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POLLUTION, HEALTH, AND AVOIDANCE BEHAVIOR:
EVIDENCE FROM THE PORTS OF LOS ANGELES

Enrico Moretti
Matthew Neidell

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ABSTRACT

A pervasive problem in the literature on the health costs of pollution is that optimizing individuals may compensate for increases in pollution by reducing their exposure to protect their health. This implies that estimates of the health effects of pollution may vastly understate the full welfare effects of pollution, particularly for individuals most at risk who have the greatest incentive to adopt compensatory behavior. Furthermore, using ambient monitors to approximate individual exposure to pollution may induce considerable measurement error. We overcome these issues by estimating the short run effects of ozone on respiratory related health conditions using daily boat arrivals and departures into the two major ports of Los Angeles as an instrumental variable for ozone levels. While daily variation in boat traffic is a major contributor to local ozone pollution, time-varying pollution due to port activity is arguably a randomly determined event uncorrelated with factors related to health. Instrumental variable estimates are significantly larger than OLS estimates, indicating the importance of accounting for avoidance behavior and measurement error in understanding the full welfare effects from pollution.

Enrico Moretti
University of California, Berkeley
Department of Economics
549 Evans Hall
Berkeley, CA 94720-3880
and NBER
moretti@econ.berkeley.edu

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

1. Introduction

Air pollution has long been recognized as a negative externality, but considerable debates have ensued over the optimal level of air quality, with few more contentious than the one surrounding ground-level ozone. A proposed 8-hour ozone standard issued by the EPA in 1997 was finally upheld by the Supreme Court in 2002, but only after endless appeals and lengthy lawsuits initiated by states and industry (Bergman, 2004). Furthermore, since ozone forms from interactions between nitrogen oxides (NOx) and volatile organic compounds (VOC) in the presence of sunlight and heat, climate change is expected to increase ozone levels (Racherla and Adams, 2009), making regulations surrounding ozone an area of increasing importance.

Ozone is presumed to have deleterious effect on health, especially for children, the elderly, and those with existing respiratory illnesses, but the exact magnitude is disputed. Part of this controversy stems from discrepancies over statistical approaches to estimate the health effects of ozone. Estimating this relationship is complicated by the fact that individuals may sort into neighborhoods based on the level of air quality, so that unobserved determinants of health may confound estimates of the relationship between health and pollution. For example, those with higher preferences for clean air or higher income may live in areas with better air quality, and these individuals may make other unobserved investments in health capital. Many quasi-experimental studies use exogenous shocks in pollution levels to overcome sorting concerns (see, for example, Chay and Greenstone, 2003a, 2003b; Chay, Dobkin and Greenstone, 2003; Friedman et al. 2001; Ransom and Pope 1995; Lleras-Muney, 2009; Jayachandran, 2009), but primarily identify long-run effects of pollution. Identifying short run effects is particularly important in the case of ozone because symptoms occur shortly after initial exposure – in as quickly as one hour[1] – and can result in serious and potentially deadly asthma attacks.

Even if one could isolate random variation in pollution, however, estimation is further complicated by the fact that individuals may adjust their exposure in response to changes in pollution. This concern is particularly relevant because individuals most at risk of being negatively affected by pollution have the greatest incentive to adopt compensatory behavior. Behavioral responses to ozone levels are unlikely to be trivial, especially in Southern California, the area we analyze. Since ozone is greatly affected by weather conditions, it is highly predictable using weather forecasts. In fact, daily ozone forecasts that inform the public of dangers from episodic ozone conditions are widely available in Los Angeles through television and newspapers. This allows individuals to engage in protective behavior by spending less time outdoors to offset some of the adverse consequences from ozone exposure, as empirical evidence supports (Neidell, 2009; Mansfield et al., 2006). If optimizing individuals compensate for changes in ambient ozone levels by reducing exposure, estimates that do not account for these responses will understate the full welfare effects of ozone.

An additional issue concerns measurement error in assigning pollution exposure to individuals. The most common approach for measuring exposure is to assign data from ambient air pollution monitors to the residential location of the individual using various interpolation techniques (see, for example, Bell et al. (2004); Dominici et al. (2000); Samoli et al. (2005)). Given the tremendous spatial variation in pollution within finely defined areas (e.g., Tin et al., 2001), this approach is likely to yield considerable measurement error. In fact, Lleras-Muney (2009) finds that not only are estimates sensitive to the interpolation technique, but the resulting

[1] Daily time-series studies that focus on short-term effects partly overcome residential sorting concerns, but, since sources of emission are not sufficient to explain the daily variation in ozone levels, it is unclear whether the factors

measurement error appears non-classical, making the direction of the bias on estimates ambiguous. While several epidemiological field studies address this concern by using personal ambient monitors (Tonne et al., 2004), these studies often involve very small samples that preclude the ability to obtain precise estimates of common outcomes of interest, such as hospitalizations.[2]

In this paper, we identify the short run effects of ozone by using daily data on boat arrivals and departures into the two major ports of Los Angeles as an instrumental variable for ozone levels. Three features make boat traffic an ideal instrument. First, boat traffic represents a major source of pollution for the Los Angeles region. The combined ports of Los Angeles and Long Beach, the largest in the US and third largest in the world, represent the single most polluting facility in the Los Angeles metropolitan area (Polakovic, 2002). Because most of these boats come from countries with much less stringent environmental regulations, they contain less sophisticated emissions technology than their U.S. counterparts and emit unusually high levels of nitrogen dioxides (NOx) – contributing to over 20% of all NOx emissions in the Los Angeles area (AQMD, 2002) – which gets carried inland to form ozone.[3] Our data confirm that boat traffic significantly affects daily ozone levels and, important for our identification, the effect of boat traffic on ozone levels declines monotonically with distance from the port.

Second, daily variation in boat traffic is arguably uncorrelated with other short run determinants of health. The overwhelming majority of boats arriving at the ports travel from

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that drive this variation have an independent effect on health.

[2] Note that one can also interpret avoidance behavior as a form of measurement error: ambient air quality from monitors will not reflect personal exposure if individuals intentionally limit their exposure as air quality worsens. Although we can not empirically distinguish between the two sources of bias, we choose to separate them because one has an economic interpretation while the other is purely statistical.

[3] In fact, emissions from the port have lead to numerous contentious debates, and a recent senate committee hearing on port pollution lead by Senator Barbara Boxer from California seeks an urgent, national response to port pollution. Evidence of the passion behind the debate is succinctly summarized by the following quote from Bob Foster, mayor

Latin America and Asia. Due to the great length of travel and unpredictable conditions at sea, these trips often take over a month's time with the exact date of arrival and departure difficult to predict. Therefore, the influx of pollution due to port activity is arguably a randomly determined event uncorrelated with factors related to health.

Third, and crucially, boat traffic is generally unobserved by local residents. The arrivals and departures are not included in ozone forecasts or reported by newspapers and local news outlets. Indeed, we find that ozone forecasts and participation in outdoor activities are empirically orthogonal to boat traffic. Therefore, it is difficult for individuals to respond to ozone levels as affected by boat traffic, suggesting that our instrumental variable estimate holds compensatory behavior fixed.

While it is in theory possible that any difference between instrumental variable estimates and OLS estimates is due to the fact the former reflect a particular local average treatment effect (LATE) – namely the impact of an increase in pollution due to port traffic on health – this possibility seems highly unlikely in our setting. The ozone created by emission from boats is chemically identical to the ozone created by emissions from all other sources (cars, power plants, etc.). Furthermore, ozone levels as affected by our instrument are quite comparable to the overall mean level of ozone at the port.[4] It is therefore plausible to think that heterogeneity in the ozone effect is limited in our setting.[5]

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of Long Beach: "We're not going to have kids in Long Beach contract asthma so someone in Kansas can get a cheaper television set." (Wald (2007)).

[4] To assess this, we computed the predicted values of ozone (at the port) if boat traffic increased or decreased by one standard deviation (SD) from the mean (based on estimating our first stage equation). The adjusted mean for a one SD decrease in boat traffic is 0.0369 and a one SD increase is 0.0420. The unadjusted mean of ozone at the port is 0.0396, which is bounded by the two adjusted means, so the IV variation is at similar levels of ozone to the overall variation

[5] Since our instrument is assumed to have a different effect on ozone depending on distance from the port, the above statement is true under the plausible assumption that effect of ozone on human health is the same for residents of Los Angeles who live close and far from the port.

Our findings are striking. OLS estimates of ozone on hospitalizations are statistically significant but small in magnitude: exposure to ozone causes less than $0.5 million per year in hospital costs in the Los Angeles region.[6] In contrast, instrumental variable estimates indicate a significantly larger effect of ozone concentrations of about $2 million per year, with several robustness checks support our main findings. These results indicate the importance of accounting for avoidance behavior and measurement error in understanding the full welfare effects from changes in pollution.

2. Background on Air Pollution and Health

Ground-level ozone is a criteria pollutant[7] regulated under the Clean Air Acts that affects respiratory morbidity by irritating lung airways, decreasing lung function, and increasing respiratory symptoms, with effects exacerbated for susceptible individuals, such as children, the elderly, and those with existing respiratory conditions like asthma. Symptoms can arise from contemporaneous exposure in as quickly as one hour of exposure. Because it may take time for exacerbation of respiratory illnesses, symptoms may arise from cumulative exposure over several days or several days after exposure.[8]

The process leading to ozone formation makes it highly predictable and straightforward to avoid. Ozone levels can be predicted using weather forecasts and ozone rapidly breaks down indoors where there are fewer surfaces to interact with (Chang et al. 2000). Since symptoms from ozone exposure can arise over a short period of time, altering short-run exposure by going indoors can reduce the onset of symptoms.

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[6] These estimates represent a lower bound of the overall costs of ozone exposure because they do not account for lost earnings, lost school days, the well-being of the affected individuals, and other health episodes that do not result in hospitalizations.

[7] Criteria pollutants are six common air pollutants with established health-based air quality standards. They include ozone, carbon monoxide, particulate matter, nitrogen dioxide, lead, and sulfur dioxide.

[8] For example, "an asthmatic may be impacted by ozone on the first day of exposure, have further effects triggered on the second day, and then report to the emergency room for an asthmatic attack three days after exposure"

Because of the potential effectiveness of avoidance behavior, two forms of public information were designed to inform the public of expected air quality conditions: the pollutant standards index (PSI) and air quality episodes. The PSI, which is forecasted on a daily basis, is computed for five criteria pollutants, and the maximum PSI across pollutants is required by federal law to be reported in major newspapers along with a brief legend summarizing the health concerns (Environmental Protection Agency 1999). California state law requires the announcement of a stage I air quality episode when the PSI is at least 200, which corresponds to 0.20 parts per million (ppm) for ozone.[9] These episodes, which also occur on a daily basis, are more widely publicized than the PSI; they are announced on both television and radio.[10]

The agency that provides air quality forecasts and issues smog alerts for Southern California is the South Coast Air Quality Management District (SCAQMD). They produce the following day's air quality forecast by noon the day before to provide enough time to disseminate the information. Because SCAQMD covers all of Orange County and the most populated parts of Los Angeles, Riverside, and San Bernardino counties – an area with considerable spatial variation in ozone – they provide the forecast for each of the 38 source receptor areas (SRAs) within SCAQMD. When an alert is issued, the staff at SCAQMD contacts a set list of recipients, including local schools and news media, who further circulate the information to the public.

Neidell (2009) provides direct evidence that people respond to information about air quality. He identifies the effect of smog alerts by using a regression discontinuity design that exploits the deterministic selection rule used for issuing alerts. When smog alerts are issued,

(Environmental Protection Agency 2006).

[9] Additionally, a stage II air quality episode is issued when the PSI exceeds 250 or ozone forecast exceeds 0.30 ppm, but this only occurred once over the time period studied.

attendance at major outdoor facilities in Los Angeles decreases by as much as 13 percent.

3. Data

Our final data set consists of several different data sets merged together at the daily level by zip code for the months April-October for the years 1993-2000 for all zip codes in SCAQMD. For health data, we use respiratory related emergency department (ED) visits from the California Hospital Discharge Data (CHDD) for the following age groups: 0-5, 6-14, 15-64, and over 64.[11] There are two practical factors that make the CHDD an attractive option. First, it includes the exact date of admission to the hospital and zip code of residence of the patient, enabling us to readily merge it to the other data. Second, it contains the entire universe of discharges and the primary diagnosis of the patient, providing a large sample for detecting respiratory related admissions at such a high frequency. Table 1 shows the daily number of ED asthma admissions per zip code for the age groups considered, as well as other independent variables used in this analysis.

We use daily pollution data maintained by the California Air Resources Board. There are roughly 35 continuously operated ozone monitors and roughly 20 for carbon monoxide (CO) and nitrogen dioxide (NO_2), two other pollutants necessary to consider because of their correlation with ozone and potential health effects.[12] We assign pollution levels to the SRA based on the

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[10] Although air quality episodes can potentially be issued for any criteria pollutant, they have only been issued for ozone. Because ozone is a major component of urban smog, this has given rise to the term "smog alerts."

[11] Respiratory related ED visits include all admissions with ICD-9-CM codes from 460-519. Because non-emergency hospitalizations can be pre-arranged and may not be an immediate reaction to ozone, we only use emergency room admissions. We find little meaningful difference in estimates, however, when using all hospitalizations.

[12] Particulate matter (PM_{10}) was not included in this analysis because measurements are not taken on a daily basis. However, it is highly correlated with NO_2 and CO (Currie and Neidell (2005)), so that controlling for these other pollutants should be sufficient.

values for the monitor in that SRA, and when no monitor is present, assign pollution values from the monitor in the nearest SRA.[13]

Data on boat traffic comes from the marine exchange of southern California. The marine exchange records daily logs of the arrival and departure dates of each individual vessel that enters the port, along with the net tons of the vessel. We aggregate this information to the total number of tons of boats arriving and departing on a daily basis. Using the latitude and longitude of the centroid of each zip code, we compute the physical distance from each zip code to the port.

Finally, data on weather is obtained from the Surface Summary of the Day (TD3200) from the National Climatic Data Center. Using the 30 weather stations available in SCAQMD, we assign daily maximum and minimum temperature, precipitation, resultant wind speed, and sun cover to each SRA in an analogous manner to pollution.[14]

4. Methodology

To fix ideas on measuring and interpreting the effect of pollution on health, assume the following short-term health production function:

(1) $h = h\,(ozone, avoid, W, S)$

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[13] Given that this may induce measurement error, we also estimated models that limit the sample to only SRAs where an ozone monitor is present, but find no considerable difference in estimates. There are considerable disagreements over how to assign pollution from monitors to individuals. For example, assigning pollution at a finer geographic level, such as the zip code, has potential to improve accuracy, but may also worsen it if people travel beyond their zip code. Using SRAs is justified on the grounds that SRAs were specifically designed to represent an area with common pollution concerns that account for geographic and population differences within SCAQMD, so there is a high degree of uniformity in ozone levels within an SRA.

[14] For maximum relative humidity and sun cover, we use data from the one weather station in SCAQMD with a complete history of this variable (Los Angeles International Airport) to assign to all of SCAQMD. Since assigning these weather variables may lead to measurement error, we also estimate models excluding all weather variables, shown in Table 4, and find this has little impact on our estimates.

where h is a measure of health, *ozone* is ambient ozone levels, and *avoid* is avoidance behavior. W are other environmental factors that directly affect health, such as weather, allergens, and other pollutants. S are all other behavioral, socio-economic and genetic factors affecting health.

There are two main approaches to estimating (1) and determining the welfare effects from changes in pollution. The first, and most common, is the dose-response approach, which does not control for avoidance behavior but instead estimates the total derivative of health with respect to ozone: $dh/dozone = \delta h/\delta ozone + \delta h/\delta avoid * \delta avoid/\delta ozone$. Since engaging in avoidance behavior may result in welfare loss, in order to measure the full welfare effects one must not only measure the utility loss associated with *dh/dozone* but also measure the costs associated with any changes in avoidance behavior (Cropper and Freeman, 1991, Deschenes and Greenstone, 2007). The second approach directly controls for avoidance behavior to estimate the partial derivate $\delta h/\delta ozone$ of the health production function, and measures the utility loss associated with the change in health (Neidell, 2004, 2009).[15]. Since both approaches require measuring changes in typically non-market behaviors, valuing the welfare effects from a change in environmental quality remains a perennial challenge.

Instead of directly observing avoidance behavior to estimate $\delta h/\delta ozone$, the strategy used in this paper is to use an instrument that shifts ozone levels but is unrelated to both avoidance behavior and other unobserved determinants of health. As described in the introduction, we use boat traffic at the ports as an instrument for ozone levels to obtain estimates of $\delta h/\delta ozone$. Below we confirm a strong partial correlation between boat traffic and ozone levels and present evidence to support the assumption that boat traffic is uncorrelated with avoidance behavior and unobserved confounders. An additional advantage of using an instrument for ozone is that it will account for measurement error in pollution exposure since boat traffic is likely to be uncorrelated with unobserved components of pollution exposure.

We estimate the following equations by two stage least squares:

$$(2) \qquad h_{azst} = \beta_1 \overline{ozone}_{st} + \beta_2 \overline{W}_{st} + \beta_3 M_t + \alpha_a + \theta_z + f(t) + u_{azst}$$

$$(3) \qquad \overline{ozone}_{st} = \gamma_1 \overline{boats}_t + \gamma_2 \overline{boats}_t * dist_s + \gamma_3 \overline{W}_{st} + \gamma_4 M_t + \theta_z + f(t) + v_{st}$$

where equation (2) is the second stage regression and (3) is the first stage regression. h_{azst} is the number of respiratory related ED admissions for age group a in zip code z of SRA s at date t. To remain consistent with previous ozone time-series studies that often found respiratory effects up to four days after exposure (Environmental Protection Agency, 2006), we compute the daily average of ozone, boats, and W over the past 5 days.[16] We average across five days because we are only interested in testing whether an effect of ozone exists, and not the particular timing of the effect. Furthermore, this lagged structure would require us to instrument for each lag of ozone, which greatly reduces the precision of our estimates.

The vector W includes maximum temperature, minimum temperature, precipitation, resultant wind speed, humidity, sun cover, carbon monoxide, and nitrogen dioxide. Since weather and other pollutants vary at a daily level and may also affect health, it is necessary to properly account for them in W. Although we argue that our instrument is uncorrelated with W, we also present evidence below that both OLS and IV estimates are insensitive to omitting observed measures of W and to interacting it with ozone, suggesting environmental conditions are not confounding factors for our analysis.

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[15] Instead of controlling directly for avoidance behavior, Neidell (2004, 2009) controls for factors that shift the demand for avoidance behavior (ozone forecasts and smog alerts). Note that the dose response and health production estimates are identical if avoidance behavior does not exist.

M_t contains day of week dummies to account for within week patterns of air quality and health. In addition to limiting the analysis to the months of April through October, *f(t)* includes year*month dummy variables and a cubic time trend to flexibly capture seasonality and long run trends in air quality and health.[17] α_a are age dummy variables. θ_z are zip code fixed effects designed to capture local time-invariant demographic factors that might affect health as well as time-invariant measurement error.[18] u is an error term that includes avoidance behavior, unobserved components of *W* and *S*, and an idiosyncratic component. By focusing on high frequency data, daily time series models have the benefit of accounting for *S* because it is unlikely that behavioral factors, such as diet and existing health capital, change daily in conjunction with ozone levels.

We instrument ozone in equation (2) using boat traffic, shown in equation (3). $boats_t$ is a measure of tons of boat arrivals and departures at date t and $dist_s$ is the distance from SRA s to the port. We interact $boats_t$ with distance to allow the effect of the boat arrivals and departure to vary depending on how far the SRA is from the port. In all the empirical models in the paper,.

If there is a homogeneous effect of ozone on health, a necessary assumption for unbiased estimates of β_1 is that $cov(boats_t, u_{azst}) = 0$ and $cov(boats_t*dist_s, u_{azst}) = 0$. Given that the boats travel from great distances and many factors affect their exact arrival date, it is reasonable to think of the timing of boat arrival as virtually random in the short run. Because of that, we have little reason to expect that short run variations in boat movements directly affect health in the short run, and below we present evidence to support that.

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[16] We also estimated models that allow for arbitrary auto-correlation of four lags, included daily weather rather than averaged weather, and included 4 or 6 day averages of all variables, and this had minimal impact on our estimates and standard errors.
[17] We also estimated models with year*week dummy variables and found this had little impact on our estimates.
[18] Note that by including zip code fixed effects we are controlling for the population at risk (to the extent it remains constant over time within a zip code), so we can interpret our estimates as the impact of ozone on the rate of hospital admissions.

5. Results

5.1. Validity of boat traffic as an instrument

Our instrument may be invalid if people can perfectly observe changes in pollution levels induced by the boats and adjust their exposure accordingly. While people may have a good sense of seasonal variation in pollution, have reliable information on current weather conditions that may affect pollution, and have easy access to pollution forecasts, we think it is unlikely they detect daily changes in pollution levels induced specifically by the boats. To probe this, we assess whether pollution forecasts – the main source of information available to the public – are based on boats movements. Shown in the first two columns of Table 2, we show estimates of the relationship between boat traffic and both smog alerts and ozone forecasts. In column 1, we regress whether a smog alert was issued anywhere in SCAQMD on our measure of boat traffic and all of the covariates in equation (3), but only using covariate data for the SRA of the port. In column 2, we repeat this regression using the ozone forecast for the SRA of the port.[19] In both analyses we only use contemporaneous levels and not a five day average since this more precisely addresses the question of whether boat traffic is incorporated into air quality forecasts. The results indicate a statistically insignificant coefficient on boat traffic for both measures of air quality information, which supports the notion that boat traffic is not used in air quality forecasts and hence is unlikely to be related to avoidance behavior.

Our instrument may also be invalid if people possess private information about boats movement and adjust their exposure based on that information. Specifically, if the information on boats movement induces people to decrease their exposure to ozone by limiting time spent outside and this in turn improves health, we will underestimate the biological effect of ozone on

[19] We can not use whether an alert was issued in the SRA of the port because this never occurred in the time period studied.

health. We assess this by estimating whether attendance at several outdoor activities is related to boat traffic. If private information is based on boat traffic, then outdoor activities will decrease when boat traffic increases. We use four measures of attendance at outdoor activities in SCAQMD: two major outdoor attractions, the Los Angeles Zoo and the Griffith Park Observatory, and two major league baseball teams, the Los Angeles Dodgers and California Angels.[20] Estimates for each venue, shown in columns 3-6, are statistically insignificant for three of the four venues. Although we find a statistically significant estimate for attendance at the Zoo, this estimate is small in magnitude: a one standard deviation increase in boat traffic is associated with a 1.4 percent increase in attendance. Furthermore, when we estimate these equations simultaneously via seemingly unrelated regression, based on a joint test of significance we find a statistically insignificant association between boat traffic and attendance. These results suggest individuals are unlikely to update their private information about pollution levels using boat traffic.

In column 7 of Table 2, we assess whether boat traffic is related to weather conditions at the port by regressing boat traffic on the weather variables and the other covariates in equation (3) using data for the SRA of the port only. The results indicate that only precipitation has a statistically significant correlation with boat traffic, but the magnitude is small. Each hundredth of an inch of precipitation is positively associated with a 757 ton increase in daily boat traffic, which is 1.4% of the standard deviation in daily boat traffic. This minimal effect for precipitation, combined with statistically insignificant estimates for the other weather variables, supports our claim that boat traffic is uncorrelated with weather conditions. Furthermore, as we

[20] For more details on these data, see Neidell (2009). The California Angels were renamed the Anaheim Angels in 1997 and the Los Angeles Angels of Anaheim in 2005.

demonstrate below, our estimates of the health effects of ozone are insensitive to the exclusion of the weather variables.

5.2. The relationship between boat movements and pollution

To assess the strength of our instrument, in Panel A of Table 3 we present evidence of the relationship between boat arrivals and departures on ozone levels in Los Angeles. It is highly statistically significant, with t-statistics that exceed 150 for both boat traffic in levels and boat traffic interacted with distance from port. Our estimates in the second column imply that each 500,000 tons of boat arrivals and departures (the approximate daily average) at the port increase ozone levels in the immediate area by .024 ppm, which is just over 60% of the mean ozone level in Long Beach of 0.039. This estimate is consistent with previous reports that suggest the port contributes to 50% of smog-forming gases (Polakovic, 2002).

The interaction term between boat movement and distance from the port allows differential effects of port activity on resultant ozone levels depending on how far the area is from the port. We expect a greater impact on ozone levels in zip codes immediately adjacent to the port, with this effect diminishing as we travel away from the port. The negative interaction term, which is also highly statistically significant, is consistent with this. Furthermore, figure 1 plots the effects of the average daily boat movement in the port on ozone levels based on the distance from the port. The results imply that the effect of the port on inland pollution levels is cut in half at 11 miles from the port and disappears at 23 miles from the port. When we add boat traffic interacted with a quadratic term in distance to allow a non-linear decay from port emissions (column 3 of Table 3), the quadratic term is statistically significant. However, Figure 1 shows that this addition does not appreciably change the spatial decay of pollution.

These results highlight the strength of our first stage: arrivals and departures at the port have a significant effect on ozone levels in the immediately surrounding areas, and this effect diminishes as one moves away from the port. Boat arrivals and departures appear uncorrelated with other factors related to health, so our second stage estimates will be consistent estimates of the biological effect of ozone on asthma hospitalizations.

5.3. The relationship between pollution and hospitalizations

Turning to estimates of the relationship between ozone and health, we present OLS and IV results in Panel B of Table 3. OLS results, shown in column 1, indicate ozone has a statistically significant relationship with respiratory related hospitalizations. A five day increase in ozone of 0.01 ppm is associated with a modest 1.2% increase in hospitalizations. When we turn to our IV estimates we find estimates that are nearly four times larger than OLS estimates. The considerably larger estimates, shown in column 2, imply a 0.01 ppm increase in the 5-day average ozone is associated with a 4.7% increase in hospitalizations. This difference is statistically significant according to a Hausman test, which has a p-value of 0.028. In column 3, when we add the quadratic in distance, we find a similar 4.5% increase in hospitalizations. These results suggest the importance of accounting for avoidance behavior and measurement error; not accounting for these factors significantly understates the effect of ozone on health.

These estimates suggest that accounting for avoidance behavior and measurement error increase estimates by a factor of 4. Neidell (2009) finds estimates are roughly 1.5 times larger when controlling only for public air quality information. This difference is due to the fact that we also correct for measurement error and other unobserved sources of information for avoidance behavior, suggesting the importance of accounting for these additional sources of bias.

Since environmental factors are an important potential source of confounding, in Table 4 we assess the sensitivity of our estimates to the weather variables and co-pollutants. If estimates are unaffected by these variables, it suggests they are unlikely to be important confounders in our analysis. Column 1 repeats our baseline estimates. Column 2 omits both the weather variables and co-pollutants. Column 3 omits only the latter while column 4 omits only the former. Lastly, in column 5 we interact ozone with all of the weather variables and co-pollutants, and compute the marginal effect of ozone on health by evaluating $\delta h/\delta ozone$ using the mean of each weather variable and co-pollutant.[21] Our estimates are clearly insensitive to these alternative specifications, suggesting the strength of our instrument in controlling for potential confounding from environmental factors.

Since we have aggregated all respiratory illnesses and ozone may have a differential effect across the type of illnesses, in Table 5 we separately explore the effects of ozone on pneumonia (ICD 480-486), bronchitis and asthma (ICD 466, 490, 491, 493, 494), and other respiratory illnesses. Pneumonia, bronchitis, and asthma are conditions more likely to be exacerbated from current exposure, as opposed to respiratory conditions like emphysema, where the effects from exposure are cumulative over time (Environmental Protection Agency, 2006). Therefore, we expect larger effects for pneumonia and bronchitis and asthma than for other respiratory conditions. The OLS results indicate fairly comparable effects across the conditions with a slightly larger effect, if anything, for other respiratory conditions. The IV results, however, paint a different picture. Consistent with expectations, the effects are largest for pneumonia, followed by bronchitis and asthma, and then a small effect for other respiratory illnesses.

[21] In this specification, we also instrument for each of the interacted terms by interacting boat traffic with the weather variables and co-pollutants.

5.4. The benefits from ozone abatement

To gauge the magnitude of our estimates, we quantify the additional hospital costs caused by ozone exposure in the Los Angeles area. We compute the additional hospital costs using the average hospital bill for any respiratory related admission, recognizing that this understates the full value because it ignores any direct effects on well-being not included in hospital costs and it ignores other health episodes that do not result in hospitalizations.[22]

Given that the mean 8-hours ozone exposure is .05 ppm (Table 1), our OLS estimates would indicate that ozone causes additional hospitalization costs of $462,000 per year in the Los Angeles region. Based on the IV estimates, the cost amounts to $1,852,000 per year. The difference, though an understatement, gives a sense of how a more simplified analysis based on OLS would incorrectly underestimate the costs of ozone exposure.

6. Conclusion

We propose a novel approach to estimate the biological effect of ozone on health. We isolate the short term effect of ozone holding constant compensatory behavior and accounting for measurement error by using boat arrivals and departures at the Ports of Los Angeles and Long Beach as an instrument for ozone levels. The ports generate significant amounts of ozone precursors that interact with environmental conditions to form ozone, and we find boat traffic significantly affects daily ozone levels in Los Angeles. Because boat traffic is unobserved by most residents, it generates an important source of variation in pollution that is difficult to avoid and can not easily be offset by residents' compensatory behavior.

We find that OLS estimates of ozone on hospitalization are small, possibly because they are contaminated by unobserved heterogeneity in avoidance behavior or measurement error. In

[22] Estimates are comparable if we aggregate the separate impacts for the different respiratory illness explored in Table 5.

contrast to OLS estimates, instrumental variable estimates indicate a considerably larger effect of ozone concentrations on respiratory related hospitalizations. We draw three main conclusions. First, the estimated effects of ozone on health are large. Second, simple correlations are significantly biased by unobserved avoidance behavior and/or measurement error. Three, our results speak to the importance of accounting for avoidance behavior and measurement error in understanding the full welfare effects from environmental quality.

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Figure 1: Effect of average daily port activity on ozone levels

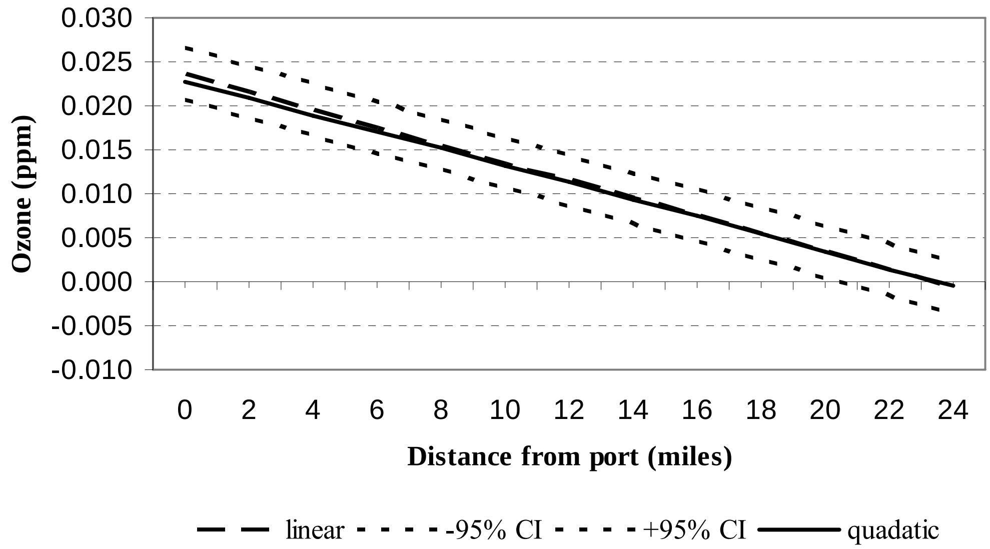

Table 1. Summary Statistics

	mean	std. dev
Dependent variables		
any respiratory illness	0.097	0.333
age 0-5	0.043	0.216
age 6-14	0.018	0.135
age 15-64	0.129	0.376
age 65+	0.199	0.465
pneumonia	0.037	0.199
bronchitis/asthma	0.033	0.186
other respiratory illness	0.027	0.167
Independent variables		
8-hour ozone (ppm)	0.050	0.018
8-hour carbon monoxide (PSI)	16.60	9.04
1-hour nitrogen dioxide (PSI)	18.30	6.92
maximum temperature (°F)	80.37	9.04
minimum temperature (°F)	59.08	5.90
precipitation (hundreths of inches)	1.02	4.63
resultant wind speed (mph)	5.74	1.48
maximum relative humidity (%)	90.24	5.09
average cloud cover sunrise to sunset (%)	4.42	1.65
boat traffic (tons / 1000)	514.49	55.58

Table 2. Relationship between boat traffic with weather, ozone forecasts, and outdoor attendance

	1 alert	2 ozone forecast	3 zoo	4 observatory	5 dodgers	6 angels	7 boats (tons/1000)
boat traffic (tons/1000)	0.00001	-0.005	-1.082*	-0.352	-0.327	1.748	
	[0.00006]	[0.004]	[0.504]	[0.388]	[3.143]	[2.891]	
maximum temperature	0.00588**	0.859**	-8.083	-11.262	-28.514	-57.273	-0.324
	[0.00181]	[0.129]	[14.204]	[11.964]	[117.318]	[85.574]	[0.810]
minimum temperature	0.00581*	0.820**	-39.151*	10.194	-125.882	74.480	-1.052
	[0.00240]	[0.170]	[17.875]	[17.976]	[137.068]	[131.567]	[1.133]
precipitation	0.00011	-0.001	-73.266**	-21.110**	-53.214*	73.280	0.757**
	[0.00040]	[0.041]	[18.010]	[7.854]	[22.753]	[96.141]	[0.237]
resultant wind speed	-0.00469	-1.045**	-27.986	35.429	-75.297	-111.110	0.096
	[0.00275]	[0.201]	[21.494]	[23.632]	[167.027]	[158.160]	[1.450]
relative humidity	0.00307**	0.129	-0.986	-10.958	-108.655	-219.546**	0.081
	[0.00090]	[0.084]	[7.428]	[8.216]	[86.961]	[68.625]	[0.481]
average cloud cover	-0.00169	-0.250	-14.511	-37.436*	86.477	-221.960	0.626
	[0.00447]	[0.262]	[18.942]	[18.547]	[214.933]	[150.598]	[1.768]
SUR joint test			$\chi_2(4) = 5.52$		P-value = 0.238		
dependent variable mean	0.08	59	4246	5469	39574	25696	516
Observations	1380	1380	916	837	464	486	1490

Notes: * significant at 5%, ** significant at 1%. All regressions include 8-hour carbon monoxide, 1-hour nitrogen dioxide, year-month dummies, day of week dummies, and cubic day trend. 'SUR joint test' is a joint test of boat traffic on zoo, observatory, dodgers, and angels attendance.

Table 3. OLS and IV regression results for effect of ozone on respiratory illnesses

	1	2	3
	OLS	IV	IV
A. First stage			
boat traffic / 100,000		4.608**	4.409**
		[0.029]	[0.044]
boat traffic / 100,000)*distance		-0.198**	-0.181**
		[0.001]	[0.003]
(boat traffic / 100,000)*distance^2)*1000			-0.293**
			[0.048]
B. Second stage			
8-hour ozone	0.113**	0.454**	0.442**
	[0.023]	[0.162]	[0.162]
Wu-Hausman F test (1,1927109)		4.820	4.485
P-value		0.028	0.034
percent effect	1.16%	4.66%	4.54%

Notes: * significant at 5%, ** significant at 1%. N=1,927,187 in all regressions. Robust standard erros clustered by date in brackets. Dependent variable is number of respiratory related hospital admissions per day, zip code, and age category. All regressions include independent variables from Table 1 (except boat arrivals and departures), age dummies, year-month dummies, day of week dummies, cubic day trend, and zip code fixed effects. 'percent effect' % change in dependent variables from .01 ppm increase in ozone (=(ozone coefficient/100)/(mean of dependent variable from Table 1)).

Table 4. Sensitivity of regression results for effect of ozone on respiratory illnesses to weather and co-pollutants

	1	2	3	4	5
OLS					
8-hour ozone	0.113**	0.130**	0.118**	0.097**	0.105**
	[0.023]	[0.020]	[0.023]	[0.022]	[0.027]
IV - First stage					
boat traffic / 100,000	4.608**	4.733**	4.959**	4.037**	24.463**
	[0.029]	[0.035]	[0.030]	[0.031]	[0.351]
(boat traffic /100,000)*distance	-0.198**	-0.192**	-0.207**	-0.187**	-0.571**
	[0.001]	[0.001]	[0.001]	[0.001]	[0.003]
IV - Second stage					
8-hour ozone	0.454**	0.465**	0.435**	0.451**	0.543**
	[0.162]	[0.168]	[0.155]	[0.171]	[0.210]
controls for weather	Y	N	Y	N	Y
controls for co-pollutants	Y	N	N	Y	Y
interactions with ozone	N	N	N	N	Y

Notes: * significant at 5%, ** significant at 1%. N=1,927,187 in all regressions. Robust standard erros clustered by date in brackets. Dependent variable is number of respiratory related hospital admissions per day, zip code, and age category. All regressions include independent variables from Table 1 (except boat arrivals and departures), age dummies, year-month dummies, day of week dummies, cubic day trend, and zip code fixed effects. For column (5), ozone is interacted with weather and co-pollutant variables, and coefficient shows marginal effect of ozone evaluated at mean of weather & co-pollutant variables. All interactions are instrumented by boat arrivals & departures interacted with weather & co-pollutant variables, but only coefficients for boat arrivals & departures & distance from first stage are shown.

Table 5. Regression results for effect of ozone by type of respiratory illness

	1 any resp. illness	2 pneumonia	3 bronchitis/ asthma	4 other respiratory illnesses
OLS				
8-hour ozone	0.113**	0.032*	0.038**	0.043**
	[0.023]	[0.014]	[0.014]	[0.011]
percent effect	1.16%	0.86%	1.15%	1.59%
IV				
8-hour ozone	0.454**	0.277**	0.145	0.032
	[0.162]	[0.101]	[0.103]	[0.080]
percent effect	4.66%	7.41%	4.39%	1.19%

See notes to Table 3. Dependent variable is number of respiratory related hospital admissions per day, zip code, and age category.