

## Defensive Investments and the Demand for Air Quality: Evidence from the NO<sub>x</sub> Budget Program<sup>†</sup>

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*The demand for air quality depends on health impacts and defensive investments, but little research assesses the empirical importance of defenses. A rich quasi-experiment suggests that the Nitrogen Oxides (NO<sub>x</sub>) Budget Program (NBP), a cap-and-trade market, decreased NO<sub>x</sub> emissions, ambient ozone concentrations, pharmaceutical expenditures, and mortality rates. The annual reductions in pharmaceutical purchases, a key defensive investment, and mortality are valued at about \$800 million and \$1.3 billion, respectively, suggesting that defenses are over one-third of willingness-to-pay for reductions in NO<sub>x</sub> emissions. Further, estimates indicate that the NBP's benefits easily exceed its costs and that NO<sub>x</sub> reductions have substantial benefits. (JEL I12, Q51, Q53, Q58)*

Willingness to pay (WTP) for well-being frequently depends on factors that enter the utility function directly (e.g., the probability of mortality, school quality, local crime rates, etc.) and compensatory investments that help to determine these factors (Grossman 1972). In a wide variety of contexts, the empirical literature has almost exclusively focused on the direct effects (e.g., health outcomes) of these factors and left the defensive investments largely unmeasured. As examples, there has been little effort to measure: the use of medications or air filters to protect against poor air quality (e.g., Chay and Greenstone 2003; Currie and Neidell 2005); parental expenditures on supplemental tutoring to improve educational outcomes for their children; or the costs of alarm systems and additional security to protect against crime. All of these defensive investments are costly and displace consumption of utility-generating goods. Indeed, economic theory suggests that these actions

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constitute a significant portion of the costs of harms, as individuals should set the marginal utility of their purchase equal to the marginal utility of avoiding the harm itself. It therefore seems reasonable to presume that the available estimates of WTP for a wide variety of factors are substantially understated.

This paper develops a measure of WTP for air quality improvements that accounts for both the direct health impacts and defensive investments. As a measure of defensive behavior, we investigate whether medication usage responds to changes in air quality. This is likely to be an especially important measure of defensive expenditures, because, for example, the annual cost of prescription medications for asthma is reported to exceed the monetized value of any other component of asthma's social cost, including mortality, emergency department admissions, or lost productivity (Weiss and Sullivan 2001). We also provide new evidence on how air pollution affects more commonly studied outcomes like mortality and hospitalizations.

The empirical application is based on a quasi-experiment that exploits three sources of variation in the introduction of an emissions market for nitrogen oxides ( $\text{NO}_x$ ). The  $\text{NO}_x$  Budget Trading Program (NBP) operated a cap-and-trade system for over 2,500 electricity generating units and industrial boilers in the eastern and midwestern United States between 2003 and 2008. Because this market had the goal of decreasing ozone pollution, which reaches high levels in summer, the market operated only between May 1 and September 30. Specifically, we use a triple-difference estimator that compares pollution, defensive expenditures, and health outcomes in the NBP participating and nonparticipating states, before versus after 2003, and summer versus winter.<sup>1</sup>

The empirical analysis produces several key results. First, there was a substantial decline in air pollution emissions and ambient concentrations. Figure 1 illustrates the dramatic effect of this market on  $\text{NO}_x$  emissions in the states participating in the NBP.<sup>2</sup> In 2001–2002, daily  $\text{NO}_x$  emissions were fairly flat throughout the calendar year, with a peak in summer. In 2005–2007,  $\text{NO}_x$  emissions were nearly 40 percent lower but almost entirely during the summer months when the NBP was in force.  $\text{NO}_x$  emissions are a primary ingredient in the complex function that produces ozone air pollution, so it is unsurprising that we find that the large reductions in  $\text{NO}_x$  led to declines in mean ozone concentrations of roughly 6 percent and reduced the number of summer days with high ozone levels (i.e., more than 65 ppb) by about 35 percent, or a third of a standard deviation.

Second, these improvements in air quality produced substantial benefits. Medication expenditures decreased by about 1.6 percent or roughly \$800 million annually in the 19 eastern and midwestern United States where the NBP was in force; this is close to an upper bound estimate of the NBP's total abatement costs. This decline in medication expenditures is evident both among short-acting respiratory medications taken in response to the presentation of respiratory symptoms and long-term control medications that are taken to prevent these episodes. Since people can engage in other defensive investments like avoiding time outdoors or purchasing air filters, medication expenditures provide a lower bound on the total

<sup>1</sup>“Winter” in this paper refers to the combined months of January–April and October–December.

<sup>2</sup>Unless otherwise noted, our data on  $\text{NO}_x$  emissions refer to emissions from power plants covered by our data (i.e., in the Acid Rain Program).

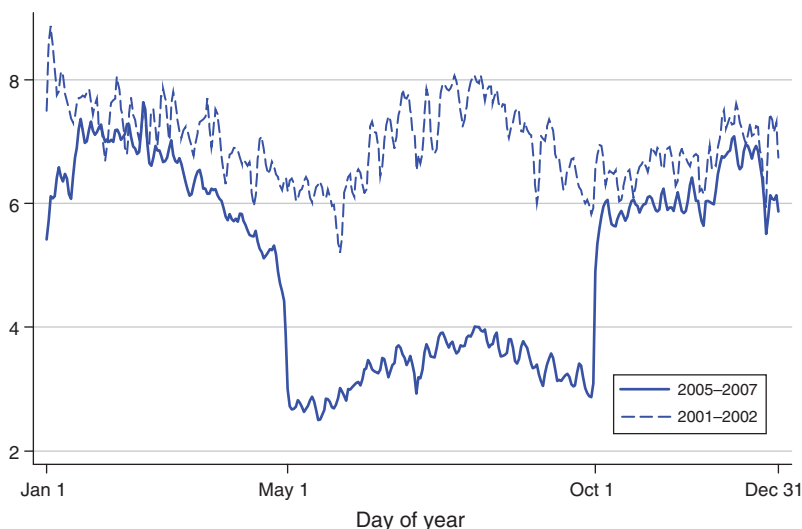


FIGURE 1. TOTAL DAILY  $\text{NO}_x$  EMISSIONS IN THE NBP-PARTICIPATING STATES

*Notes:* Figure 1 shows average total daily  $\text{NO}_x$  emissions in the NBP participating states in 2001–2002 and 2005–2007. These estimates are obtained from an OLS regression of  $\text{NO}_x$  emissions on six day-of-week indicators and a constant. The values in the graph equal the constant plus the regression residuals, so that the graph depicts fitted values for the reference category (Wednesday). Total daily  $\text{NO}_x$  emissions on y-axis are measured in thousands of tons. The sample includes emissions from all the Acid Rain Units. NBP participating states include: Alabama, Connecticut, Delaware, Illinois, Indiana, Kentucky, Maryland, Massachusetts, Michigan, Missouri, New Jersey, New York, North Carolina, Ohio, Pennsylvania, Rhode Island, South Carolina, Tennessee, Virginia, and West Virginia, and Washington, DC. The NBP operated only in northeastern states on May 1 of 2003, and expanded to the other states on May 31 of 2004. See the text for more details.

defensive costs associated with air pollution. Further, the summertime mortality rate declined by up to 0.4 percent, corresponding to 1,975 fewer premature deaths per summer in the NBP states, mainly among individuals 75 and older. The application of age-adjusted estimates of the value of a statistical life (Murphy and Topel 2006) implies this reduced mortality is valued at about \$1.3 billion annually. The mortality estimates are less precise than the medication ones, and the results must be interpreted accordingly. Additionally, there is little systematic evidence of an effect of the NBP on hospitalization charges. Overall, it is striking that defensive investments account for more than one-third of our estimate of total WTP for reductions in  $\text{NO}_x$  emissions.<sup>3</sup>

Third, the paper provides the first instrumental variables estimates of the effect of  $\text{NO}_x$  emissions on health and defensive investments. Such information is an essential determinant of air quality policy since  $\text{NO}_x$  is a pollutant that can be controlled directly by regulation, whereas ozone cannot be. Ambient ozone is determined by a complex function based on several factors including  $\text{NO}_x$  emissions, volatile organic compound emissions (VOCs), and temperature. These estimates suggest a significant causal link between  $\text{NO}_x$  emissions, medication purchases, and mortality. For example, we find that a 10 percentage point (pp) reduction in  $\text{NO}_x$  emissions leads

<sup>3</sup> $\text{NO}_x$  emissions can influence crop yields (through ozone), visibility, the value of outdoor activities, the purchase of air filters, and other factors. A complete measure of WTP for reductions in  $\text{NO}_x$  emissions, as well as the defensive expenditures' (e.g., air filter purchases) share, would include all of these factors.

to 0.06 to 0.011 pp reduction in medication purchases and also an 0.04 to 0.07 pp reduction in mortality. Further, it may be appropriate to conclude the reductions in ozone concentrations stemming from the reductions in  $\text{NO}_x$  emissions are the primary channel for these health improvements; we cautiously report instrumental variable estimates that reveal positive relationships between ambient ozone concentrations and medication expenditures and mortality rates, respectively.

In addition to providing new evidence on the empirical importance of defensive expenditures in the context of air pollution, this paper makes several contributions.<sup>4</sup> First, the results may be useful for the ongoing and contentious academic and policy debates about the regulation of  $\text{NO}_x$  emissions as a means to reduce ambient ozone concentrations. The recent controversy surrounding Volkswagen's use of a "defeat device" that understated  $\text{NO}_x$  emissions and ongoing litigation about the health consequences reflects the dearth of reliable information on the health consequences of  $\text{NO}_x$  emissions. More broadly, ozone is one of the six "criteria" pollutants that the Clean Air Act targets, but, unlike the other pollutants, it has remained persistently high. Further, the Obama Administration tightened the national ambient air quality standard in 2015 from 75 to 70 ppb, following a long political and legal battle between the White House, EPA, Congress, and industry; as of 2015, 126 million Americans, or about 40 percent of the population, live in areas that violate this new air quality standard for ozone. These ozone standards are contentious at least partly because much of the previous evidence comes from observational studies where there is a substantial risk of confounding air pollution and other determinants of health.<sup>5</sup> The central role of  $\text{NO}_x$  emissions in controlling ozone concentrations is underscored by the fact that the regulatory impact analysis for the new ozone standard requires a 65 percent reduction in  $\text{NO}_x$  emissions between 2011 and 2025 (EPA 2015). The paper speaks directly to this debate and can contribute to the regular updating of cost-benefit analyses of the Clean Air Act.<sup>6</sup>

Second, this study is an important step forward in moving closer to the ideal of credibly measuring the consequences of *sustained* exposure to air pollution. Much of the literature relating human health and ozone concentrations focuses on daily or weekly variation in ozone and on specific states or groups of cities; studies based on

<sup>4</sup> An emerging empirical literature aims to measure behavioral responses, including defenses, to health-reducing environmental factors (Graff Zivin and Neidell 2009; Neidell 2009; Deschênes and Greenstone 2011; Graff Zivin, Neidell, and Schlenker 2011; Barreca et al. 2015; Barreca et al. 2016; Ito and Zhang 2016). An older theoretical literature analyzes defenses and WTP (Courant and Porter 1981; Bartik 1988). A small epidemiological literature, largely using samples of under 100 asthma patients, shows that asthmatics increase medication use on polluted days (Menichini and Mudu 2010). As we discuss later, our focus on summer versus rest-of-year seasonal expenditures improves over existing work, which focuses on same-day effects. Same-day regressions can suffer from short-term displacement if pollution merely shifts the day on which a person uses medication but does not change total medium-run medication use. At the same time, we note that medications may be stored across seasons.

<sup>5</sup> Key papers about the relationship between health and ozone include Bell et al. (2004), Currie and Neidell (2005), NRC (2008), Jerrett et al. (2009), Neidell (2009), Lleras-Muney (2010), Moretti and Neidell (2011), and Dominici, Greenstone, and Sunstein (2014).

<sup>6</sup> The results are also important because they fill a knowledge gap in recent research on the NBP regulation. Economic research has found that the NBP led firms to install costly abatement technologies, and that regulated electricity generating units were especially likely to install highly effective "Selective Catalytic Reduction" abatement technology (Fowlie 2010). Engineering estimates suggest that the marginal abatement cost of  $\text{NO}_x$  through this program is much larger than the marginal abatement cost of  $\text{NO}_x$  from vehicles (Fowlie, Knittel, and Wolfram 2012). An integrated assessment model simulating costs and benefits of this market finds that the NBP as actually implemented was more cost-effective than an alternative design which recognized that damages vary across space because actual abatement costs exceeded ex ante expectations (Fowlie and Muller 2013).

daily and/or weekly variation are especially subject to concerns about “harvesting,” or temporal displacement of mortality (and medication expenditures), and that the true loss of life expectancy is small (Deschênes and Greenstone 2011). In contrast, the NBP provides quasi-experimental variation in emitted and ambient air pollution at the 5-month level (i.e., May 1 through September 30); in the case of ozone, this is effectively annual variation because ozone is only considered a health risk during the higher concentration summer months. For these reasons, the paper is less subject to concerns about harvesting and is well suited to shed light on efforts to control  $\text{NO}_x$  emissions and ozone concentrations. Additionally, recent research has emphasized the importance of using quasi-experimental variation to obtain reliable estimates of the relationship between human well-being and air pollution, and the NBP provides variation that is plausibly unrelated to other determinants of well-being (Dominici, Greenstone, and Sunstein 2014).

Third, we are unaware of other studies that demonstrate the impact of an emissions market on ambient pollution and human health with real world data. Most evaluations of emissions markets combine engineering models of emissions abatement, chemistry models of pollution transport, and epidemiological dose-response models (e.g., Muller and Mendelsohn 2009). The limitations of this approach are underlined by our failure to find consistent evidence of an impact of the NBP market on particulates air pollution, which the models (and the EPA) projected as the primary channel for any health benefits. In contrast, this paper’s analysis is conducted with the most comprehensive data file ever compiled on emissions, pollution concentrations, defensive expenditures, and mortality rates.

The rest of this paper is organized as follows. Section I reviews ozone formation and the NBP. Section II presents a simple model of defensive investments. Section III describes data sources and the analysis sample. Section IV discusses the econometric models. Section V reports the results and Section VI conducts a cost-benefit analysis of the NBP and develops a measure of WTP for ozone reductions. Section VII concludes.

## **I. Ambient Pollution and the NBP Emission Market**

### *A. Ambient Pollution*

The Clean Air Act was designed to control ambient levels of ozone and five other pollutants that harm health. Ozone differs from the other pollutants in two important ways. First, polluters do not emit ozone directly. Instead, ambient ozone concentrations are governed by complex nonlinear photochemistry that depends on two chemicals precursors,  $\text{NO}_x$  and VOCs, and sunlight and heat. The market we study operates only in summer because winter ozone levels in the eastern United States are low, and ozone spikes to high peaks on hot and sunny days.

Second, the health consequences of ozone are believed to occur from short-term exposure to high levels (Lippman 2009). Ozone regulation has targeted these peak exposures, rather than focusing on mean ozone levels. For example, the National Ambient Air Quality Standards for ozone primarily reflect the highest few readings of the year. Most epidemiological studies focus on very short-term effects, though some evidence suggests that medium- or long-run effects are larger (Jerrett et al.

2009). Hence, this market is most likely to affect health if it truncates the right tail of the ozone distribution.

### B. The $\text{NO}_x$ Budget Trading Program

As detailed in online Appendix IV, an initial version of the NBP operated in 1999–2002 and produced small declines in summer  $\text{NO}_x$  emissions that are unlikely to confound our analysis of the 2003–2008 NBP (see online Appendix IV). A more stringent version of the NBP then began in 2003 and operated until 2008.<sup>7</sup> This market included 2,500 electricity generating units and industrial boilers, though the 700 coal-fired electricity generating units in the market accounted for 95 percent of all NBP  $\text{NO}_x$  emissions (EPA 2009b).

The NBP market was implemented partially in 2003 and fully in 2004–2005. The 2003–2008 emissions market originally aimed to cover the 8 northeastern states plus Washington, DC, plus 11 additional eastern states. Litigation in the Midwest, however, delayed implementation in the 11 additional states until May 31, 2004.<sup>8</sup> Online Appendix Figure 1 shows the division of states by NBP participation status in the subsequent analysis.<sup>9</sup>

Accordingly, the EPA allocated about 150,000 tons of  $\text{NO}_x$  allowances in 2003, 650,000 tons in 2004, and about 550,000 tons in each of the years 2005–2008.<sup>10</sup> Each state received a set of permits and chose how to distribute those permits to affected sources. Once permits were distributed, affected sources could buy and sell them through open markets. A single emissions cap affected the entire market region, though firms could bank allowances for any future year. Many firms banked allowances: In each year of the market, about 250,000 tons of allowances were saved unused for subsequent years (EPA 2009a). At the end of each market season, each source had to give the EPA one allowance for each ton of  $\text{NO}_x$  emitted. Seventy percent of units complied by using emissions controls (e.g., low  $\text{NO}_x$  burners or selective catalytic reduction), and the remainder complied exclusively by holding emissions permits (EPA 2009b). The mean resulting permit price in the emissions market was \$2,523 per ton of  $\text{NO}_x$  US\$(2015). This reflects the marginal abatement cost of the last unit of  $\text{NO}_x$  abated, and we use it to develop an upper bound on the aggregate abatement cost of the NBP.

<sup>7</sup>In 2009, the Clean Air Interstate Rule (CAIR) replaced this market. In 2010, the EPA proposed a Transport Rule which would combine this  $\text{NO}_x$  market with a market for  $\text{SO}_2$  emissions. In July 2011, the EPA replaced this proposal with the Cross-State Air Pollution Rule, which regulates power plant emissions in 27 states with the goal of decreasing ambient ozone and particulate levels.

<sup>8</sup>The 1999–2002 Ozone Transport Commission Market included Connecticut, Delaware, Maryland, Massachusetts, New Jersey, New York, Pennsylvania, Rhode Island, and Washington, DC. On May 1, 2003, the NBP emissions cap applied to the exact same set of states. On May 31, 2004, it also began applying to Alabama (excluding a southern region of the state), Illinois, Indiana, Kentucky, Michigan, North Carolina, Ohio, South Carolina, Tennessee, Virginia, and West Virginia. Missouri entered the market in 2007.

<sup>9</sup>The main results define all states in the NBP cap-and-trade region as treated, they exclude states that are adjacent to NBP states, and they define remaining states as comparison (non-treated). We exclude states adjacent to the NBP region from the main results because their treatment status is ambiguous (see online Appendix IV for details).

<sup>10</sup>All tons in the paper refer to short tons and not metric tons.



## II. Model of Willingness-to-Pay (WTP)

We build upon the canonical Becker-Grossman health production function to highlight the role of defensive investments in the measurement of WTP for clean air (Becker 1965; Grossman 1972; Freeman 2003). This model shows that accurate measurement of WTP requires knowledge of both how pollution affects health outcomes such as mortality and how it affects defensive investments that maintain health but otherwise generate no utility, such as medications.

Assume the sick days  $s(d)$  which a person suffers depends on the dose  $d$  of pollution she is exposed to. The effective dose  $d(c, a)$  depends on the ambient concentration  $c$  of the pollutant and on the defensive behavior  $a$ . Defensive behaviors can be taken before or after pollution is ingested—in the terminology of Graff Zivin and Neidell (2013), defenses include both averting and mitigating activities. Substituting provides the following health production function:

$$(1) \quad s = s(c, a).$$

People gain utility from consumption of a general good  $X$  (whose price is normalized to 1), leisure  $f$ , and health. Budgets are constrained by nonlabor income  $I$ , the wage rate  $p_w$ , available time  $T$ , and the price  $p_a$  of defensive investments:  $\max_{X,f,a} u(X, f, s)$  subject to  $I + p_w(T - f - s) \geq X + p_a a$ . Assuming an interior solution to the maximization problem, we can rearrange the total derivative of the health production function (1) to give the following expression for the partial effect of ambient pollution on sick days:

$$(2) \quad \frac{\partial s}{\partial c} = \frac{ds}{dc} - \left( \frac{\partial s}{\partial a} \frac{\partial a^*}{\partial c} \right).$$

This expression is useful because it underscores that the partial derivative of sick days with respect to pollution is equal to the sum of the total derivative and the product of the partial derivative of sick days with respect to defensive behavior (assumed to have a negative sign) and the partial derivative of defensive behavior with respect to pollution (assumed to have a positive sign). In general, complete data on defensive behavior is unavailable, so most empirical investigations of pollution on health (see, e.g., Chay and Greenstone 2003) reveal  $ds/dc$ , rather than  $\partial s/\partial a$ . As equation (2) demonstrates, the total derivative is an underestimate of the desired partial derivative. Indeed, it is possible that virtually all of the response to a change in pollution comes through changes in defensive behavior and that there is little impact on health outcomes; in this case, an exclusive focus on the total derivative would lead to a substantial understatement of the health effect of pollution. The full impact therefore requires either estimation of  $\partial s/\partial da$ , which is almost always infeasible, or of  $ds/dc$  and  $\partial a^*/\partial c$ . We emphasize that defenses used both before and after pollution is ingested (i.e., averting and mitigating activities) are indistinguishable in the WTP expression (2). From the view of social welfare, the distinction between them is not relevant.

To express the marginal WTP for clean air  $w_c$  in dollars, we manipulate the previous expressions to obtain the following decomposition:

$$(3) \quad w_c = \left( p_w \frac{ds}{dc} \right) + \left( p_a \frac{\partial a^*}{\partial c} \right) - \left( \frac{\partial u / \partial s}{\lambda} \frac{ds}{dc} \right).$$

Expression (3) shows that the marginal WTP for clean air includes three terms. The first is the effect of pollution on productive work time, valued at the wage rate. The third is the disutility of sickness, valued in dollars. This third component includes mortality. The second is the cost of defensive investments, valued at their market price. This second component is the aspect of WTP that existing research has not measured. It is important to note that medications are not a complete measure of defensive investments against air pollution. The paper's primary empirical goal is to develop a measure of marginal WTP that is based on  $ds/dc$  and  $\partial a^* / \partial c$ .

Our setting has two important deviations from this neoclassical model: markups and moral hazard. Branded medications generally have low marginal cost and high markups that reflect intellectual property rights. Hence, it is natural to question whether changes in medication purchases amount to a transfer from consumers to drug firms, and not a social cost. In the short-run, this is indeed the case. However, pharmaceutical firms must invest socially valuable resources to develop medications that treat conditions exacerbated by air pollution. With lower levels of air pollution, fewer resources would be spent to develop these medications. Thus over the long run, there is a social benefit (see Finkelstein 2004) for a similar induced innovation process.

The second important deviation is that the marginal cost to the consumer is smaller than the price, by 80 percent in our data, because consumers with insurance generally pay a copayment or deductible for medications. We report medication cost results both using the full transacted price for medications (which is more accurate than the published or wholesale price) and using the copayment. The copayment may provide the best measure of a person's private WTP for her own medications. Since an insurer must pay the remainder of the medication cost, the full cost of the medication may more accurately represent social-WTP for the cost of the medications.

### III. Data

This analysis has compiled an unprecedented set of data files to assess the impacts of the NO<sub>x</sub> Budget Program. Although market-based instruments are viewed as among the most important contributions of economics to environmental policy, to the best of our knowledge this study represents the first time any analysis has linked ex post health measurements directly to emissions and air quality measures in order to evaluate an emissions market.

*Medications.*—We use confidential data on medication and hospital admissions from MarketScan. MarketScan contracts with large employers to obtain all insurance-related records for their employees and their dependents including children. The data report the purchase county, date, the medication's National Drug Code (NDC), and the money paid from consumer and insurer to the medication provider.



We use data from all persons in the 19 covered firms which appear in all years, 2001–2007, of MarketScan, which is the largest panel the data allow us to obtain with these firms. This extract includes over 22 million person-season year observations, and over 100 million separate medication purchases. Because the distribution of persons across counties is skewed, we report all values as the log of rates per 1,000 people, and use generalized least squares (GLS) weights equal to the relevant MarketScan population.<sup>11</sup> Because the other datasets become available in 1997 but medication data become available in 2001, for non-medication results we report parameter estimates both with data for the period 1997–2007 and for the period 2001–2007.

Medications are not linked to a single International Classification of Disease (ICD) code. In the subsequent analysis, we follow the convention in the pollution-health literature and treat respiratory and cardiovascular related episodes as most likely to be affected by air pollution. We define an NDC as respiratory if it satisfies any of three criteria: (i) if it is listed in the Third Treatment Guidelines for Asthma (NHLBI 2007); (ii) in a recent *New England Journal of Medicine* guide to asthma treatment (Fanta 2009); or (iii) in the standard industry publication for medication characteristics (Lippincott, Williams, and Wilkins 2004) as indicated for asthma, emphysema, bronchitis, or chronic obstructive pulmonary disorder. Online Appendix III provides additional details on the steps taken to define respiratory medications. We identify cardiovascular medications by their corresponding therapeutic group in Red Book (PDR 2006).<sup>12</sup>

This broad approach to identifying respiratory and cardiovascular drugs is the most appropriate we can discern. Nonetheless, because doctors prescribe medications to treat conditions for which the medications are not indicated, some of these medications were probably prescribed for non-respiratory and non-cardiovascular conditions. Moreover, it is also likely that medications prescribed for respiratory and cardiovascular conditions are not in this list. For example, the three sources mentioned above that we use to define respiratory medications have somewhat different categorization of which medications are respiratory. Internet searches for respiratory medications also find medications which can be used for respiratory conditions, but which are not listed as respiratory in any of the sources above.<sup>13</sup> Additionally, Red Book identifies a single therapeutic group for each NDC. Since a medication may be used to treat multiple conditions, medications in non-cardiovascular therapeutic groups may also be used to treat cardiovascular conditions.

*Hospitalizations.*—We count hospital admission costs as including all inpatient episodes plus all emergency outpatient episodes. When a hospital visit has several associated procedures each with its own ICD9 code, we take the mode procedure. Our measure of hospital costs includes all charges from the hospital to the insurer and patient.

<sup>11</sup> Few county-season medication values equal zero, though these observations are dropped in logs.

<sup>12</sup> Red Book has no category for respiratory medications.

<sup>13</sup> For example, dexamethasone is not listed as respiratory in any of our sources, but medical websites like the Mayo Clinic's list it as used to treat asthma along with many other conditions including inflammation, allergies, arthritis, blood or bone marrow problems, kidney problems, skin conditions, and multiple sclerosis. Similarly, isoflurane is not listed as a respiratory condition in our data, as it is primarily used for anesthesia, but many medical journal articles and other sources document its use for asthma.

*Mortality.*—To measure mortality, we use restricted-access data on the universe of deaths in the 1997–2007 period. These Multiple Cause of Death files (MCOB) come from the National Center for Health Statistics (NCHS) and were accessed through an agreement between NCHS and the Census Research Data Centers. These files contain information on the county, cause of death, demographics, and date of each fatality.

*Pollution Emissions.*—To measure pollution emissions, we extract daily totals of unit-level  $\text{NO}_x$ ,  $\text{SO}_2$ , and  $\text{CO}_2$  emissions for all states from the EPA's Clean Air Markets Division. The  $\text{NO}_x$  emissions almost entirely come from continuous emissions monitoring systems (CEMS) and are quite accurate. Units which are part of the Acid Rain Program must report  $\text{NO}_x$  emissions throughout the year, while units in the NBP must report  $\text{NO}_x$  emissions only in the May 1–September 30 period. Because we compare summer versus winter and east versus west, estimates in the paper use only data from Acid Rain Units. However, in the examined period, units in the NBP and not in the Acid Rain Program make a very small share of  $\text{NO}_x$  emissions.

*Ambient Pollution.*—We use a few criteria to select ambient pollution monitoring data from the EPA's detailed Air Quality System. Many EPA monitors operate for limited time spans and may change reporting frequency in response to pollution (Henderson 1996). The main analysis uses a fairly strenuous selection rule of limiting to monitors which have valid readings for at least 47 weeks in all years 1997–2007. Online Appendix Table 1 shows that we obtain similar results with a weaker monitor selection rule. For ozone, we focus on a concentration measure the EPA regulates: for each day, we calculate an “8-hour value” as the maximum rolling 8-hour mean within the day. We also calculate the number of days on which this 8-hour value was equal to or greater than 65 ppb, which is an indicator of high-ozone days.

*Weather.*—We compiled daily maximum and minimum temperature, total daily precipitation, and dew point temperature data from records of the National Climate Data Center Summary of the Day files (File TD-3200). Online Appendix III explains the procedure chosen to ensure accurate and complete weather readings.

*Summary Statistics.*—Table 1 shows the means, standard deviations, and county representation for the main variables in our analysis. Of the 2,539 counties in our preferred sample, medication and hospitalization data are available for 96 percent of these counties, which had a population of 261 million in 2004.<sup>14</sup> Ambient ozone data are available for only 168 counties, but these counties are heavily populated and their 2004 population was 97 million. Data on particulates less than 2.5 micrometers ( $\text{PM}_{2.5}$ ) are available in 298 counties (population 144 million) and data on

<sup>14</sup> While the United States has about 3,000 counties, our working sample is smaller since, as discussed in online Appendix IV, the main sample excludes several states adjacent to the NBP region since their treatment status is ambiguous.

TABLE 1—MEAN SUMMER VALUES OF THE POLLUTION, WEATHER, AND HEALTH VARIABLES, 2001–2007

	Counties with data (1)	Mean (2)	Standard deviation (3)
<i>Pollution emissions (000's of tons/summer)</i>			
NO <sub>x</sub> emissions	2,539	0.52	(1.99)
SO <sub>2</sub> emissions	2,539	1.50	(6.52)
CO <sub>2</sub> emissions	2,539	384	(1,299)
<i>Air quality (ambient pollution)</i>			
Ozone 8-hour value (ppb)	168	48.06	(9.28)
Ozone days $\geq$ 65 (ppb)	168	23.60	(22.64)
NO <sub>2</sub> (ppb)	110	11.45	(5.39)
CO (ppm)	125	0.44	(0.24)
PM <sub>2.5</sub> ( $\mu\text{g}/\text{m}^3$ )	298	13.33	(4.19)
PM <sub>10</sub> ( $\mu\text{g}/\text{m}^3$ )	39	27.28	(6.26)
SO <sub>2</sub> (ppb)	150	3.26	(2.27)
<i>Weather</i>			
Temperature (°F)	2,539	70.57	(5.79)
Precipitation (1/100")	2,539	11.47	(5.36)
Dew point temp. (°F)	2,539	57.72	(7.95)
<i>Medication costs (\$ per person)</i>			
All	2,435	377.56	(338.66)
Copayment	2,435	79.10	(59.21)
Respiratory or cardio.	2,435	99.59	(108.74)
<i>Hospitalizations (\$ per person)</i>			
All	2,435	601.76	(2,503.76)
Respiratory or cardio.	2,435	150.29	(972.70)
<i>Mortality (deaths per 100,000 people)</i>			
All	2,539	399.07	(118.90)
Respiratory or cardio.	2,539	179.30	(68.77)

*Notes:* Medication and hospitalization costs are reported in 2015 dollars and deflated using the US CPI for urban consumers. Emissions, medications, and deaths are totals per summer. Ambient pollution and weather are mean summer values. Means are across counties (i.e., not weighted). All data are for the period 2001–2007.

particulates less than 10 micrometers (PM<sub>10</sub>) are available for 39 counties (population of 26 million).

The summary statistics in Table 1 also provide a benchmark to measure the economic importance of medications and the emissions market. In summer, ozone averages 48 ppb. The 2010 proposed EPA air quality standard stipulated that a county could have no more than 3 days over a total of 3 years which exceed 60–70 ppb. Table 1 shows that during the sample period, 24 days every summer exceed 65 ppb in the typical county. On average during this time, the average person spent \$378 per summer on medications, and about \$600 on hospital admissions.

In unreported results, we also investigated potential unobserved variables in the observational associations between ozone and health. We divided all counties with ozone data into two sets—one set with mean summer ozone above the national median (“high ozone”), and another with mean summer ozone below the national median (“low ozone”). All ambient pollutant measures except carbon monoxide have significantly higher levels in the high-ozone counties. Temperature, precipitation, and dew point temperature are lower in high-ozone counties. The finding that

so many of these observed county characteristics covary with ozone suggests that an observational association of ozone with health is likely to reflect the contributions of other unobserved variables and may explain the instability of the estimated health-ozone relationship that has plagued the previous literature. It is apparent that the estimation of the causal effect of  $\text{NO}_x$  emissions and ozone on health and defensive expenditures requires a research design that isolates variation in  $\text{NO}_x$  and ozone that is independent of potential confounders.

#### IV. Econometric Model

We use a differences-in-differences-in-differences (DDD) estimator to isolate the causal effects of the emissions market on pollution, defensive investments, and health, and use instrumental variables to measure the “structural” effect of  $\text{NO}_x$  emissions and ozone on the same outcomes. The DDD estimator exploits three sources of variation in the emission and health data. First, we compare the years before and after the NBP’s operation. Eight states plus Washington, DC initiated this market in 2003, while 11 other states joined in 2004. This market did not operate before 2003. Second, 19 states plus Washington, DC participated in the NBP while 22 other states did not participate and were not adjacent to a NBP state (see online Appendix Figure 1). Third, the NBP market only operated during the summer, so we compare summer versus winter.

Specifically, we estimate the following model:

$$(4) \quad Y_{cst} = \gamma_1 1(\text{NBP Operating})_{cst} + W'_{cst} \beta + \mu_{ct} + \eta_{st} + \nu_{cs} + \varepsilon_{cst}.$$

Here,  $c$  references county,  $s$  indicates season, and  $t$  denotes year. The year is divided into two seasons, summer and winter. Summer matches the NBP’s operation period of May 1–September 30. The variables  $Y_{cst}$  are pollution emissions, ambient pollution concentrations, medication costs, hospitalization costs, and mortality rates. Because the NBP market started partway in 2003, we define  $\text{Post} = 0.5$  in 2003 and  $\text{Post} = 1.0$  in 2004 through 2007. Online Appendix Tables 1, 2, and 4 show qualitatively similar results for all natural alternatives to this definition. All regressions limit the sample to a balanced panel of county-season-years. Our main results cluster standard errors by state-season, but the online Appendix reports alternative levels of clustering, with similar conclusions.

Since temperature has nonlinear effects on health, it is important to adjust for weather flexibly. The matrix of weather controls,  $W_{cst}$ , includes measures of precipitation, temperature, and dew point temperature (a measure of humidity). For temperature and humidity, we calculate 20 quantiles of the overall daily distribution.<sup>15</sup> For each county-season-year observation in the data, we then calculate the share of days that fall into each of the 20 quantiles.

To operationalize the DDD estimator, the specification includes all three sets of two-way fixed effects. The vector  $\mu_{ct}$  is a complete set of county by year fixed effects, which account for all factors common to a county within a year (e.g., local

<sup>15</sup>The lower quantiles of the precipitation distribution all equal zero, so for simplicity we specify the precipitation control as the mean level of precipitation in each county-year-summer.

economic activity and the quality of local health care providers). The season-by-year fixed effects,  $\eta_{st}$ , control for all factors common to a season and year: for example, they would adjust for the development of a new drug to treat asthma that was sold in NBP and non-NBP states. Finally, the county-by-season fixed effects,  $\nu_{cs}$ , allow for permanent differences in outcomes across county-by-seasons. This specification estimates the difference in outcomes between a world with all  $\text{NO}_x$  regulations including the NBP (including the Ozone Transport Commission market, RECLAIM, ozone non-attainment designations, and others) versus a world with all  $\text{NO}_x$  regulations except the NBP. Other regulations did apply to  $\text{NO}_x$  emissions from power plants in this period; for example, the Massachusetts State Implementation Plan adopted strict annual (though not summer-only)  $\text{NO}_x$  emissions standards for power plants in 2001, which began applying between 2004 and 2008. Such policies help explain the downward trends observed in both winter and in non-NBP states in online Appendix Figure 2. Our identifying assumption is that such policies did not change differentially in NBP versus non-NBP states, in winter versus summer, over this period.

The parameter of interest is  $\gamma_1$ , associated with the variable  $1(\text{NBP Operating})_{cst}$ . As noted earlier, this takes the value of 0.5 for all NBP states in 2003, when the market was operating in 8 states plus Washington, DC, and a value of 1 in 2004 and all subsequent years. The 2003 value is assigned to all NBP states, rather than just states which entered the market in 2003, because  $\text{NO}_x$  and ozone travel far and emissions reductions in one NBP state affected ambient ozone in other NBP states. After adjustment for the fixed effects,  $\gamma_1$  captures the variation in outcomes specific to NBP states, relative to non-NBP states, in years when the NBP operated, relative to before its initiation, and in the summer, relative to the winter. This only leaves variation in the outcomes at the level at which the market operated.

Separate measures of the market's effect in each year provide additional useful information. Hence, for most outcomes, we also report the parameters  $\alpha_{1997}, \dots, \alpha_{2007}$  from the following model:

$$(5) \quad Y_{cst} = \sum_{t=1997}^{2007} \alpha_t 1(\text{NBP State and Summer})_{cs} + W'_{cst} \beta + \mu_{ct} + \eta_{st} + \nu_{cs} + \varepsilon_{cst},$$

where  $1(\text{NBP State and Summer})_{cs} = 1$  for all summer observations from NBP states, regardless of the year. We plot the  $\alpha_t$ s in event study style figures to provide visual evidence on the validity of the conclusions from the estimation of equation (4).<sup>16</sup> Importantly, the event study style graphs provide an opportunity to assess whether there were pre-NBP trends in outcomes that were specific to NBP states after nonparametric adjustment for all county by year, season by year, and county by season factors. Online Appendix Figure 4 reports 20 separate event study graphs that cover all main outcomes in the paper.

Finally, we report on the results from the estimation of instrumental variables versions of

$$(6) \quad Y_{cst} = \delta \text{NOx}_{cst} + W'_{cst} \phi + \lambda_{ct} + \pi_{st} + \gamma_{cs} + \nu_{cst},$$

<sup>16</sup>The data on medication purchases and hospitalization begins in 2001, so for these outcomes, the event-study graphs are for the period 2001–2007.

where the subscripts have the same meaning as in equations (4) and (5) and the equation includes the same set of fixed effects. Here,  $Y_{cst}$  is restricted to measures of medication purchases and mortality rates. The key difference is that  $\text{NO}_x$  emissions in county  $c$ , season  $s$ , and year  $t$  is an endogenous regressor and  $1(\text{NBP Operating})_{cst}$  from equation (4) is used as an instrumental variable. We demonstrate below that there is a strong first-stage in that the instrumental variable predicts  $\text{NO}_x$  emissions. The exclusion restriction is the other necessary condition for a valid instrumental variable and, conditional on the full set of two-way fixed effects, we believe that it is credible to assume that  $1(\text{NBP Operating})_{cst}$  only affects medication purchases and mortality rates through  $\text{NO}_x$  emissions.

The case for the validity of the exclusion restriction when ozone is the endogenous variable is plausible. However, it is less clear cut for two reasons: (i) the link between the NBP and ambient ozone is less direct since it is mediated by complex nonlinear photochemistry and this can make for a noisy relationship; and (ii) air quality models show that atmospheric  $\text{NO}_x$  can transform into particulates air pollution that is harmful to human health (Pandis and Seinfeld 2006). Nevertheless, there is a straightforward channel and we also report on versions of equation (6) where ambient ozone, instead of  $\text{NO}_x$  emissions, is the endogenous variable, and  $1(\text{NBP Operating})_{cst}$  is the instrumental variable. Reliable estimation of either, or both, versions of equation (6) would be of tremendous practical value for policy and, more broadly, so that this paper's results can be applied to other settings.

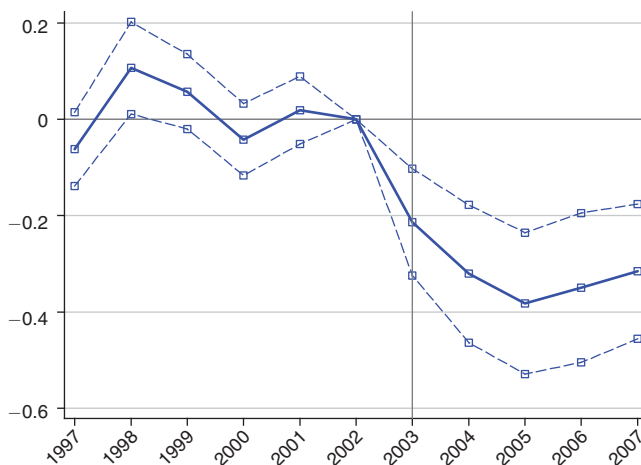
## V. Results

### A. Emissions

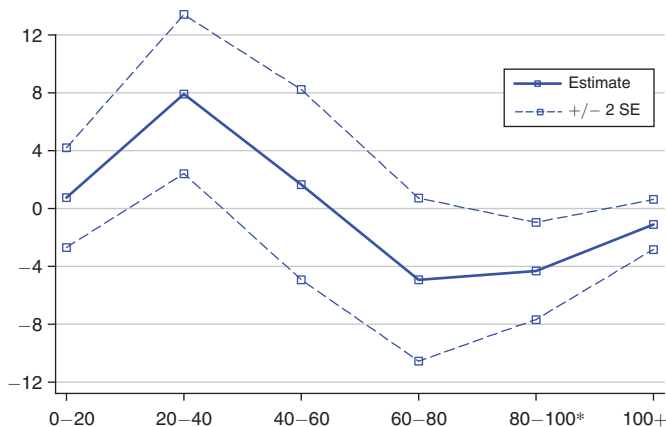
The NBP required affected units to reduce  $\text{NO}_x$  emissions during the summer. Figure 2, panel A, shows an event study graph measuring the difference between  $\text{NO}_x$  emissions in the eastern and western United States and in summer versus winter, separately by year, with the year 2002 normalized to take the value zero. The value for 2001 is almost exactly equal to zero, which is consistent with a lack of pre-trends in  $\text{NO}_x$  emissions. Figure 2, panel A, shows that in the year 2003, when the NBP market began,  $\text{NO}_x$  emissions fell by 0.2 thousand tons per county-season-year; and by the later years of the NBP,  $\text{NO}_x$  emissions had fallen by a total of 0.3 to 0.4 thousand tons per county-season-year. Panel A of Table 2 reports estimates of several versions of equation (4) for pollution emissions measured at the county by season by year level. Column 1 includes county-by-season, season-by-year, and state-by-year fixed effects. Column 2 adds binned weather controls. Column 3 replaces the state-by-year fixed effects with county-by-year fixed effects, which causes the parameters of interest to be identified from comparisons of summer and winter emissions within a county by year. Column 4 restricts the sample to 2001–2007, which are the years when medication and hospitalization data are available. Since emissions readings are totals rather than averages, the regressions (excepting column 5) are unweighted.

The entries in row 1 report the parameter estimate and standard error associated with the variable  $1(\text{NBP Operating})_{cst}$ . The results suggest that the NBP market



Panel A. Event study for NO<sub>x</sub> emissions, 1997–2007

Panel B. NBP market impact on the number of summer days in six ozone bins

FIGURE 2. NBP MARKET IMPACT ON NO<sub>x</sub> EMISSIONS AND AMBIENT OZONE POLLUTION

*Notes:* The estimates in Figure 2, panel A, are from an event study regression for NO<sub>x</sub> emissions (measured in thousand of tons and observed at the county  $\times$  year  $\times$  season) where the estimates for year 2002 are restricted to have a value of 0. The regression includes detailed weather controls and a full set of county  $\times$  year, season  $\times$  year, and county  $\times$  season fixed effects. The standard errors underlying the confidence intervals (dashed lines) are clustered at the state-season level. In panel B, the bins represent 6 categories of ozone 8-hour values, corresponding to the number of days per summer where the ozone 8-hour value is 0–20 ppb, 20–40 ppb, 40–60 ppb, etc. The ozone 8-hour value is measured as the maximum rolling 8-hour mean of hourly values within each day. The coefficients reported in Figure 2, panel B, are from a separate regression for each bin that includes detailed weather controls and a full set of county  $\times$  year, season  $\times$  year, and county  $\times$  season fixed effects. The regression is weighted by the number of ozone readings in each county  $\times$  season  $\times$  year. The asterisk on the 80–100 category denotes the non-attainment air quality standard during the NBP years (85 ppb).

decreased NO<sub>x</sub> emissions in the average county by 330–440 tons. This corresponds to a total decrease of between 391,000 and 521,000 tons of NO<sub>x</sub> per summer.

It is informative to compare these statistics against other reports of the NBP's impacts on NO<sub>x</sub> emissions. The EPA (2008) estimates that a combination of the NBP and its smaller predecessor, the NO<sub>x</sub> SIP Call, decreased ozone season NO<sub>x</sub> emissions by a larger amount, 750,000 tons. Their estimate comes from a time-series

TABLE 2—EFFECT OF THE NBP MARKET ON EMITTED AND AMBIENT POLLUTION

	(1)	(2)	(3)	(4)	(5)
<i>Panel A. Pollution emissions (000s of tons per summer)</i>					
1. NO <sub>x</sub>	−0.36 (0.05)	−0.38 (0.06)	−0.36 (0.07)	−0.33 (0.07)	−0.44 (0.12)
2. SO <sub>2</sub>	−0.08 (0.04)	−0.14 (0.07)	−0.07 (0.05)	−0.07 (0.03)	−0.04 (0.12)
3. CO <sub>2</sub>	−2.66 (4.33)	−24.58 (15.89)	−3.56 (5.60)	−12.66 (6.48)	−83.05 (50.54)
<i>Panel B. Air quality (ambient pollution)</i>					
4. Ozone 8-hour value (ppb)	−2.92 (0.76)	−3.58 (0.86)	−2.87 (0.72)	−3.38 (0.56)	−3.37 (0.54)
5. Ozone days ≥ 65 (ppb)	−7.33 (2.44)	−8.28 (2.43)	−7.79 (2.77)	−9.55 (2.63)	−10.26 (2.46)
6. CO: carbon monoxide (ppm)	−0.05 (0.02)	−0.05 (0.04)	−0.05 (0.03)	−0.02 (0.02)	0.00 (0.02)
7. SO <sub>2</sub> : sulfur dioxide (ppb)	0.16 (0.12)	0.25 (0.24)	0.13 (0.17)	0.16 (0.19)	0.32 (0.19)
8. NO <sub>2</sub> : nitrogen dioxide (ppb)	−1.13 (0.21)	−0.18 (0.82)	−0.98 (0.38)	−0.78 (0.39)	−0.90 (0.51)
9. PM <sub>2.5</sub> : particulates less than 2.5 micrometers (μg/m <sup>3</sup> )	— —	— —	— —	−0.45 (0.32)	−1.03 (0.27)
10. PM <sub>10</sub> : particulates less than 10 micrometers (μg/m <sup>3</sup> )	— —	— —	— —	−1.20 (1.17)	−0.99 (1.48)
County-by-season fixed effects	X	X	X	X	X
Summer-by-year fixed effects	X	X	X	X	X
State-by-year fixed effects	X	X			
County-by-year fixed effects			X	X	X
Detailed weather controls		X	X	X	X
Data begins in 2001				X	X
Weighted by emission/pollution monitors (panel B only)	X	X	X	X	
Weighted by population					X

*Notes:* The entries in Table 2 are the coefficient estimates from the DDD estimator described in equation (4). Each coefficient is from a separate regression that includes a full set of county × year, season × year, and county × season fixed effects. Additional control variables are listed in the text. The reported standard errors are clustered at the state-season level. Emitted pollutant variables (panel A) are measured in thousand of tons and ambient pollutant variables (panel B) are mean values. Unless otherwise noted, the sample period begins in 1997. Ambient pollution regressions (panel B) are GLS weighted by the number of underlying pollution readings unless otherwise noted. For emissions, the number of observations is 55,858 in columns 1 to 3 and 35,546 for columns 4–5. For ambient pollution, the number of observations for each pollutant based on 1997–2007 sample is 3,146 (Ozone); 2,244 (CO); 2,684 (SO<sub>2</sub>); 1,782 (NO<sub>2</sub>). For ambient pollution based on 2001–2007 sample, the number of observations is 2,352 (Ozone), 1,750 (CO), 2,100 (SO<sub>2</sub>), 1,540 (NO<sub>2</sub>), 4,172 (PM<sub>2.5</sub>), and 546 (PM<sub>10</sub>). For emissions, share of population covered is 100 percent. For ambient pollution, share of population covered is 28–40 percent for ozone, CO, SO<sub>2</sub>, and NO<sub>2</sub>; 55 percent for PM<sub>2.5</sub>; and 10 percent for PM<sub>10</sub>.

comparison of the years 2000 and 2007. Reconciling this with our smaller estimate is straightforward. A time-series comparison of the years 2002 and 2007 implies a smaller decrease of somewhat over 500,000 tons. Accounting for secular trends in emissions, which were present in both summer and winter seasons and in the NBP and non-NBP states (online Appendix Figure 2), suggests an estimate within our range of 391,000 to 521,000 tons.<sup>17</sup>

<sup>17</sup> Our emissions totals are not numerically equal to those of at least the EPA's (2008) NBP report for a few reasons: their report describes Missouri sources as regulated by an NBP in all years (whereas in reality those sources were only regulated in 2007; we exclude Missouri from our main analysis); we treat all of Alabama as in the NBP

We also measure whether the NBP market affected emissions of pollutants other than  $\text{NO}_x$ . Two economic reasons explain why the market might have affected emissions of such co-pollutants. If permits for  $\text{NO}_x$  emissions cost enough that the market caused natural gas units to displace electricity generation from relatively dirty coal-fired units, then the market could have decreased emissions of pollutants other than  $\text{NO}_x$ . Second, complementarity or substitutability of  $\text{NO}_x$  with other pollutants in electricity generation could lead units to change emissions of other pollutants. Rows 2 and 3 in panel A of Table 2 indicate that NBP did not substantially affect  $\text{SO}_2$  or  $\text{CO}_2$  emissions. Our preferred estimates in column 3 are not statistically significant, though some of the other estimates are. However, all of the estimates are economically small; for example, the point estimates in the preferred specifications in columns 3 and 4 are about 1 percent to 5 percent of the mean from years 2001–2002, or a tenth of our proportional estimate for  $\text{NO}_x$ . Event study graphs in online Appendix Figure 4, panel B, suggest quantitatively similar conclusions. The  $\text{SO}_2$  graph, for example, suggests a decrease of 0.1 thousand tons per county-season, and the  $\text{CO}_2$  graph suggests a decrease of approximately 20,000 tons per county-season.

### B. Ambient Pollution

Panel B in Table 2 reports on how the NBP affected ambient concentrations of ozone and the other pollutants that are most heavily regulated under the Clean Air Act. Columns 1–4 have identical specifications to those in panel A, except that they are weighted by the number of pollution readings from the EPA's ambient air quality monitors in a given year by county. The column 5 estimates are from the same specification as in column 4, except that they are weighted by county population, which will be the relevant weight in the analysis of the impact of the NBP market on health outcomes (though defensive investments are weighted by the population in the MarketScan survey).

Rows 4 and 5 of Table 2, panel B, reveal large and precisely estimated effects of the emissions market on ground-level ozone concentrations (as measured by the maximum 8-hour value). The richest specifications in columns 3–5 indicate that the NBP decreased mean summer ozone by about 3 ppb (or 6 percent relative to the baseline average). Importantly, the NBP also decreased the number of high-ozone days (days where the 8-hour value equals or exceeds 65 ppb) by 7.8 to 10.3 days per summer (or 33 percent–44 percent of the baseline average). The corresponding event study figure for the 8-hour ozone reading (online Appendix Figure 3, panel C) exhibits some evidence of differential preexisting trends in summer ozone concentrations in NBP states. Accounting for these differences increases the magnitude of the NBP's estimated reduction on ozone concentrations, although these models are more demanding of the data and so the estimates are less precise, but remain significant at the conventional level.

Given the large effect of the NBP on the number of days with ozone equaling or exceeding 65 ppb, we also analyze the market's impact on the density function for

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while they exclude some sources in southern Alabama; and we include only Acid Rain Units in the analysis since they have high-quality CEMS data for summer and winter and pre-NBP years, while the EPA's reports, which focus only on summer emissions, also include the fairly few NBP sources that are not in the Acid Rain Program.

daily ozone concentrations to explore where in the daily ozone distribution the NBP affected concentrations. Figure 2, panel B, reports these results; the main finding is that the market reduced the number of summer days with relatively high-ozone concentrations (i.e., greater than 60 ppb) and increased the number of days with ozone concentrations less than 60 ppb. It is noteworthy that the EPA has experimented with daily ozone standards of 65, 75, and 85 ppb in recent years and that the identifying variation in ozone concentrations comes from this part of the distribution where there is great scientific and policy uncertainty.<sup>18</sup>

Rows 6–8 in panel B of Table 2 test for impacts of NBP on carbon monoxide (CO), sulfur dioxide (SO<sub>2</sub>), and nitrogen dioxide (NO<sub>2</sub>). Online Appendix Figure 4, panel B, shows the corresponding event study graphs. CO emissions come primarily from transportation, so it is not surprising that the regressions fail to find evidence that the NBP affected CO emissions; the graphs bear this out. Further, there is no regression evidence of an impact on SO<sub>2</sub>, though the event study graph has some evidence of pre-trends differences for this outcome. NO<sub>x</sub> is a standard term used to describe a mix of two compounds: nitric oxide (NO) and NO<sub>2</sub>, a pollutant subject to its own regulations. Row 8 shows that the NBP decreased ambient NO<sub>2</sub> levels by 2–10 percent relative to the baseline, though NO<sub>2</sub> has limited or possibly no effect on health (Lippman 2009). The event study graph shows some decrease though is less clear than for ozone.

The impact of the NBP on particulates concentrations is of special interest because particulates can result from NO<sub>x</sub> emissions and are widely believed to be the most dangerous air pollutant for human health (Chay and Greenstone 2003; Pope, Ezzati, and Dockery 2009; Chen et al. 2013). Further, before its implementation, the EPA estimated that 48–53 percent of the projected health benefits from the NBP would come through the channel of reduced particulates concentrations (EPA 1998). The impact of emitted NO<sub>x</sub> on ambient particulate matter is theoretically ambiguous and depends on the level of other chemicals in the atmosphere (see online Appendix I).

Rows 9 and 10 in panel B of Table 2 empirically examine the impact of the NBP market on the concentrations of particles smaller than 10 micrometers (PM<sub>10</sub>) and 2.5 micrometers (PM<sub>2.5</sub>), both of which are small enough to be respirable. The PM<sub>10</sub> and PM<sub>2.5</sub> monitoring networks were just being erected in the late 1990s, so to have meaningful samples it is necessary to focus on the 2001–2007 period as in columns 4 and 5. Column 4, where the equation is weighted by the number of monitoring observations, provides limited evidence that the NBP affected particulate matter. Alternatively, when the equation is weighted by population, as is the case in the preferred defensive expenditures and health outcomes equations, the NBP is associated with an 8 percent reduction in PM<sub>2.5</sub>. Because PM<sub>2.5</sub> is believed to create substantial health damages, however, the implications of this number for human health may be larger than this modest change in ambient concentrations might suggest. In the smaller sample of counties with PM<sub>10</sub> monitors, we fail to find evidence of a statistically significant change in PM<sub>10</sub>. The results in rows 9 and 10 are inconclusive

<sup>18</sup>Online Appendix Figure 3, panel A, shows the number of days with ozone in each of six bins in the years before the NBP program began. Online Appendix Figure 3, panel B, shows event study graphs of the change in these counts due to the NBP. These graphs also show that the change in ozone was largely among days with 60–100 ppb, which are exactly the set of days that regulation targets.

about whether the NBP affected particulates concentrations. The event study graphs in online Appendix Figure 4, panel B, also show no clear evidence of a decrease in ambient particulates.

Overall, the large reduction in  $\text{NO}_x$  emissions caused by the NBP market and the rest of the evidence in Table 2 is generally supportive of the premise that the effect of the NBP on health occurs primarily through its effect on ozone concentrations (see additional sensitivity analyses in online Appendix V). Pollutants with important effects on health such as CO and  $\text{SO}_2$  were unaffected by the NBP. However, the mixed estimates of the effect of the NBP on  $\text{PM}_{2.5}$  (some statistically significant, some not) suggest that the subsequent 2SLS estimates of the effects of ozone on defensive expenditures and health outcomes derived from the variation in ozone induced by the NBP should be interpreted cautiously, because they may reflect the impact of ozone or particulates, or a combination of the two pollutants. We therefore focus more on the instrumental variables estimates of the effects of  $\text{NO}_x$  emissions on medication purchases and mortality.

### C. Defensive Investments

Table 3 statistically summarizes the reduced-form effect of the NBP market on log medication costs. The richest specification in columns 3 and 4 indicates that the NBP reduced total medication costs by 1.6 percent. The estimate is precise with the full set of controls and has similar magnitude but less precision with less detailed controls.<sup>19</sup> Finally, it is worth noting that the column 4 estimate is derived from the subsample of counties with ozone pollution monitors, which is used for the instrumental variables estimation below; this reduces the sample size from 30,926 to 2,338.

Figure 3, panel A, shows the event study graph for log of respiratory and cardiovascular medication expenditures from the estimation of equation (5). The event study suggests that the NBP market decreased medication expenditures in these categories by nearly 2 percentage points. This impact was roughly constant and is marginally significant in individual years. Importantly, there is no evidence of meaningful differences in the trend in summertime medication purchases between NBP and non-NBP states in advance of the market's initiation. The picture is broadly similar though less precise for the smaller set of firms available over the period 2000–2007 (online Appendix Figure 4, panel B).

We also measure medication purchases separately by cause. As discussed above, the allocation of medications to causes is inexact; doctors can prescribe a medication for many purposes, and the MarketScan data do not identify the cause for which a specific medication was prescribed. The goal of this exercise is to test whether the decline in medication purchases was evident among respiratory and cardiovascular medications (although the imprecision of the assignment of causes to medications means that there are good reasons to expect an impact in other categories). The

<sup>19</sup>County-by-year fixed effects add precision in these estimates. Because the medication data are from MarketScan and represent workers in the balanced panel of firms, county-by-year fixed effects address both local labor market shocks and firm- and factory-specific events like layoffs or mass hiring. Consistent with GLS providing an efficient response to heteroscedasticity, the unweighted estimate for log medication costs per capita is similar but less precise, at  $-0.011$  (0.017).

TABLE 3—EFFECT OF THE NBP MARKET ON LOG MEDICATION COSTS PER CAPITA

	(1)	(2)	(3)	(4)
<i>Panel A. All medications</i>				
1. All medications	−0.008 (0.010)	−0.015 (0.008)	−0.016 (0.006)	−0.016 (0.007)
MarketScan as share of total population	0.007	0.007	0.007	0.002
Counties as share of total population	0.994	0.994	0.994	0.370
<i>Panel B. Specific types of medications</i>				
2. Respiratory or cardiovascular	−0.009 (0.013)	−0.019 (0.011)	−0.023 (0.008)	−0.019 (0.009)
3. Non-respiratory and non-cardiovascular	−0.009 (0.010)	−0.015 (0.008)	−0.015 (0.006)	−0.017 (0.007)
County-by-season fixed effects	X	X	X	X
Summer-by-year fixed effects	X	X	X	X
State-by-year fixed effects	X	X		
County-by-year fixed effects			X	X
Detailed weather controls		X	X	X
Only counties with ozone monitors				X
Weighted by population	X	X	X	X

*Notes:* Medication costs are reported in 2015 dollars and deflated using the US CPI for urban consumers. The entries in Table 3 are the coefficient estimates from the DDD estimator described in equation (4) when the dependent variable is the log of medication costs per person-season-year in a county. Each coefficient is from a separate regression that includes a full set of county  $\times$  year, season  $\times$  year, and county  $\times$  season fixed effects. Additional control variables are listed in the bottom of Table 3. The reported standard errors are clustered at the state-season level. The regressions are GLS weighted by the MarketScan population in a given county-year-season. The reported standard errors are clustered at the state-season level. Total population refers to the 2,539 counties in the main sample. Number of observations is as follows: Row 1, columns 1–3: 30,926. Row 1, column 4: 2,338. Row 2, columns 1–3: 28,910. Row 2 column 4: 2,324. Row 3 columns 1–3: 30,730. Row 3 column 4: 2,338.

column 3 estimate in row 2 indicates that the NBP decreased expenditures on respiratory and cardiovascular medications by a statistically significant 2.3 percent. In the smaller sample of counties with ozone monitors in column 4, the point estimate is similar to column 3. Expenditures on all other medications also declined in all specifications. In the richest specification of column 3, this decline is 1.5 percentage points and is modestly smaller than the decline for respiratory and cardiovascular medications. Event study graphs for non-cardiovascular and non-respiratory medications show less evidence of a change than is apparent for cardiovascular and respiratory medications (online Appendix Figure 4, panel B).

An important question is the extent to which medications are a defense rather than just another health expense. Almost all of the previous literature reports on direct health outcomes (e.g., mortality rates, incidence of asthma attacks, and lung functioning). Following guidance from the medical literature (e.g., Fanta 2009), our paper's argument is that all of these health conditions are a function of ambient pollution *and* compensatory adaptations or defenses that include pharmaceutical purchases and a wide range of other costly actions. The share of WTP accounted for by these defenses has essentially been unmeasured previously across a wide variety of settings; as a result, current measures of WTP are incomplete and downward biased by an unknown magnitude. Online Appendix II discusses this question in detail, and while we argue that all medications are defensive, online Appendix Table 2 reports results indicating that the NBP led to reductions in purchases of both short-acting



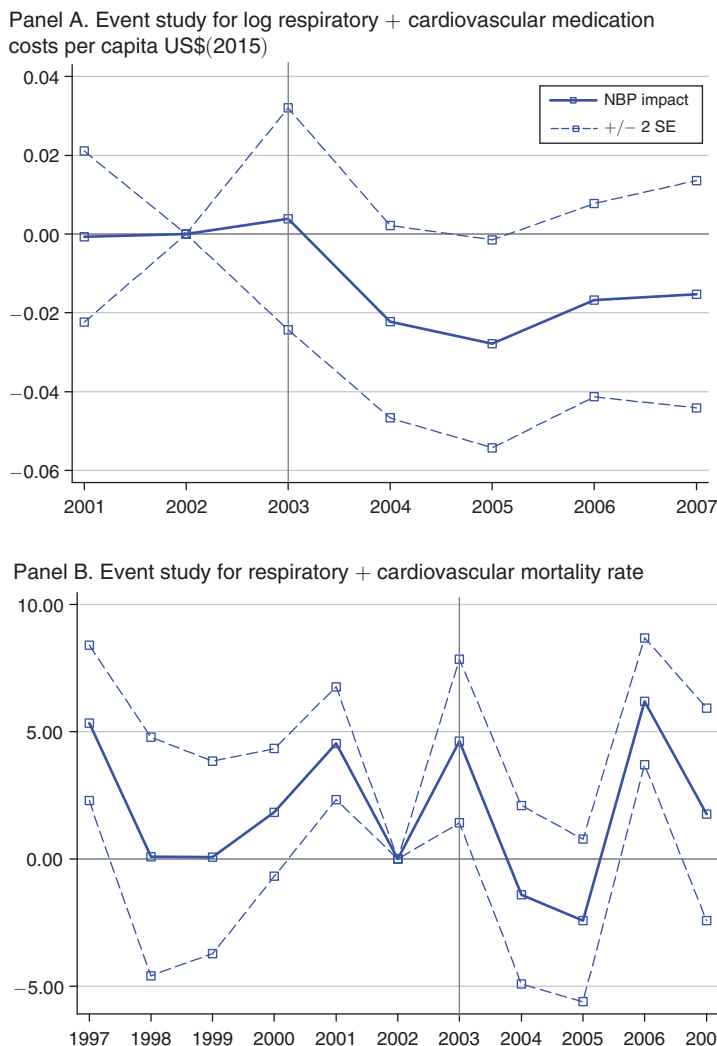


FIGURE 3. IMPACT OF THE NBP MARKET ON DEFENSIVE EXPENDITURES HEALTH OUTCOMES

*Notes:* The estimates in Figure 3, panel A, are from an event study regression for log medication costs per capita (respiratory and cardiovascular medications only), and the estimates in Figure 3, panel B, are from an event study regression for mortality rates per 100,000 population (respiratory and cardiovascular causes only). In both regressions, the estimates for year 2002 are restricted to have a value of 0. The regressions include detailed weather controls and a full set of county  $\times$  year, season  $\times$  year, and county  $\times$  season fixed effects, and are weighted by county population (panel A uses MarketScan population and panel B uses total population). The standard errors underlying the confidence intervals (dashed lines) are clustered at the state-season level. Medication costs are in 2015 dollars, deflated using the BLS CPI for urban consumers.

acute and long-acting control respiratory medications; both are negative though the long-term control estimate is more precisely estimated.

#### D. Hospital Visits and Mortality

*Hospital Visits.*—Because we seek to compare defensive costs against direct health costs, we also measure how the market affected hospital visits and mortality. Due to

the large number of county-year-season observations with zero hospitalization costs, we focus on the level rather than the log of per capita hospitalization costs.

Overall, our conclusion from these results is that most specifications do not detect meaningful effects of the NBP on hospitalization costs and we do not pursue this outcome further (online Appendix Table 3). We emphasize however that the MarketScan data exclude uninsured, Medicare, and Medicaid patients. These groups are included in some studies which find effects of ozone on hospitalization (Currie and Neidell 2005, Lleras-Muney 2010), and are believed to experience the largest impacts from high ambient ozone levels (ALA 2013). For these reasons, estimates of the effect of the NBP market on hospitalization (and potentially medications) could significantly understate population average effects.<sup>20</sup>

*Mortality.*—In most analyses of air pollution, mortality accounts for the largest share of the regulatory benefits. The richest specifications in columns 3–5 row 1 of Table 4 suggest that the NBP market decreased the all-cause, all-age summertime mortality rate by about 1.7 to 3.0 deaths per 100,000 population, depending on the sample, and would generally be judged to be statistically significant. The effect in the subsample of counties with ozone monitors is larger (see column 4), indicating a reduction of 6.0 deaths per 100,000 population.

Rows 2 through 4 of Table 4 divide the overall mortality rate by cause of death. Reading across columns 3 and 4 of row 2, it is apparent that 24 percent to 41 percent of the decline in overall mortality is concentrated among cardiovascular/respiratory deaths. Row 3 finds that the NBP also significantly decreased death from non-respiratory and non-cardiovascular causes. The difference between the effects in row 2 and row 3 is generally around one standard error, and the estimates are statistically indistinguishable.

Research on other pollutants finds that most of the health consequences of particulate matter are concentrated among respiratory and cardiovascular causes, although pathways for ozone are less well understood. The finding that the NBP affected respiratory and cardiovascular in addition to other causes is consistent with two hypotheses. One is that the NBP was correlated with unobserved shocks which affected mortality, and the second hypothesis is that the NBP itself caused these changes in mortality. Row 4 of Table 4 provides an important fact in support of the second hypothesis. Row 4 shows that the market had no effect on external (primarily accidental) deaths, which is a reassuring placebo test.

Panel C of Table 4 breaks the entire population into four age groups and separately estimates the effect of the NBP on each group's mortality rate using the full sample. The richest sample and specification in column 3 detects no statistically significant effect on the mortality of persons aged 74 and below, although the point estimates imply that the market prevented 700 deaths within this group. The largest point estimate for mortality occurs among people aged 75 and older. These results suggest that the NBP market prevented about 1,300 deaths each summer among people 75 and older. This finding is confirmed visually by the event study graph in

<sup>20</sup> At the same time, the MarketScan medication and hospitalizations data include insured groups that may be more prone than uninsured individuals to incur expenditures in response to health risks (Finkelstein et al. 2012).

TABLE 4—EFFECT OF THE NBP EMISSIONS MARKET ON MORTALITY RATES

	(1)	(2)	(3)	(4)	(5)
<i>Panel A. All deaths</i>					
1. All deaths	-2.07 (0.94)	0.90 (3.58)	-1.70 (0.83)	-5.97 (1.77)	-3.03 (1.53)
<i>Panel B. Specific causes of death</i>					
2. Respiratory or cardiovascular	-0.71 (0.50)	0.45 (1.65)	-0.41 (0.67)	-2.42 (1.17)	-1.08 (0.95)
3. Non-respiratory and non-cardio.	-1.36 (0.57)	0.46 (2.15)	-1.29 (0.46)	-3.55 (0.79)	-1.95 (0.91)
4. External	0.32 (0.20)	0.22 (0.32)	0.22 (0.30)	-0.18 (0.62)	0.15 (0.38)
<i>Panel C. All causes of death, by age group</i>					
5. Age 0 (infants)	-1.66 (3.84)	-5.92 (6.06)	-5.48 (6.03)	-3.58 (10.61)	-11.64 (9.23)
Estimated change in 2005 deaths	-29	-102	-94	-62	-201
6. Ages 1–64	-0.05 (0.32)	-0.77 (0.82)	-0.26 (0.43)	-0.58 (1.13)	-0.56 (0.86)
Estimated change in 2005 deaths	-58	-897	-303	-676	-653
7. Ages 65–74	-3.16 (4.35)	-1.73 (10.22)	-3.40 (6.18)	-12.38 (9.94)	-6.61 (6.25)
Estimated change in 2005 deaths	-281	-154	-303	-1,103	-589
8. Ages 75+	-29.10 (8.14)	-2.37 (21.48)	-16.08 (9.82)	-82.56 (16.88)	-28.83 (15.91)
Estimated change in 2005 deaths	-2,496	-2,340	-1,275	-7,647	-1,509
County-by-season fixed effects	X	X	X	X	X
Summer-by-year fixed effects	X	X	X	X	X
State-by-year fixed effects	X	X			
County-by-year fixed effects			X	X	X
Detailed weather controls		X	X	X	X
Counties with ozone monitors				X	
Data begins in 2001					X

*Notes:* The entries in Table 4 are the coefficient estimates from the DDD estimator described in equation (4) where the dependent variable is deaths per 100,000 population in each county-year-season. Each coefficient is from a separate regression that includes a full set of county  $\times$  year, season  $\times$  year, and county  $\times$  season fixed effects. Additional control variables are listed in the bottom of Table 4. The reported standard errors are clustered at the state-season level. The regressions are GLS weighted by the relevant population in a given county-year-season. Unless noted otherwise, the data begin in 1997. Number of observations is 55,858 for columns 1–3; 3,146 for column 4; and 35,546 for column 5.

Figure 3, panel B, although the estimates from individual years are noisy, and by the age-specific analyses in online Appendix Figure 4, panel B.

The age-group decomposition implies that the NBP prevented 1,975 summer deaths annually. About 65 percent of these were among people aged over 75. By contrast, the overall share of all summer deaths which occur among people aged over 75 is 55 percent, suggesting that the elderly disproportionately benefited from the NBP.

An important question that Table 4 leaves unanswered is the gain in life expectancy associated with these delayed fatalities. Indeed, the question of the magnitude of gains in life expectancy is unanswered in almost all of the air pollution and health literature because it is largely based on changes in mortality rates over relatively short periods of time (e.g., a few days or a week). The difficulty is that it is possible and perhaps likely that the relatively sick benefited and that their lifespans were

extended only modestly, given their age. In the extreme, the NBP might merely have moved the date of these deaths to the winter months immediately following the market.<sup>21</sup>

We explored two approaches to investigate the empirical relevance of this short-term “seasonal” displacement hypothesis. First, we experimented with redefining each “year” to begin on May 1 of one calendar year and conclude on April 30 of the following calendar year. This redefined “year” compares each summertime season against the seven following months. Second, we estimated differences-in-differences regressions where each observation represents a calendar year (as opposed to a calendar-season-year), and where we measure the change in mortality rates by NBP status pre versus post. We also combined these two approaches to estimate differences-in-differences models with the restructured year.

These approaches do not provide strong support for the short-term displacement hypothesis. In most cases, the estimated effect of the market on mortality was negative and had similar magnitude to the models reported in the paper, but these estimates were imprecise and we could not reject the null hypothesis that the NBP had no long-run impact on mortality. Overall, we conclude that this research design lacks power to measure the effect of ozone on life expectancy beyond the five month length of the NBP’s summer season. Nevertheless, this paper’s focus on the summertime mortality rate is an advance from the previous literature that has primarily estimated how ozone affects same-day or same-week mortality rates.<sup>22</sup>

### E. *Instrumental Variables (IV)*

The preceding sections measure the reduced-form effects of the NBP market on pollution, defenses, and health. We now turn to an IV approach to measuring the effect of NO<sub>x</sub> emissions and ozone on defensive expenditures and mortality rates. The interpretation of the IV estimates of the effect of NO<sub>x</sub> generated by the NBP market variation as causal is straightforward: NO<sub>x</sub> is a pollutant controlled by regulation, and the estimated effects on health and defenses are a direct result of the quasi-experimental change in NO<sub>x</sub> emissions. Table 2 showed that changes in NO<sub>x</sub> are the primary channel for the large changes in ambient ozone concentrations. However, changes in NO<sub>x</sub>—depending on the model specification—also can lead to changes in other ambient pollutants, including PM<sub>2.5</sub>. Thus we underscore that instrumental variable estimates of the effect of ozone should be interpreted more cautiously.

We report IV estimates for the effect of NO<sub>x</sub> on health for two different geographic samples: all counties, and the 24 percent of NBP counties with positive NO<sub>x</sub> emissions in summer 2002 that account for 44 percent of these states’ population. We refer to the second sample as “Counties with NO<sub>x</sub> Emissions.” Most counties lack power plants that produce NO<sub>x</sub> emissions so the NBP could not have affected emissions in these counties, except by deterring entry; relatedly, the first-stage

<sup>21</sup>The paper’s triple difference estimator compares summer and winter deaths within a year. If some of the deaths are displaced from summer to October–December of the same year, then the estimator will overstate the decline in mortality.

<sup>22</sup>Currie and Neidell (2005) is an exception since they estimate monthly and quarterly mortality regressions.

estimate of the NBP's effect on  $\text{NO}_x$  is more powerful when excluding these zeros. We emphasize estimates from the second sample, since it is *ex ante* expected to have more statistical power. Of course we can only estimate IV regressions for ozone using the counties with ozone monitors.

The first row of panel A of Table 5 reports fixed effects estimates of the association between  $\text{NO}_x$  emissions and medication purchases (columns 1–2) and between measures of the all-age mortality rate (columns 3–4). Rows 2 and 3 repeat the exercise for two different measures of ambient ozone. The estimates are from separate regressions of the outcome on alternative measures of  $\text{NO}_x$  emissions (or ozone concentrations) and are adjusted for county-by-season fixed effects, county-by-year fixed effects, season-by-year fixed effects, detailed weather controls, and each observation represents a county-year-season as in the prior analysis. Most of these estimates are statistically insignificant, and exhibit sign and magnitude variability (including perversely signed coefficients on mortality), suggesting little evidence of systematic effects on medication purchases or mortality rates.

Panel B reports on the two-stage least squares (2SLS) or instrumental variables (IV) estimates that are adjusted for the same controls as in the fixed effects specifications but the endogenous variables (i.e.,  $\text{NO}_x$  emissions in thousands of tons, average 8-hour ozone concentration, and the number of days equaling or exceeding 65 ppb) are instrumented using the quasi-experimental variation generated by the NBP market. That is, we use the variable  $1(\text{NBP Operating})_{\text{cst}}$  as an instrument for  $\text{NO}_x$  emissions, and for ambient ozone concentrations.

The entries indicate a strong relationship between  $\text{NO}_x$  emissions and medication purchases. For example, the estimates based on the sample of counties with positive  $\text{NO}_x$  emissions imply that a 10 percentage point decline in  $\text{NO}_x$  emissions relative to the Table 1 mean of 0.52 leads to a 0.06 percentage point reduction in spending on all medications. The estimates including all counties are larger, at 0.11 percentage points, though less precise. Both estimates are substantially larger in magnitude than the analogous OLS ones in panel A, which is consistent with the possibilities that the OLS estimates are plagued by substantial confounding and that  $\text{NO}_x$  emissions are measured with error. The ozone entries imply that a 10 percentage point decline in the average 8 hour ozone measure and a 10 percentage point decline in days with ozone concentrations exceeding 65 ppb reduces all medication spending by 2.7 and 0.36 percentage points, respectively (both statistically significant).

The IV mortality estimates in columns 4a and 4b also imply large mortality effects of  $\text{NO}_x$  emissions and ozone concentrations.<sup>23</sup> The estimates based on counties with  $\text{NO}_x$  emissions suggest that a 1 million ton increase in  $\text{NO}_x$  emissions leads to 2.7 additional summertime deaths per 100,000 people, an estimate which borders on statistical significance, or that a 10 percentage point decline in  $\text{NO}_x$  leads to a reduction in the mortality rate of 0.04 percentage points. The estimates for all

<sup>23</sup>The first-stage regression for column 3 of Table 5 corresponds to column 5 of Table 2. For the other columns of Table 5, the first stage regressions are as follows: for column 1,  $-0.74$  (0.27); for column 2a,  $-1.55$  (0.44); for column 2b,  $-2.87$  (0.63) and  $-10.50$  (2.14); for column 4a,  $-0.70$  (0.26); for column 4b,  $-2.91$  (0.62) and  $-8.27$  (2.05). The sample and weighting (MarketScan versus total population) varies across these regressions. Online Appendix Table 7 reports 2SLS estimates separately for respiratory and cardiovascular causes (panel A), and for non-respiratory and non-cardiovascular causes (panel B). These 2SLS estimates by cause are somewhat less precise than the all-cause estimates in Table 5, though they still show that a substantial portion of the health consequences of  $\text{NO}_x$  emissions or ozone exposure come through respiratory and cardiovascular causes.

TABLE 5—EFFECT OF NO<sub>x</sub> EMISSIONS AND AMBIENT OZONE CONCENTRATIONS ON MEDICATION PURCHASES AND MORTALITY: OLS AND IV ESTIMATES

	log medication costs			All-cause mortality		
	All counties	Counties with NO <sub>x</sub> emissions	Ozone monitored counties	All counties	Counties with NO <sub>x</sub> emissions	Ozone monitored counties
	(1)	(2a)	(2b)	(3)	(4a)	(4b)
<i>Panel A. OLS</i>						
NO <sub>x</sub> emissions	0.08 (1.13)	0.48 (1.06)	— —	−0.02 (0.29)	−0.02 (0.29)	— —
8-hour ozone	— —	— —	0.39 (0.86)	— —	— —	0.10 (0.15)
Days ≥ 65 ppb	— —	— —	0.20 (0.14)	— —	— —	−0.01 (0.03)
<i>Panel B. 2SLS</i>						
NO <sub>x</sub> emissions	21.68 (13.59)	12.28 (5.77)	— —	5.28 (3.65)	2.73 (1.84)	— —
8-hour ozone	— —	— —	5.65 (2.05)	— —	— —	1.96 (1.01)
Days ≥ 65 ppb	— —	— —	1.54 (0.58)	— —	— —	0.69 (0.37)

*Notes:* The coefficient estimates in columns 1, 2a, and 2b are multiplied by 1000 for readability. Columns 1, 2a, and 2b are based on the 2001–2007 sample; columns 3, 4a, and 4b are based on the 1997–2007 sample. NO<sub>x</sub> emissions are measured in thousand tons per county. All regressions include county × year, season × year, and county × season fixed effects, as well as the detailed weather controls. The reported standard errors are clustered at the state-season level. The regressions are GLS weighted by the relevant population in a given county-year-season (MarketScan or full population). In panel B, the endogenous variable is NO<sub>x</sub> or ozone and the excluded instrument is Summer × Post × NBP interaction (see equation (6)). Number of observations is 30,926 for medication regressions including all counties, 7,616 for medication regressions including counties with NO<sub>x</sub> emissions, 2,338 for medication regressions including only counties with ozone monitors, 55,858 for mortality regressions including all counties, 12,320 for mortality regressions including counties with NO<sub>x</sub> emissions, and 3,146 for mortality regressions only including counties with ozone monitors. The sample of counties with NO<sub>x</sub> emissions include those with nonzero emissions in the summer of 2002. The sample is smaller for medications than for mortality due to counties without no medication data or zero expenditures.

counties are somewhat larger. The estimates also indicate that a 1 ppb increase in the 8 hour ozone concentration or 1 additional day with a concentration exceeding 65 ppb lead to 1.96 and 0.69 additional summertime deaths per 100,000 people, respectively; correspondingly, a 10 percentage point increase in 8-hour ozone or in days with ozone concentrations exceeding 65 ppb leads to a 2 or an 0.41 percentage point increase in summertime deaths per 100,000 people, respectively.<sup>24</sup>

To the best of our knowledge, this is the first paper to develop plausibly causal estimates of the relationships between NO<sub>x</sub> emissions with health and defensive investments.<sup>25</sup> Further, if it is appropriate to interpret the IV ozone estimates as

<sup>24</sup>For mortality, the values in Table 5 include the full period 1997–2007. We can also estimate results restricted to years 2001–2007, and obtain generally similar results. With this shorter time period, the 2SLS estimate for the mortality effect of NO<sub>x</sub> emissions in all counties is 4.66 (4.03), or 4.72 (3.07) in counties with NO<sub>x</sub> emissions. The 2SLS estimates for 8-hour ozone and ozone days above 65 ppb are 2.15 (1.31) and 0.71 (0.48), respectively.

<sup>25</sup>Holland et al. (2016) apply the Muller and Mendelsohn (2009) model to simulate the consequences of emissions from Volkswagen vehicles. They do not estimate regressions directly linking observed NO<sub>x</sub> emissions to observed outcomes like health.



causal, they would substantially alter our understanding of the welfare consequence of exposure to ozone. For example, the most prominent ozone-mortality study (Bell et al. 2004) finds an elasticity of weekly ozone with respect to daily mortality rates that is smaller than the elasticity implied by Table 5.

## VI. Calculating Welfare Impacts

This paper's results allow us to conduct a simple cost-benefit analysis for the entire NBP, with the caveat that data restrictions prevent us from measuring all health outcomes and defensive expenditures. The estimates in Table 2 imply that the NBP market decreased  $\text{NO}_x$  emissions by 427,000 tons per summer on average and the average cost of a  $\text{NO}_x$  permit was \$2,523/ton.<sup>26</sup> The permit price should reflect an upper bound on abatement costs per ton, because firms should only use abatement technologies that cost less than the permit price. Thus, an upper bound estimate is that the market caused firms to spend US\$(2015)1,076 million annually to abate  $\text{NO}_x$  (Table 6, column 5). Defining 2003 to have half a year of typical abatement costs, we obtain an upper bound on 2003–2007 total abatement costs of \$4.8 billion ( $= 1,076 \times 4.5$ ).

We now turn to estimating the NBP's social benefits. As we discussed above, it may seem natural to assume that a change in pharmaceutical purchases are simply a transfer from consumers to pharmaceutical firms and thus have zero social cost. However, reductions in air pollution concentrations decrease the demand for medications that protect individuals from air pollution. Dynamically, this decline in demand will reduce the resources used to develop these medication types and will allow these resources to be applied to more productive uses. We are unaware of an empirically validated approach to socially valuing this reduction in drug purchases but believe that it is defensible to assume that it is valued at their full cost, especially over long time horizons; Table 6 adopts this assumption, and also reports the total change in copayments.

Column 1 of Table 6, panel B, reports the average annual reduction in medication expenditures, as well as the sum over the NBP's life. Specifically, we take the estimated 1.6 percent reduction in medication purchases from the regression result in column 3 and row 1 of Table 3 and multiply that by the annual mean medication purchases. This calculation suggests that the NBP market led to a decrease in medication expenditures of \$820 million per year or \$3.7 billion when summed over the 4.5 years that the NBP operated. It is unclear whether this extrapolation from the MarketScan population under- or over-states the effect on the full population.

The Table 4 panel C mortality estimates imply that the market prevented about 1,975 deaths per summer. The value of a statistical life (VSL) determines the monetary value assigned to these deaths. To provide one approach to monetization, we use Ashenfelter and Greenstone's (2004) upper bound VSL of US\$(2015)2.27 million for a prime age person and Murphy and Topel's (2006) method to develop estimates of the VSL for each age group in our analysis. This adjustment is especially consequential in this setting where the avoided fatalities are largely among individuals

<sup>26</sup>The effect on  $\text{NO}_x$  emissions is calculated by multiplying the estimated impact of NBP on  $\text{NO}_x$  emissions (−0.36) in thousand tons from Table 2, column 3, by the number of counties in the NBP (1,185).

TABLE 6—THE WELFARE IMPACTS OF THE NBP AND THE SOCIAL BENEFITS OF NO<sub>x</sub> AND OZONE REDUCTIONS

	Medication costs (\$ million)	Medication copayments (\$ million)	Mortality:		Total using (1) (\$ million)	Total using (2) (\$ million)
			Number of deaths (3)	Monetized value (\$ million) (4)		
	(1)	(2)	(3)	(4)	(5)	(6)
<i>Panel A. An upper bound estimate of NBP's social costs</i>						
Upper bound per year	—	—	—	—	\$1,076	\$1,076
Upper bound, 2003–2007 total	—	—	—	—	\$4,843	\$4,843
<i>Panel B. Estimates of the NBP's benefits</i>						
Total per year	\$820	\$161	1,975	\$1,319	\$2,139	\$1,480
Total 2003–2007	\$3,690	\$725	8,887	\$5,935	\$9,625	\$6,660
<i>Panel C. The annual social benefits of NO<sub>x</sub> reductions in NBP states (million tons)</i>						
Regressions based on . . .						
All counties	\$938	\$178	6,048	\$4,039	\$4,977	\$4,217
Counties with NO <sub>x</sub> emissions	\$531	\$103	3,127	\$2,088	\$2,620	\$2,192
<i>Panel D. The social benefits of ozone reductions in NBP states (ppb)</i>						
1 ppb ozone decrease	\$290	\$41	2,660	\$1,777	\$2,066	\$1,818
1 Less day with ozone > 65 ppb	\$79	\$11	937	\$625	\$704	\$637

Notes: All dollar amounts are in 2015 constant dollars deflated using BLS CPI for urban consumers. The mortality impact estimates without dollar signs are number of deaths. The monetized mortality impact uses the VSL of \$2.27 million (2015 dollars) from Ashenfelter and Greenstone (2004) and the age adjustments from Murphy and Topel (2006, p. 888). The implied VSLs are as follows: \$2.26 million (infants); \$1.78 million (age 1–64); \$0.7 million (age 65–74); and \$0.3 million (age 75+). Total 2003–2007 decrease due to NBP assumes impact is for half of 2003 summer and for all of summers 2004–2007. NBP cost upper bound is based on the permit price of about \$2,523/ton US\$(2015) and estimated total abatement quantity of 427,000 tons. The numbers in panel A comes from multiplying together the mean NBP allowance price per ton, the effect of the NBP on county-level NO<sub>x</sub> emissions (Table 2, column 4), and the number of counties in the NBP states (1,185). The numbers in panels B–D come from multiplying together regression estimates of how the NBP, NO<sub>x</sub>, or ozone affects medication costs or mortality by the total number of people in the NBP states in the year 2005 (136 million people). Specifically, panel B, column 1, uses the estimate from Table 3, column 3. Panel B, columns 2–3 use the estimates from Table 4, column 3, panel C. Panel C, column 1 uses the estimate from Table 5, column 1, panel B. Panel C, columns 2–3 use the estimate from Table 5, panel B, column 3. Panel D, column 1 uses the estimate from Table 5, panel B, column 2. Panel D, columns 2–3 use the estimate from Table 5, panel B, column 4. Panel D estimates are based on regressions using counties with ozone monitors. All estimates apply to the full population in NBP states. See the text for further details.

75 and over. The implied VSLs are as follows: \$2.3 million (infants), \$1.78 million (ages 1–64), \$0.7 million (ages 65–74), and \$0.3 million (ages 75+). The application of this approach implies that the value of the mortality avoided by the NBP is \$1.3 billion per year, or \$5.9 billion in the period 2003–2007 (Table 6, panel B).<sup>27</sup>

The entries in panels A and B of Table 6 provide the basis for a comparison of the costs and benefits. The upper bound on the NBP's aggregate abatement costs is \$4.8 billion, but by themselves the value of the reduced medication purchases of \$3.7 billion nearly equals these costs. At least in this context, defensive investments are economically important. Once the value of the reduced rates of mortality is added in, the benefits of the market are nearly twice as large as the upper-bound of

<sup>27</sup> We thank Kevin Murphy and Bob Topel for sharing the data underlying Figure 3 of their paper. The VSL used here is lower than the \$8.7 million VSL (\$2015) used by the EPA, which is not age-adjusted. Our primary goal is not to endorse a specific VSL value, but to demonstrate the results that come from one choice of VSL and age-adjustment. Using the \$8.7 million VSL rather than the \$2.27 million VSL implies that the mortality benefits of NBP were larger: \$5.0 billion per year, or \$22.7 billion for the 2003–2007 total.

its abatement costs (i.e., \$9.6 billion in benefits and \$4.8 billion in costs). Thus the NBP's social benefits easily exceeded its abatement costs.

We consider two alternatives to the benefit-cost analysis in column 5, Table 6. An alternative measure of medication costs is copayments. The log change in copayments is almost identical to the log change in total medication expenditures (see online Appendix Table 2). Copayments represent 21 percent of the total payment for medications (Table 1). Total copayment savings per year are \$160 million, or \$725 million over the 2003–2007 NBP. A second alternative is to calculate the upper-bound on the NBP market cost using other measures of the NBP allowance price. An alternative estimate of market costs of \$3,000 per ton implies an upper bound cost of \$1.3 billion per year, or \$5.8 billion total over the 2003–2007 period.<sup>28</sup> Under both alternatives, medication costs represent an economically important proportion of the benefits and costs of the NBP market, and excluding medication expenditures (which are only one component of defensive investments) would substantially understate the market's benefits. The magnitude of the understatement varies with these assumptions.

Estimates of WTP for reductions in NO<sub>x</sub> emissions have considerable policy relevance since NO<sub>x</sub> is the pollutant that policymakers can regulate directly, whereas ozone is only formed through complicated chemical reactions involving other pollutants. Table 6, panel C, reports on estimated WTP for a reduction of one million tons of NO<sub>x</sub> emissions and its component parts.<sup>29</sup> Based on estimates from counties with NO<sub>x</sub> emissions, each 1 million ton decrease in summertime NO<sub>x</sub> emissions in the NBP states annually saves about \$0.5 billion in medication expenditures and roughly 3,100 premature summertime deaths, with an estimated value of \$2.1 billion in mortality benefits (Table 6, panel C); the total WTP is thus about \$2.6 billion. The alternative estimated WTP of \$5.0 billion, based on estimates from the full sample of counties, is also quite large. These figures are underestimates if other categories of well-being or defensive expenditures respond to changes in NO<sub>x</sub> emissions.

Table 6 also reports estimates of WTP for a reduction in ozone, but they must be interpreted cautiously due to uncertainty about the validity of the exclusion restriction.<sup>30</sup> The IV ozone results suggest that each 1 ppb decrease in the mean 8-hour summer ozone concentration in the NBP states is worth approximately \$2.1 billion in social benefits annually. Similarly, one fewer day per summer in the NBP states with an ozone concentration exceeding 65 ppb would yield roughly \$700 million of benefits annually (Table 6, panel D).

<sup>28</sup>This value slightly exceeds the average allowance price in the years 2002–2006. When the market opened in May 2003, allowance prices of different vintages ranged from \$2,250 to \$5,000 per ton. Prices of all vintages fell rapidly during the first year to below \$3,000. In the following years 2004–2006, allowance prices were fairly stable at between \$2,000 and \$3,000 per ton. In 2007 all allowance prices declined to \$1,000 per ton.

<sup>29</sup>The value in column 2 comes from multiplying together the IV estimates of the effect of NO<sub>x</sub> emissions on log medication costs (Table 5), the mean medication expenditure per person-season (Table 1), and the population in the NBP states in the year 2005 (136 million). The value in column 3 comes from multiplying the IV estimates of the effects of NO<sub>x</sub> emissions on the mortality rate (Table 5) by the total population in the NBP states. The value in column 4 comes from by taking the mean WTP per death prevented from panel B of Table 5, and multiplying it by the change in number of deaths from column 3 of Table 6.

<sup>30</sup>The approach for calculating the ozone benefits in panel D of Table 6 is similar to the methodology for calculating the NO<sub>x</sub> benefits in panel C of Table 6, and is described above. It is worth noting that estimates of the benefits of the NBP, NO<sub>x</sub> emissions, and ozone for mortality and medications do not have identical samples since not all counties have ozone monitors, and that medications data are available only beginning in 2001.

## VII. Conclusions

Theoretical models make clear that WTP for well-being in a variety of contexts is a function of factors that enter the utility function directly (e.g., the probability of mortality, school quality, etc.) *and* the costly investments that help to determine these factors. One approach to developing measures of WTP is to find a single market that captures individuals' full valuation, as can be the case with property markets under some assumptions (see, e.g., Chay and Greenstone 2005; Greenstone and Gallagher 2008; Keiser and Shapiro 2017). All too frequently though, the data and/or a compelling research design for the key market outcomes are unavailable, making it necessary to develop measures of WTP by summing its components.

However, across a wide variety of applied literatures, the empirical evidence on WTP has almost exclusively focused on the factors that enter the utility function directly. The resulting measures of WTP are thus generally underestimated and the extent of this underestimation is unknown. This paper has demonstrated that defensive expenditures are an important part of WTP for air quality. Indeed, in the context of the NO<sub>x</sub> Budget Program, the improvement in air quality generates reductions in medication purchases that are close to an upper bound estimate of the abatement cost. A fruitful area for research is to explore whether individuals' compensatory behavior and resulting defensive investments account for such a large fraction of WTP in other settings.

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