93
CO	0.461	180	2.97

The correlation column presents the average of pairwise correlations between monitors within a MSA and quarter.

Up to 5 monitors per MSA were randomly chosen to create correlation coefficient matrices.

1.11.3 Additional Details on the Source-Receptor Instrument

The source-receptor matrix we use to construct our instrument was originally developed in the mid-1990s by EPA and its contractors for use in evaluating how source-specific reductions in emissions translate into changes in ambient air quality across space. The dispersion coefficients in the matrix were predicted using the Climatological Regional Dispersion Model (CRDM), which translates emissions from a specific source to particulate matter concentrations in downwind counties (Abt Associations, 2000; Heo et al. 2016, p. 6062). The original model included 5,905 emission sources across the US, which were categorized along four dimensions: area sources linked to the 3,080 counties in the lower 48 states, and low, medium, and high effective stack height sources. The 565 high stack (>500m) sources were included as individual units, while county-level aggregates of low (<250m) and medium (250 to 500m) stack sources constituted the remaining 1,887 and 373 sources, respectively. Each of the 5,905 sources is linked to 3,080 receptor counties.

Dispersion coefficients are available for four pollutants: directly emitted particulates, sulfur dioxide (SO_2), nitrous oxides (NO_X), and ammonia (NH_3). Each dispersion coefficient measures the incremental contribution to average ambient $PM_{2.5}$ concentration at a specific receptor, from one ton of emissions at a specific source. To construct our instruments we use quarterly emissions data of SO_2 and NO_X obtained from EPA's Air Markets Program Data (AMPD), which provides data for thousands of facility units at a quarterly frequency. Since the matrix was originally designed for use with National Emissions Inventory (NEI) data, steps were taken to match the newer AMPD information to the matrix. Among the 565 tall stack units, we were able to directly match 496 facility units (88 percent). Using additional information from the NEI, we obtained physical stack characteristics for a subset of the additional non-matched units. For these, we develop a county/stack height measure and use it to group stacks above 250 meters into the medium stack group.[7] All other units observed in the AMPD are

[7] Effective stack height (ESH) is a function of ground elevation, physical stack dimensions, stack gas velocity and temperature, wind speed, and ambient air temperature. For our matching we use ground elevation plus physical

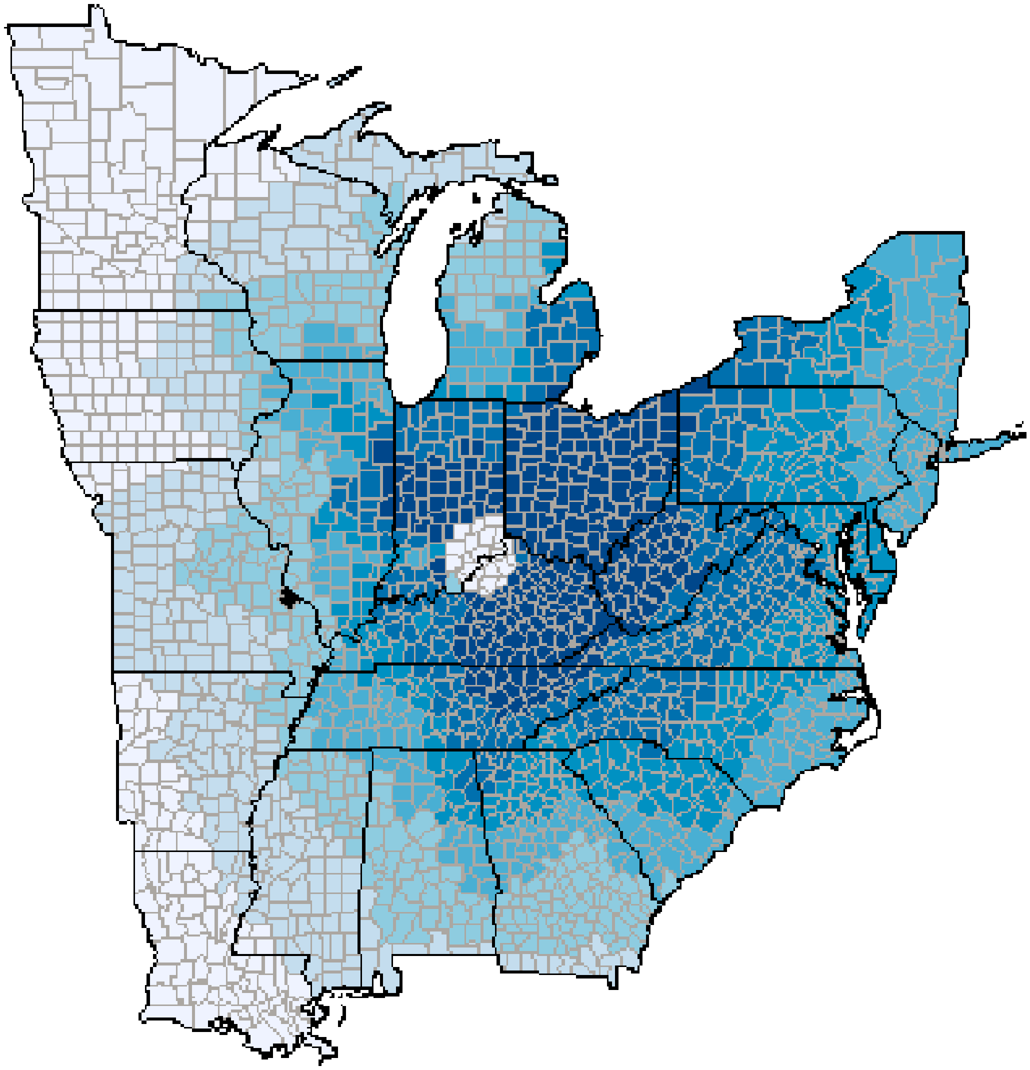
assumed to be short. Short stack transfer coefficients are generally smaller than medium stack coefficients because emissions are carried further at higher altitudes. Our matching process therefore ensures a conservative approach in which sources will generally have a transfer coefficient smaller than or equal to what was intended, when the source-receptor matrix was created. With these manipulations, we are able to use 4,489 sources of SO_2 and NO_X in our calculations. We use the matrix operation shown in equation (14) to aggregate the distant source contributions to a particular receptor county, and then average this across all counties within an MSA to create the final instrument. Specifically, for our main models, if a source is within 50 km of the population centroid of its receptor county we set its transfer coefficient to zero, so that nearby emission sources do not contribute to the final measure.

Figure C1 gives sample output from the source-receptor. For purposes of illustration, SO_2 emissions from a single facility, Clifty Creek Power Plant in southern Indiana, are used as inputs in the model. Counties with darker shades of blue receive larger $PM_{2.5}$ deposits from the power plant. The white circle surrounding Clifty Creek includes all counties whose population centroid is within 50 km of the facility. The figure illustrates that SO_2 emissions in southern Indiana have a measureable impact on ambient $PM_{2.5}$ concentrations as far away as New York and South Carolina.

It is important to note that the CRDM model, and the source-receptor matrix derived from it, does not represent the current state of the art in atmospheric dispersion modeling, and so it is no longer a preferred method for predicting ambient pollution changes in response to specific emission reductions (see Heo et al., 2016, for a recent discussion of competing approaches and CRDM's limitations). For our more modest objective, however, it is a good choice: we only need the predictions from the model to be correlated with local pollution concentrations. As such, our static linking of emissions from a non-exhaustive subset of distant sources to county predictions can constitute a poor level prediction, while still serving as a valid instrument.

stack height to proxy for ESH. This is an admittedly crude approximation that omits plume rise, but it provides a lower bound on the true ESH, which leads to conservative transfer coefficients when constructing the distant source instrument.

Figure: Ambient $PM_{2.5}$ Contributions from Clifty Creek Power Plant SO_2 Emissions



1.11.4 Additional IV Results

First stage IV estimates

Table R1 presents the first stage of the jointly estimated IV regression from Table 5, Column 1. SO_2 and NO_x Emissions are the distant source excluded instruments. The coefficients imply that a one standard deviation increase in the distant SO_2 emission instrument increases local $PM_{2.5}$ concentrations by 148 percent. The instrument values are relatively small with much variability, which explains the large magnitude of this coefficient. Interestingly, the NO_x instrument, which measures contributions to local $PM_{2.5}$ levels from distant NO_x emissions, is a stronger predictor for SO_2 concentrations than the distant SO_2 emissions instrument.

Table R1: First Stage Estimation Results

	(1) $PM_{2.5}$	(2) SO_2
SO_2 Emissions	1.479***	-0.211
	(0.268)	(0.238)
NO_x Emissions	-0.258	1.585***
	(0.255)	(0.240)
O_3	0.245***	-0.200***
	(0.059)	(0.061)
NO_2	0.461***	0.173
	(0.121)	(0.119)
CO	0.483***	-0.076
	(0.118)	(0.088)
Observations	48,626	48,626
Households	6,137	6,137

OLS estimator with log-transformed total spending on asthma and COPD as dependent variable. Pollution units, including those of the instruments, are standard deviations of mean concentrations. Both regressions include household fixed effects, household, weather controls, quarter of year fixed effects, and region-year fixed effects. Household level probability weights are used. Standard errors, clustered at the MSA/survey cohort level, are included in parentheses. *** p<0.01, ** p<0.05, * p<0.1

Ozone

Column 1 of Table R2 presents results from an IV regression where O_3 is over-identified using the two distant source instruments. In this specification, we find a statistically significant effect of 7.4 percent from a one standard deviation increase in ozone concentrations. SO_2 again is found to have a positive and significant sign. Column 2 attempts to jointly instrument for O_3 and $PM_{2.5}$, but the instruments are very weak in this setting and lead to very imprecise coefficient estimates. The third column in the table jointly instruments for O_3 and SO_2, and results in estimates similar to those in column 1. Our main results suggest that $PM_{2.5}$ has the largest impact on medical expenditures, and SO_2 may again be serving as a proxy for this in Table R2. Because we are not able to estimate particulate matter jointly with O_3 in our setting, our main results in this paper do not focus on the effect of O_3. However, the results shown here do give some evidence of this effect and motivate future research on it.

Table R2: Using Distant Source Instruments to Identify Ozone Effect

Pollutant	(1) O_3 Instrumented		(2) O_3 & $PM_{2.5}$ Instrumented		(3) O_3 & SO_2 Instrumented	
O_3	0.0740**		0.445		0.0815**	
	(0.0345)		(0.938)		(0.0355)	
$PM_{2.5}$	-0.0170		-0.313		-0.0181	
	(0.0133)		(0.753)		(0.0136)	
SO_2	0.0559***		0.136		0.0393**	
	(0.0117)		(0.204)		(0.0176)	
First Stage	O_3		O_3	$PM_{2.5}$	O_3	SO_2
F-Stat	20.21		0.40	0.31	29.22	74.13
Observations	48,626		48,626		48,626	
Households	6,137		6,137		6,137	

Contributions from distant SO_2 and NOx emissions to local predicted particulate matter concentration are used as instruments for all regressions. All regressions include household fixed effects, household, co-pollutant and weather controls, quarter of year fixed effects, and region-year fixed effects. The dependent variables are log-transformed expenditures on Asthma and COPD. Pollution units are standard deviations of mean concentrations. Household level probability weights are used. Standard errors, clustered at the MSA/survey cohort level, are included in parentheses. *** p<0.01, ** p<0.05, * p<0.1

Chapter 2: Using Residential Sorting Behavior to Better Understand the Relationship between Urban Greenspace and Health

2.1 Introduction

The high percentage of Americans who classify as overweight or obese continues to raise public health concerns in the United States. Obesity rates have steadily increased over the past two decades, with recent estimates indicating that 69 percent of adults in the U.S. are overweight, and 35 percent are obese (Expert Panel Report, 2014). Though the trend has started to level off in recent years, the high rates may have long term health consequences. Clinical evidence suggests that obesity increases an individual's risk of morbidity from a wide range of conditions including hypertension, heart disease, and diabetes. Finkelstein et al. (2009) estimate that obese individuals incur $1,429 more in medical expenses annually than normal weight individuals. This translates to $147 billion dollars in additional expenditures in the United States, or 9.1 percent of total spending. Further, costs of obesity may include decreased worker productivity and other morbidity costs beyond those captured by medical expenditures.

Due to the high costs of obesity, understanding its underlying causes is important for designing policies to address it. While genetic differences explain why some individuals are more likely to gain weight than others, these differences do not explain the recent trends in obesity rates. At its core, obesity is a product of too much caloric intake relative to caloric expenditure. Potential underlying causes of this caloric gap include changes in relative food prices that encourage more food consumption, occupational changes from labor intensive jobs in manufacturing to jobs in the service or technology sectors, and built environment changes that discourage physical activity (Rosin 2008).

Even though decreasing caloric intake is the best way to manage weight (Expert Panel Report, 2014), a recent wave of research in public health finds associations between the proximity of an individual to built environment characteristics related to physical activity, which I will refer to broadly as greenspace, and obesity. Since physical activity has health benefits beyond weight loss, this literature relates greenspace to a broad range of health outcomes. Access to public parks is often the environmental characteristic of interest, but greenspace can also include other measures such as local tree coverage. Studies have found correlations between greenspace and obesity rates, cardiovascular illness, stress, depression, anxiety, and self-reported health (Lee and Maheswaran 2010; Beyer et al. 2014). Though the evidence specific to park-obesity associations has generally been weak or mixed (Coombes et al. 2010, Potestio et al. 2009), a stronger relationship is often found between parks and physical activity measures.

There are several hypothesized causal pathways that link greenspace to health outcomes. Close proximity to a park reduces the cost of utilizing it and may encourage individuals to engage in more healthy activities, such as walking, running, or playing sports. Even if the park is not used for vigorous exercise, walking to and from the park alone may constitute an activity increase. Over time, this increase in physical activity can lead to health improvements. Further, greenspace may be negatively correlated with other environmental "bads", such as localized air pollution (McPherson et al. 1994). Finally, it is possible that natural scenery is intrinsically good for mental health and offers a refuge from otherwise stressful urban environments.

My study addresses one of the biggest empirical issues prevalent in this literature: people sort themselves into neighborhoods based on the characteristics of those neighborhoods and their personal preferences. Much of the association between parks and health outcomes or physical activity measures likely comes from the fact that people who are physically active will choose to live in neighborhoods with amenities that cater to active recreation. There is strong evidence that housing prices implicitly include the values of local amenities, such as air pollution and open space (Klaiber and Phaneuf 2010; Bayer, Keohane, and Timmins 2009). These studies yield insight into how much people are willing to pay for different levels of an amenity. In the current setting, greenspace valuations may vary by individual preferences, which will impact residential location choices. This is the key mechanism through which heterogeneous preferences may lead to observed associations between health characteristics and local greenspace. For instance, a physically fit person may have preferences that induce them to seek out amenities that support their healthy lifestyle. This would lead to estimates which overstate the effect of greenspace on health. Alternatively, if unfit individuals seek out healthy amenities in order to improve their own health, then sorting will bias results in the opposite way.

To address this concern, some authors have implemented within person estimators to control for individual preferences that are constant over time. If residential sorting is based on unchanging unobserved characteristics, then a within person estimator resolves the issue. Using a first difference estimator, Eid et al. (2008) find no significant relationship between obesity status and urban sprawl. Boone-Heinonen et al. (2010) apply an individual fixed effects estimator to data on built environment characteristics and physical activity rates. They find a small but positive and significant impact of private recreation facilities (e.g. private gym or athletic club), but no effect from other characteristics including public park facilities, street connectivity, and landscape diversity. In addition, Baum and Chou (2015) implement a fixed effects estimator and find that urbanization impacts obesity. These studies provide more reliable results of health/environment relationships, but they do not account for sorting based on time-varying unobservables.

Other studies use instrumental variables (IV) to address the endogeneity issue. Courtemanche and Carden (2011) use an instrument based on Walmart store location decisions to explore how the spread of Walmart stores impacts obesity rates. They find that an additional Walmart in a community increases an individual's probability of obesity by 2.3 percent, an effect that operates through gaining access to cheaper food. Similarly, Dunn (2010), explores how access to fast food impacts obesity. Using the number of interstate exits to instrument for fast food restaurants, Dunn finds evidence of a positive effect in counties with medium population density. Also using an IV strategy, Zhao and Kaestner (2010) find that urban sprawl contributed to 13 percent of the recent increase in obesity rates in the U.S. Finally, multiple papers have used IV estimators when studying the impact of the built environment on travel behaviors (Boarnet and Sarmiento 1998, Khattak and Rodriguez 2005).

I use data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) to estimate the relationship between parks and obesity in a more comprehensive way. My study utilizes both fixed effects and IV to identify the impact of parks on obesity, which allows me to compare the impact of both strategies in an empirical setting. Add Health follows students from grades 7-12 into young adulthood and combines data on both person-level characteristics and detailed neighborhood features, including the number of parks in proximity to a person's home. For a given residential address, park counts within 1 km do not change much over time, so my estimates rely on spatial variability in built environment characteristics. Because I use individual fixed effects to control for unobserved heterogeneity, I restrict my sample to individuals that move between survey waves to ensure sufficient within-person variability in neighborhood attributes. While this sample restriction invokes selection concerns, this is less of a problem in my setting, because most children move away from home in the years following high school graduation.

To address time-varying unobserved variables, I use logic from the residential sorting literature to select valid instruments in my setting. In particular, I use neighborhood characteristics that are likely to be correlated with parks, but unrelated to health outcomes, conditional on other covariates. Including person fixed effects decreases the magnitude of the estimates when compared to pooled OLS, but the IV strategy more than compensates for this reduction. In my preferred specification, I find that one additional park within 1 km reduces residents' BMI by 1.25 percent.

This study provides one of the most comprehensive examinations of the relationship between urban greenspace and health to date. I identify and address additional difficulties for causal inference in this setting. This paper provides new evidence of the benefits of built environment amenities and can inform public policy that addresses current health challenges in the United States.

2.2 Data

My data comes from Add Health, which combines data on person-level characteristics with detailed neighborhood features. Add Health follows a cohort from grades 7-12 into early adulthood, beginning with a nationally representative sample of this age group in the first wave of the survey. Adolescents undergo a number of significant life transitions during this period, so time-varying unobservables are of particular concern for this population. I focus on Waves 1 (1994-95) and 3 (2001-02) of the survey, because they include comprehensive neighborhood amenity characteristics. These characteristics come from a number of sources, including government and proprietary data sets, and were merged with person-level survey data before being released for use. Geographic data is not explicit, but masked identifiers down to the Census block group level are observed by the researcher. Because of this, I do not know where the survey participants live, but I do know if two participants live in the same census block group, tract, county, or state. Sampling in Wave 1 is clustered on school systems, but participants are relatively dispersed by Wave 3. Specifically, the subsample of participants that move between waves live in 972 distinct census tracts (in 138 counties) in Wave 1, and 5062 census tracts (in 876 counties) by Wave 3. Change in residential address between survey waves is identified by geocoded residential addresses. If the distance between addresses is less than one quarter of a mile, this change is not thought to be significant and is coded in the data as though the individual did not move. Approximately 79 percent of participants change residence between these two interview periods, and 70 percent live independently (no longer with parents) by Wave 3. Conditional on moving, mean distance moved is 169.2 miles, while median distance moved is only 8.1 miles. Participants who change residence by Wave 3 are of particular interest because moving ensures sufficient variation in environmental characteristics, which tend not to change much over time. Concerns about movers being a selected sample are in part alleviated by the fact that most individuals move away from home during this period of their life, and are therefore fairly representative of this age group.

A measure of Body Mass Index (BMI) will be the main health outcome utilized in my empirical analysis. BMI is a function of height and weight, and serves as a proxy for obesity. This is not a perfect health measure, as it does not take body fat percentage into account, but it is widely used in the literature and the best measure available in the data. A number of individual level demographic variables are available in addition to the health measures. Table 1 presents summary statistics for these variables, including education level, marriage status, and if the respondent has children. By Wave 3, participants are aged between 18 and 27. Around 21 percent are obese (classified as having a BMI of 30 or greater), over 20 percent are married, and 27 percent are enrolled as full time students.

Most residential locations are identified by geocoded address or GPS measurement, so contextual variables in the data are precisely measured. Neighborhood characteristics come from outside sources and do not rely on respondent recall or estimates, and they are merged at the census block group level or the exact residential address. For example, information on median housing value and housing unit density are known by block group. Other variables, such as number of parks, are measured as counts within a 1 kilometer street network or Euclidean distance of an individual's residence. The network distance best represents how far an individual must travel to access a park and offers the best measure of access cost, so it is used when available. Previous work has found the strongest association between built environment features and physical activity within 1-3 km buffers (Boone-Heinonen 2010), so I follow this in my preferred specification. These measures are also available at 5 and 8 km distances, and using alternative measures does not qualitatively impact my results. Additional neighborhood variables are summarized in Table 2. The alpha index measures street connectivity, which proxies for the walkability of a neighborhood. Higher values indicate higher connectivity. The mean fractal dimension index (MFDI) serves as a second land use control. Values near one are indicative of an urban environment, while higher values are associated with more natural settings. These two variables control for urban sprawl/urbanization, which is known to be associated with obesity. Further, I control for economic, weather, additional urbanicity, and health variables that may impact BMI and/or how people interact with parks.

If parks are heterogeneous in terms of quality, a simple park count may not be the best measure of the amenities offered. To address this, I use additional park measures that may be more indicative of quality or accessibility. These include categories of parks or facilities associated with physical activity that are grouped based on primary Standard Industrial Classification (SIC) codes, which implies they have some sort of commercial operations. For example, I compare the effect of parks that require a membership, like a private country club, to public parks without fees or other entry restrictions. Definitions and examples of these alternative park definitions can be found in Table 3.

Table 4 presents summary statistics by obesity status and income indicators. Columns 1 and 2 compare park counts for individuals living in block groups with housing values below and above the median in my sample. Low housing value neighborhoods have fewer overall, membership, outdoor, and public parks. However, there are significantly more YMCA facilities in these neighborhoods, and the difference in means for the membership category is not significantly different from zero. The lack of significance for this category may indicate that private membership facilities, which are not public goods, are not reflected in nearby housing prices. Still, this generally supports the idea that parks are positive amenities whose value is reflected in housing prices. An obesity comparison yields the expected results, in that obese individuals live near fewer parks across all types.

2.3 Empirical Strategy

2.3.1 Neighborhood Choice

To better understand the difficulties in estimating the causal impact of built environment features on health outcomes, it is useful to detail how neighborhood decisions are made in a discrete choice framework. A neighborhood can be thought of as a bundled good containing multiple quality attributes. Since individuals only choose one neighborhood location, they do not choose the level of each attribute separately; they must choose from the bundles available to them. Given J available alternatives, individuals choose the neighborhood that maximizes their utility. The utility individual *i* receives from choosing location *j* can be written as

$$U_{ij} = V_{ij}\left(X_i, N_j, \epsilon_{ij}, \beta, \alpha\right) \tag{1}$$

where indirect utility *V* is a function of person-level characteristics *X* and neighborhood characteristics *N*. The term ϵ_{ij} can be thought of as unobserved (to the econometrician) heterogeneity, and it includes structural or neighborhood characteristics omitted from *N* as well as unobserved characteristics that impact choices such as personal preferences. β and α are parameters that link observed characteristics to utility outcomes. In the empirical model, I parameterize *V* as follows:

$$V_{ij} = \beta N_j + \sum_{k}^{K} \sum_{l}^{L} \alpha_{kl}\left(N_{jk} \times X_{il}\right) + \epsilon_{ij} \tag{2}$$

with interactions between the individual and neighborhood variables. The econometrician does not observe ϵ_{ij}, but makes an assumption about the distribution from which it is drawn in order to operationalize the model. The parameters in (2) can be estimated using a multinomial logit model, which assumes the error term to be distributed Type 1 Extreme Value. Because estimation relies on differences in utilities, the *X* variables are not included separately from their interaction terms. These interaction terms are of central interest in the discrete choice exercise. They yield information on how valuations of neighborhood characteristics vary based on observed heterogeneity. For example, if the coefficient on the interaction between number of parks and obesity status is negative, then an obese individual receives less utility from a park than a non-obese individual. If this is true, then an obese individual will be relatively less likely to choose a neighborhood with many parks, driving a sorting process that results in spatial correlations between parks and obesity.

From this set up, it is clear that person level characteristics play an important role in neighborhood choice, and the level of amenities in a person's chosen neighborhood are not independent of their observed characteristics and unobserved preferences. This choice mechanism causes inference difficulties when assessing causal relationships between built environment features and health outcomes, something that is discussed in more detail in the next section.

2.3.2 Built Environment Impact on Health

To be clear about the empirical challenges faced when estimating the relationship between neighborhood characteristics and health outcomes, consider estimating the health equation

$$Health_i = \beta_0 + \beta_1 Parks_i + \beta_2 N_i + \beta_3 X_i + \epsilon_i \ , \tag{3}$$

where *N* includes other neighborhood characteristics and *X* is a vector of person-level controls. I am interested in the effect of an additional park on health, but estimates will be biased if the error term contains unobservables that impact both the level of parks near one's residence and health. More precisely, bias occurs if the error term can be written as

$$\epsilon_i = c_i + \eta_i \tag{4}$$

and $E[c_i|parks_i, N_i, X_i] \neq 0$. This will be the case if unobservable preferences for healthy lifestyles determine both the number of parks near one's chosen residence and level of fitness.

Within-Person Estimator

With panel data, the issue outlined above can be resolved. The estimating equation becomes

$$Health_i = \beta_1 Parks_{it} + \beta_2 N_{it} + \beta_3 X_{it} + c_i + \eta_{it} \tag{5}$$

and the unobserved heterogeneity c_i can be removed through a first difference transformation or by including individual fixed effects. However, if the error term in (5) contains a time varying component d_{it} such that $E[d_{it}|parks_{it},\ N_{it},\ X_{it}, c_i] \neq 0$, a within-person estimator will not yield consistent results.

Instrumental Variables Estimator

If an appropriate instrument is available, then an IV strategy will allow for time-varying unobserved variables that are correlated with parks. Some examples of using this approach to address endogeneity caused by residential sorting come from the transportation literature. Boarnet and Sarmiento (1998) estimate how land use influences transportation behavior. They use neighborhood racial composition and age of the housing stock as instruments for land-use characteristics, such as population density and street connectivity. Validity of the IV strategy requires unobserved preferences for the instruments, which are also taken into account by the individual when choosing a residential location, to be unrelated to preferences for the land use variables. For instance, in the aforementioned study, preferences for racial composition must be unrelated to preferences for population density.

I propose a novel instrumental variables strategy using information from housing markets. The general idea is that greenspace will be correlated with other neighborhood amenities and housing characteristics that are plausibly unrelated to health. For instance, houses located near parks may also have more square footage or be located in better school districts. The data set includes a number of characteristics that may work well as instruments. First, I use median housing value in the respondent's block group. An increase in neighborhood quality will raise both demand for that location and the price of housing units there. In equilibrium, the value of parks, as well as unobserved neighborhood amenities, will be incorporated in median housing price. Through this mechanism, housing prices will be correlated with parks and also contain information on the unobserved quality of a neighborhood. Next, I use number of schools within 1 km. Schools may be associated with positive amenities such as safety or social cohesion, and they are often located adjacent to parks. Finally, I use number of housing units per square mile as an instrument. This serves as a measure of lot size, a positive characteristic. This variable may be higher in more suburban block groups, which may attract higher BMI residents, but controlling for land use characteristics in *N* should help alleviate these concerns. The Add Health data contains many health-related variables that will help absorb variation in my instruments that may be related to health outcomes.

Despite my careful controls, there may be channels through which an instrument may impact health apart from its relationship with parks. To address this concern, I use attributes from other block groups in the same county as an alternative set of instruments. The reasoning for these new instruments is similar to that presented in Bayer and Timmons (2007). When making a residential location decision, individuals consider all available alternatives in the market and choose a location that maximizes their utility conditional on their preferences and budget constraint. Demand for a location may also influence the neighborhood attributes themselves. For example, if many people have preferences for living near city centers, then housing density and prices will be higher in these areas. More schools may be built in these neighborhoods to serve demand. They will also tend to have higher population densities, which could be seen as a negative amenity. Bayer and Timmons (2007) focus on this congestion effect in their paper. Housing market equilibriums result from this complex interplay between supply and demand, and it is therefore reasonable to assume that certain neighborhood characteristics will be a function of available amenities in other neighborhoods in the same market. However, the attributes of other neighborhoods are unlikely to have a direct impact on health.

I propose using instruments *Z* for greenspace in the following specification:

First Stage:

$$Parks_i = \alpha_0 + \alpha_1 Z_i + \alpha_2 N_i + \alpha_3 X_i + \zeta_i \tag{6}$$

Second Stage:

$$Health_i = \beta_0 + \beta_1 Parks_i + \beta_2 N_i + \beta_3 X_i + \epsilon_i \tag{7}$$

The success of this IV strategy depends on two main requirements: (a) that *Z* is correlated with parks, and (b) that *Z* does not relate to health except through its association with controls in *N*. The first condition should be satisfied due to the nature of housing market equilibriums, and is verified in my analysis. The second requirement relies on sufficiently controlling for health-related characteristics and preferences. As demonstrated section 3.1, the residential sorting process implies that neighborhood amenities will be related to unobserved preferences. Requirement (b) can alternatively be satisfied under certain preference separability assumptions. This is reasonable if, for example, the unobserved determinants of choosing to live near a school are very different from the reasons an individual chooses to live near a park.

2.4 Results

2.4.1 Preliminary Evidence: Discrete Choice Estimates

To investigate how person-level characteristics influence neighborhood amenity preferences, I estimate a multinomial logit model of specification (2). In Wave 3 of the data, participants are spread over hundreds of different cities in the United States, and neighborhood characteristics are only observed if a survey participant chooses to live there. This creates difficulties when selecting a choice set. For this exercise, I choose to restrict the sample to the metropolitan area with the most participants in the data, which provides a relatively complete set of choices. Each observed location represents an available choice, and I assume the set of choices observed in the data were the only alternatives available.

Neighborhood characteristics include number of parks, schools, median housing value, housing unit density, diet resources, and measures of street connectivity. Variables in X include BMI measured prior to the move, and Wave 3 (post-move) education, marriage status, number of children, and income. Table 5 presents estimated coefficients of the neighborhood variables and their interactions. The interaction term between parks and BMI is marginally significant, indicating that individuals with higher BMIs receive relatively lower utility from being located near parks. Higher incomes groups are more likely to locate near parks, suggesting that parks are a positive amenity, though the main effect is negative and insignificant. Interestingly, the coefficient on BMI interacted with median value is also significant. This could indicate that higher BMI individuals dedicate less of their income on housing, or are more price sensitive with respect to housing. Alternatively, this could indicate that BMI is partially serving for some other unobserved determinant of housing choices. In a similar way, more highly educated people are found to choose more expensive housing. The income variable in Add Health relies on self-reporting, which may mean it is a noisy or biased measure of actual wealth, and other variables such as BMI and education are picking up its effect.

The results of this exercise illustrate the potential importance of unobserved preferences in residential location decisions. The significant coefficient on the parks and BMI interaction term is consistent with sorting behavior driving observed correlations between the two. However, this does not rule out a causal or biological relationship. Sorting may explain only a small portion of the association, or, as previously noted, BMI may be picking up the effect of alternative unobserved variable in this setting. The fact that the utility of schools and housing density is not found to vary with BMI supports the validity of the instruments described in section 3.2. However, a cause for concern is that utility from housing values does significantly vary on BMI.

Still, the magnitude of the interaction term coefficient is relatively small, and it may be unreliable for the same reasons mentioned for the parks and BMI interaction term.

2.4.2 Reduced Form Estimates

I first offer some preliminary evidence of this relationship between greenspace and health. Although I observe each individual twice in the data, I first treat all observations as independent and estimate a pooled OLS regression, the results of which are presented in the first column of Table 6. As hypothesized, an increase in number of parks within one kilometers an individual's residence is found to have a small but significant association with reduced BMI. My log-linear specification implies that one additional park decreases BMI by 0.36 percent. Though it should not be interpreted as causal, finding a significant relationship here is interesting in itself, given the mixed results from previous studies that have attempted to find direct associations between parks and BMI. Being married, having children, and higher education levels are also positively correlated with BMI, while being a full time student is associated with a significantly lower BMI. Column 2 in this table regresses number of parks in wave 3 of the survey on number of parks in wave 1 of the survey. Even though I restrict my sample to individuals who move between these two survey ways, there is still a strong positive association between the numbers of parks near an individual over time. This suggests that individuals move to neighborhoods with characteristics similar to those where they lived previously, a reminder that these characteristics are not randomly distributed. Further, higher BMIs in Wave 1 are associated with fewer parks in Wave 3, justifying the concern that sorting is driving observed park/health relationships. Somewhat surprisingly, participants with children in the third wave are less likely to live near parks. This could be due to budget constraints, as raising children is expensive and parks are positive amenities that are reflected in higher housing values. In column 3 I regress log-transformed Wave 3 BMI on Wave 3 parks and Wave 1 BMI. Unsurprisingly, I find that BMI is highly persistent over time. However, even after controlling for previous BMI, the number of parks is still contemporaneously correlated with BMI. This initial evidence could suggest that, although sorting on health characteristics does occur, it may not fully explain correlations between parks and BMI.

2.4.3 Main Results

Comparing how estimates vary across specifications gives a more nuanced understanding of the factors that contribute to observed associations between greenspace and health. In column

1 of Table 7, I estimate a panel model that includes individual fixed effects. This approach accounts for individual level unobserved characteristics that are not changing over time. To the extent that sorting behavior is explained by these characteristics, fixed effects estimates are more representative of the causal relationship between parks and BMI. Similar to previous findings, the significance of the parks coefficients disappears in this setting. This result presents more evidence that sorting is driving the observed associations, and it implies that living near a park has no discernible impact on obesity.

Though fixed effects estimates control for unobservables that do not change over time, time-varying variables that are left unaccounted for may still bias results. This is of particular concern in my setting, as subjects move from adolescence into young adulthood. This is a period of immense change for many individuals, and it is reasonable that health attitudes or health-related behaviors may be changing during this transitional period. To address this concern, I implement the instrumental variables strategy described above. Columns 2 and 3 of Table 7 instrument for *parks* using the following instruments: median house value, housing unit density, and number of schools within 1 km. In all of the IV models, *parks* has a significant and negative impact on BMI. Consistent with the difference observed between the pooled OLS and Fixed Effects estimates, inclusion of individual fixed effects in column 3 leads to a smaller estimated coefficient than in column 2. This pattern is consistent with negative bias from unobserved time-constant variables, but the opposite-signed bias from time-varying unobservables. The latter may result from higher BMI individuals seeking out physical activity amenities in order to improve their fitness. Similar to Eid et al. (2008), I find no significant effect from land-use controls when fixed effects are included.

2.4.4 Robustness

Even after including health controls, there may be hypothesized channels through which each individual instrument impacts health. However, since my three instruments measure dissimilar amenities, there is not likely to be a common mechanism that influences BMI. Appendix Table A3, columns 3-5, shows results from the Fixed Effects IV model when each instrument is used separately. The striking similarity of results across instruments helps alleviate concerns that the instruments fail the required exclusion restriction. As a formal test, I use multiple instruments to run tests of overidentifying restrictions. As reported in Table 7, in each IV specification I fail to reject the null hypothesis that the instruments are valid. First stage F-statistics are also reported, and full first and second stage are presented in the appendix.

The final column in Table 7 uses my alternative set of instruments. Instead of using own location attributes to instrument for parks, I use the mean level of attributes in other block groups in the same county. That is, I use leave-one-out averages of block group median home price, housing unit density, and school counts. The coefficient estimate in column 4 is negative and significant, though somewhat smaller in magnitude than in column 3, which uses the same Fixed Effects IV specification. This is strong evidence that my results are not being driven by invalid instruments, since it is unlikely that amenities in other neighborhoods will have a direct impact on BMI.

A direct way in which parks may impact health is through encouraging physical activity. Increased physical activity can lower BMI, but this benefit may take months or years to fully accrue. For this reason, one would not expect number of parks in a neighborhood to have a measurable effect on BMI for individuals who have recently moved. In Wave I of the survey, participants are asked what age they moved to their current residence, so I only identify the move date at an annual level. However, in Wave III of the survey, I observe the year and month of the move. Because of this, I use only use the latter round of the survey when investigating the impact of parks on recent movers. In column 1 of Table 8, I estimate an IV regression similar to the one in column 2 of Table 7, but only on the Wave III cross section. The result is very similar; one additional park is found to decrease BMI by about 2.4 percent. In column 2 of Table 8, I include a term that interacts number of parks with recent mover status. A participant is labeled a recent mover if they moved to their current address within one month of their interview date. 283 individuals in the sample meet this criteria. To implement the IV estimator, I interact my instruments with recent mover status and use the resulting variables as additional instruments. Column 2 presents the results of this estimation. The coefficient on the interaction term is slightly larger, but opposite in sign, than that of the main effect. This indicates that, for recent movers, the effect of parks is close to zero. This aligns well with the hypothesis that the health benefits take time to accrue, and it supports the causal interpretation of my results.

The results from Table 7 use a count of all parks as the key independent variable. However, parks may vary widely in quality, accessibility, and amenities offered, so being more specific about park type gives a more nuanced understanding of the mechanisms through which parks impact health. Table 9 show results using alternative park definitions with the Fixed Effects IV strategy. All categories have negative and significant coefficients on the park measure. The coefficient on Membership facilities has the lowest magnitude. Since these types of parks have the highest barriers to entry, this result is consistent with the idea that increasing park access will increase use and therefore maximize health benefits. This may also be indicative of how these parks are used, since the impact of a private golf course on BMI likely differs significantly from a neighborhood park with a walking path or sports fields. The magnitude of the estimates for the public and outdoor park categories are similar to the estimates in column 3 of Table 7. Having a YMCA within 1 km of your residence is found to decrease BMI by over 4 percent. This large effect may relate to the types of physical activity amenities, such as swimming pools and gym equipment, often offered at a YMCA. These results suggest that the intensity of physical activities associated with a park facility matters for weight loss.

Column 1 of Table 10 uses number of parks between 1 and 3 km from the participant's residence as the dependent variable. Parks in this outer perimeter are still found to have a significant impact on BMI, but the coefficient magnitude is substantially smaller than that of a park within 1 km. This supports the idea that proximity to a park influences its use and resulting health benefits. I next explore two measures of park access that don't explicitly rely on the count of nearby parks. Using the Euclidean distance to the nearest park, I find that as this distance rises, so does BMI. This corroborates the cost-of-access story and suggests that longer travel distances discourage the use of parks. In column 3, park area is also found to have an impact on BMI, though the coefficient is only weakly significant. There are multiple ways to interpret the impact of park area. First, similar to the minimum distance finding, more park area within a 1 km radius means that the distance needed to reach a park will be lower on average. Second, larger parks may allow for types of physical activity, such as running or hiking, that a small park cannot accommodate. It is likely that the health benefits from a park come not only from walking to access it, but also from physical activity that occurs once there.

Small parks are likely more common in dense urban areas, where open space is scarcer. If parks are smaller but more frequent in these areas, one concern is that a basic count of parks is partially capturing the health benefits of living in a very walkable neighborhood. Finding a significant effect from park area, and controlling for urbanicity and street connectivity measures, helps alleviate this concern. Still, this issue warrants further investigation. Wave 3 of the survey includes additional data on food resources, which are not directly associated with physical activity. Interestingly, living near a restaurant could impact BMI in multiple ways: (a) by encouraging increased walking to the location or (b) by increasing caloric consumption because of easier food access. Estimates of the impact of food resources are presented in Table 11. Since these new variables are only available for Wave 3 of the survey, I present IV estimates on this cross section. For sake of comparison, column 1 estimates the effect of parks using just this subset of the data. Column 2 similarly instruments for parks, but includes a count of all food resources as a control. By including food resources, I can better control for urbanicity factors that are not captured by my other covariates, such as if the individual lives near a strip mall. The coefficient on parks remains significant and increases by half of a percentage point, which suggests that unobserved urbanicity is not driving the effect on parks. Next, I use the same set of instruments to measure the effect of food resources on BMI. One might expect a fast food restaurant to increase BMI, while a health food store may lower BMI. Surprisingly, I find that both fast food restaurants and health food stores decrease BMI. This finding could mean that living near either food resource encourages enough walking to offset the effect of increased consumption, or that the count of food resources is serving as a proxy for urbanicity or walkability. To address this, I specify the fast and health food resources as a percentage of all food resources available. This highlights the fact that the mix, not just count, of available food resources is important. In columns 4 and 6, I find that a larger share of fast food restaurants has no impact on BMI, while larger health food shares have a large negative impact on BMI. The last two columns in Table 11 extend this exercise to YMCAs, which were found to have the largest impact on BMI. As expected, a higher share of the resource associated with relatively higher physical activity intensity is found to negatively impact BMI. This again demonstrates that the effects found for physical activity amenities are not entirely driven by their association with urban density.

Next, I investigate how climate influences park use. In places with relatively harsh winters, outdoor parks can only be utilized during part of the year. Theoretically, this should dampen the impact the park has on health. Columns 1 and 2 of Table 12 compare the effect of outdoor facilities in locations with January temperatures below and above the sample median. I find significant and negative effects on both subsamples, but, consistent with my hypothesis, the magnitude of the coefficient is almost 8 times larger in the warmer climates. As a further robustness check, I carry out the same comparison for the YMCA category. These facilities are more likely to have indoor physical activity amenities that can be used year round regardless of climate. The warmer climate coefficient in column 4 is large, but not statistically different from zero. I do find a significant effect for the colder climate subgroup. It is possible that people living in these climates must rely more heavily on indoor facilities, thus explaining why I only find a statistically significant effect for this group.

2.5 Conclusion

Using panel data with rich information on residential location choices, I estimate the relationship between greenspace amenities and health. I rely on an understanding of residential sorting behavior to find valid instruments for neighborhood amenity levels in order to address a standard endogeneity problem. My findings show that time-variant unobserved variables bias downward the estimated effect of access to greenspace on health. This implies that simply adding fixed effects in a panel setting may not be sufficient for identification.

OLS regressions on the pooled sample show a negative association between parks and BMI. Although this cannot be interpreted as a causal, finding a significant relationship in the cross section is promising, as previous cross-section studies have struggled to find direct associations between parks and BMI. Including individual fixed effects reduces the magnitude and significance of this finding. However, when jointly utilizing a fixed effects and instrumental variable estimation strategy, my preferred specification, I find that the addition of a generic park to a neighborhood reduces BMI by over 1 percent.

The policy implications of this finding depend on the costs of constructing and maintaining a park relative to the monetized health benefits of BMI reductions. A back of the envelope benefits calculation illuminates this trade off. A 3 km radius of a residential address is close to the geographic size of the average block in the sample, and I find that one additional park within 3 km decreases obesity rates by one third of a percent. On average, just over 2000 people live in the block groups in my sample, so this reduction equates to about 6.5 fewer obese individuals. If each obese individual incurs $1,429 in additional healthcare spending annually, then the monetized benefit of adding a park is $9,377 annually. Although this is a crude estimate, it demonstrates that health benefits alone likely cannot justify the costs of constructing and operating a park. However, my results suggest a much larger effect of health clubs, such as a YMCA, on BMI. Subsidizing gym memberships may be a more effective strategy if obesity reductions are the central policy objective.

2.6 References

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2.7 Tables

Table 1: Wave 3 Person-Level Characteristics

Variable	Individuals	Mean	Std. Dev.	Min	Max
BMI	9093	26.167	6.151	12.293	66.130
Obese	9093	0.216	0.412	0	1
Education	9093	13.199	2.033	6	22
Married	9093	0.209	0.406	0	1
Children	9093	0.431	0.761	0	9
Full Time Student	9093	0.274	0.446	0	1
Age	9093	22.070	1.762	18	27

Statistics based on subsample of movers. Obese defined by having a BMI of 30 or greater.

Table 2: Summary Statistics of Neighborhood Characteristics

Variable	Definition	Obs	Mean	Std. Dev.	Min	Max
Park Measures						
Park Count 1k	Within 1 km of residence[a]	17239	0.911	1.425	0.000	16.000
Park Count 1-3k	Between 1-3 km of residence[a]	17239	4.812	6.428	0.000	54.000
Membership	Within 1 km[b]	17239	0.294	1.066	0.000	34.000
Outdoor	Within 1 km[b]	17239	0.145	0.512	0.000	15.000
Public	Within 1 km[b]	17239	0.142	0.484	0.000	11.000
YMCA	Within 1 km[b]	17239	0.050	0.293	0.000	7.000
Instruments						
Median Value	Median House Value[c] (BG) in $10,000s	14667	11.313	8.996	0.000	100.000
Unit Density	Housing Units per sq. km[c] (BG)	17232	598.038	2003.490	0.000	56482.500
Schools	Within 1 km[b]	17239	6.966	12.763	0.000	200.400
Geographic/Economic						
Area	Sq. km[c] (BG)	17239	26.501	107.052	0.012	4816.599
Alpha	Street Connectivity[a] (1 km)	17239	0.314	0.638	-8.000	10.000
MFDI	Landscape Diversity[d]	17239	1.071	0.028	1.004	1.182
MHI	Median Household Income[b] (BG)	17239	39094.950	20161.530	0.000	200001.000
Unemployment	Rate for >16 population[e] (BG)	17239	0.075	0.070	0.000	0.955
COLI	Cost of Living Index[f]	17239	1.083	0.219	0.850	2.370
Health						
Birthweight	Low birth weight proportion[a] (C)	17239	0.076	0.017	0.036	0.144
Medicaid	Spending per beneficiary[a] (S)	17239	3650.383	1281.579	441.714	7725.138
Mortality	Per 1,000[a] (C)	17239	8.336	2.015	1.488	18.338
Infant Mortality	White, Per 10,000[a] (C)	17239	55.668	57.618	0.000	240.000

Adult Arrests	Per 100,000[g] (C)	17239	714.260	394.695	0.000	9403.547
Juvinile Arrests	Per 100,000[g] (C)	17239	271.920	162.807	0.000	2310.586
Weather						
Precipitation	Mean total rainfall, July[h]	17239	3.371	2.057	0.000	9.110
Sun	Mean sunshine total hours, Annual[h]	17239	2785.888	412.815	1488.000	4015.000
Summer Temp	Mean daily max temp, July[h]	17239	86.996	6.415	63.800	108.700
Winter Temp	Mean daily min temp, January[h]	17239	28.119	12.406	-9.000	65.000
Snowfall	Mean total snowfall, Annual[h]	17239	17.167	20.812	0.000	86.900

BG indicates measure at Block Group level, C at the County level, and S at the State level. Weather norms come from the nearest weather station with non-missing data. Statistics based on subsample of movers. Data origin: a) ESRI StreetMap Pro; b) Dun and Bradstreet; c) U.S. Census; d) National land cover dataset; e) U.S. Bureau of Labor Statistics; f) American Chamber of Commerce Research Association; g) Uniform Crime Reporting data; h) Climate Atlas of the United States

Table 3 : Additional Park Measures

Park Type	Definition	Example
Membership	Require a membership	Country club, boating club, health club
Outdoor	Are “outdoor” in nature.	Campgrounds, ski slope, golf course, riding stable
Public	Free, public access	Tennis courts, community center, recreation center, public beach
YMCA	Non-profit aimed at improving community health and well-being	YMCA, YWCA

Categories based on Dun & Bradstreet primary Standard Industrial Classification. There is potential for overlap between categories. For example, a private golf course would be included in both the Membership and Outdoor categories.

Table 4: Wave 3 Parks by Income and Obesity Status

	(1) Low Value	(2) High Value	(3) Non-Obese	(4) Obese
Park Count	0.686	1.227	1.007	0.772
Membership	0.387	0.409	0.434	0.267
Outdoor	0.179	0.229	0.220	0.149
Public	0.168	0.196	0.192	0.147
YMCA	0.075	0.0566	0.070	0.050
Observations	4,546	4,540	7,122	1,964

Movers subsample. Park measures are counts within 1 km of residence in Wave 3. High value indicates greater than the median housing value ($96,300, block group level) in the sample. All value comparison means statistically different at the 5 percent level except for the membership category. For Obese comparison, YMCA means statistically different at 5 percent level, all other categories significant at 1 percent level.

Table 5: Discrete Choice Estimates

	Main Effect	×BMI	×Education	×Married	×Children	×Income
Parks	-0.0857	-0.0127*	0.0232	-0.0014	0.0187	0.0296**
Schools	-0.0138	-0.0012	0.0033	-0.0351*	0.0040	0.0011
Median Value	-0.02445	-0.0021**	0.0055***	-0.0046	-0.0081	-0.0003
Unit Density	0.0652	0.0037	0.0002	-0.07134	-0.1433***	-0.0301*
Diet Resources	-0.0143	0.0002	0.0000	0.0117**	0.0044*	0.0010

Parks, Schools, and Dietary Resources measured as counts within 1 km of residence. Median home value, at the block group level, is in $10,000s. Housing unit density is measured in 1,000s of units per sq. km. Several street connectivity measures were included in estimation but their coefficients are omitted from the table due to lack of significance.

Table 6: Preliminary Evidence

	(1) Log(BMI)	(2) Parks Wave 3	(3) Log(BMI) Wave 3
Parks	-0.00359**		
	(0.00161)		
Parks Wave 1		0.227***	
		(0.0195)	
Parks Wave 3			-0.00306***
			(0.000999)
BMI Wave 1		-0.00971***	0.0366***
		(0.00361)	(0.000448)
Full Time Student	-0.0551***	0.00343	0.00128
	(0.00681)	(0.0451)	(0.00352)
Children	0.0187***	-0.0391**	0.00499*
	(0.00543)	(0.0152)	(0.00263)
Education	0.00889***	0.0513***	-0.00245***
	(0.00120)	(0.00952)	(0.000802)
Married	0.0301***	-0.153***	0.0385***
	(0.00795)	(0.0340)	(0.00422)
Constant	2.899***	12.72***	2.375***
	(0.183)	(1.348)	(0.125)
Weather Controls	yes	yes	yes
Health Controls	yes	yes	yes
Time F.E.	yes	no	no
Observations	16,191	6,797	8,867
R-squared	0.148	0.229	0.565

Movers sample. OLS estimates, column titles are dependent variables. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

Table 7: Comparison of Fixed Effects and Instrumental Variable Approaches

	(1) FE	(2) Pooled IV	(3) FE IV	(4) FE IV
Park Count 1 km	-0.000184	-0.0239***	-0.0125**	-0.00784*
	(0.00161)	(0.00536)	(0.00529)	(0.00451)
F-Stat		50.78	14.79	33.37
Hansen J Stat (p-val)		0.5854	0.9159	0.9094
Observations	16,191	13,768	10,016	13,548
R-squared	0.511	0.133	0.525	0.502
Number of ID	8,949		5,008	6,774

Movers sample. Log-transformed BMI is the dependent variable. Park count measured as number of parks within 1 km Euclidean distance of residence. Median housing value, housing unit density, and school count of own location used as instruments for columns 2 and 3. Average median housing value, housing unit density, and school count of other locations in same county used as instruments for columns 4. Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 8: Impact of Parks by Residence Length

	(1) WIII Sample IV	(2) Recent Mover Interaction
Park Count 1k	-0.0242***	-0.0258***
	(0.00665)	(0.00694)
Parks X Recent Move		0.0299**
		(0.0126)
Parks F-Stat	34.73	19.52
Interaction F-Stat		6.19
Hansen J Stat (p-val)	0.9201	0.9919
Observations	8,542	8,542

Movers sample. Recent Move indicates participant has moved within 1 month of being surveyed. Log-transformed BMI is the dependent variable. Median housing value, housing unit density, and school count of own location used as instruments for park count. In column 2, interactions of these instruments with recent mover status as additional instruments. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

Table 9: Alternative Park Categories

	(1) Membership	(2) Outdoor	(3) Public	(4) YMCA
Park Measure	-0.00466**	-0.0161**	-0.0173**	-0.0414**
	(0.00217)	(0.00754)	(0.00729)	(0.0200)
F-Stat	77.19	23.26	48.54	7.17
Hansen J Stat (p-val)	0.4123	0.3974	0.6956	0.5461
Observations	10,016	10,016	10,016	10,016
Number of ID	5,008	5,008	5,008	5,008

Movers sample. Membership, Outdoor, Public, and YMCA counts within 1 km network distance of residence. Median housing value, housing unit density, and school count of own location used as instruments for facility count. Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 10: Alternative Park Measures

	(1) Parks ∈ (1,3] km	(2) Minimum Distance	(3) Total Area
Park Measure	-0.00212**	0.0226**	-0.284*
	(0.000871)	(0.0104)	(0.172)
F-Stat	27.41	11.57	3.38
Hansen J Stat (p-val)	0.8722	0.5597	0.4914
Observations	10,016	5,946	10,016
R-squared	0.526	0.494	0.469
Number of ID	5,008	2,973	5,008

Movers sample. Log-transformed BMI is the dependent variable. Minimum distance indicates distance to nearest park. Total area includes are of all parks within 1 km of residence. Median housing value, housing unit density, and school count of own location used as instruments for the park measure. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

Table 11: Assessing the Impact of Urban Density on BMI

	(1) Wave 3 Park Count	(2) Park Count W/ Diet Control	(3) Fast Food Count	(4) Fast Food Share	(5) Health Food Count	(6) Health Food Share	(7) YMCA Count	(8) YMCA Share
Built Environment Measure	-0.0238***	-0.0288***	-0.0268***	-0.00138	-0.0216***	-0.1104***	-0.101***	-.05808**
	(0.00608)	(0.00733)	(0.00749)	(0.00416)	(0.00688)	(.03778)	(0.0328)	(.02687)
Observations	8,542	8,542	8,517	8,517	8,517	8,517	8,517	8,517

Movers sample. Log-transformed BMI is the dependent variable. Column names indicate built environment measure used in estimation. Column 2 includes controls for count of dietary resources within 1 km network distance of residence. Median housing value, housing unit density, and school count of own location used as instruments for the built environment measures. Robust standard errors in parentheses. *** $p<0.01$, ** $p<0.05$, * $p<0.1$

Table 12: Climate's Influence on Park Utilization

	(1) Cold Outdoor	(2) Hot Outdoor	(3) Cold YMCA	(4) Hot YMCA
Park Count 1 km	-0.0113*	-0.0862**	-0.0303*	-0.167
	(0.00672)	(0.0397)	(0.0179)	(0.109)
Observations	4,498	4,874	4,498	4,874
R-squared	0.553	0.481	0.552	0.495
Number of ID	2,249	2,437	2,249	2,437

Log-transformed BMI is the dependent variable. Movers sample. Cold defined as having January minimum temperature below sample median (29 degrees Fahrenheit). Median housing value, housing unit density, and school count of own location used as instruments for park count. Robust standard errors in parentheses. *** $p<0.01$, ** $p<0.05$, * $p<0.1$

2.8 Appendix

Table A1: Full Results

	(1) FE	(2) Pooled IV	(3) FE IV
Park Count 1 km	-0.000184	-0.0239***	-0.0125**
	(0.00121)	(0.00536)	(0.00529)
Full Time Student	0.00257	-0.0522***	0.00487
	(0.00549)	(0.00693)	(0.00642)
Children	0.00915***	0.0170***	0.0106***
	(0.00332)	(0.00535)	(0.00394)
Precipitation	0.00218	-0.00421**	0.000487
	(0.00222)	(0.00192)	(0.00281)
Sun	4.06e-06	-5.98e-07	4.88e-06
	(9.46e-06)	(7.68e-06)	(1.14e-05)
Summer Temp	0.000632	0.000798	0.000163
	(0.000437)	(0.000493)	(0.000521)
Winter Temp	-0.00177***	0.000198	-0.00149**
	(0.000466)	(0.000417)	(0.000592)
Snowfall	-0.000351	0.000110	-0.000336
	(0.000229)	(0.000211)	(0.000285)
Alpha	0.00102	0.00367	0.00149
	(0.00212)	(0.00380)	(0.00314)
Median Income	4.57e-08	-5.78e-07***	-2.75e-08
	(1.00e-07)	(1.27e-07)	(1.23e-07)
Education	0.00383***	0.00743***	0.00537***
	(0.00126)	(0.00132)	(0.00151)
Married	0.0371***	0.0259***	0.0288***
	(0.00593)	(0.00813)	(0.00712)
MFDI	-0.0436	-0.368*	-0.00676
	(0.132)	(0.193)	(0.160)
COLI	0.0151	0.00453	-0.00569
	(0.00973)	(0.0175)	(0.0181)
Birthweight	0.158	0.195	0.190
	(0.184)	(0.191)	(0.232)
Unemployment	0.0146	0.131***	0.0241
	(0.0272)	(0.0370)	(0.0363)
Medicaid	-2.97e-06	2.23e-06	-6.61e-06*
	(3.07e-06)	(3.35e-06)	(3.74e-06)
Mortality	0.00112	0.00165	0.00115
	(0.00127)	(0.00139)	(0.00161)
Infant mortality	-6.05e-05	-3.10e-05	-7.32e-05
	(7.93e-05)	(0.000140)	(0.000107)
Adult Arrests	6.07e-06	2.26e-05**	8.95e-06

	(7.68e-06)	(1.04e-05)	(8.98e-06)
Juvenile Arrests	-1.43e-05	-1.81e-05	-4.90e-06
	(1.74e-05)	(2.38e-05)	(2.09e-05)
Time	0.114***	0.154***	0.116***
	(0.0118)	(0.0182)	(0.0150)
Constant	3.053***	3.310***	
	(0.146)	(0.214)	
Observations	16,191	13,768	10,016
R-squared	0.511	0.134	0.523
Number of ID	8,949		5,008

Movers sample. Log-transformed BMI is the dependent variable. Median housing value, housing unit density, and school count of own location used as instruments for columns 2 and 3. Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table A2: First Stage Results

	(1) Park Count
Median Housing value	0.108*
	(.065)
Housing Unit Density	0.041
	(0.044)
Schools	0.0231***
	(0.00485)
Full time student	-0.129*
	(0.0782)
Children	0.0389
	(0.0413)
Precipitation	-0.150***
	(0.0313)
Sun	4.10e-05
	(0.000152)
Summer Temp	0.000265
	(0.00727)
Winter Temp	0.0285***
	(0.00652)
Snowfall	0.0128***
	(0.00301)
Alpha 1 km	0.0408**
	(0.0162)

Median Income	-7.40e-06***
	(2.02e-06)
Education	0.0427**
	(0.0167)
Married	-0.0676
	(0.0632)
MFDI	-5.276***
	(1.399)
COLI	0.272
	(0.210)
Birthweight	7.087***
	(2.194)
Unemployment	-0.312
	(0.606)
Medicaid	-1.76e-05
	(4.44e-05)
Mortality	-0.0353**
	(0.0174)
Infant mortality	0.000537
	(0.000943)
Adult Arrests	0.000257**
	(0.000102)
Juvenile Arrests	-0.000214
	(0.000212)
Time	0.302**
	(0.148)
Constant	4.645***
	(1.770)
Observations	13,985
Number of ID	8,813
R-squared	0.140

First stage results for the specification in Column 3 of Table 6. Housing unit density in 1,000s of units per sq. km. Median housing value is in $10,000s.
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table A3: Alternate IV Specifications

	(1) Obesity	(2) Log(BMI)	(3) Value	(4) Density	(5) Schools
Park Count 1k	-0.0215*	-0.0125**	-0.0143	-0.0141***	-0.0112**
	(0.0119)	(0.00529)	(0.0215)	(0.00463)	(0.00449)
F-stat	13.79	14.79	6.06	24.50	87.83
Observations	10,016	10,016	10,022	14,478	14,484
R-squared	0.143	0.525	0.520	0.499	0.503
Number of ID	5,008	5,008	5,011	7,239	7,242

Dependent variable is obesity status in column 1 and log-transformed BMI in columns 2-5. Median housing value used as instrument in column 3, housing unit density used as instrument in column 4, schools used as instrument in column 5, and all three are used in columns 1 and 2. Robust standard errors in parentheses *** p<0.01, ** p<0.05, * p<0.1

2.9 Acknowledgements

This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due to Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the AddHealth data files is available on the AddHealth Web site (http://www.cpc.unc.edu/a ddhealth). No direct support was received from grant P01-HD31921 for this analysis.

Chapter 3: Responding to Environmental Risk: The Micro-Determinants of Defensive Behaviors

3.1 Introduction

Damages from air pollution take many forms, including worsened health outcomes, increases in premature mortality, and costs related to living with chronic conditions. Government agencies regulate environmental pollutants, but this regulation does not fully protect the public from harm. Individuals compensate by managing their own exposure levels to pollution, though these defensive actions also have associated costs which should be accounted for when crafting optimal environmental policy. This paper explores the micro-determinants of the decision to engage in defensive behaviors when facing environmental degradation.

Defensive behavior can take a variety of forms. Common strategies are spending less time outdoors, where exposure rates are higher, or wearing protective gear such as particulate-filtering masks. Longer term avoidance behaviors can include investing in high quality air filters or purifiers to increase the differential between outdoor and indoor levels of pollution, or moving to an area that is known to have better average air quality. Further, some medications can be taken to offset potential damage from pollution exposure. For water pollution, common avoidance behaviors include using water treatment devices such as filters or consuming bottled water rather than tap water. This paper will use the terms “defensive behavior” and “avoidance behavior” almost interchangeably, but there is a distinction. “Avoidance behavior” will reference actions taken to limit exposure to a pollutant, while “defensive behavior” will apply generally to actions aimed at mitigating damage caused by pollution. In this sense, the former is a subset of the latter.

Individuals become aware of environmental risk in a number of ways. For air pollution, the government creates an air quality index (AQI) that is distributed through local media on poor air quality days. People may seek out further information about general air and water pollution levels where they live through a variety of online resources. Further, experience with certain diseases, such as being asthmatic, may prompt an individual to be more knowledgeable about certain causes and irritants of the disease, including pollution exposure.

In this study, I use data from the National Health and Nutrition Examination Survey (NHANES), which asks respondents about their participation in a range of avoidance behaviors. I estimate how a wide range of personal characteristics influence these behaviors, controlling for environmental factors that may influence these decisions. This paper adds to the current literature in a number of ways. Previous work, discussed in more detail in the next section, has demonstrated the importance of accounting for defensive behavior when measuring the health costs of pollution. Since these behaviors respond to pollution and improve health, not controlling for these behaviors will lead to underestimates of the relationship between ambient pollution levels and health outcomes. Thus, better understanding the extent of avoidance behaviors will inform future studies that wish to estimate these relationships. Further, avoidance behaviors themselves are costly, and should be included when considering the total welfare costs of pollution. This study provides additional evidence that people engage in protective behaviors, and unlike previous studies, it is able to identify which types of behaviors are more prevalent. Further, it provides a more comprehensive set of personal characteristics that may influence avoidance behavior. Understanding which groups are more likely to engage in avoidance behaviors, and their motivations for doing so, will help us better understand a piece of the costs of pollution and which groups bear a disproportionate burden of these costs.

3.2 Background

A health production framework, introduced in Grossman's seminal 1972 paper, offers a precise way to think about the demand for defensive behaviors. In this model, avoidance behaviors are costly actions that improve one's health stock. The cost of avoidance behavior can take many forms. For commodities, like air filters and medication, costs come from the ticket price and associated time costs (visiting a doctor, filling a prescription, etc.). For behaviors like cancelling outdoor activities, the cost depends on the substitutability of indoor activities. For elderly individuals, a walk through the mall may be a close substitute for walking outdoors in a park, so the cost of making that leisure time trade-off may be small. The costs could be larger, however, for cancelling a planned group sports activity or a trip to the zoo. The optimal level of avoidance behavior depends on its price, how it contributes to health, i.e. how efficient it is at reducing exposure or protecting respiratory health, and how it impacts other direct utility inputs such as leisure time.

The costs of air pollution extend beyond just those associated with defensive behaviors. At some point, the marginal benefit of engaging in these behaviors will be surpassed by their costs, and individuals will suffer from the untreated health consequences of pollution exposure. Further, decreased health will impact worker productivity and increase sick days. At the extreme, medical technologies may not exist to fully alleviate damage caused by pollution, leading to premature mortality. A number of papers investigate how defensive expenditures relate to true willingness to pay (WTP) to reduce pollution levels (Courant and Porter 1981, Bockstael and McConnell 1983, Harrington and Portney 1987, Bartik 1988). Under reasonable assumptions on the structure of utility and health production functions, this literature generally finds that defensive expenditures form a lower bound on WTP. Many empirical studies attempt to measure costs associated with pollution. Gerking and Stanley (1986) find a WTP of about $24 to reduce outdoor ozone exposure by 30 percent. Dickie and Gerking (1991) find that individuals living in high ozone areas are WTP $170 annually to prevent spikes in ozone, and they note that this estimate is 2-4 times larger than costs associated with medical expenditures alone. According to Dickie (2005), parents are WTP $100-150 to avoid one illness induced school day lost. Further, Blomquist et al. 2011 estimates the annual value of asthma control to be from $1700-4000. With respect to water quality, Jakus et al. (1997) find that contaminated reservoirs, which prompt fishers to choose new sites or reduce the number of fishing trips taken, result in personal economic losses of $47 per season in East Tennessee. Finally, Graff Zivin et al. (2011) find that costs of avoidance behavior related to contaminated tap water were roughly $60 million in 2005. The scope of these studies varies widely, and these estimates are generally lower bounds on the true costs of pollution. Though other costs of pollution, such as premature mortality increases, are clearly important, this paper focuses on avoidance behavior, which is often ignored when assessing relationships between ambient pollution levels and observed health outcomes.

Recent work in Economics has verified the fact that people engage in avoidance behaviors. Smith et al. (1995) find that people invest in structural modifications to prevent radon exposure. Other studies find changes in fishing habits and purchases of canned fish products after fish consumption advisories (May and Burger 1996, Jakus et al. 1997, Jakus et al. 1998, Shimshack et al. 2007). Graff Zivin et al. (2011) find increases in bottled water purchases as a result of tap water violations. Berger et al. (1987) and Sun et al. (2017) show that purchases of PM2.5 filtering masks and air filters/purifiers increase in response to air pollution warnings. Additional studies have found a response in outdoor activities on high pollution days (Bresnahan et al. 1997, Graff Zivin and Neidell 2009, Janke 2014). Though these papers often document some heterogeneity in responses, my setting is ideal for comprehensively investigating which types of individuals have higher propensities of engaging in avoidance behaviors.

Studies repeatedly find differences in defensive actions between susceptible, or vulnerable, groups and non-susceptible groups (Mullahy 1999, Wu 2003, Shimshack et al. 2007, Graff Zivin and Neidell 2009, Neidell 2009, Graff Zivin et al. 2011). Pollution advisories often target specific groups, such as children, the elderly, or people with pre-existing chronic conditions. One way in which the Environmental Protection Agency (EPA) attempts to inform the public of changes in environmental quality is through the reporting of an Air Quality Index (AQI). The AQI presents an easily interpretable level of air quality ranging from “healthy” to “hazardous”. An AQI is calculated for each of 5 criteria pollutants: ozone (O3), particulate matter (PM2.5 or PM10), carbon monoxide (CO), nitrogen dioxide (NO2), and sulfur dioxide (SO2). Often only the highest these AQIs is reported as the relevant air quality metric. AQIs at certain levels are targeted to sensitive groups. For example, an O3 AQI above 100 is said to be unhealthy for people with lung disease, children and the elderly, and people who are active outdoors. These types of individuals are most susceptible to the negative health effects of air pollution, and therefore have the most to gain by engaging in defensive behaviors to reduce their exposure level.

From a health production perspective, susceptible subpopulations have different health technologies, so their marginal change in health with respect to a change in pollution is relatively higher than for the non-susceptible group. Alternatively, vulnerable groups may be more likely to be aware of changing environmental conditions and its health impacts, and this information differential could result in wider response gaps. The fact that education is sometimes found to be positively correlated with defensive behaviors further supports this information argument (Bresnahan et al. 1997, Wu 2003). Shimshack et al. (2007) find that educated individuals respond more strongly to advisories, but only if they are part of a targeted group. Information may also affect valuations for improvements in environmental quality. Krupnick and Cropper (1992) find that having relatives with a history of respiratory disease increases an individual's WTP to prevent chronic bronchitis. For some types of defensive behaviors, like preventive medical procedures, higher participation rates by unhealthy groups may simply be a function of them coming into more frequent contact with the healthcare system (Mullahy 1999, Wu 2003). For example, unhealthy individuals may be more likely to get a flu shot during a previously planned doctor appointment, decreasing the time costs of receiving the shot. In this case, the decision to receive preventive care may be determined more by convenience than by being a member of a targeted vulnerable group. Further, since WTP for environmental improvement is a function of income, I expect wealthier individuals to generally engage in more avoidance behaviors, especially those for which the time costs are minimal, such as purchasing air filters or water treatment devices. Several studies provide evidence of this income effect. Krupnick and Cropper (1992) find that WTP to avoid chronic bronchitis increases with income. Additional studies find that consumption of protective goods increases with income (Smith et al. 1995, Zivin et al. 2011, Sun et al. 2017).

Time preferences and costs are other likely predictors of avoidance behavior. For behaviors that have a non-negligible time component, the value of time should be taken into consideration. Often wage is used to proxy for this, indicating that high income individuals will have higher costs associated with the defensive behavior. Thus, if high wage individuals have to take time off work make a doctor's appointment, they will be less likely to seek out a preventive procedure. However, if the procedure lowers the probability of getting sick and missing more work time later, higher wages increase the probability of engaging in defensive behaviors. This trade-off highlights the potential importance of time preferences in predicting healthy behaviors. Courtemanche et al. (2014) looks at how time preferences impact obesity, and they find that impatient individuals are more likely to gain weight when food prices drop. In other words, people with high discount rates are willing to trade-off short run benefits with longer run health risks. In another paper, Courtemanche and Carden (2011) find a significant impact of access to cheap food on obesity levels. Time preferences also have implications for how people manage their respiratory health. Two common forms of asthma medication can be categorized as (a) rescue inhalers, which are taken in response to an asthma attack to lessen its severity, and (b) long-acting inhalers, which are prescribed to be taken daily in order to lower the probability of an asthma attack occurring. In the language of Ehrlich and Becker (1972), taking these types of medications can be thought of as self-insurance and self-protection, respectively. If long-acting inhalers are used preventively, then higher discount rates may be associated with lower rates of usage. However, Deschenes et al. (2016) and Williams and Phaneuf (2017) find long-acting inhalers to be more responsive to changes in air pollution, consistent with evidence from the health literature that these types of medications are used in a reactive rather than preventive manner (Stempel et al. 2005).

Some characteristics will impact initial air pollution exposure rates. People employed in industries like construction and agriculture spend more time outdoors and cannot easily substitute away from outdoor activities. People who engage in more active outdoor recreation also face higher rates of exposure. For this group, work constraints do not restrict their ability to substitute away from outdoor activities, but doing so may be more costly than for someone that prefers indoor leisure time activities. Occupation and leisure time choices may also reflect underlying risk preferences. Anderson and Mellor (2008) find risk aversion to be negatively correlated with unhealthy behavior, including smoking and heavy drinking, and health outcomes including obesity.

A simple health production framework assumes individuals have perfect knowledge of their health technology, i.e. they know how much health benefit they will receive from a marginal increase in defensive behavior. In the real world, information is incomplete, leaving open the possibility that individuals engage in too much or too little defensive behavior. Shimshack et al. (2007) find that some groups who are not at risk still change their consumption habits. If these individuals receive no health benefit from reducing consumption, then the welfare benefits of a consumption advisory are diminished by this unintended response. Risk averse individuals may engage in defensive behaviors even if they are uncertain about the associated health consequences. On the other hand, some groups may fail to respond to an advisory because of overconfidence in their own knowledge and experience or mistrust of the information source (May and Burger 1996, Busch 2009). Other factors, such as anxiety about knowing one's true health risks, may lower the probability of preventive procedures including cancer screenings (Wu 2003).

Other behaviors are more altruistic in nature, aimed at generating public environmental benefits rather than private health benefits. Advisories from the 'Spare the Air" program in the San Francisco Bay area encourage increased use of public transportation and ride-sharing on high ozone days. Cutter and Neidell (2009) find that total traffic is reduced by 2.5-3.5% on days when advisories are issued. Although the individual health gains from this type of behavior is minimal, people may derive "warm glow" utility from helping others. Still, Owen et al. (2012)

find evidence that people tend to overestimate the public environmental benefit from their actions. Interestingly, if people were better informed about the marginal environmental benefits from behaviors such as recycling and energy conservation, they may actually be less likely to engage in them.

Although decisions to engage in defensive behaviors are complex and depend on a multitude of factors, several trends emerge from this review of the literature. First, vulnerable or targeted groups, such as children, the elderly, or those with preexisting conditions, are more likely to engage in defensive behaviors. Second, more educated and/or more informed individuals are more prone to take these actions. Finally, males, or people with higher risk tolerance, are less likely to engage in such behaviors. In this paper, I use survey responses about a variety of defensive behaviors to comprehensively study their determining factors and motivations, and I investigate how these factors compare across different types of defensive actions.

3.3 Data

3.3.1 Publicly Available Data

Variables for the main analysis come from the 2007-2008 and 2009-2010 waves of the National Health and Nutrition Examination Survey (NHANES). NHANES uses a nationally representative sample of individuals and contains health and demographic information, some of which is summarized in Table 1. My baseline estimates include information on 11,337 unique individuals. Not all variables are asked to all participants in both survey periods, so the addition of some covariates significantly reduces the sample size. I will treat such regressions as robustness checks and focus the analysis on the larger sample. Demographic variables include age, racial minority status, sex, and education. Education level is grouped into one of five categories: (1) less than 9th grade, (2) some high school, (3) high school graduate or GED equivalent, (4) some college, or (5) college graduate. Education may also serve as a proxy for being informed about air pollution or how effectively one can protect against it.

Health variables include indicators of insurance, respiratory health, blood lead levels, and obesity. An individual is considered asthmatic if they report having an asthma event in the past year. A second measure of respiratory health, *spirometry*, comes from the examination portion of the NHANES. *Spirometry* is constructed as the ratio of forced expiratory volume (FEV) in the first second of the spirometry test to forced vital capacity (FVC), a measure of lung capacity. Lower values of *spirometry* indicate lower expiratory air flow and worse respiratory health. This measure is frequently used to diagnose asthma and chronic obstructive pulmonary disease (COPD). Blood lead levels, measured in micrograms per deciliter (ug/dL) come from the laboratory section of the NHANES. Major sources of lead contamination are lead paint and lead pipes, both of which are more common in older houses. Housing age is defined on a 6 point scale, with (1) being the youngest (built after 1990) and (6) being the oldest (built before 1940). *Well water* is an indicator variable that equals one if the source of tap water is a private or public well, which may impact the amount of lead found in tap water.

Leisure time activities are summarized in the *active hours* and *inactive hours* variables. These indicate the average number of hours spent on active (biking, jogging, playing sports, etc.) and inactive (playing video games, watching television, etc.) in a typical week. For active recreation, participants are asked how many days per week and how many minutes each day they spend on these activities. For inactive recreation, they are asked how many minutes are spent on the activity in a typical day. I use these responses to create weekly measures, in hours, to compare active and inactive activity levels. Because of the level of detail requested, recall bias may be of particular concern for these variables, but they should still be a good signal for recent activity levels. *Healthy diet* records respondents' perceptions of the healthiness of their diet, from (1) excellent to (5) poor. *Smoker* equals one if the participant has consumes at least 100 cigarettes in his lifetime, which also yields evidence of health preferences and tolerance for risk.

Economic variables include *hours worked*, *family income*, *savings*, and an indicator of the physical intensity of work. Annual family income is binned into twelve categories, with higher numbers indicating higher incomes. These categories range from (1) less than $5,000 to (12) greater than $100,000. Family savings are divided into 7 categories, ranging from (1) less than $500 to (7) greater than $5,000. The variable *Savings/Income* roughly describes how much savings a family has relative to monthly earnings. This savings ratio variable may also serve as a proxy for risk/time preferences. More risk averse people may save more in case of emergency events, while people with higher discount rates will tend to save less. *Vigorous Work* equals one if a person's job requires vigorous physical activity, which might also influence active leisure choices outside of work.

Several additional variables reveal if the participant engages in risky behaviors. The variable *condom* describes how frequently a condom is used during intercourse, (1) being "never" and (5) indicating "always". Getting a Hepatitis A vaccine is a preventive health behavior which demonstrates concern about potential negative health outcomes. The last two variables in Table 1 indicate whether or not the individuals have used various illegal drugs recreationally. For some sensitive questions, the eligible sample is restricted by age. For instance, condom use is only asked to the 20-59 age group. Further, some variables have a high number of missing values even among the eligible sample. This could be due to refusal to answer or not knowing/having the answer ready. Results that include these additional variables come from estimation on smaller sample sizes and should be interpreted with caution in case the missing values are not randomly distributed.

Although other years are available, I restrict my sample to the 2007-2010 NHANES because these waves of the survey ask about a range of defensive behaviors. In particular, the survey asks, “During the past 12 months, when you thought or were informed air quality was bad, did you do anything differently?” Follow up questions ask the respondents *how* they changed their behavior, including: wearing a mask, spending less time outdoors, avoiding roads that have heavy traffic, doing less strenuous activities, taking medication, closing windows in your house, driving your car less, canceling outdoor activities, exercising indoors instead of outdoors, using buses, trains, or subways, changing air filter/air cleaner, and other. I have grouped the types of avoidance behavior into four categories. *Change* is just a measure of if the participant made any changes in response to being informed about poor air quality. *Expenditure* includes the set of responses that directly require spending money: buying a mask, medication, or new air filter. *Outdoors* indicates some change in outdoor activities: spending less time outdoors, cancelling outdoor activities, and exercising indoors instead of outdoors. *Altruism* indicates a response that is geared towards lowering one’s own contribution to the pollution problem: driving your car less and switching to public transportation. Finally, another question asks participants if they use some sort of water treatment device for their tap water at home. Water treatment devices can include Brita filters, charcoal filters, water softeners, aerators, and reverse osmosis technology. This serves as a defense against water, rather than air, pollution, and allows me to compare defensive behaviors across different types of environmental risk. Table 2 lists the number of individuals that engage in each type of behavior.

3.3.2 Restricted Access Data

In the public-use NHANES data files, I am not able to identify an individual's residential location. Other variables not included in NHANES may be important for explaining the microeconomic determinants of defensive behaviors. For this reason, I use restricted date-of-interview and geographic data accessed through a Federal Statistical Research Data Center to merge additional information on air quality, weather, and unemployment at the county level. Pollution data comes from the U.S. EPA and measures the severity of air pollution in a county over the past year. The EPA collects data on air pollutants that are known to impact human health, including ozone, particulate matter, sulfur dioxide, nitrogen dioxide, and carbon monoxide. A daily index value is created that indicates if the measured level of the pollutant is good, unhealthy for sensitive groups, unhealthy, or very unhealthy. The highest daily index among all the pollutants measured in a county becomes the air quality index (AQI) ascribed to the county for that particular day. I count the number of days that fall into each of the AQI health categories over the year leading up to an individual's interview date, and these counts are used as the measure of air pollution severity associated with a person's county of residence. Metropolitan Statistical Areas with populations over 350,000 are generally required to report daily AQI measures to the general public through local media sources (EPA 2006). Since this information is distributed at a city-wide basis, there is little benefit of obtaining this variable at a finer degree of geographic detail.

Temperature, precipitation, and humidity data come from the North American Land Data

Assimilation System (NLDAS) accessed through the CDC's WONDER online databases. These variables are relevant because they affect the relative value of outdoor recreation activities. For example, on very hot days people will not be spending much time outside regardless of the air quality on that day. Since the Physical Activity Questionnaire includes questions that reference short term (a "typical" day or week) and longer term (past 12 months) activities, both annual and monthly weather measures will be utilized.

Annual county level unemployment rates for 2007-2010 will come from the Bureau of Labor Statistics. Similar to Mullahy (1999), I use unemployment rate as an instrumental variable for labor market characteristics (hours worked, employment status) to control for unobserved characteristics that are both associated with the labor market measure and the decision to engage in avoidance behavior.

Table 3 presents some summary statistics for these variables. Due to the sensitive nature of the data, minimum and maximum values are not included. Many counties do not have air quality monitoring, so my restricted sample size is reduced to 7,819 individuals. Because of this significant data reduction, I primarily use these additional data to check the robustness of my results and ensure that important unobservables are not influencing estimated relationships.

3.4 Empirical Strategy

I estimate how individual characteristics influence the decision to engage in defensive behaviors. Baseline regressions are of the form

$$Y_i = \beta_1 leisure_i + \beta_2 X_i + \beta_3 environment_i + \epsilon_i$$

where *leisure* is a measure of active leisure time choices, X are other individual level variables including health and insurance status, and *environment* includes air pollution and weather controls for individual *i*'s residential location. Y is a binary indicator for having engaged in at least one avoidance behavior type in the past year, or it can be defined for a given behavior, such as spending less time outdoors ($Y_i = 1$ if individual *i* spent less time outdoors because of an air quality alert, 0 otherwise).

3.4.1 Instrumental Variables

The *leisure* variable indicates the amount of time spent on outdoor leisure activities during a typical week. One concern is that this measure will be correlated with unobserved health preferences that co-determine both the amount of time spent on healthy leisure choices as well as the decision to engage in avoidance behavior. This endogeneity may bias the coefficient estimate of β_1 because individuals with high values for health will both spend more time on outdoor leisure activities and be more likely to take defensive actions against air pollution exposure. A positive estimate of β_1 may therefore be misinterpreted as high outdoor leisure types being more likely to engage in avoidance behavior, which is counterintuitive if it is more costly for these individuals to substitute away from outdoor activities.

To address this empirically, I implement an instrumental variables estimator. Valid instruments are (a) strongly correlated with the amount of time spent on outdoor leisure in the past week, but (b) unrelated to having engaged in avoidance behavior in the past year. Instruments come from monthly weather variation. I rely on the timing of the NHANES questions to justify the validity of these instruments. Avoidance behavior questions are specific to the past year, but recreation questions refer a typical week, so responses will likely refer to activity levels in the past few weeks. Monthly weather deviations from annual averages will influence recent recreation decisions but not avoidance behavior decisions. For example, if an individual is surveyed in an unusually rainy month, they may have spent relatively little time engaged in outdoor activities. Avoidance behavior decisions will depend on precipitation levels over the past 12 months, so conditional on controlling for these annual averages, the monthly measures will be valid instruments.

I am also interested in the impact of labor market variables on avoidance behavior choices. Workers may engage in more defensive behaviors to reduce the probability of costly illnesses that require them to take time off work. Alternatively, since their time is more constrained or valuable, workers may be less likely to engage in behaviors that require them to alter their leisure time choices. However, the decision to work may be related to unobserved leisure, health, and risk preferences. For this reason, I use county level unemployment rate to instrument for hours worked. Unemployment rates will influence the probability that an individual has a job, but should not be directly related to avoidance behaviors.

3.5 Results

3.5.1 Main Results

Since protecting health is a central reason for engaging in defensive behaviors, I first estimate reduced form relationships between avoidance behaviors and health outcomes in the data. Columns 1 and 2 of Table 4 present the impact of avoidance behavior, smoking, and physical activity on respiratory outcomes. *Asthma Year* indicates having had an asthma attack in the past year, and *Spirometry* records the outcome of a test of lung functioning, where lower values correspond to lower respiratory health. The results are consistent in for both outcomes: *change*, an indicator for avoidance behavior, and *smoker* are associated with worse respiratory health, while physical activity has the opposite effect. Though avoidance behaviors are aimed at improving health, the coefficient on *change* likely reflects the fact that unhealthy individuals are more likely to engage in these behaviors. The coefficient on *active hours* must be interpreted in a similar way; healthier people may engage in more physical activity, physical activity may improve respiratory health, or the coefficient reflects a combination of these two effects.

Column 3 investigates factors that contribute to blood lead levels. *Water treatment*, a defensive behavior, significantly decreases lead levels, while older houses increase levels. An indicator for getting tap water from a well has a positive but insignificant effect. In contrast to the respiratory outcomes, a selection based on health is unlikely for blood lead levels. An important distinction between these two health outcomes is their observability. People with asthma know they have respiratory problem, while most people do not observe their level of lead exposure. Rather, water treatment is done because of knowledge of potential health risks and for non-health reasons. For example, one common water treatment, water softening, is aimed at reducing limescale build-up. The coefficient on *water treatment* could represent a large benefit from the subset of treatment options that do decrease lead levels, or it could be driven by an unobserved factor related to both lead levels and water treatment decisions. The positive effect of *housing age* is sensible, since older houses are more likely to contain lead pipes or lead-based paint.

Next, I examine the motivating factors behind defensive behaviors. OLS estimates for various defensive behavior types are presented in Table 5[8]. The dependent variables in each column come from the binary indicators listed in Table 2. Many of these initial results reinforce previous findings in this literature. For responses to air pollution, in columns 1-4, having an asthma attack in the past year has the strongest impact on behavior change. Individuals with recent asthma experience are about 13 percent more likely to change their behavior in response to an air quality warning. Unsurprisingly, this is not true for the *water treatment* category. *Education* has the most consistent effect across each category, with higher levels of *education* increasing the probability of engaging in defensive behaviors. The determinants of *expenditures* are quite different from the other air pollution behaviors. *Asthma year* still has a significant effect, but it is much smaller than in the *outdoors* category. Interestingly, *expenditures* is the only air pollution category in which *income* is statistically significant. This is consistent with previous literature that find positive associations between wealth and defensive behaviors, but suggests that other types of avoidance behaviors are not as sensitive to income. Still, the magnitude of this effect is small; rising into a higher income bracket only increases probability of *expenditure* by 0.16 percent. *Income* is also significant for *water treatment*, but the magnitude is much larger for this category: an increase in income bracket increases likelihood of *water treatment* by over 2 percent.

Racial minorities are found to engage in more air pollution defensive behaviors, but the estimated effects are only marginally significant. Contrastingly, minorities are 12 percent less likely to have some form of water treatment. Obesity is also only found to have a strong

[8] Disaggregated defensive behavior category results are included in the Appendix.

relationship in the water treatment category, with obese individuals being 3.7 percent less likely to engage in the behavior.

Excluding *expenditures*, the determinants of the other air pollution defensive behaviors are qualitatively similar. The impact of *insurance* and *active hours* is comparable between the *water treatment* and non-expenditure air pollution categories. For these behavior types, having insurance and a higher education level increases the likelihood of participating. These variables may be serving as proxies for risk or health preferences. For example, if market insurance induces moral hazard, one might instead expect a negative coefficient on insurance status. However, unobserved risk aversion or health attitudes may be related to the decision to buy health insurance, and their exclusion from the regression may induce the positive *insurance* coefficient.

The impacts of *active hours* and *hours worked* are strongest in the *outdoors* category. Individuals with longer work hours are more time constrained and less flexible in changing or delaying planned activities. The expected coefficient sign on *active hours* is ambiguous. Spending more time outdoors results in higher air pollution exposure, so those that frequently engage in active recreation may be more likely to engage in avoidance behavior to avoid the additional risk. Alternatively, preferences for active outdoor recreation may make substituting away from outdoor activities more costly, resulting in a negative coefficient on *active hours*. As in the case of insurance, correlations with unobserved health preferences may result in the positive coefficient found in column 2.

Air quality levels are an important determinant of engaging in air pollution related avoidance behaviors. If an individual lives in an area that never experiences air pollution spikes and no air quality alerts are issued, then she will be less likely to take defensive action. Further, weather characteristics are an important determinant of recreation choices and may be correlated with air quality measures. For these reasons, controlling for pollution and weather characteristics may be important for fully understanding avoidance behavior decisions. Using a Federal Statistical Research Data Center, I merge air quality, temperature, humidity, and precipitation data with the NHANES survey data at the county level. Many counties, particularly in rural areas, do not have air quality monitoring, so including the additional controls decreases the sample size to 7,819 individuals. For this reason, I treat the models with these controls as robustness checks. For consistency in comparisons, column 1 in Table 6 replicates column 1 from Table 5, but on the smaller sample. Columns 2 and 3 of Table 6 include the additional pollution and weather controls. A comparison of columns 1 and 2, which both use *change* as the dependent variable, shows that the addition of these controls has little impact on the coefficient estimates. The controls themselves generally have insignificant coefficients. The impact of high pollution days is positive, but insignificant, in column 2. *Precipitation* consistently has a negative effect but again is insignificant at the 5 percent level. The ex-ante expected signs on the weather variables are ambiguous. Worse weather may lower the cost of cancelling outdoor activities, therefore increasing the likelihood of avoidance behavior. However, people in areas with harsh climates may spend less time on outdoor recreation in general, decreasing the need to engage in avoidance behaviors. The dependent variable in column 3 indicates whether or not the individual is made aware of a poor air quality day in the past year. Surprisingly, the covariates, including number of high pollution days, asthma status, and education, are not predictive of being informed of poor air quality. This raises the concern that avoidance behaviors are not responding to actual risk levels, resulting in too little or even too much defensive behavior.

3.5.2 The Role of Unobserved Preferences

Unobserved health attitudes or risk preferences may confound some of the main result estimates. As previously mentioned, *active hours* variable may be serving as a proxy for health attitudes. To separate the impact of active leisure time choices from that of unobserved preferences for health, I use an instrumental variables strategy. To operationalize this strategy, I find variables that are correlated with active leisure choices but unrelated to engaging in avoidance behaviors. Access to the FSRDC allows me to include instruments that exploiting the timing of the interview questions. Air pollution avoidance behavior questions asked in the NHANES are in reference to the past year, while recreation questions refer to a “typical” week, which will likely reflect recent activity levels. After controlling for annual weather variables, monthly weather variables will be correlated with recreation responses but not related to annual avoidance behavior choices. For example, if the interview happens during an especially cold or rainy month, active leisure hours reported for an individual may be lower than if that same individual were interviewed during a dry summer month. However, it is unlikely that this short term weather variation will impact defensive behavior choices over the previous year. Table 7 uses monthly precipitation and humidity to instrument for active leisure hours for engaging in an air pollution avoidance behavior. In each column of Table 7, the coefficient on *active hours* is now negative, but insignificant. The instruments in each specification are very weak, so no definitive conclusions can be made from this analysis.

When assessing the impact of labor market variables on avoidance behavior, one must consider that the decision to work is not random. Employment choices may be related to defensive behaviors in unobserved ways. For this reason, I instrument for *hours worked* using county level unemployment rate. The supply of jobs near one’s residence will influence her probability of being employed, but should be unrelated to individual level unobserved determinants of employment decisions. Table 8 presents the results of this exercise. The estimated impact of *insurance*, which is often closely tied to employment status, is now strongly significantly in all categories other than *expenditure*; there is no evidence of a moral hazard effect in this context. The sign on *hours worked* is positive and weakly significant only for the expenditure category. This suggests that the main influence of labor market decisions on avoidance behavior works through an income channel rather than through leisure time trade-offs, though *income* itself does not have a significant coefficient.

An alternative strategy to account for time and risk preferences is to use additional proxy variables from the NHANES dataset. Many candidate proxy variables have missing values for a subset of participants, so including them results in a reduced sample size. Each model in Table 9 uses *change* as the binary dependent variable. The models specifications are the same as in the first column of Table 5, with additional control variables included. Column 1 includes a measure of savings. Families with higher savings may be more risk averse or have lower discount rates. The negative sign on this variable is unexpected, since engaging in avoidance behavior is a way to reduce the risk of future health problems.

The specification in column 2 of Table 9 includes additional controls that may indicate risk preferences, including an indicator for having a healthy diet, using various illegal drugs, receiving a Hepatitis A vaccination, frequency of condom use, and smoker status. The healthy diet indicator provides the only significant effect. As expected, the coefficient is negative, which proxy for health preferences that would increase the likelihood of engaging in avoidance behavior. An alternative explanation in relies on diet and outdoor recreation being substitute for improving health. In the data, these variables are negatively correlated, perhaps implying that

Individuals with healthy diets feel less need to engage in active recreation for weight maintenance. Adding these additional controls greatly reduces the sample size, so column 3 includes the controls while excluding the savings measure, which adds over 3,000 observations, and the results do not change substantially.

3.6 Conclusion

This study provides a comprehensive evaluation of the micro-determinants of avoidance behavior, controlling for weather and pollution levels and accounting for unobserved preferences. The analysis in this paper highlights the importance of considering unobserved risk, health, and time preferences when interpreting relationships between person-level characteristics and the decision to engage in defensive behaviors. The results of instrumental variables estimates suggest that hours worked are positively related to some types of avoidance behaviors. This illustrates the importance of accounting for endogenous covariates. I find no conclusive results when investigating a similar relationship between leisure time choices and avoidance behavior.

Many of my results are consistent with previous findings. For instance, I find that higher education, working fewer hours, having insurance, and asthma status are positively associated with avoidance behavior. A novelty in my approach comes from being able to distinguish between types of defensive behaviors. Looking at a multitude of defensive behavior types helps identify which ones are more prevalent and therefore contribute more to pollution welfare costs. Estimated differences among types are often intuitive; for example, asthma status does not have a clear impact on water treatment choices, but is a very strong predictor of air pollution avoidance behavior. *Income* is only found to be related to defensive behaviors that require monetary expenditures, such as buying an air or water filter. Because of this, income inequality does not appear to exacerbate pollution exposure inequities for many common avoidance behaviors, such as cancelling outdoor activities.

Defensive behaviors are costly, and this paper provides insights into which groups are bearing more of these costs. It also highlights the essential tradeoffs and motivations related to engaging in these behaviors. Individuals who value healthy lifestyles must weigh the benefits of outdoor exercise against the costs of higher pollution exposure. Elevated pollution levels over the long run may discourage active leisure choices, leading to additional health costs. In addition, fully understanding behavioral reactions to pollution helps inform future studies that attempt to measure dose-response relationships.

3.7 References

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3.8 Tables

Table 1: Individual Level Health, Demographic, and Economic Summary Statistics

Variables	Obs	Mean	Std. Dev.	Min	Max
Demographic					
Age	11337	46.729	19.468	16	80
Non-white	11337	0.527	0.499	0	1
Male	11337	0.494	0.500	0	1
Education	11337	3.170	1.278	0	5
Housing					
Age of Home	9279	3.085	1.768	1	6
Well Water	11337	0.119	0.324	0	1
Health					
Insurance	11337	0.761	0.427	0	1
Asthma Year	11337	0.040	0.195	0	1
Spirometry	8991	0.788	0.086	0.253	1
Blood Lead (ug/dL)	10752	1.679	1.538	0.18	33.1
Obese	11337	0.416	0.493	0	1
Active Hours	11337	2.462	4.762	0	69
Inactive hours	11310	37.809	23.062	0	140
Healthy Diet	11333	2.965	0.999	1	5
Smoker	10244	0.473	0.499	0	1
Economic					
Hours Worked	11337	20.605	22.596	0	130
Family Income	11337	6.949	3.287	1	12
Savings/Income	4974	0.581	0.725	0.083	7
Vigorous Work	11337	0.184	0.388	0	1
Risky Behaviors					
Condom	4793	3.383	1.731	1	5
Hepatitis A Vaccine	11337	0.203	0.402	0	1
Cocaine, Meth, or Heroine	7632	0.172	0.378	0	1
Marijuana	6208	0.548	0.498	0	1

Summary statistics conditional on observations not missing values for the variables included in baseline models: *Age, Non-white, Male, Education, Insurance, Asthma Year, Obese, Active Hours, Hours Worked, Family Income,* and *Vigorous Work*.

Table 2: Prevalence of Defensive Behaviors

Defensive Behavior	Description	Count	Percent
Air pollution			
Change	Changed behavior in anyway	1,344	11.85
Outdoors		1,053	9.29
Time Outdoors	Spent less time outdoors	948	8.36
Strenuous	Did less strenuous activities	161	1.42
Cancelled	Cancelled outdoor activities	197	1.74
Exercise	Exercised indoors instead of outdoors	112	0.99
Expenditure		217	1.91
Filter	Purchases and air filter/purifier	52	0.46
Medication	Bought medication	63	0.56
Mask	Wore a mask	114	1.01
Altruism		154	1.36
Traffic	Avoided roads with heavy traffic	85	0.75
Car	Drove car less	140	1.23
Bus	Used bus, train, or subway	23	0.20
Windows	Closed windows	327	2.88
Other		83	0.73
Water Pollution			
Water Treatment	Any household water treatment device	2,903	25.65

Counts conditional on having non-missing values for controls used in baseline estimates. Change indicates having engaged in at least one of the air pollution behaviors.

Table 3: Restricted-Access Variable Summary Statistics

Variable	Obs	Mean	Std. Dev.
Unhealthy Sensitive Days	7,819	16.71	26.24
Unhealthy Days	7,819	2.40	6.68
Very Unhealthy Days	7,819	0.42	1.25
90th Percentile Temp	7,819	85.92	6.43
10th Percentile Temp	7,819	45.59	13.07
Annual Humidity	7,819	89.42	3.92
Annual Precipitation	7,819	2.52	1.06
Monthly Humidity	7,819	84.71	4.86
Monthly Precipitation	7,819	2.58	1.77
Unemployment Rate	7,819	7.62	2.57

Counts conditional on having non-missing values for controls used in baseline estimates. Variables merged using Federal Statistical Research Data Center. Minimums and Maximums excluded due to sensitive nature of the data.

Table 4: Defensive Behaviors and Health Outcomes

	(1) Asthma Year	(2) Spirometry	(3) Lead
Change	0.0494***	-0.00861***	
	(0.00772)	(0.00313)	
Smoker	0.0184***	-0.0368***	
	(0.00359)	(0.00229)	
Active Hours	-0.000974*	0.000671***	
	(0.000535)	(0.000192)	
Water Treatment			-0.105**
			(0.0406)
Well Water			0.0711
			(0.0533)
Housing Age			0.0785***
			(0.0139)
Constant	0.0232***	0.796***	1.267***
	(0.00283)	(0.00254)	(0.0584)
Observations	12,131	9,033	13,213
R-squared	0.011	0.056	0.013

Coefficients come from OLS estimates and account for NHANES' complex survey design (primary sampling units, strata, and probability weights). Standard errors in parentheses *** p<0.01, ** p<0.05, * p<0.1

Table 5: Impact of Individual-Level Characteristics on Defensive Behaviors

	(1) Change	(2) Outdoors	(3) Expenditure	(4) Altruism	(5) Water Treatment
Active Hours	0.00158	0.00150*	-0.000422	0.000415	0.00113
	(0.000993)	(0.000839)	(0.000320)	(0.000251)	(0.00124)
Asthma Year	0.131***	0.128***	0.0497***	0.0236***	-0.000604
	(0.0180)	(0.0190)	(0.0143)	(0.00772)	(0.0205)
Age	0.00135***	0.00139***	0.000158	6.31e-05	0.000433
	(0.000240)	(0.000198)	(0.000100)	(5.96e-05)	(0.000540)
Non-White	0.0221*	0.0189*	0.00235	0.00471*	-0.120***
	(0.0113)	(0.0112)	(0.00414)	(0.00277)	(0.0247)
Male	-0.0436***	-0.0507***	0.00259	-0.00892***	-0.00128
	(0.00756)	(0.00610)	(0.00292)	(0.00265)	(0.00610)
Education	0.0195***	0.0145***	0.00215	0.00609***	0.0282***
	(0.00441)	(0.00416)	(0.00137)	(0.000933)	(0.00594)
Insurance	0.0186*	0.0154*	-0.00460	0.00715**	0.0488***
	(0.00972)	(0.00857)	(0.00620)	(0.00279)	(0.0124)
Obese	0.0102	0.00718	-0.00248	-1.03e-05	-0.0374***
	(0.00673)	(0.00579)	(0.00379)	(0.00227)	(0.0132)
Vigorous Work	0.0168	0.0136	0.00402	0.00619**	0.0144
	(0.0114)	(0.00892)	(0.00515)	(0.00273)	(0.0182)
Hours Work	-0.000409**	-0.000436**	3.57e-05	-2.52e-05	-0.000361
	(0.000180)	(0.000161)	(7.79e-05)	(5.62e-05)	(0.000300)
Income	0.00104	0.00119	0.00156**	-0.000570	0.0222***
	(0.00165)	(0.00132)	(0.000757)	(0.000388)	(0.00336)
Constant	-0.0263	-0.0288	-0.00641	-0.0116**	0.0436
	(0.0222)	(0.0194)	(0.00932)	(0.00558)	(0.0382)
Observations	11,337	11,337	11,337	11,337	12,382
R-squared	0.025	0.031	0.007	0.008	0.076

Dependent variables are binary indicators of engaging in each category of defensive behavior. Coefficients come from OLS estimates and account for NHANES' complex survey design (primary sampling units, strata, and probability weights). Standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1

Table 6: Defensive Behavior Determinants- Weather and Pollution Controls

	(1) Change	(2) Change	(3) Informed
Active Hours	0.00203	0.00198	0.000136
	(1.4)	(1.4)	(0.17)
Asthma year	0.141***	0.143***	0.0111
	(5.67)	(5.83)	(0.8)
Age	0.00172***	0.00168***	-0.00011
	(6.61)	(7.07)	(-0.64)
Non-White	0.0227	-0.0086	(0.0017
	(1.5)	(-0.51)	(-0.18)
Male	-0.0489***	-0.0487***	-0.00412
	(-5.46)	(-5.27)	(-0.97)
Education	0.0213**	0.0204***	0.000212
	(3.49)	(4.01)	(0.06)
Insurance	0.0213	0.0284*	-0.00589
	(1.75)	(2.45)	(-0.66)
Obese	0.0138	0.0180*	0.00655
	(1.65)	(2.16)	(1.03)
Vigorous Work	0.0312*	0.0344*	0.0152
	(2.3)	(2.69)	(1.8)
Hours Worked	-0.00045	-0.00034	-3.2E-05
	(-2.00)	(-1.38)	(-0.20)
Income	0.00117	-0.00023	0.000657
	(0.68)	(-0.14)	(0.57)
Unhealthy Sensitive Days		0.0011	-9.7E-05
		(1.58)	(-0.35)
90th Pctl Temp		-0.00188	-6.1E-05
		(-0.44)	(-0.02)
10th Pctl Temp		0.00212	1.43E-05
		(1.71)	(0.02)
Humidity		-0.00167	-0.00028
		(-0.41)	(-0.07)
Precipitation		-0.0282	-0.00553
		(-1.61)	(-0.67)
Constant	-0.045	0.248	0.999***
	(-1.42)	(0.87)	(6.72)
Observations	7819	7819	7819

Restricted-access sample. Dependent variables in columns 1 and 2 are binary indicators of engaging in each category of defensive behavior in response to an air quality warning. Dependent variable in column 3 is an indicator of having been informed of a poor air quality day. Coefficients come from OLS estimates and account for NHANES' complex survey design (primary sampling units, strata, and probability weights). T statistics in parentheses. *** p<0.001, ** p<0.01, * p<0.05

Table 7: Determinants of Defensive Behavior-Instrumental Variable Estimates

	(1) Change	(2) Change	(3) Change
Active Hours	-0.0827	-0.0293	-0.235
	(-1.24)	(-0.40)	(-0.60)
Asthma Year	0.157***	0.148***	0.182
	(4.15)	(5.77)	(1.58)
Age	-0.00295	-3.2E-05	-0.0113
	(-0.78)	(-0.01)	(-0.52)
Non-White	-0.023	-0.0139	-0.0489
	(-1.10)	(-0.76)	(-0.64)
Male	0.0515	-0.0117	0.232
	(0.64)	(-0.13)	(0.5)
Education	0.0542	0.0329	0.115
	(1.96)	(1.14)	(0.73)
Insurance	0.0743	0.0453	0.157
	(1.9)	(1.21)	(0.65)
Obese	-0.0294	0.000492	-0.115
	(-0.81)	(0.01)	(-0.50)
Vigorous Work	0.0828	0.0523	0.17
	(1.77)	(1.13)	(0.72)
Hours Worked	-0.00235	-0.00108	-0.00598
	(-1.39)	(-0.60)	(-0.63)
Income	0.00539	0.00185	0.0155
	(1.04)	(0.36)	(0.59)
Unhealthy Sensitive	0.00129	0.00117	0.00162
Days	(1.5)	(1.47)	(1.09)
90^{th} Pctl Temp	-0.00296	-0.00228	-0.00491
	(-0.59)	(-0.53)	(-0.51)
10^{th} Pctl Temp	0.000788	0.00163	-0.00161
	(0.42)	(0.8)	(-0.26)
Humidity	-0.00162	-0.00165	-0.00153
	(-0.30)	(-0.37)	(-0.16)
Precipitation	-0.0411	-0.033	-0.0644
	(-1.78)	(-1.69)	(-0.99)
Constant	0.674	0.405	1.442
	(1.49)	(1.03)	(0.73)
Observations	7819	7819	7819

F-stat	2.29	2.85	1.31
Over ID p-value	0.006		

Dependent variables is a binary indicator of engaging in any category of defensive behavior. Coefficients come from IV estimates, where monthly weather variables instrument for active hours, and account for NHANES' complex survey design (primary sampling units, strata, and probability weights).In column 1, monthly humidity and precipitation are used as instruments, in columns 2 and 3, precipitation alone and humidity alone, respectively, are used as instruments. Standard errors in parentheses: *** p<0.001, ** p<0.01, * p<0.05

Table 8: Labor Market Instrumental Variables Estimates with Weather and Pollution Controls

	(1) Change IV	(2) Outdoors IV	(3) Expenditure IV	(4) Altruism IV
Active Hours	0.00185	0.000943	0.000888	0.000085
	(0.61)	(0.39)	(1.07)	(0.14)
Asthma Year	0.143***	0.145***	0.0626**	0.0231**
	(5.02)	(5.28)	(3.14)	(2.56)
Age	0.00161	0.000982	0.000857**	8.32E-05
	(1.16)	(0.82)	(2.36)	(0.33)
Non-White	-0.00824	-0.00506	-0.00189	-0.00163
	(-0.38)	(-0.26)	(-0.29)	(-0.43)
Male	-0.0467	-0.0361	-0.0165	-0.00813
	(-1.09)	(-0.98)	(-1.56)	(-1.07)
Education	0.0213	0.0232	-0.00546	0.00605**
	(1.17)	(1.45)	(-1.17)	(2.11)
Insurance	0.0282**	0.0245**	-0.00386	0.00883***
	(2.38)	(2.66)	(-0.47)	(3.43)
Obese	0.0183*	0.0172*	-0.00729	0.0016
	(1.89)	(1.83)	(-1.40)	(0.48)
Vigorous Work	0.0363	0.0484	-0.0121	0.00831
	(0.91)	(1.3)	(-1.16)	(1.13)
Income	0.000156	0.00361	-0.00246	-0.00037
	(0.02)	(0.47)	(-1.21)	(-0.30)
Hours Worked	-0.0006	-0.00289	0.00259*	-0.00017
	(-0.11)	(-0.59)	(1.99)	(-0.19)
Unhealthy Sensitive Days	0.0011	0.00113	0.000355***	-0.00014
	(1.61)	(1.64)	(2.82)	(-1.37)
90th Pctl Temp	-0.00188	-0.00227	-0.00167*	0.00036

	(-0.43)	(-0.53)	(-1.94)	(0.44)
10^{th} Pctl Temp	0.0021	0.00182	0.0000908	0.000792***
	(1.39)	(1.24)	(0.37)	(3.06)
Humidity	-0.00163	0.000706	0.00207*	-0.00224**
	(-0.37)	(0.19)	(1.89)	(-2.50)
Precipitation	-0.0281	-0.0188	-0.00825**	-0.00578
	(-1.55)	(-1.06)	(-2.66)	(-1.61)
Constant	0.246	0.0795	-0.0609	0.142**
	(0.8)	(0.26)	(-1.30)	(2.22)
Observations	7819	7819	7819	7819

Restricted-access sample. Dependent variables are binary indicators of engaging in each category of defensive behavior. Coefficients come from IV estimates, where unemployment rate instruments for hours worked, and account for NHANES' complex survey design (primary sampling units, strata, and probability weights). The F-stat for instruments is 43.99. T statistics in parentheses: *** p<0.01, ** p<0.05, * p<0.1

Table 9: Additional Health, Time, and Risk Preference Controls

	(1)	(2)	(3)
Active Hours	0.00294*	0.00393**	0.00190
	(0.00153)	(0.00185)	(0.00177)
Asthma Year	0.168***	0.129***	0.139***
	(0.0323)	(0.0468)	(0.0360)
Age	0.00120***	0.00159**	0.00218***
	(0.000300)	(0.000729)	(0.000493)
Non-White	0.0160	0.0232	0.0360**
	(0.0150)	(0.0192)	(0.0136)
Male	-0.0341***	-0.0177	-0.0315***
	(0.00836)	(0.0184)	(0.0115)
Education	0.0175***	0.0165*	0.0129
	(0.00487)	(0.00935)	(0.00809)
Insurance	0.0330***	0.0408**	0.0181
	(0.0101)	(0.0163)	(0.0147)
Obese	0.00519	0.00900	0.00951
	(0.0104)	(0.0137)	(0.0103)
Vigorous Work	0.0534***	0.0353**	0.0160
	(0.0151)	(0.0166)	(0.0142)
Hours Worked	-0.000556**	-0.000842**	-0.000614*
	(0.000260)	(0.000360)	(0.000326)
Income	-0.00142	-0.00367	0.00189
	(0.00261)	(0.00441)	(0.00251)
Savings Ratio	-0.0178***	-0.0383***	
	(0.00612)	(0.0118)	
Healthy Diet		-0.0112*	-0.0124***
		(0.00660)	(0.00409)
Marijuana		-0.0195	0.0159

		(0.0183)	(0.0106)
Cocaine, Meth, Heroin		-0.0109	0.0219
		(0.0232)	(0.0152)
Hepatitis A Vaccine		-0.0262	-0.00453
		(0.0200)	(0.0140)
Condom		-0.00124	-0.00167
		(0.00499)	(0.00379)
Smoker		0.0122	-0.0138
		(0.0215)	(0.0171)
Constant	-0.00311	0.0569	-0.00702
	(0.0209)	(0.0517)	(0.0365)
Observations	4,974	1,899	4,612
R-squared	0.038	0.040	0.027

Restricted-access sample. Dependent variables are binary indicators of engaging in each category of defensive behavior. Coefficients in columns 1-3 come from OLS estimates. All models account for NHANES' complex survey design (primary sampling units, strata, and probability weights). Standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1

3.9 Appendix

Table A1: OLS Full Sample, by Avoidance Behavior Category

	(3) Time Outdoors	(4) Strenuous	(5) Cancelled	(6) Exercise
Active Hours	0.00149*	0.000493*	0.000178	0.00101***
	(0.000848)	(0.000289)	(0.000227)	(0.000257)
Asthma Year	0.121***	0.0445***	0.0197*	0.00757
	(0.0180)	(0.0144)	(0.0101)	(0.00780)
Age	0.00133***	0.000330***	0.000104	6.14e-05
	(0.000191)	(0.000106)	(7.25e-05)	(6.40e-05)
Non-White	0.0150	-0.00196	0.00998***	0.00487*
	(0.0105)	(0.00252)	(0.00322)	(0.00263)
Male	-0.0497***	-0.00422*	-0.00784**	-0.00717***
	(0.00588)	(0.00240)	(0.00302)	(0.00190)
Education	0.0126***	0.00489***	0.00369***	0.00459***
	(0.00369)	(0.00154)	(0.00119)	(0.00101)
Insurance	0.0103	0.00261	0.00970***	0.00442**
	(0.00826)	(0.00203)	(0.00214)	(0.00189)
Obese	0.0117*	-0.00271	-0.00484	-0.00253
	(0.00605)	(0.00278)	(0.00307)	(0.00196)
Vigorous Work	0.0103	0.00276	0.00707	0.000261
	(0.00792)	(0.00269)	(0.00475)	(0.00310)
Hours Work	-0.000451***	-0.000128*	-0.000115	7.96e-07
	(0.000141)	(6.70e-05)	(6.91e-05)	(4.91e-05)
Income	0.000871	0.000503	0.000621	0.000370
	(0.00121)	(0.000501)	(0.000501)	(0.000360)
Constant	-0.0214	-0.0205**	-0.0118	-0.0147***
	(0.0167)	(0.00968)	(0.00823)	(0.00450)
Observations	11,337	11,337	11,337	11,337
R-squared	0.031	0.013	0.007	0.008

Dependent variables are binary indicators of engaging in each category of defensive behavior, listed in Table 2. Coefficients come from OLS estimates and account for NHANES' complex survey design (primary sampling units, strata, and probability weights). Standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1

Table A2

	(2) Air Filter	(3) Medication	(4) Mask	(6) Traffic
Active Hours	-3.75e-05	-3.39e-05	-0.000336	0.000159
	(0.000148)	(0.000120)	(0.000224)	(0.000138)
Asthma Year	-0.000588	0.0305***	0.0218*	0.0188***
	(0.00462)	(0.0102)	(0.0121)	(0.00645)
Age	-3.78e-05	0.000135***	7.75e-05	0.000143***
	(5.02e-05)	(4.36e-05)	(7.03e-05)	(3.35e-05)
Non-White	-0.00251	0.00111	0.00389	0.00744***
	(0.00166)	(0.00116)	(0.00369)	(0.00170)
Male	0.00101	-0.00294**	0.00374	-0.00248
	(0.00160)	(0.00130)	(0.00228)	(0.00200)
Education	0.000579	0.00161***	4.00e-05	0.00227***
	(0.000695)	(0.000583)	(0.00132)	(0.000737)
Insurance	-0.000908	-0.00256	-0.000661	0.00104
	(0.00226)	(0.00239)	(0.00401)	(0.00208)
Obese	0.000243	0.000522	-0.00329	0.000658
	(0.00210)	(0.00157)	(0.00244)	(0.00207)
Vigorous Work	-0.00266	0.00215	0.00482	0.00206
	(0.00221)	(0.00204)	(0.00443)	(0.00150)
Hours Work	-3.76e-05	-4.99e-05	0.000119*	8.68e-06
	(2.94e-05)	(3.10e-05)	(6.07e-05)	(4.22e-05)
Income	0.000348	2.18e-05	0.00117**	6.46e-05
	(0.000320)	(0.000233)	(0.000472)	(0.000292)
Constant	0.00500	-0.00445	-0.00708	-0.0122***
	(0.00469)	(0.00330)	(0.00732)	(0.00337)
Observations	11,337	11,337	11,337	11,337
R-squared	0.001	0.010	0.005	0.005

Dependent variables are binary indicators of engaging in each category of defensive behavior, listed in Table 2. Coefficients come from OLS estimates and account for NHANES' complex survey design (primary sampling units, strata, and probability weights). Standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1

Table A3

	(1) Car	(2) Bus	(3) Windows	(4) Other
Active Hours	0.000291	0.000186*	0.000102	0.000231
	(0.000253)	(9.91e-05)	(0.000425)	(0.000159)
Asthma Year	0.0233***	0.000378	0.0182*	0.00767**
	(0.00732)	(0.00140)	(0.0100)	(0.00297)
Age	6.49e-05	9.39e-06	0.000435***	7.90e-05**
	(5.31e-05)	(1.93e-05)	(0.000117)	(3.27e-05)
Non-White	0.00446*	0.00123	0.00818*	0.00322
	(0.00250)	(0.000836)	(0.00430)	(0.00227)
Male	-0.00958***	0.000455	-0.0162***	-0.00111
	(0.00263)	(0.000970)	(0.00387)	(0.00167)
Education	0.00552***	0.000710*	0.00595***	0.00239**
	(0.000957)	(0.000387)	(0.00209)	(0.000885)
Insurance	0.00606**	0.000987	0.00642	-0.000674
	(0.00269)	(0.000815)	(0.00425)	(0.00260)
Obese	7.21e-05	0.000160	0.00395	0.000564
	(0.00232)	(0.000408)	(0.00364)	(0.00149)
Vigorous Work	0.00574**	0.000998	0.00208	0.00106
	(0.00240)	(0.00119)	(0.00409)	(0.00207)
Hours Work	-1.36e-05	1.96e-06	-0.000157*	-6.12e-06
	(5.34e-05)	(1.39e-05)	(7.76e-05)	(3.93e-05)
Income	-0.000290	-0.000267**	0.000296	-0.000533
	(0.000385)	(0.000118)	(0.000752)	(0.000319)
Constant	-0.0113**	-0.00167	-0.0122	-0.00251
	(0.00550)	(0.00155)	(0.0116)	(0.00326)
Observations	11,337	11,337	11,337	11,337
R-squared	0.008	0.002	0.009	0.002

Dependent variables are binary indicators of engaging in each category of defensive behavior, listed in Table 2. Coefficients come from OLS estimates and account for NHANES' complex survey design (primary sampling units, strata, and probability weights). Standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1

3.10 Acknowledgements

This paper uses data from the National Center for Health Statistics, accessed through a Federal Statistical Research Data Center. The findings and conclusions in this paper are those of the author(s) and do not necessarily represent the views of the Research Data Center, the National Center for Health Statistics, or the Centers for Disease Control and Prevention.