# AIR POLLUTION, CHILDREN'S HEALTH, AND SOCIO-ECONOMIC STATUS: THE EFFECT OF OUTDOOR AIR QUALITY ON ASTHMA

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# AIR POLLUTION, HEALTH, AND SOCIO-ECONOMIC STATUS: THE EFFECT OF OUTDOOR AIR QUALITY ON CHILDHOOD ASTHMA

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**Abstract**

This paper examines the effect of air pollution on child hospitalizations for asthma using a unique zip code level panel data set. The effect of pollution is identified using naturally occurring seasonal variations in pollution within zip codes. I also improve on past work by analyzing how the effect of pollution varies by age, by including measures of avoidance behavior, and by allowing the effect to vary by socio-economic status (SES). Of the pollutants considered, carbon monoxide has a significant effect on asthma hospitalizations among children ages 1 to 18. In addition, households respond to information about pollution with avoidance behavior, especially high SES families, suggesting that it is important to account for these endogenous responses when measuring the causal effect of pollution on health. Finally, the net effect of pollution is much greater for children of lower SES, indicating that pollution is one potential mechanism through which SES affects health.

*JEL Classifications*: I12, J13, J15, Q25

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## 1. Introduction

A primary objective of air quality policies around the world is to protect human health. However, many critics argue that air quality standards are set somewhat arbitrarily with inconclusive evidence of the specific health benefits and with inadequate considerations of the costs to producers. Given that substantial costs to industry have been widely demonstrated,[1] in order to determine optimal policy intervention it is crucial to identify the associated benefits from improvements in air quality.

While many studies have focused on estimating a relationship between pollution and health, they have largely neglected to consider that pollution exposure is endogenously determined if individuals make choices to maximize their well-being. People with high preferences for clean air may chose to live in areas with better air quality. People can respond to a wide range of readily available information on pollution levels by adjusting their exposure. Failing to appropriately account for such actions can yield misleading estimates of the causal effect of pollution on health.

This paper focuses on developing an empirical strategy for measuring the effect of pollution on health. Specifically, I look at the effect of air pollution on children's hospitalization for asthma. Childhood asthma is of particular interest for two reasons: 1) asthma is the leading chronic condition affecting children; and 2) current pollution standards are based on adult health responses to pollution and children face a greater risk from pollution exposure due to the sensitivity of their developing biological systems.

This study builds on earlier work in five ways. First, I develop a unique, quarterly, zip code level data set by matching information about all individual hospitalizations in California between 1992 and 1998 to ambient pollution levels, meteorological data, and various demographic data. Second, I identify the effect of pollution using naturally occurring seasonal variations within zip codes. Since zip codes are a finely defined geographic area and the seasonal patterns in pollution are remarkably strong and diverse throughout California, this controls for many confounding factors that might affect asthma hospitalization rates. Third, I allow the effect of pollution to differ with the age of the child, as biological models suggest it might. Fourth, I collect data about public announcements of health advisories in order to show empirically that it is

important to account for the endogeneity of household responses to pollution. Fifth, to assess if the effect of pollution varies across different segments of the population, I allow the effect of pollution to differ with socio-economic status (SES), as measured by education levels in the zip code.

The primary finding of this paper is that carbon monoxide (CO) has a significant effect on hospitalizations for asthma among children ages 1 to 18, while none of the pollutants considered has a clear impact on hospitalizations for infants. These findings for CO are robust to various sensitivity checks. The decline in CO levels from 1992 to 1998 explains between 4 and 14 percent of the decrease in asthma hospitalization rates, resulting in savings of approximately $4.3 million in hospital expenses for asthma admissions in California in 1998 alone.

Second, I find that families display avoidance behavior by responding to health advisories, especially high SES families. The announcement of health advisories decreases asthma hospitalizations by roughly 3 to 6 percent. This indicates the importance of accounting for the endogeneity of family behavior when measuring the causal effect of pollution on health.

Third, not only are the coefficients measuring the effect of pollution larger for low SES children, but these children are also exposed to considerably higher levels of pollution. As a result, they suffer greater harm from pollution and this suggests that pollution is a potential mechanism for the well-known relationship between SES and health -- poorer families are unable to afford to live in cleaner areas, and their children's health suffers as a result.

The paper is laid out as follows. Section 2 provides some background information on asthma and its potential association with pollution. Section 3 presents the economic framework for this analysis. Section 4 presents the estimation strategy. Section 5 describes the data used for the analysis. Section 6 presents the econometric results. Section 7 concludes with a discussion.

## 2. Background

Approximately 5 million children in the U.S. have asthma. It is the leading specific reason for school absence and the most frequent cause of pediatric emergency room use and hospital admission (NIEHS

[1] See, for example, Greenstone (1999) for estimates on the costs of the Clean Air Acts on industrial activity in the

(1999)). Asthma disproportionately attacks children of lower SES, and continues for most well beyond childhood (AAP (2000)). Most disconcerting is that reported asthma rates for children age 18 and younger have increased by more than 70 percent from 1982 to 1994 (AAP (2000))[2].

Despite mounting public concern, the factors influencing this illness are not fully understood, especially for children. Medical research has demonstrated that asthma is both a chronic and acute illness. In the chronic aspect, an individual's airways are persistently inflamed and their immune system is hyper-responsive, but the causes of this remain largely unknown (American Lung Association (2000)). During an acute response, an irritant is inhaled that causes three changes to occur: muscular bands around the bronchioles constrict, the linings of the airway become inflamed, and excess mucus is produced. The irritants are believed to cause this because, by being recognized by the immune system as foreign, immunoglobin E (IgE), an antibody, is produced in response. IgE binds with mast cells -- particular cells filled with chemical mediators – causing the release of some of the mediators in the mast cells (AAP (2000)). As a result of these changes in lung functioning, the airways are severely narrowed, making it difficult to breathe.

Such potential irritants, or asthma "triggers", include molds, pollens, animal dander, tobacco smoke, weather, exercise, and outdoor air pollution. However, much mystery surrounds these triggers. It is unclear which, if any, of these triggers cause asthma in the first place. It is unclear how these triggers interact to affect the airways. For example, diesel fumes and ozone, while both potential triggers, may increase an individual's reaction to pollens and mold (AAP (2000)). With respect to outdoor pollutants, it is not known whether constant levels of sustained exposure or short periods of peak exposure are more likely to lead to asthma attacks. Finally, it is not known if a "threshold" effect for various pollutants exist, such that pollution levels below a specific point do not lead to asthma attacks (AAP (2000)).

Many researchers have attempted to link air pollution and childhood asthma, but with mixed results.[3] Most studies have been short time-series that focus on a given city and track the daily number of hospital or

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United States.

[2] There is, however, much debate regarding this apparent rise in asthma. I discuss this is more detail below.

emergency room (ER) admissions for asthma and the average daily levels of various criteria pollutants.[4] A wide range of estimated correlations between admissions for asthma and carbon monoxide (CO), ozone (O3), particulate matter (PM10), and nitrogen dioxide (NO2) have been reported, with no clear patterns or magnitude of effects evident.[5]

Due to the inconclusive findings and the fact that ambient air pollution levels have declined in most parts of the country while the reported incidence of asthma has risen[6], many researchers have begun to question the link between ambient air pollution and asthma (von Mutius (2000a, 2000b), Vacek (1999), Duhme et. al. (1998)). For example, the Committee on the Medical Effects of Air Pollution concluded that "overall evidence is small that non-biological outdoor air pollution has an important effect on the initiation and [provocation] of asthma" (2000). As a result, alternative theories have sprung up recently. One theory proposes that children are "too clean" because they often use antibiotics to combat minor illnesses. As a result, their immune systems do not develop properly and attack many harmless substances that enter the body (AAP (2000)). A second competing theory is that the changing lifestyles of children – poorer diets, less exercise, more time indoors – has led to the increase in asthma related illnesses (von Mutius (2000a)).

However, not all researchers have dismissed the role that pollution may play. There is a debate as to whether asthma rates have actually increased. Better detection of asthma and different classifications of illness could explain some of the increases in individual and doctor reports. For example, what was long labeled wheezy bronchitis is now classified as asthma (Speizer (2001)). Recent expansions in Medicaid could also explain part of the increase in reported cases -- as children's access to health care increases, there is a greater chance of early detection and treatment.

Many researchers have also questioned the methodological approaches used to identify the

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[3] Some representative studies include Desqueyroux and Momas (1999), Gouveia and Fletcher (2000), Fauroux et. al. (2000), and Norris et. al. (1999). I have reviewed over 100 articles about similar studies.

[4] Criteria pollutants are non-toxic air pollutants considered most responsible for urban air pollution and are known to be hazardous to health. They include SO2, NO2, O3, CO, PM10, and lead.

[5] Other studies that have attempted to link pollution and general health use data that follow the same individuals over a short period of time to control for permanent health-related factors, such as smoking rates and exercise habits (Alberini and Krupnick (1998), Portney and Mullahy (1986, 1990)). However, most of these studies focus on adults, and the results may not be directly applicable to children. Furthermore, a general limitation of these studies is that, given the limited number of observations over a short period of time, it is unlikely that there is enough variation in specific health outcomes to obtain precise estimates.

relationship between pollution and asthma (Nystad (2000), Eggleston et. al. (1999), von Mutius (2000b), Bjorksten (1999)). Since air pollution is not randomly assigned, most studies have been largely unsuccessful in disentangling pollution from other confounding factors that affect health. Additionally, these studies do not account for direct responses to ambient levels of pollution. Furthermore, these studies tend to group all children into just one category, and we might expect a number of biological and behavioral factors to vary for children of different ages. Lastly, most studies conduct single pollutant analyses, which does not provide clear policy implications if pollutants are highly correlated.

A final reason to believe a connection between pollution and asthma might exist is that studies with more convincing empirical designs have found consistent effects of pollution on children's health. Chay and Greenstone (2001) use declines in pollution that resulted from the 1980-82 recession and find a strong link between total suspended particles and infant mortality. Since most infant mortality is due to respiratory failure, it is reasonable to suspect that pollution could be related to other respiratory illnesses, such as asthma. Ransom and Pope (1995) use changes in pollution that resulted from the opening and closing of a steel mill due to a labor strike and find a large effect on bronchitis and asthma in children. Their study, however, does not identify the effect of specific pollutants, only the effect of the mill being opened or closed.[7]

### 3. Economic Theory

One approach to understanding the impact of pollution on health would be to assume that everyone is unaware of the amount of pollution in the air. Therefore, ambient levels of pollution would serve as an unbiased proxy for an individual's exposure to pollution and pollution levels would not be correlated with any types of behavior. One could then estimate a relationship between health and pollution by regressing health outcomes on ambient levels of pollution as well as other exogenous factors that are related to both pollution and health, such as weather conditions.

However, this approach is oversimplified because individuals can undertake avoidance activities to

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[6] See footnote 1.

[7] Another study (Friedman et. al. (2001)) that attempts to use a "natural experiment" caused by changing traffic patterns in Atlanta during the 1996 Olympics also does not identify the effects of particular pollutants. Moreover, this study does not consider the changing behavior of families in response to the Olympics in general.

reduce the effect of externalities, which makes an individual's exposure to pollution an endogenously determined variable.[8] This introduces two issues. First, there are many tools available to inform people when air pollution levels pose a threat to health. Home devices, such as peak expiratory flow (PEF) meters, can be used to measure lung functioning on a given day (if the individual is already known to have respiratory illness). California State law requires the announcement of health advisories when air quality exceeds certain limits (Air Resources Board (1990)). State and local agencies are required to report a daily measure of air quality in large metropolitan areas, with newspapers a common source (U.S. EPA (1999a)). Many regional air quality offices, such as the California Air Resources Board, provide web pages with up-to-the-minute pollution details and e-mail notifications of dangerous pollution levels.[9] Many pollutants are directly visible -- on high-smog days in Los Angeles, whitish clouds often cover the sky or a reddish-brown haze is visible around the horizon. If people directly respond to this information, then ambient pollution levels will not accurately represent their exposure to pollution.

A second issue arises because air quality, like many local public goods, is capitalized into housing prices, making it an attribute of a home that people can demand (Chay and Greenstone (2000)). Therefore, families with a higher value for cleaner air can locate in areas with better air quality.[10] These families may also make additional investments in their children's health -- they may be less likely to smoke or more likely to seek preventative health care. As a result, there are many confounding behavioral factors related to both pollution and health, making it difficult to identify the effect of pollution on health.[11]

To understand the empirical implications of such actions for estimating the effect of pollution on hospitalizations for childhood asthma, it is useful to think of health endpoints occurring as the result of a two-stage decision process: Parents first invest in their child's health, and then decide the type of health care

[8] For a detailed description of avoidance (or averting) behavior, see Zeckhauser and Fisher (1976).
[9] For example, visit http://www.arb.ca.gov/adam/welcome.html to find daily pollution levels in California.
[10] Families do not need to have direct preferences for this attribute. However, because air quality is an input in the health production function, people with preferences regarding health will have implicit tastes for air quality.
[11] This is analogous to the confounding that arises in estimating the effect of school quality on test scores. Parents who choose to live in areas with better school quality may also make additional investments in their children, making it difficult to identify the effect of school quality.

to use if their child's health condition needs medical attention.[12]

Investing in Health

This description follows Cropper's (1977) model closely in spirit, which extends Grossman's (1972) model by incorporating pollution. The main differences here are that parents invest in their child's health, rather than their own, and housing purchases enter the model.

A child's health is determined by the following health production function:

$$H = H(P, A, M, W; E) \quad (1)$$

where $P$ is ambient air pollution, $A$ is contemporaneous avoidance behavior that directly affects the child's exposure to pollution, $M$ are other investments in health (such as indoor air filters, medical care, diet, exercise, and smoking)[13], $W$ are exogenous factors that affect health (such as weather and technology), and $E$ is a family specific endowment (such as the child's existing health stock or the parents' knowledge of health production).

Note that this is a slightly different treatment of avoidance behavior than in the previous literature. I distinguish between contemporaneous and permanent avoidance behavior by considering contemporaneous avoidance behavior a direct response to pollution levels, while permanent avoidance behavior need not be a direct response. For example, the decision to keep a child inside on a high pollution day is a contemporaneous response, while the use of an air filtration system on a regular basis (regardless of daily or seasonal fluctuations in pollution levels) is a permanent response. This introduces an important empirical implication that is discussed below.

Assume the family's objective is to maximize utility defined over consumption ($C$), housing consumption, and the health of the child. Based on hedonic price methods, we can replace housing consumption in the utility function with the attributes of the house, defined here as $P$ and $O$, where $O$ are

[12] While hospital data are not ideal for estimating the effect of pollution – it does not include cases where children use other sources of care instead – it allows two notable advantages over other reported measures. First, ER admissions are an objective measure of asthma. Second, it provides a large number of observations with narrow geographic identifiers to allow the identification strategy (described below) to work. Since ER admissions do not represent all asthma cases, this will underestimate the total effect of pollution on asthma.

[13] These factors could also be components of consumption that enter into the utility function of the parent, such as smoking.

attributes of the home other than pollution. Parents choose *C, P, O, A,* and *M* to maximize utility subject to (1) and the following budget constraint[14]:

$$I = p_C C + F(P, O) + p_A A + p_M M \tag{2}$$

where $I$ is (exogenously determined) income, $p_j$ is the time-inclusive price of commodity $j = \{C, A, M\}$, and $F(\bullet)$ is the (possibly non-linear) price function of the housing attributes.

The first order conditions for utility maximization for the three choice parameters of interest *(P, A,* and *M)* imply:

$$\left(\frac{\partial U}{\partial P} + \frac{\partial U}{\partial H}\frac{\partial H}{\partial P}\right) / \mu = \frac{\partial F(\bullet)}{\partial P} \tag{3}$$

$$\left(\frac{\partial U}{\partial H}\frac{\partial H}{\partial A}\right) / \mu = p_A \tag{4}$$

$$\left(\frac{\partial U}{\partial H}\frac{\partial H}{\partial M}\right) / \mu = p_M \tag{5}$$

where $\mu$, the Lagrange multiplier for the budget constraint, represents the marginal utility of income. As indicated, parents choose the amounts of *P, A,* and *M* that equates their benefits and costs on the margin.

There are three items worth noting from this model. First, an exogenous increase in pollution (that does not induce people to move) will increase the amount of contemporaneous avoidance behavior. This can arise in two ways. First, if pollution increases such that public warnings are announced, this information will lower the price of avoidance behavior and increase its demand. Second, if avoidance behavior is a binary choice (to stay inside or go outside), people will choose to avoid pollution if the costs from it are less than the costs from exposure. Therefore, as pollution increases, the costs from exposure increase while the costs of avoiding remain the same, leading to an increase in avoidance behavior. [15]

A second implication of this model, obtained by dividing equation (3) by (5), is that while the parents' choice of air quality is clearly related to choices of *M*, the direction of this relation depends on the functional form of *U, H,* and *F*. To see the intuition behind this, we can imagine two situations that invoke

[14] Letting leisure, parental health, and sick time enter into the model will not affect the main implications given here.

different responses. On one hand, since $P$ and $M$ are normal goods, wealthier families consume higher levels of both. On the other hand, if $P$ is bundled with other components, such as school quality and crime rates (the non-linearity of $F$), then in order to purchase lower levels of air quality they must compromise by choosing less $M$.

The third insight is that families that are more knowledgeable in health production face a lower price for health ($p_A$ or $p_M$). As a result, they will invest larger amounts in their children's health by choosing "better" quantities of $A$ or $M$, such as less tobacco smoke, better indoor air quality, or healthier diets. Similarly, parents will make larger investments in children with lower health stock, such as younger children. This arises because younger children face a greater risk from pollution exposure than older children ($\frac{\partial H}{\partial P}$ is higher) and/or it is less costly to monitor the behavior of younger children ($p_A$ and/or $p_M$ is lower). For example, it is not uncommon for parents to insist on keeping tobacco smoke away from their infant, only to become more yielding about limiting tobacco smoke as the child grows older. This finding, combined with the second prediction described above, suggests that a child's exposure to pollution is correlated with the family specific endowment.

Health Care Utilization

If the child's health has crossed a certain threshold ($h$) and some type of health care is required, the parent must decide how to manage the situation. In the case of asthma, if the child has already been diagnosed as asthmatic and has the necessary medication, the family may be able to manage the attack successfully and need no further attention. If they do not have medication, or the attack is severe enough that it requires additional medical attention, the family must decide on the type of care to use. If the family has an existing relationship with a private doctor, they may initiate care through the doctor. However, if the family has little or no prior contact with a doctor, their only option is to go to the hospital.

If these choices depend on the characteristics of the family or the health of the child ($E$) and families choose the type of care that maximizes utility, we expect heterogeneous responses to asthma attacks to arise.

[15] This assumes that levels of outdoor pollution are not perfectly correlated with levels of indoor pollution.

For example, infants have fewer avoidance options[16] and are less likely to have been diagnosed with asthma and thus have the proper medication at home. Furthermore, parents typically bring their infant to the hospital for many acute illnesses, such as asthma attacks, regardless of their general health investment strategy. As a result, we expect younger children to be more likely to go to the hospital for an asthma attack, holding all else equal. Additionally, parents who are more efficient investors in health may be more likely to seek preventative care, increasing the odds of diagnosing asthma. We therefore might expect them to be more likely to manage an attack themselves or to have an existing relationship with a doctor, reducing their likelihood of using a hospital for an asthma attack. Since the characteristics of the family are related to the child's exposure to pollution (as shown above), this suggests that the choice of hospitalization is also potentially correlated with the child's exposure to pollution.

Combining the decision process, we can view the probability of going to the hospital for an asthma attack, *Pr (Y)*, as:

$$Pr(Y) = Pr(H > h) * Pr(HP \mid H > h) = f(P, A, M, W; E) \quad (6)$$

where $Pr(H > h)$ is the probability an asthma attack has occurred and $Pr(HP \mid H > h)$ is the probability of using the hospital as the source of care given that an attack has occurred.

**4. Estimation Strategy**

The above section suggests that all variables in (6) are potentially related to the child's exposure to pollution, indicating that there will be an omitted variable bias if they are not observed. Given that these variables are difficult to observe, I instead propose to control for these variables using the following innovations. First, I look at the effect of air pollution separately for children of different age groups. These groups correspond with both biological development and the type of care that families typically display towards children. I define the age categories of interest as follows: children age 0-1 (lung "branching" occurring at rapid rate; infants most protected by parents and most likely to use hospital for illness); 1-3 (alveoli develop and mature; children spend more time in day care); 3-6 (children more likely to enroll in preschool/kindergarten); 6-12 (elementary school); and 12-18 (secondary school). This will allow for

[16]For example, PEF meters are unavailable for infants (AAP (2000)).

different potential biological and behavioral responses to pollution by the age of the child.

Second, by creating quarterly time-series data at the zip code level, I define the unit of observation as the zip code/quarter and specify a zip code fixed effect (FE). This will capture permanent observed and unobserved factors within a zip code that affect health, such as average smoking rates, average indoor pollution levels, and average health care decisions to the extent that they are constant over time or do not change in ways that are correlated with pollution. Since the zip code is a finely defined geographic area with frequent social interactions amongst residents, the zip code FE will capture a large share of potentially omitted characteristics.

The third innovation comes from using the diverse seasonal variation in pollution in California that arises from local microclimates and geography. For example, levels of ozone increase in the summer at a greater rate because ozone is formed in the presence of sunlight. Particulate matter is trapped by fog in winter weather. CO levels increase in cold, stagnant weather. Figure 1 shows the strong seasonal patterns of these pollutants. Furthermore, these seasonal patterns vary for different parts of the state depending on the unique physical characteristics of each area (U.S. EPA (2000)). For example, ozone increases at a greater rate in the summer in hotter and sunnier areas, such as southern and central California. PM10 increases in drier areas in the summer and fall, but increase in colder areas in the winter because of increased use of combustion sources (Nystrom (2001)). To highlight some of this diversity, figure 2 shows quarterly pollution levels for coastal counties in southern California. For example, Ventura, Los Angeles, and San Diego all have comparable mean levels of O3; however, the quarterly variation in Los Angeles is considerably greater than the other two. Orange County has a lower mean level of O3 than San Diego, but the variation in Orange is greater. Since these diverse patterns in pollution are naturally occurring, it is reasonable to assume that it is independent of many investments in health.

In sum, I will compare how seasonal changes in pollution within a given zip code affect changes in seasonal asthma rates for a specific age group.[17] The following example of smoking rates and outdoor pollution highlights how the empirical strategy works. Failing to control for smoking is only a problem if

smoking behavior is related to both pollution and asthma. By looking at separate age groups, I circumvent the need to control for how parents monitor tobacco smoke around their children based on the age of the child. By using zip code fixed effects, I look at whether changes in pollution are linked to changes in asthma within a zip code. If smoking either doesn't change with changes in pollution, or if it changes in a way that is unrelated to changes in pollution, then the fixed effect would control for smoking behavior. Smoking behavior, however, may change over time or within a year. If this is the case, the fixed effect will not capture the changing smoking patterns. However, if smoking patterns do not change from one season to the next in a way that is correlated with the seasonal changes in pollution unique to that area, then I will not need to explicitly control for smoking behavior.

While this identification strategy overcomes many problems, there is one main source of endogeneity that remains -- contemporaneous avoidance behavior. Since people can directly respond to daily pollution, this will not be captured by the identification strategy. Although I include some measures of avoidance behavior, these measures only capture part of avoidance behavior and only as it relates to ozone. However, as shown in the economic model, contemporaneous avoidance behavior is positively related to pollution levels. If avoidance behavior lowers the likelihood of having an asthma attack, omitting it will yield a lower bound of the true effect.

To see the identification strategy more formally, assume (6) can be represented by the following linear approximation:

$$E(Y_i) = \beta_0 P_i + \beta_1 A_i + \beta_2 M + \beta_3 W + \beta_4 E \qquad (7)$$

However, since $Y_i$ is only observed when it equals one, aggregate to the zip code level and calculate a hospitalization rate:

$$E\left(\frac{Y_z}{N_z}\right) = \frac{\sum_{i=1}^{N} E(Y_i)}{N_z} = \beta_0 \frac{\sum P_i}{N_z} + \beta_1 \frac{\sum A}{N_z} + \beta_2 \frac{\sum M}{N_z} + \beta_3 \frac{\sum W}{N_z} + \beta_4 \frac{\sum E_i}{N_z} \qquad (8)$$

where $N_z$ is the number of children in zip code $z$. Replacing the summations with their average values yields:

[17] By using seasonal changes in pollution, this will only test short to medium-term responses to pollution and not long-term health effects.

$$E\left(\frac{Y_z}{N_z}\right) = \beta_0 P_z + \beta_1 A_z + \beta_2 M_z + \beta_3 W_z + \beta_4 E_z \tag{9}$$

The main problem in estimating this equation is that $A_z$, $M_z$, $W_z$, and $E_z$ are difficult to fully observe. However, given that there are repeated observations for a zip code over time, assign the following:

$$\alpha_z = \beta_{11} A_z + \beta_{21} M_z + \beta_{31} W_z + \beta_{41} E_z \tag{10}$$

This zip code fixed effect will capture permanent observable and unobservable components of these variables. Since $A_z$, $M_z$, $W_z$, and $E_z$ also have contemporaneous components, rewrite (10) as:

$$E\left(\frac{Y_{zt}}{N_{zt}}\right) = \beta_0 P_{zt} + \beta_1 A_{zt} + \beta_{22} M_{zt} + \beta_{32} W_{zt} + \beta_{42} E_{zt} + \alpha_z \tag{11}$$

While some measures for $A_{zt}$, $M_{zt}$, $W_{zt}$, and $E_{zt}$ exist, it is unlikely that I can adequately measure all of them. However, using seasonal variation in pollution assumes the following:

$$\begin{aligned} &\rho\left(P_{zt}, M^*_{zt} \mid \alpha_z\right) = 0 \\ &\rho\left(P_{zt}, W^*_{zt} \mid \alpha_z\right) = 0 \\ &\rho\left(P_{zt}, E^*_{zt} \mid \alpha_z\right) = 0 \end{aligned} \tag{12}$$

where * is the unobserved component. That is, after controlling for permanent factors via a zip code fixed effect, seasonal changes in pollution are unrelated to unobserved seasonal changes in $M_{zt}$, $W_{zt}$, and $E_{zt}$. This is the fundamental identification assumption of this model.

Additionally, using the first prediction from the model, we expect the following to hold:

$$\begin{aligned} &\rho\left(P_{zt}, A^*_{zt} \mid \alpha_z\right) \geq 0 \\ &\beta_{12} \leq 0 \end{aligned} \tag{13}$$

That is, contemporaneous avoidance behavior is positively related to pollution and improves health (by lowering the likelihood of an asthma attack). It is straightforward to show that $\widehat{\beta_0} \leq E\left(\beta_0\right)$, meaning the estimate for $\beta_0$ will be a lower bound of the true effect.

It is worth highlighting the potential impact from omitting contemporaneous avoidance behavior because responses are likely to vary by the pollutant – some pollutants are more "recognized" than others. For example, ozone has been a pollutant of major focus because its concentration often exceeds the National

Ambient Air Quality Standards (NAAQS) as outlined in the Clean Air Acts. As a result, these exceedances are reflected in various media sources, raising public awareness of ozone levels. The following chart lists the main pollutants considered in this analysis[18] and their sources for recognition.[19]

| Pollutant | Emission Sources | Violations of NAAQS | Direct Detection |
|---|---|---|---|
| O3 | Automobiles and industrial sources, reacts in sunlight and heat | Frequent violations | Major component of visible urban smog |
| CO | Automobiles | Some violations | Odorless and colorless |
| PM10 | Directly emitted and formed from other pollutants | Some violations | Reduces visibility |
| NO2 | Automobiles and stationary fuel combustion sources | Little or no violations | Odor and visible at moderate levels |

To proceed with estimation, to insure that asthma rates are bounded below by 0, I adjust the functional form of (11) using the log of the asthma rate:

$$E\ln\left(\frac{Y_{zt}}{N_{zt}}\right) = \beta_0 P_{zt} + \beta_1 A_{zt} + \beta_{22} M_{zt} + \beta_{32} W_{zt} + \beta_{42} E_{zt} + \alpha_z \tag{14}$$

Distributing the log and parameterizing population gives:

$$E\ln(Y_{zt}) = \beta_0 P_{zt} + \beta_1 A_{zt} + \beta_{22} M_{zt} + \beta_{32} W_{zt} + \beta_{42} E_{zt} + \beta_5 \ln N_{zt} + \alpha_z \tag{15}$$

However, since $Y_{zt}$ can take on the value of zero, instead exponentiate (15) to get:

$$E(Y_{zt}) = \exp\{\beta_0 P_{zt} + \beta_1 A_{zt} + \beta_{22} M_{zt} + \beta_{32} W_{zt} + \beta_{42} E_{zt} + \beta_5 \ln N_{zt} + \alpha_z\} \tag{16}$$

This is now equivalent to a Poisson regression with arrival rate $\lambda_{zt} = E(Y_{zt})$.[20] $\beta_0$ is the coefficient vector of interest. The main hypothesis to test is whether $\beta_0 = 0$, namely that pollution has no effect on asthma hospital admissions.

[18] In an earlier version of this paper, I included sulfur dioxide (SO2) in the analysis. I omit SO2 in this analysis because 1) there are very few monitors for SO2, which makes it difficult to accurately assign exposure to SO2 without significant reductions in sample size and 2) current levels of SO2 are widely believed to be low enough such that they do not pose a threat to health. Since SO2 primarily comes from stationary sources and as a result is not highly correlated with the other pollutants considered, omitting SO2 did not affect estimates of the other pollutants.

[19] In addition to sources that target a wide range of audience, there are individual specific avoidance possibilities. For example, PEF meters are a widely prescribed part of asthma treatment plans (AAP (2000)). Families can use these devices to gauge lung functioning on any given day, regardless of what they may know about pollution levels. However, since PEF meters are unavailable for infants, they should not interfere with estimation for this age group.

[20] There are alternative ways to motivate this as a Poisson regression. See Portney and Mullahy (1986) for one alternative.
I also relax the Poisson assumption to test the validity of this restriction.

## 5. Data

Sources

The California Hospital Discharge Data (CHDD) is a rich source of individual health outcomes. This data set records the principal diagnosis of the patient upon release from the hospital[21], the month of admission,[22] the zip code of residence, as well as the sex, race, age, and the expected source of payment for all individuals discharged from a hospital in the state of California. Data are available from 1992 to 1998 and each year contains on average over 800,000 hospital discharges for children under age 18 (not including newborns).

While hospital data does not include information on all asthma attacks, the CHDD offers three key advantages over self-reported surveys. First, hospital discharges, in particular ER admissions, are a more objective measure of asthma and are less likely to be sensitive to reporting biases.[23] Second, there are a large number of observations available each year in the CHDD. Third, having the zip code of the patient enables me to specify a zip code fixed effect and to merge other key data sources at the zip code level.

The key data merged are atmospheric pollution levels from Environmental Protection Agency (EPA) air monitoring stations throughout California. The monitor data are readily available from 1982 until the present and are the most detailed data recording ambient levels of criteria pollutants. Furthermore, they contain the exact location of the monitor, enabling them to be merged with the CHDD. Figure 3 shows O3 monitors in California in 1999 along with county outlines. These monitors are mainly located in the more densely populated areas (shaded in gray). Figure 4 highlights Los Angeles County, showing again O3 monitors and now the outlines of zip codes. Since Los Angeles is a diverse county both demographically and geographically and there are many monitors to capture local pollution levels, assigning pollution at the zip code level should produce more reliable measures than from assigning it at a broader level.

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[21] This is assigned according to the International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM) by the U.S. Department of Health and Human Services.

[22] The exact day of the month is censored in the version of the data that has already been released to me. Only an indicator for the day of the week is available.

[23] ER admissions account for approximately 67% of all hospital admissions for asthma.

I also merge other data sources at the zip code level. Monthly meterological data from the National Climatic Data Center contains various measures from more than 1000 weather stations in California as well as their exact location.[24] The California Association of Realtors provides monthly zip code level information on the number of homes and average and median sales price from 1991 to the present.[25] Using 1990 Census estimates of population counts by age for each zip code and annual county estimates by age from the Demographic Research Unit of the California Department of Finance, I have approximated the annual population for each zip code and age group.

As proxies for avoidance behavior, I merge the number of health advisories announced in each quarter. Health advisories are required by California law to be issued by local air quality management districts[26] when criteria pollutants exceed levels as specified by the California Air Resources Board. When this occurs, schools are directly contacted and are urged to limit physical activities for children until pollution levels ease, while other sensitive people are advised to avoid the pollution by remaining indoors (Air Resources Board (1990)). While these advisories are required to be announced for all of the criteria pollutants, historically announcements have only be made for ozone levels, and as a result the advisories are commonly referred to as "smog alerts."

Linking Pollution

To approximate a quarterly time-series of pollution at the zip code level, I first calculated the coordinates for the centroid of each zip code in California. Using the reported coordinates of the EPA monitors, I then measured the distance between each centroid and each monitor. Finally, I calculated the level of pollution for a zip code by averaging reported values from all monitors within 20 miles of the centroid, weighting by the inverse of the distance from the centroid to the monitor.[27] Therefore, I define pollution in zip code $z$ at time $t$ as:

$$P_{zt} = \sum_j \left( P_{jt} * \frac{1}{D_j \mid D_j \leq 20} \right) / \left( \frac{1}{D_j \mid D_j \leq 20} \right) \tag{17}$$

[24] The meterological data are merged using the same inverse-distance weighted technique used to approximate zip code levels of pollution (described below).

[25] Since both the meterological and housing data are available monthly, I average them to a quarterly level.

[26] There are currently 17 air quality management districts in California.

where $D_j$ is the distance from monitor $j$ to the centroid of zip code $z$ and $P_{jt}$ is the pollution measure at monitor $j$ at time $t$.

Four immediate issues arise in measuring pollution in this way. First, many monitors have been added or removed over the time period studied. This occurs because pollution monitors are installed in areas where pollution exceeds NAAQS, but can also be removed from an area if it falls below NAAQS (U.S. EPA (1999b)). As a result, monitors are more likely to be placed in areas where pollution levels have been increasing, and less likely to exist in areas where pollution has been declining. To assess the implication of this, I estimate (17) in two ways: using all monitors from 1992 to 1998 and using only continuously operated monitors from 1992 to 1998. Appendix table 1 shows the number of monitors over time for both methods and the correlation between quarterly zip code levels of each pollutant calculated by each method. The overall number of monitors has not changed considerably and the correlations for all are at least 0.98, indicating that the sampling technique used for monitors should not interfere with inference.[28]

Second, while it is crucial to control for multiple pollutants simultaneously, trying to separately identify the effect of each pollutant can be difficult if pollutants are highly correlated. Many pollutants originate from similar sources, as the preceding chart indicated. Appendix table 2 shows the correlation matrix for the pollutants considered here. O3 does not appear highly correlated with any other pollutants, while NO2 appears highly correlated with CO and PM10. This may make it difficult to obtain precise estimates for NO2.[29]

Third, there are many factors that affect how pollutants travel, such as wind, rain, and the size of the pollutant particle, and this may affect how well (17) measures the actual pollution concentration[30]. For example, particulate matter, such as PM10, settles to the ground at a much quicker rate than do gaseous pollutants (Wilson and Spengler (1996)). To get a sense of how accurate the above approach is, I estimate the level of pollution at each monitor (as opposed to zip code) using the above formula as if the monitor of

[27] I also changed the radius to 10 and 5 miles to test the sensitivity of this assumption, but the results were similar.
[28] For SO2, the number of monitors fell from 62 to 38 over this period, with 35 continuously operated.
[29] When including SO2 in the correlation matrix, the correlation between SO2 and O3, CO, PM10, and NO2 are .01, .34, .20, and .36, respectively. The other rows of the correlation matrix remain nearly identical.
[30] While I obtained measures of precipitation to include in the analysis, wind data is not as widely available. Furthermore, it is unclear exactly how to incorporate wind data.

interest were not there. Therefore, I estimate the amount of pollution at a given monitor based on the pollution levels at monitors less than 20 miles away. I do this for all monitors and then calculate the correlation between the estimated pollution and the actual pollution, shown in appendix table 3. The correlation for O3 and NO2 are remarkably high. This is not surprising since both pollutants are formed in the atmosphere, as opposed to being direct products of emission. For PM10 and CO, the correlations are slightly lower, but are still high enough that it does not appear to be a major concern.[31]

Fourth, since monitors tend to exist in more polluted and populated areas, it is important to understand how the characteristics of the population in these areas differ from those that are excluded from the analysis. Appendix table 4 shows various demographic characteristics for zip codes that are within 20 miles of a monitor for each of the pollutants and zip codes that are not. While all of the variables shown are statistically different, the driving force behind these differences appears to be the percent of the population of the zip code that lives in urbanized areas. This coincides with the monitor locations shown in figure 3. Since rural areas represent a much lower fraction of the population, omitting them is not likely to affect the results considerably.

Trends and Descriptive Statistics

Table 1a shows the descriptive statistics of the data used in the analysis, including the "between" and "within" zip code variation of each variable.[32] For the pollutants, it is not unusual for the seasonal within zip code variation to exceed the between zip code variation, as is the case for O3 and CO. For asthma admission rates[33], younger children have a greater likelihood of visiting the ER[34], with infants approximately 6 times more likely to visit the ER than children over 6 and 1-6 year old 1.5 times more likely to visit than children over 6. Most of the variation in asthma rates comes from within the zip code. The patterns in variation for asthma and pollution suggest ample variation for obtaining precise estimates using the identification strategy described above.

Table 1a also shows variables that represent $A_{zt}$, $M_{zt}$, $W_{zt}$, and $E_{zt}$. House prices are designed to

[31] For SO2, the correlation is only 0.59, indicating the potential mismeasurement that arises in using SO2.
[32] The "between" standard deviation is calculated using $\bar{x}_i$ and the "within" is calculated using $x_{it} - \bar{x}_i + \bar{x}$.
[33] Asthma is labeled as ICD-9-CM 493.

reflect changes in asset wealth and are a "sufficient" statistics for many demographics of a given area, such as school quality and crime rates. The percentage of newborns with government sponsored health insurance (calculated from the CHDD) is used as a measure of changes in income.[35] The percentage of normal newborns (calculated from the CHDD[36]) is used to approximate the health stock for infants. Average maximum temperature and inches of precipitation both affect the likelihood of being outdoors and may directly exacerbate asthma symptoms (American Lung Association (2001)). Additional controls not shown in the table are seasonal dummies, which attempt to capture children's time outdoors as dictated by school schedules, and annual dummies, designed to capture general changes in factors that affect asthma that are common to all groups, such as technological changes in prevention, treatment, and labeling of asthma.

Since asthma disproportionately attacks children of low SES, table 1b shows pollution levels and ER asthma rates for various SES groups. I define SES groups as above and below the median for the percent of adults over 25 years old in a zip code without a high school diploma. The average levels of all pollutants except O3 are higher for the low SES groups. Asthma rates are almost twice as high for children under age 6, and approximately 50% higher for children over age 6. These differences in pollution and asthma rates by SES are statistically significant.[37]

Table 1c shows cumulative counts of ER asthma admissions by age group. For every age group, most of the counts are either 0,1, or 2, and 99% percent of the counts are under 6. The highest count for any age group is 20. These numbers support the appropriateness of a count-data regression model, such as the Poisson model.

In turning to annual trends, figure 5 shows annual ER asthma rates for the various age groups. The admission rates appear relatively stable over time for infants and 6-12 year olds except for upward spikes in 1995 and 1997. For the other groups and averaged across all age groups, rates have generally gone down

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[34] ER admissions are distinguished from other admissions according to the "source of admission" variable from the CHDD.
[35] There was only one expansion in medicaid eligibility that affected newborns during the time period studied. In February of 1995, eligibility was extended from 185 to 200 percent of the federal poverty level. Although Access to Infants and Mothers (AIM) also increased during this period, less than 0.6% of all births in California are paid for by AIM (Managed Risk Medical Insurance Board (2001)).
[36] In the CHDD, newborns are classified into one of the following seven categories: 1) died or transferred 2) extreme immaturity or respiratory distress syndrome 3) prematurity with major problems 4) prematurity without major problems 5) full term with major problems 6) neonate with other significant problems and 7) normal newborn.

over time, also with spikes in 1995 and 1997. To compare asthma patterns in California with those elsewhere in the U.S., figure 6 shows all hospital admissions for asthma for children in California, the entire U.S., and each region of the U.S. using the National Hospital Discharge Survey (NHDS).[38] The northeast has the highest admission rate, followed by the Midwest, the south, and then the west. California rates follow the same pattern as the entire west but appear slightly lower. The pattern for California is remarkably comparable to that for the entire U.S. In turning to quarterly patterns, figure 7 shows asthma patterns over time for each age group separately. Immediately evident are the strong seasonal patterns for admissions for all age groups. Rates for each age group increase on average anywhere from 1.5 to 2.5 times from the lowest quarter to the highest. Furthermore, the seasonal patterns differ across age groups. The high season for infants is the 1st quarter, whereas high season for teens is the 4th quarter. These striking patterns demonstrate the importance of looking at age groups separately and the potential value in exploiting seasonal variation.

Before turning to the estimation, a case study of a specific zip code highlights the main findings of this analysis. Figure 8 plots quarterly standardized pollution levels and asthma counts for children ages 1-3 in zip code 92410 (San Bernardino). A strong pattern between asthma and CO emerges, with peaks and trough occurring at roughly the same time throughout the entire time period. While at times asthma follows the patterns of other pollutants, the pattern tends not to persist for the entire time period, indicating a potential link between CO and asthma.

## 6. Results

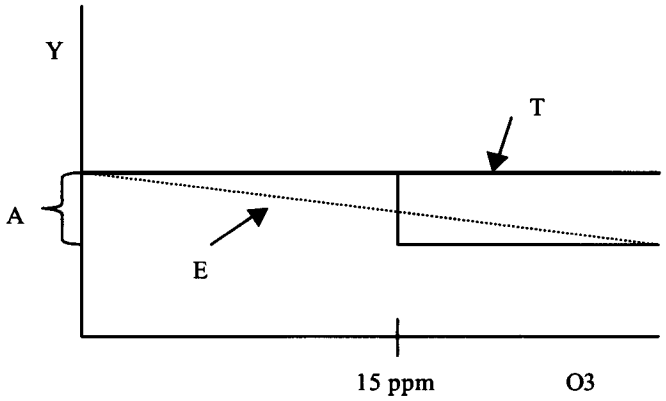
Main Results

The first set of results is fixed effect estimates of equation (16) without any direct controls for avoidance behavior, shown in table 2a.[39] The results indicate that pollution has a different effect on infants as compared to older children. NO2 is significant and positively related to asthma ER hospitalizations for infants. However, for all older age groups, CO is positive and significantly correlated with asthma. In terms of the control variables, temperature does not appear to be significantly related to asthma, while precipitation

[37] These patterns are also present when SES is defined by race or income.
[38] The NHDS does not provide information to separately identify emergency and non-emergency hospital admissions and the only geographic identifier is the region.

is generally negatively correlated with asthma, suggesting that increases in rain may lower children's exposure to pollution by increasing their amount of time spent inside or by "cleaning" the air (Wilson and Spengler (1996)). The coefficients for the demographic variables are almost always imprecisely estimated. Since these variables often have significant effects on health outcomes, this suggests that the zip code fixed effects appear to control for a large amount of observed as well as unobserved heterogeneity.

While the presence of negative coefficients for O3 in table 2a may at first seem surprising, not controlling for contemporaneous avoidance behavior yields estimates that are lower bounds of the true effect. Furthermore, if people respond to an increase in pollution by increasing avoidance behavior to the point that health actually improves, it can induce a negative effect. The following diagram illustrates how negative effects could arise for O3.




T = true dose-response
E = estimated dose-response
A = avoidance behavior

When ozone exceeds 15 ppm, a health advisory is announced. If schools or parents respond by keeping their children inside, children may exercise less as a result. Since exercise is believed to induce asthma and is not directly observed, by omitting *A* I would estimate line *E* instead of *T*, yielding a spurious negative effect of O3 on asthma hospitalizations.[40]

To test the impact from omitting avoidance behavior, I add to the model the number of health advisories announced in each quarter. The results from including this variable, reported in table 2b, show

[39] For ease of interpretation, all pollutants have been standardized to have a mean of zero and standard deviation of one.

that health advisories have a strong negative effect on asthma admissions for all age groups except the oldest, supporting the notion that avoidance behavior is actively undertaken. Meanwhile, the negative effect for O3 almost entirely disappears and there are no qualitative changes in the other pollutants. This supports that omitting avoidance behavior induces a lower bound of the true effect of pollution on health, and the other pollutants considered might have a larger effect on asthma hospitalizations if more controls for avoidance behavior were available.

An important issue to explore is whether the effect of pollution on asthma is the same for all subsets of the population. Some groups, such as children with low health stock, may face different risks from comparable levels of pollution. Additionally, as table 1b shows, pollution levels and asthma rates are higher for children of low SES. To assess the importance of this, I run separate regressions for the SES groups as defined in table 1b.[41] There are two items worth noting from these results, shown in table 3. First, although the effect of CO is only statistically different for children ages 3-6, the results suggest that the impact of CO is in general larger for children of low SES, providing one possible explanation for some of the differences in asthma rates by SES. Second, the effect of health advisories is smaller for children of low SES, with statistically significant differences for children ages 6-12 and 12-18. This suggests that avoidance behavior is less actively undertaken by low SES families, and could also explain some of the difference in asthma rates by SES.

Sensitivity Analysis[42]

While the identification strategy used appears successful in capturing a large share of unobservable characteristics, there are remaining potential sources of endogeneity. If trends have occurred during the 90s that do not affect all subsets of the population equally, such as smoking or health care patterns, then the annual dummy variables will not suffice. To account for these and other possible trends, I specify the fixed

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[40] Note that this diagram assumes that ozone has no effect on asthma. However, the same effect could occur if ozone (or other pollutants) has a positive effect on asthma.

[41] I also performed this analysis by defining SES according to minority population and income, and the results were comparable.

[42] An ideal test of the overall validity of this empirical strategy would be to replace ER asthma admissions with certain hospitalizations that are unlikely to be significantly related to pollution. If I find a significant effect of pollution on these outcomes, this would suggest the model is misspecified. However, such outcomes must meet the following

effect as a zip code/year interaction. This strategy compares the effect of seasonal changes in pollution within a given zip code in a given year on asthma. The results from this strategy are shown in table 4. For all age groups, the only considerable change is that the effect of NO2 on infants now disappears.

I also perform checks on the sensitivity to the distributional assumption. The Poisson model, by accommodating the count structure of the data, implies a non-linear conditional expectation function (CEF). To test if this CEF is reasonable, I estimate a linear model, which provides the best linear approximation to the true CEF, and evaluate the coefficients comparable to those produced by the Poisson model ($\beta = \delta lny / \delta x$). Additionally, a common concern with the Poisson model is that it cannot accommodate the excess of mass at zero.[43] If we view the number of asthma ER hospitalizations not as actual counts but as a proxy for some true unobserved variable, then the ordered probit is an appropriate specification that will not be affected by the excess zeros.[44] While the coefficients from the ordered probit are not directly comparable to those from the Poisson, they provide a qualitative test of the distributional assumption. These result, shown in table 5, do not indicate any considerable differences for the linear or ordered probit models, and support the appropriateness of the Poisson distribution.

Magnitude of Effect

To get a sense of the magnitude of these findings, we can measure the percentage change ($\delta$) in asthma hospitalizations that has resulted from changes in pollution levels over time:

$$\delta = (\lambda_p - \lambda_c) / \lambda_p \qquad (18)$$

where $\lambda_p$ is the arrival rate for asthma hospitalizations with previous levels of pollution and $\lambda_c$ is the arrival rate with current levels of pollution. Using equation (16) for $\lambda$, we can rewrite (18) as:

$$\delta = exp\{\beta_0 * (P_p - P_c)\} - 1 \qquad (19)$$

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requirements: 1) they must not be related to pollution via avoidance behavior; 2) the outcomes must be of count nature; and 3) the outcomes must show seasonal variation. I was unable to readily identify outcomes that met these criterion.

[43] Although cross-sectional count models have been developed to accommodate the excess zeros, none are currently available for fixed effect models, largely due to an incidental parameter problem posed by the individual fixed effects and ill-behaved likelihood functions. I attempted to estimate the cross-sectional model with a full set of zip code dummy variables, but the models would not converge.

[44] Although there is an incidental parameters problem in FE probit models in general, Heckman (1981) has shown that at T=8 the incidental parameters do not considerably affect the parameters of interest. Here, $T \approx 28$. I censor the number of counts at 5 for the ordered probit since over 99 percent of counts are no more than 5 for each age group.

where $\beta_0$ is the coefficient of the effect of pollution on admissions and $P_p$ and $P_c$ are the pollution levels in previous and current years, respectively. If we treat the above estimates as causal, replacing $\beta_0$ with its estimated coefficient and $P_p$ and $P_c$ with their corresponding average pollution levels will provide an estimates of $\delta$.

Table 6a shows the results for this exercise on ER admissions for asthma using $P_p$ first as 1992 and then as 1980, and $P_c$ as 1998. The declines in pollution since 1992 have decreased hospitalizations for the various age groups from 3.6% to 13.8%, with more pronounced affects for the older age groups. The declines in pollution since 1980 have more dramatic effects, leading to decreases in hospitalizations that range from 13.8% to 45%, also with larger effects for older children.

To get a rough idea of some of the annual economic benefits associated with these lower levels of pollutants, I multiply the number of asthma hospitalizations in 1998 by $\delta$ to get an estimate of the change in the number of asthma cases. Then I multiple this by the average cost of hospitalization for asthma in 1998. Thus, the fall in pollution levels from those experienced in 1992 has saved approximately $4.4 million in ER admissions for asthma in California in 1998 alone, while the fall in pollution from 1980 has saved approximately $14.5 million in 1998. These numbers, however, represent a lower bound of the true social benefits associated with reductions in asthma attacks. They only include emergency room admissions and their expenses in California, thus ignoring the rest of the U.S., other sources of care for asthma attacks, follow-up treatment, lost wages for the family, lost human capital development of the child, psychic costs to the family, and any long-term link to health problems for the child.[45]

Since avoidance behavior as measured by health advisories has a significant effect on hospitalizations for asthma, it is useful to approximate the magnitude of these advisories.[46] To measure the percent reduction from an additional advisory conditional on O3 exceeding 15 ppm, specify (19) as:

$$\delta = exp\ \{\beta_1 * Health\ Advisory/O3 \geq 15\ ppm)\} - 1 \qquad (20)$$

[45] See Harrington and Portney (1987) for a more detailed description of these additional costs. Unfortunately, there are no readily comparable measures of the costs to industry from increased pollution. See Greenstone (1998) for an excellent and up-to-date study of this subject.

[46] Although the empirical strategy does not explicitly attempt to identify the effect of avoidance behavior, I include these estimates to obtain a ballpark sense of the magnitude of health advisories.

Shown in table 6b, replacing $\beta_1$ with its estimated coefficient, the announcement of a health advisory reduces asthma hospitalizations by 3 to 6% for children under age 12.[47]

Although the coefficient estimates by SES are comparable, table 1b indicated that low SES children face considerably higher levels of pollution. To get a sense of the impact of these higher levels of pollution, I approximate the proportional effect of higher pollution levels in low SES areas on asthma by alternatively specifying equation (19) as:

$$\delta = exp\{\beta_0 * (P_L - P_H)\} - 1 \qquad (21)$$

where $P_L$ and $P_H$ are the pollution levels for the low and high SES groups, respectively. By replacing $P_L$ and $P_H$ with average pollution levels reported in table 1b, we can get an estimate of $\delta$. These effects, shown in table 6c, indicate that higher levels of pollution explain as much as 4% of the difference in asthma ER hospitalizations.[48] This suggests that although the increased presence of pollution in low SES areas puts these children at a higher risk for hospitalization for an asthma attack, there are still many other factors that affect hospitalizations.

**7. Discussion**

There are three main findings in this paper. First, CO increases asthma hospitalizations for children ages 1-18. Although NO2 appears to have an effect on infants in certain specifications, this finding is not as robust as those for CO. How plausible are these results? Since infants typically spend more time inside, they may be less affected by outdoor pollution. For older children, the plausibility of a direct effect of CO on asthma is unlikely because CO mainly affects the brain and heart by travelling through the bloodstream, rather than the lungs (World Health Organization (2000)). However, because CO mainly comes from vehicle exhaust, a likely explanation is that CO functions in these models as a proxy for vehicle emissions (U.S. EPA (2000)). Furthermore, since these estimates are lower bounds of the true effect of pollution on health, the possibility of an effect for other pollutants can not be ruled out.[49]

[47] While it would be ideal to get a measure of the hospitalization costs associated with this, it is not feasible in this case because the percent reductions are conditional on O3 exceeding 15 ppm.

[48] This does not necessarily imply that pollution is more likely to induce asthma in low SES children. High SES children could use sources of care other than the hospital.

[49] Additionally, effects from daily peaks may be "smoothed out" in a seasonal analysis.

A second finding that emerged is that avoidance behavior appears to play a significant role in reducing the effect of pollution on childhood asthma, as indicated by the negative effect of health advisories on admissions. Furthermore, avoidance behavior appears to be less actively undertaken by low SES families. Given these findings, it is important to understand the effects of other potential sources for avoidance behavior, as it can suggest other policies to improve health outcomes. Moreover, the costs associated with avoidance behavior cannot be ignored in a welfare analysis.

A third finding is that the net effect of pollution appears to be larger for children of lower SES, suggesting that pollution may be responsible for some of the gradient in incidence of asthma by SES. Furthermore, neurobiological and economic research has suggested that early shocks to a child's health can persist for many years (Shonkoff and Marshall (1990), Case et. al. (2001), Currie and Hyson (1999)), and asthma itself has been associated with later health conditions, including lung cancer (Ernster (1996)). Therefore, if poorer families are unable to afford to live in cleaner areas and as a result their children's health development suffers, this would suggest that pollution is one potential mechanism by which SES affects health.

Since current pollution standards are based on adult health responses, understanding the link between pollution and children's health has become increasingly important to a wide audience, and particularly to the EPA. The next step in this project is to look at the links between air pollution and other health outcomes, such as the incidence of low birthweight and other respiratory illnesses. The empirical strategy developed here appears to be fruitful for finding these links and developing more comprehensive measures of some of the health benefits from improvements in air quality.

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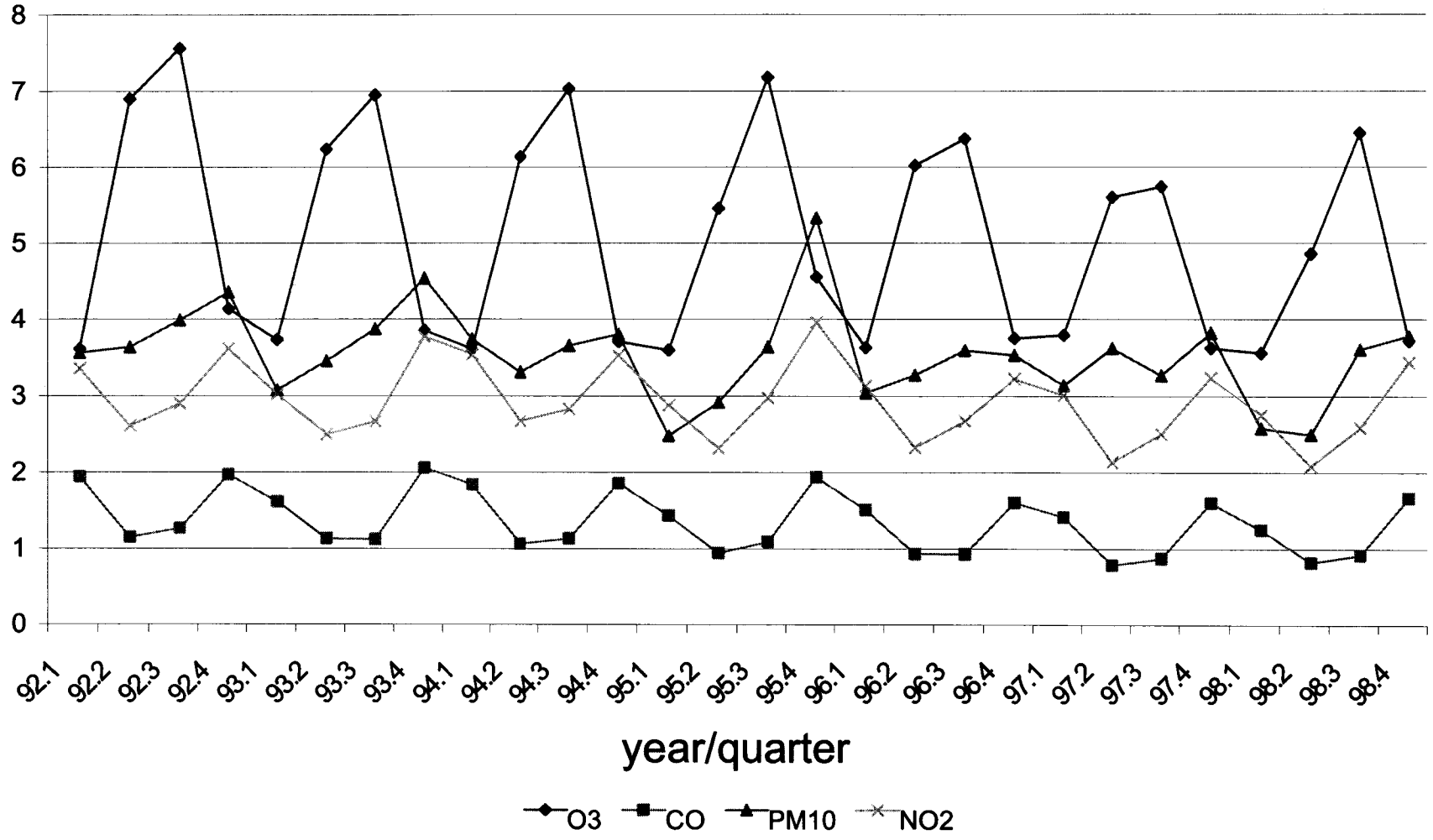
# Figure 1: Quarterly Pollution



# Figure 2. Seasonal Variation in Pollution by County

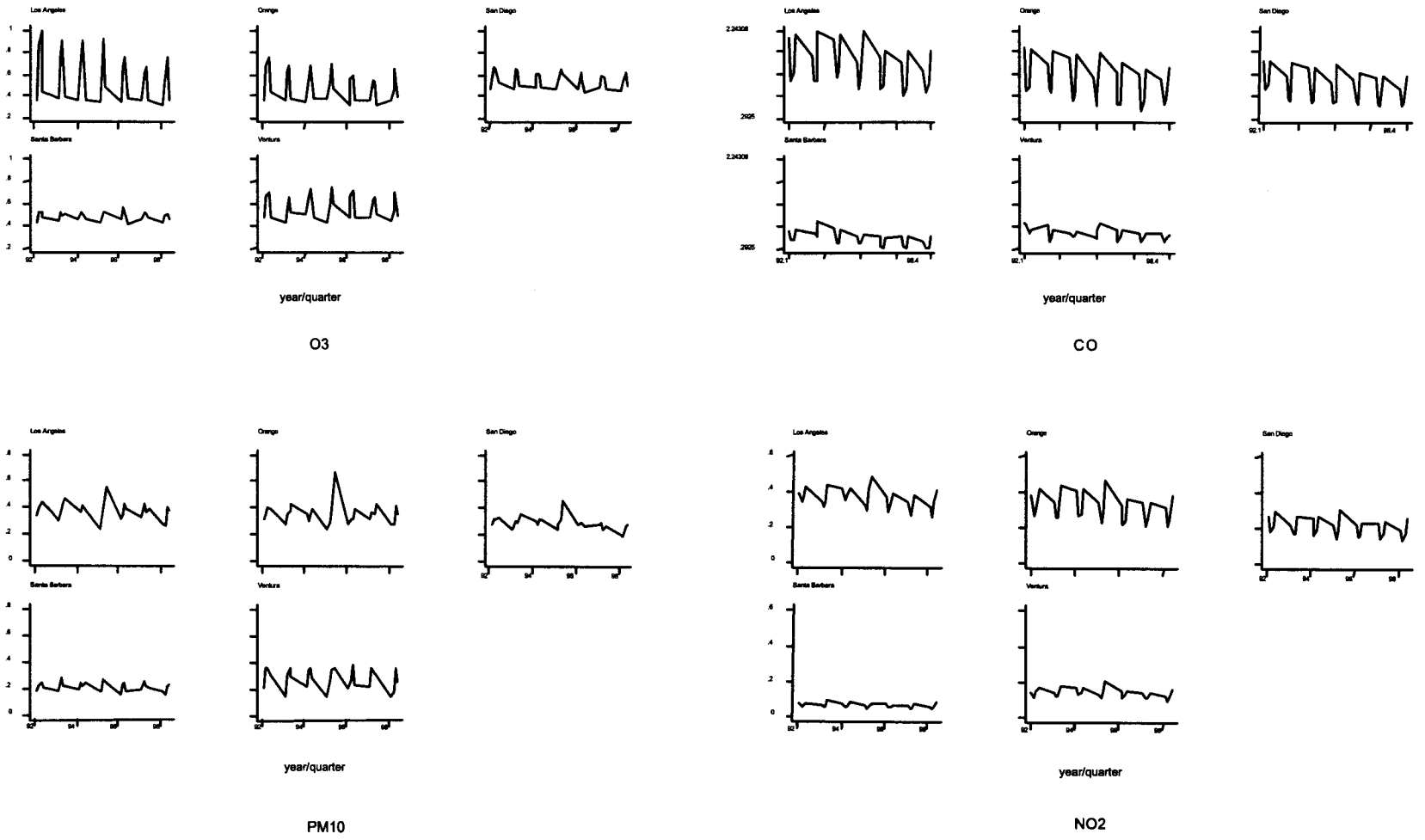


**Figure 3. Ozone Monitors in California**

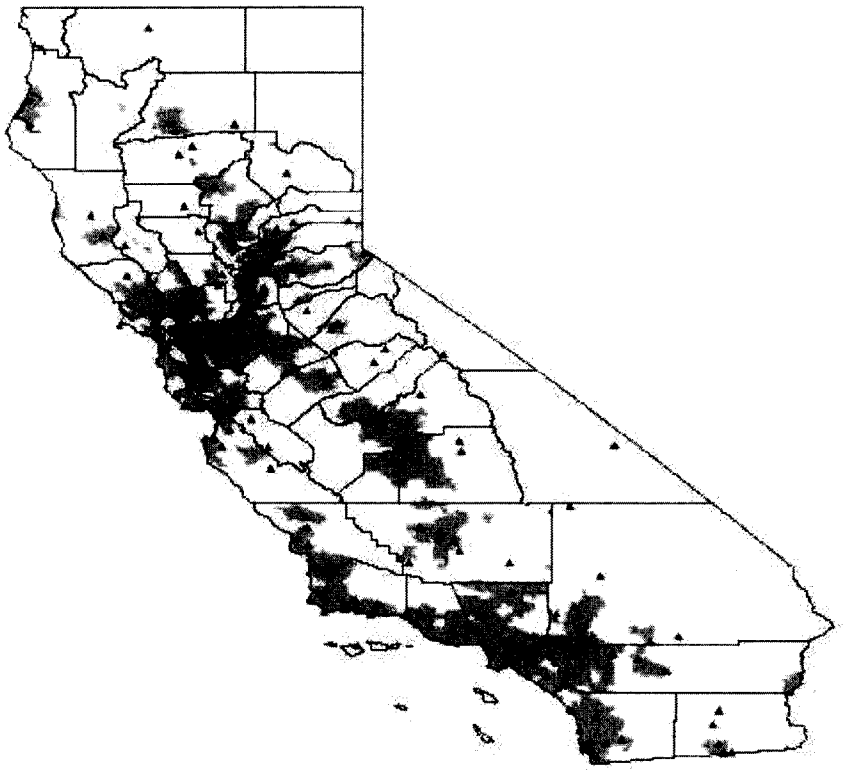


**Figure 4. Ozone Monitors in Los Angeles County**

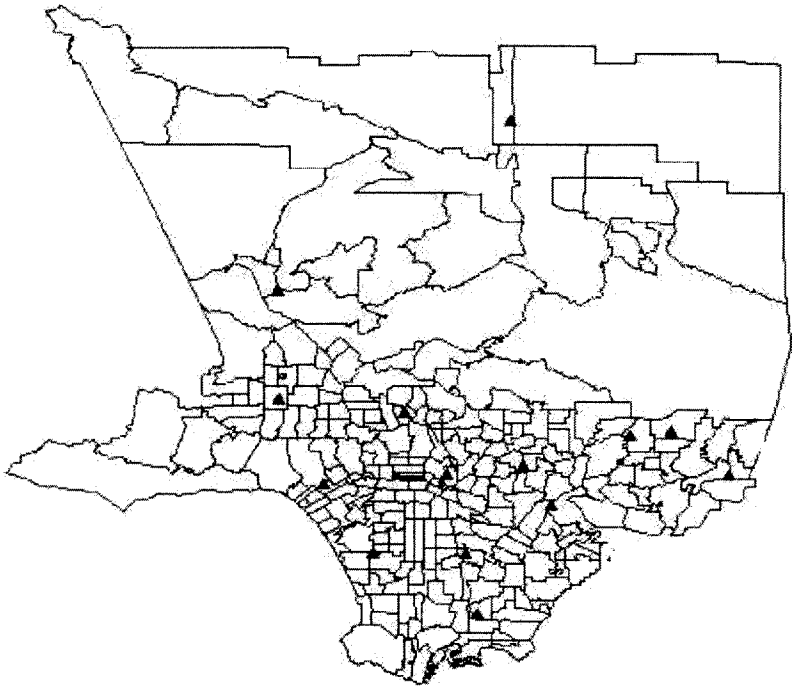


**Figure 5. Asthma ER Admission Rates by Age**

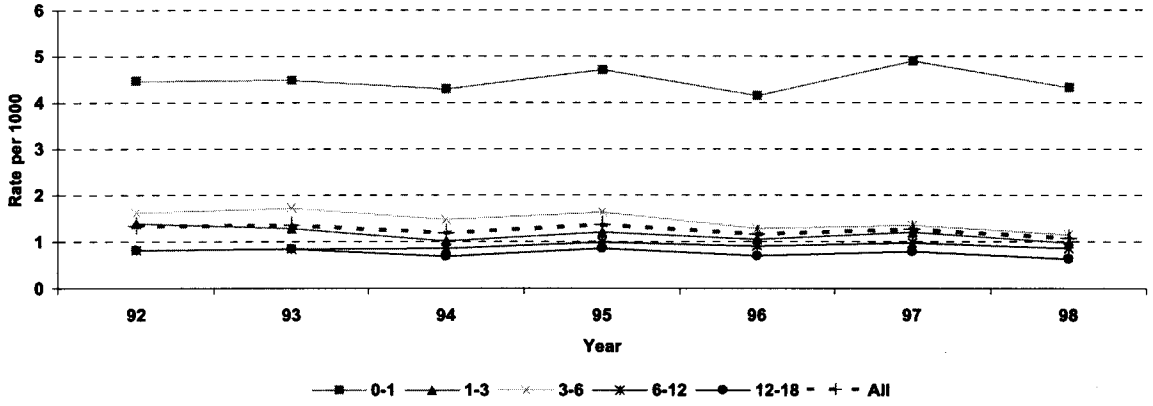



**Figure 6: All Hospital Admissions for Asthma for Children in U.S.**

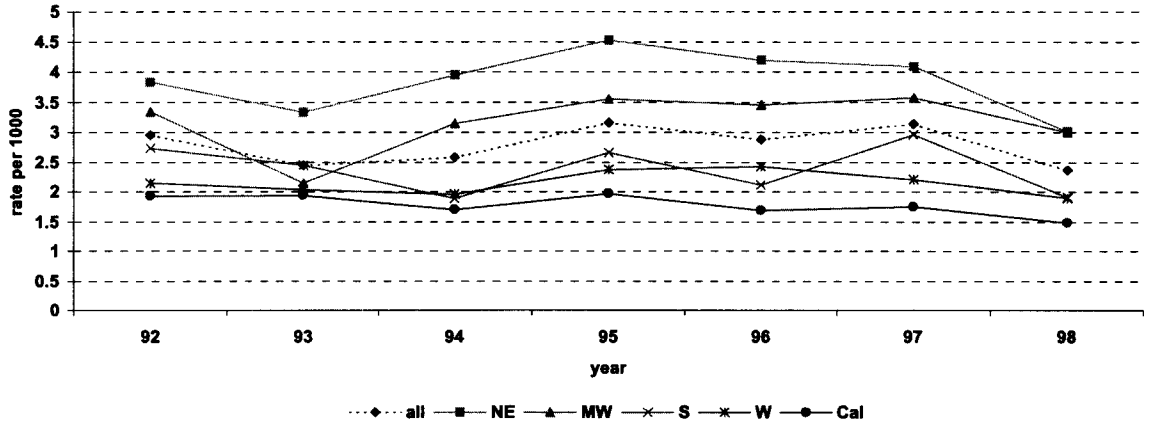
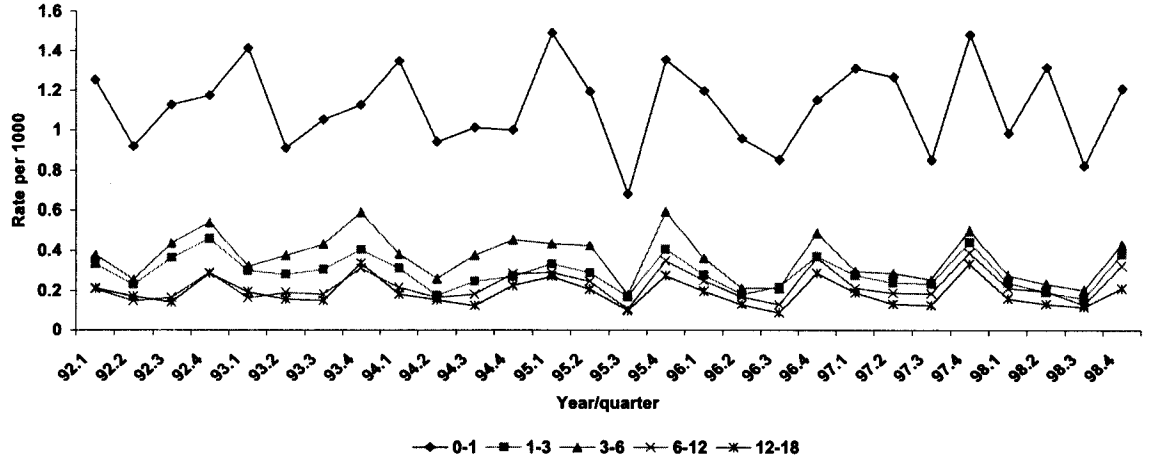



**Figure 7. Quarterly ER Asthma Rates by Age**



# Figure 8. Pollution and Asthma for Ages 1-3 in Zip 92410

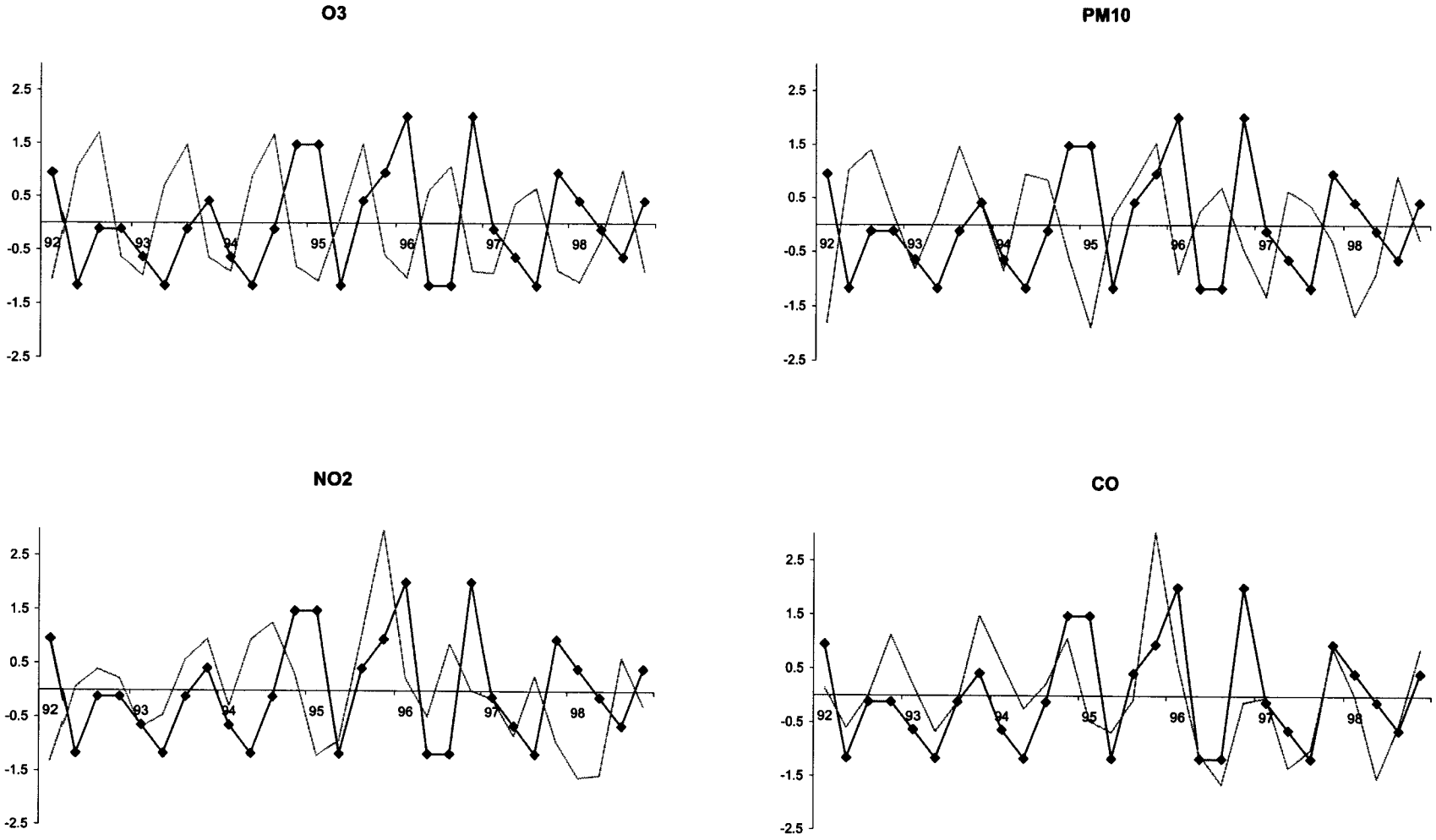


**Table 1. Summary Statistics**

**A. Descriptive Statistics**

| | Obser-vations | Groups | Mean | Std. Dev. | 'Between' Zip SD | 'Within' Zip SD |
|---|---|---|---|---|---|---|
| O3 (ppm*10) | 23345 | 842 | 0.491 | 0.196 | 0.113 | 0.160 |
| CO (ppm) | 23345 | 842 | 1.090 | 0.541 | 0.374 | 0.392 |
| PM10 ($\mu g/m^3/100$) | 23345 | 842 | 0.325 | 0.120 | 0.090 | 0.079 |
| NO2 (ppm*10) | 23345 | 842 | 0.254 | 0.107 | 0.093 | 0.053 |
| % normal neonates | 23226 | 842 | 0.701 | 0.083 | 0.064 | 0.053 |
| % government insurance | 23323 | 842 | 0.383 | 0.224 | 0.217 | 0.058 |
| ave. max. temperature (°F) | 23345 | 842 | 7.38 | 0.92 | 0.35 | 0.84 |
| total precipitation (in.) | 23345 | 842 | 5.81 | 7.37 | 1.80 | 7.15 |
| semi-annual house price/10,000 | 17646 | 735 | 21.69 | 14.44 | 14.56 | 6.89 |
| health advisories | 23345 | 842 | 0.76 | 1.71 | 0.68 | 1.57 |
| ER asthma rate age 0-1 | 23345 | 842 | 1.08 | 3.33 | 1.42 | 3.02 |
| population age 0-1 | 23345 | 842 | 491 | 397 | 395 | 34 |
| ER asthma rate age 1-3 | 23623 | 853 | 0.27 | 1.38 | 0.45 | 1.30 |
| population age 1-3 | 23623 | 853 | 1774 | 1446 | 1442 | 112 |
| ER asthma rate age 3-6 | 23623 | 853 | 0.36 | 2.76 | 1.02 | 2.58 |
| population age 3-6 | 23623 | 853 | 1654 | 1334 | 1321 | 192 |
| ER asthma rate age 6-12 | 23600 | 852 | 0.21 | 0.95 | 0.31 | 0.90 |
| population age 6-12 | 23600 | 852 | 2898 | 2271 | 2263 | 218 |
| ER asthma rate age 12-18 | 23577 | 851 | 0.18 | 1.04 | 0.28 | 1.01 |
| population age 12-18 | 23577 | 851 | 1546 | 1193 | 1192 | 101 |

Notes: The "between" standard deviation is calculated using $\bar{x}_i$ and the "within" is calculated using $x_{it} - \bar{x}_i + \bar{x}$.

**B. Pollution and Asthma by SES**

| | Low | High | Age | Low | High |
|---|---|---|---|---|---|
| O3*100 | 4.764 | 5.114 | 0-1 | 0.771 | 1.631 |
| | 0.018 | 0.021 | | 0.028 | 0.039 |
| CO | 1.053 | 1.163 | 1-3 | 0.217 | 0.395 |
| | 0.005 | 0.006 | | 0.007 | 0.019 |
| PM10 | 30.346 | 35.466 | 3-6 | 0.268 | 0.538 |
| | 0.105 | 0.124 | | 0.015 | 0.037 |
| NO2*100 | 2.400 | 2.774 | 6-12 | 0.157 | 0.296 |
| | 0.010 | 0.011 | | 0.005 | 0.013 |
| N | 10834 | 10315 | 12-18 | 0.171 | 0.261 |
| | | | | 0.006 | 0.015 |

Notes: Standard errors in parenthesis. Low SES is defined as zip code percentage of high school dropouts less than median.

**C. Asthma Counts by Age**

| Counts | Age 0-1 | Age 1-3 | Age 3-6 | Age 6-12 | Age 12-18 |
|---|---|---|---|---|---|
| 0 | 14044 | 14454 | 14270 | 14031 | 15477 |
| 1 | 4247 | 4406 | 4335 | 4514 | 3528 |
| 2 | 1571 | 1461 | 1669 | 1653 | 814 |
| 3 | 628 | 546 | 688 | 736 | 265 |
| 4 | 306 | 244 | 302 | 382 | 78 |
| 5 | 154 | 109 | 158 | 199 | 29 |
| >5 | 199 | 119 | 169 | 223 | 22 |
| Total | 21149 | 21339 | 21591 | 21738 | 20213 |

**Table 2. Main Results**

**Panel A. Fixed Effect Estimates by Age Group**

| | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| | Age 0-1 | Age 1-3 | Age 3-6 | Age 6-12 | Age 12-18 |
| O3 | -0.078** | -0.102** | -0.092** | -0.099** | -0.036 |
| | (0.026) | (0.027) | (0.026) | (0.025) | (0.036) |
| CO | -0.046 | 0.096** | 0.104** | 0.128** | 0.186** |
| | (0.033) | (0.034) | (0.032) | (0.031) | (0.045) |
| PM10 | -0.020 | -0.025 | -0.002 | -0.014 | -0.036 |
| | (0.022) | (0.024) | (0.023) | (0.021) | (0.031) |
| NO2 | 0.121* | -0.032 | -0.083 | -0.015 | -0.030 |
| | (0.054) | (0.057) | (0.052) | (0.050) | (0.073) |
| ave. max. temp./10,000 | -1.844 | -3.368 | 1.823 | -4.297 | -0.834 |
| | (3.077) | (3.442) | (3.253) | (3.078) | (4.464) |
| total precip./10,000 | -0.400 | -0.850** | -0.758** | -0.022 | 0.430 |
| | (0.257) | (0.276) | (0.261) | (0.241) | (0.347) |
| log (house price/10,000) | -0.035 | -0.063 | -0.061 | -0.136* | -0.147 |
| | (0.055) | (0.057) | (0.056) | (0.056) | (0.081) |
| % gov't health insurance | 0.333 | 0.126 | 0.257 | 0.067 | 0.740* |
| | (0.254) | (0.263) | (0.245) | (0.236) | (0.343) |
| % normal neonates | -0.015 | | | | |
| | (0.223) | | | | |
| Observations | 21075 | 21331 | 21567 | 21715 | 20207 |
| Number of groups | 759 | 768 | 778 | 784 | 727 |

* significant at 5%; ** significant at 1%

Notes: Standard errors in parenthesis. Pollutants are normalized to have a mean of zero and standard deviation of one. All columns contain seasonal and annual dummy variables, log of population and an indicator variable if house price is missing.

**Panel B. Fixed Effect Estimates by Age Group with Controls for Avoidance Behavior**

| | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| | Age 0-1 | Age 1-3 | Age 3-6 | Age 6-12 | Age 12-18 |
| O3 | -0.033 | -0.067* | -0.016 | -0.053 | -0.041 |
| | (0.028) | (0.030) | (0.028) | (0.027) | (0.040) |
| CO | -0.063 | 0.087* | 0.072* | 0.117** | 0.200** |
| | (0.034) | (0.034) | (0.032) | (0.032) | (0.046) |
| PM10 | -0.022 | -0.028 | -0.003 | -0.019 | -0.043 |
| | (0.022) | (0.025) | (0.023) | (0.021) | (0.031) |
| NO2 | 0.117* | -0.032 | -0.087 | -0.016 | -0.026 |
| | (0.054) | (0.057) | (0.052) | (0.050) | (0.073) |
| # of health advisories | -0.035** | -0.026** | -0.065** | -0.031** | 0.010 |
| | (0.008) | (0.010) | (0.009) | (0.009) | (0.012) |
| Observations | 21075 | 21331 | 21567 | 21715 | 20207 |
| Number of groups | 759 | 768 | 778 | 784 | 727 |

* significant at 5%; ** significant at 1%

Notes: Standard errors in parenthesis. Pollutants are normalized to have a mean of zero and standard deviation of one. All columns include maximum temperature, precipitation, log of population, seasonal and annual dummy variables, log of semi-annual house price, % gov't health insurance, and an indicator if health advisory information missing. Column (1) includes % normal neonates.

**Table 3. Fixed Effect Estimates by Age Group with SES Interactions**

| | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| | Age 0-1 | Age 1-3 | Age 3-6 | Age 6-12 | Age 12-18 |
| | **A. High SES** | | | | |
| O3 | -0.059 | -0.042 | -0.088 | -0.056 | -0.044 |
| | (0.057) | (0.057) | (0.056) | (0.055) | (0.071) |
| CO | -0.007 | -0.039 | -0.109 | 0.060 | 0.167 |
| | (0.077) | (0.074) | (0.073) | (0.071) | (0.090) |
| PM10 | -0.104* | -0.119* | -0.040 | -0.060 | 0.009 |
| | (0.053) | (0.052) | (0.050) | (0.046) | (0.061) |
| NO2 | 0.210 | -0.010 | 0.179 | 0.100 | -0.082 |
| | (0.112) | (0.108) | (0.106) | (0.101) | (0.134) |
| # of health advisories | -0.045* | -0.043* | -0.073** | -0.069** | -0.039 |
| | (0.020) | (0.020) | (0.020) | (0.020) | (0.026) |
| Observations | 10797 | 11014 | 11272 | 11347 | 10338 |
| Number of groups | 389 | 397 | 407 | 411 | 372 |
| | **B. Low SES** | | | | |
| O3 | -0.016 | -0.062 | 0.014 | -0.050 | -0.023 |
| | (0.032) | (0.035) | (0.032) | (0.032) | (0.049) |
| CO | -0.073 | 0.117** | 0.104** | 0.148** | 0.215** |
| | (0.038) | (0.040) | (0.037) | (0.036) | (0.055) |
| PM10 | -0.000 | 0.001 | 0.009 | -0.010 | -0.076* |
| | (0.025) | (0.028) | (0.026) | (0.024) | (0.036) |
| NO2 | 0.079 | -0.050 | -0.172** | -0.063 | -0.008 |
| | (0.062) | (0.067) | (0.061) | (0.058) | (0.088) |
| # of health advisories | -0.032** | -0.019 | -0.063** | -0.021* | 0.026 |
| | (0.009) | (0.011) | (0.010) | (0.010) | (0.014) |
| Observations | 10278 | 10317 | 10295 | 10368 | 9869 |
| Number of groups | 370 | 371 | 371 | 373 | 355 |

* significant at 5%; ** significant at 1%

See notes to Table 2B. Low (high) SES is defined as zip code percentage of high school dropouts less than (above) the median.

**Table 4. Zip Code/Year Fixed Effect Estimates by Age Group**

| | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| | Age 0-1 | Age 1-3 | Age 3-6 | Age 6-12 | Age 12-18 |
| O3 | 0.016 | -0.041 | 0.003 | -0.054 | -0.079 |
| | (0.031) | (0.033) | (0.030) | (0.030) | (0.044) |
| CO | -0.028 | 0.090* | 0.094* | 0.094** | 0.136** |
| | (0.038) | (0.039) | (0.037) | (0.036) | (0.053) |
| PM10 | -0.006 | 0.002 | 0.008 | -0.020 | -0.029 |
| | (0.025) | (0.028) | (0.026) | (0.024) | (0.035) |
| NO2 | -0.008 | -0.120 | -0.147* | -0.016 | 0.014 |
| | (0.065) | (0.068) | (0.063) | (0.060) | (0.088) |
| # of health advisories | -0.033** | -0.029** | -0.068** | -0.025** | 0.013 |
| | (0.009) | (0.010) | (0.010) | (0.009) | (0.013) |
| Observations | 13796 | 13817 | 14322 | 14703 | 11235 |
| Number of groups | 3463 | 3471 | 3597 | 3694 | 2821 |

* significant at 5%; ** significant at 1%

Notes: Standard errors in parenthesis. Pollutants are normalized to have a mean of zero and standard deviation of one. All columns include maximum temperature, precipitation, log of population, seasonal dummy variables, log of semi-annual house price, and % gov't health insured. Column (1) includes % normal neonates.

**Table 5. Fixed Effect Estimates with Different Distributional Assumption**

**Panel A. Linear Fixed Effect Estimates by Age Group**

| | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| | Age 0-1 | Age 1-3 | Age 3-6 | Age 6-12 | Age 12-18 |
| O3 | -0.067* | -0.105** | -0.077** | -0.145** | -0.122** |
| | (0.030) | (0.031) | (0.029) | (0.029) | (0.040) |
| CO | -0.031 | 0.168** | 0.165** | 0.203** | 0.281** |
| | (0.037) | (0.038) | (0.036) | (0.036) | (0.050) |
| PM10 | -0.023 | -0.028 | 0.003 | -0.012 | -0.033 |
| | (0.026) | (0.027) | (0.025) | (0.025) | (0.035) |
| NO2 | 0.099 | -0.035 | -0.065 | -0.001 | -0.041 |
| | (0.056) | (0.058) | (0.054) | (0.054) | (0.075) |
| # of health advisories | -0.028** | -0.018* | -0.048** | -0.025** | 0.011 |
| | (0.009) | (0.009) | (0.008) | (0.008) | (0.012) |
| Observations | 21109 | 21331 | 21567 | 21715 | 20207 |
| Number of groups | 761 | 768 | 778 | 784 | 727 |

**Panel B . Ordered Probit Fixed Effect Estimates by Age Group**

| | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| | Age 0-1 | Age 1-3 | Age 3-6 | Age 6-12 | Age 12-18 |
| O3 | -0.022 | -0.060* | -0.027 | -0.058* | -0.029 |
| | (0.028) | (0.028) | (0.028) | (0.027) | (0.031) |
| CO | -0.023 | 0.076* | 0.096** | 0.119** | 0.176** |
| | (0.035) | (0.034) | (0.034) | (0.034) | (0.038) |
| PM10 | -0.004 | -0.032 | -0.009 | -0.022 | -0.029 |
| | (0.023) | (0.024) | (0.024) | (0.023) | (0.026) |
| NO2 | 0.081 | -0.047 | -0.074 | -0.019 | -0.026 |
| | (0.053) | (0.053) | (0.052) | (0.052) | (0.059) |
| # of health advisories | -0.038** | -0.027** | -0.058** | -0.039** | 0.004 |
| | (0.008) | (0.008) | (0.009) | (0.008) | (0.009) |
| Observations | 21109 | 21331 | 21567 | 21715 | 20207 |
| Number of groups | 761 | 768 | 778 | 784 | 727 |

* significant at 5%; ** significant at 1%

Notes: Standard errors in parenthesis. Pollutants are normalized to have a mean of zero and standard deviation of one. All columns include maximum temperature, precipitation, log of population, seasonal and annual dummy variables, log of semi-annual house price, and % gov't health insured. Column (1) includes % normal neonates. In panel A, the reported coefficients are semi-elasticities ($\delta ny/\delta x$) evaluated at the means of the independent variables. In panel B, counts are censored at 5.

**Table 6. Magnitude of Effects**

**A. Effect Over Time**

| i. Change in Pollution Levels Over Time | | | | |
|---|---|---|---|---|
| Year* | O3*100 | CO | PM10 | NO2*100 |
| 1980 | 5.92 | 1.79 | n/a | 3.11 |
| 1992 | 5.43 | 1.13 | 31.55 | 2.07 |
| 1998 | 4.98 | 0.78 | 24.30 | 1.74 |

Notes: 1980 uses data from all monitors; 1992 and 1998 use data from continuously operated monitors ony.

| ii. Annual Effect for Emergency Room Admissions for Asthma in 1998 | | | | | | |
|---|---|---|---|---|---|---|
| Age | Average charge | Number of Admissions | $\delta_{92}$ | cost($\delta_{92}$) | $\delta_{80}$ | cost($\delta_{80}$) |
| 0-1 | 6,819 | 1,899 | 3.6% | $469,477 | 16.1% | $2,078,542 |
| 1-3 | 5,915 | 1,528 | 4.1% | $371,438 | 13.8% | $1,246,494 |
| 3-6 | 6,608 | 1,865 | 4.7% | $584,099 | 14.3% | $1,761,698 |
| 6-12 | 8,272 | 2,556 | 7.9% | $1,663,603 | 24.4% | $5,164,411 |
| 12-18 | 9,207 | 1,017 | 13.8% | $1,287,787 | 45.0% | $4,216,980 |
| Total | | | | $4,376,404 | | $14,468,125 |

Notes: $\delta_{92}$ ($\delta_{80}$) is the percentage decrease in ER admissions for asthma in 1998 only if pollution levels were at their 1992 (1980) levels using pollutants significantly estimated at the 5% level. Cost($\delta_y$) = average charge × number of admissions × $\delta_y$. Coefficient estimates used to obtain $\delta_y$ are from the fixed effect specification reported in table 2B without standardizing pollutants.

**B. Effect of Health Advisories**

| Age | δ |
|---|---|
| 0-1 | -3.4% |
| 1-3 | -2.6% |
| 3-6 | -6.3% |
| 6-12 | -3.0% |
| 12-18 | 1.0% |

Notes: δ is the percentage decrease in ER admissions for asthma from the announcement of a health advisory conditional on O3 exceeding 15 ppm. Coefficient estimates used to obtain δ are from the fixed effect specification reported in table 2B.

**C. Effect by SES**

| Age | δ |
|---|---|
| 0-1 | 4.2% |
| 1-3 | 1.8% |
| 3-6 | 1.5% |
| 6-12 | 2.4% |
| 12-18 | 4.2% |

Notes: δ is the percentage increase in ER admissions for asthma from higher pollution levels in low SES areas using pollutants significantly estimated at the 5% level. Low SES is defined as zip code percentage of high school dropouts less than median. Coefficient estimates used to obtain δ are from the fixed effect specification reported in table 2B without standardizing pollutants.

**Appendix Table 1. Number of Monitors Over Time and Correlations by Monitor Sampling**

| | 1992 | 1998 | Continuously Operated | Correlation |
|---|---|---|---|---|
| O3 | 171 | 178 | 138 | .9955 |
| CO | 91 | 88 | 75 | .9893 |
| PM10 | 125 | 149 | 98 | .9807 |
| NO2 | 109 | 108 | 87 | .9928 |

**Appendix Table 2. Pollution Correlation Matrix**

| | O3 | CO | PM10 | NO2 |
|---|---|---|---|---|
| O3 | 1 | | | |
| CO | -0.22 | 1 | | |
| PM10 | 0.44 | 0.52 | 1 | |
| NO2 | 0.10 | 0.86 | 0.70 | 1 |

**Appendix Table 3. Correlation Between Actual and Estimated Pollution Levels**

| Pollutant | Correlation | Observations | Monitors |
|---|---|---|---|
| O3 | 0.9245 | 3141 | 106 |
| CO | 0.7847 | 1524 | 53 |
| PM10 | 0.7651 | 1718 | 57 |
| NO2 | 0.9016 | 2035 | 71 |

Notes: Weighted pollution levels at each monitor are calculated using an inverse-distance weighted sum of all monitors within 20 miles.

**Appendix Table 4. Characteristics of Zip Codes Inside and Outside 20 Miles from Monitors for All Pollutants**

| | Far | Near | t |
|---|---|---|---|
| median HH income | 28,703 | 38,848 | 14.52 |
| % urban | 14% | 84% | 39.25 |
| % white | 85% | 72% | 13.77 |
| % black | 2% | 7% | 12.64 |
| % < HS degree | 26% | 23% | 3.97 |
| % college degree | 16% | 25% | 12.82 |
| total population < 18 | 1,510,589 | 6,490,109 | 2.74 |
| average ER asthma rate | 0.234 | 0.283 | 27.98 |