NBER WORKING PAPER SERIES

CONGESTION PRICING, AIR POLLUTION AND CHILDREN'S HEALTH

Emilia Simeonova
Janet Currie
Peter Nilsson
Reed Walker

Working Paper 24410
http://www.nber.org/papers/w24410

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
March 2018, Revised September 2019

We would like to thank Nicholas Sanders and participants at the 2017 AEA meetings, as well as seminar participants at Karolinska Institute, Uppsala University, the Institute for International Economic Studies, University of Oslo, University of Bergen, University of Umea, and the Stockholm School of Economics. We are grateful to three anonymous referees for helpful comments. We gratefully acknowledge financial support from the Swedish Research Council. Jenny Jans provided invaluable help as a research assistant for this project. The data used in this study are confidential and protected by Swedish privacy laws. They cannot be accessed by anyone not approved under the original data agreement signed by the authors. The data can be purchased from Statistics Sweden, the Swedish National Board for Health and Welfare, and the Swedish Tax registry after the appropriate ethics and privacy board approvals have been secured from these agencies. Dr. Simeonova and Nilsson are willing to assist those interested in purchasing registry data. The authors have no financial conflicts of interest to disclose. The views expressed herein are those of the authors and do not necessarily reflect the views of the National Bureau of Economic Research.

NBER working papers are circulated for discussion and comment purposes. They have not been peer-reviewed or been subject to the review by the NBER Board of Directors that accompanies official NBER publications.

Congestion Pricing, Air Pollution and Children's Health
Emilia Simeonova, Janet Currie, Peter Nilsson, and Reed Walker
NBER Working Paper No. 24410
March 2018, Revised September 2019
JEL No. H23,I18

ABSTRACT

This study examines the effects of a congestion tax in central Stockholm on ambient air pollution and the health of local children. We demonstrate that the tax reduced ambient air pollution by 5–15 percent and the rate of acute asthma attacks among young children. We do not see corresponding changes in accidents or hospitalizations for nonrespiratory conditions. As the change in health was more gradual than the change in pollution, it may take time for the full health effects of changes in pollution to materialize if the mechanism is pollution. Hence, short-run estimates of pollution reduction programs may understate long-run health benefits.

Emilia Simeonova
JHU Carey School of Business
100 International Drive
Baltimore, MD 21202
and NBER
emilia.simeonova@gmail.com

Janet Currie
Department of Economics
Center for Health and Wellbeing
185A Julis Romo Rabinowitz Building
Princeton University
Princeton, NJ 08544
and NBER
jcurrie@princeton.edu

Peter Nilsson
Assistant Professor
IIES, Stockholm University
peter.nilsson(a)iies.su.se
nilssonjanpeter@gmail.com

Reed Walker
Haas School of Business
University of California, Berkeley
2220 Piedmont Ave
Berkeley, CA 94720
and NBER
rwalker@haas.berkeley.edu

I. Inroduction

Economists have long recognized the negative externalities associated with traffic congestion and have suggested congestion pricing as a possible solution (Vickrey 1969). Typically, research and policy making surrounding congestion pricing has been concerned with time lost commuting, fuel costs, and business activity (Leape 2006).[1] Recent research suggests that congestion also significantly contributes to local air pollution levels and negatively impacts the health and well-being of nearby residents (see for example, Currie and Walker, 2011; Knittel, Miller, and Sanders 2016).

As traffic congestion in cities has increased, rates of respiratory illnesses such as asthma have risen (Centers for Disease Control 2011). Asthma is now the leading cause of hospitalization among children in the United States. The increase in asthma rates is most pronounced amongst individuals of low socioeconomic status, who disproportionately live in densely populated areas with severe traffic congestion. The highest rates of asthma in the United States are in the Bronx, New York (Garg et al. 2003). This area of New York City is bisected by five major highways that rank among the most congested in the United States (Bruner 2009). With both traffic congestion and children's asthma rates trending upward in recent years, it is plausible that the two are causally related.

This study is the first to examine the link between congestion pricing and inpatient and outpatient visits for child asthma. We use data from Stockholm, Sweden. Since August 2007, Stockholm has levied charges on most vehicles entering the city center. The charge was permanently implemented after a seven-month trial period between January and July 2006.[2] We

[1] Travel delays and extra fuel consumption cost the United States an estimated $70 billion each year (Schrank and Lomax 2007). An urban motorist lost an estimated 62 hours to congestion during peak hours in 2000.
[2] See Eliasson (2017) for a survey of the effects of congestion pricing, including several studies of Stockholm's experience. Anas and Lindsey (2011) also offer an overview of this literature.

combine the variation in congestion fees with data on ambient air pollution and administrative data on all inpatient and outpatient health visits. As a counterfactual for what would have happened in the absence of a congestion fee program, we compare outcomes within the Stockholm city center to outcomes in other city centers within Sweden that did not implement congestion pricing.

Consistent with studies of congestion pricing in other locales, we show that congestion pricing in Stockholm reduced pollution from automobiles significantly.[3] Nitrogen dioxide (NO2) and particulate matter (PM10) levels fell by 15– 20 and 10–15 percent relative to pre congestion pricing levels, respectively. For this period, there are no reliable data on changes in other pollutants from cars, such as PM2.5, but because PM2.5 is formed through combustion or atmospheric reactions to traffic pollution, it is likely that the concentration of fine particles also decreased.

This policy-induced reduction in air pollution levels accompanied significant reductions in the incidence of childhood asthma in Stockholm in the months and years after the program was implemented. We estimate that during the trial, the rate of acute asthma visits fell by 16 percent. After congestion pricing became permanent, the rate fell by about 50 percent relative to rates in the precongestion pricing period.

Investigating the link between congestion pricing and children's health is important for several reasons. First, although congestion pricing has been evaluated largely in the context of its effects on traffic and commuting times, it may also have significant benefits in terms of health,

[3] Wolff (2014) and Gibson and Carnovale (2015) examine the effects of congestion pricing on pollution in Germany and in Milan, respectively. Other studies such as Danel and Bekka (2000) simulate the potential effects of congestion pricing on pollution.

and it is important to quantify those benefits.[4] More generally, a large body of literature has investigated the effects of pollution on child mortality (see Almond, Currie, and Duque (2018) for a review), but less attention has been paid to child morbidity, which is arguably a more common and sensitive indicator.

Second, because a trial period preceded the permanent Stockholm congestion pricing zone (CPZ) program, the program offers a unique opportunity to assess both the immediate effects of a drop in pollution and the somewhat longer-term effects. As health is a stock, it would not be surprising if the longer-term effects, which allow for adjustment of the underlying health stock, are larger than the shorter-term effects. Simply put, if a child never develops asthma at all, then he or she will be less susceptible to asthma attacks. However, there is limited empirical evidence documenting whether and to what extent short-run changes in pollution have different effects in the short and long runs.

Third, the Stockholm congestion pricing program took place in a context with low ambient pollution levels, even by the standards of other developed countries. Hence, it offers insight into whether pollution levels that are well below current U.S. regulatory standards can have negative effects on children's health and if so, whether those effects can be mitigated through measures such as congestion pricing.

Although we show that congestion pricing in central Stockholm reduced both pollution and children's asthma attacks, it is less clear whether the reduction in pollution caused all of the reduction in asthma. Congestion pricing could have encouraged outdoor play, walking, or even

[4] Johansson et al. (2009) examine the effect of Stockholm's program on PM10 and NO2 and then use estimates from the literature to estimate the likely health effects. One study that looks at health effects directly is Green et al. (2016) who examine the effect of London's congestion pricing policy on accidents.

reduced stress in the affected zone relative to other parts of Stockholm.[5] It also could have impacted the demographics of people in the central city. We show that congestion pricing had little impact on acute care visits for non-respiratory conditions and that demographics did not change in a way that was likely to reduce asthma rates. Our work is limited by the fact that we are unable to compare people in central Stockholm to people in other parts of Stockholm as most pollution monitors were clustered in the central area that became the congestion pricing zone (CPZ).

The rest of the paper is arranged as follows. The next section reviews the institutional framework of the congestion pricing experiment and permanent implementation as well as the relevant prior literature. In the third section we describe the data. Section 4 presents the empirical framework, and Section 5 shows the estimation results. Section 6 concludes.

II. Background

The purpose of the Stockholm CPZ was to reduce congestion by reducing traffic entering the central city. The CPZ tax goes up to $2.60 USD per vehicle, depending on the time of the day. There are no charges at night, on weekends, on public holidays, or during July. The toll is collected automatically as cars cross the perimeter of the congestion zone, using license plate scanning technology.

The tax started with a seven-month trial period called the Stockholm Congestion Trials (Stockholmsförsöket*).* The trial period ran from January 3, 2006 through July 31, 2006. Government estimates suggested that inner-city traffic was reduced by around 20–25 percent

[5] The effects can be quite pervasive. For example, Daunfeldt et al. (2009) examine the effects on retail sales in the CPZ. If we thought that the only effects were through pollution reduction, then we could use the policy as an instrument for pollution to identify its effects on asthma. But given the possibility of other effects, we focus on reduced-form estimates of the effects of congestion pricing on health.

during the trial (Stockholmsförsöket, 2006b). Based on the success of the trial program, the Swedish government decided to make the program permanent.[6] The tolls were imposed again starting August 1, 2007, and have been collected ever since.

Automobile exhaust contains carbon monoxide, nitrogen dioxide, particulate matter, and other harmful pollutants. In urban areas, motor vehicle emissions are the leading cause of ambient air pollution. The medical literature shows that particulate matter (PM10 and PM2.5) affects lung growth and lung function in children (Gauderman et al. 2000; Yu et al. 2000; Hoek et al. 2000), and the biological channels are well-understood. Exposure to nitrogen dioxide (NO2) also worsens asthma symptoms and is associated with inferior respiratory health (Lipsett et al. 1997; Shima et al. 2000).

Asthma is a chronic inflammatory disorder of the airways. In susceptible individuals, this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and coughing. These episodes are usually associated with widespread but variable airflow obstruction. The inflammation also causes bronchial hyperresponsiveness to a variety of stimuli. After the removal of an initial stimulus, the airflow limitation may not be fully reversible. Stated differently, once a child has asthma, it is harder to return to baseline functioning even after the removal of triggers, like allergens. The onset of asthma in childhood is associated with significant, permanent deficits in lung growth. Therefore, if asthma can be avoided, the child's lung growth and health will be better than it would have been in the presence of asthma.[7]

Previous research asking how air pollution affects children's respiratory health has generally focused on the impacts of short-term variations in air pollution that last between

[6] Although the county held a referendum on the issues, and the vote was overwhelmingly negative except inside the CPZ, the government decided to proceed nevertheless.
[7] The discussion in this section is based on the U.S. National Asthma Education and Prevention Program Third Expert Panel on the Diagnosis and Management of Asthma (2007)

several hours and a few days (Friedman et al. 2001; Neidell, 2004; Neidell and Moretti, 2011; Schlenker and Walker, 2016; Jans et al. 2016). For example, Schlenker and Walker (2016) use variation in daily airport congestion rates as a cause of increased carbon monoxide emissions that are then linked to hospital admissions for respiratory conditions. Bauernschuster et al. (2015) show that on days that German public transport workers are on strike, there is increased pollution due to heavier car traffic into major cities, as well as increased rates of hospitalizations for respiratory conditions among the elderly and children younger than five. Friedman et al. (2001) show how changes in transportation and commuting behaviors during the 1996 Atlanta Olympics affected both air quality and childhood asthma. Each of these studies relies on high-frequency short-term (day-to-day) variation in pollution levels to isolate congestion effects on health. In contrast, we examine both the effect of a six-month reduction in air pollution (during the congestion pricing trial) and the effects of the later longer-term reduction in pollution after the congestion tax was permanently implemented. [8]

The mechanisms through which children are affected by traffic-generated pollution may differ from those in adults: children spend more time outdoors and engage in more physical activity. Furthermore, lung development continues post-natally until the adolescent years and is susceptible to negative environmental shocks (Pinkerton et al. 2000; Dietert et al. 2000). Children, and young children in particular, may find it harder to control asthma and thus present with acute symptoms more often. For all these reasons, standards for air pollution that adequately protect adults may not adequately protect children.

[8] The most closely related study regarding the effect of permanent congestion pricing may be Currie and Walker (2011), which examines the effect of implementing Electronic Toll Collection technology on air pollution and infant health near highway toll plazas. The study, however, only was able to examine the effects of in-utero exposure, not exposure after birth.

There is considerable evidence in both the medical and epidemiology literature that both the level of pollution and the length of exposure to it affect the severity of asthma. Avol et al. (2001) show that children who relocated from areas with high air pollution to areas with lower air pollution experienced improved lung functioning, with the effects being larger for children who relocated three to five years prior compared to those who had moved within the previous one to two years. Similarly, among individuals who had lived close to a major road in Los Angeles for several years, there was a strong correlation between distance from the road and asthma prevalence, but there was no correlation between distance and asthma prevalence among people who had just moved to the neighborhood (McConnell et al. 2006).

Crucially for our study, these studies suggest that it may take some time after an initial change in pollution levels for a new equilibrium level of asthma to be reached. Because pollution has a cumulative effect on asthma that is not immediately reversed, the effect of a short-run decrease in pollution may be quite different than the longer-term effect of a permanent decrease in pollution.

The implementation of congestion pricing and the ensuing traffic reduction in central Stockholm also may have affected children's health in ways not directly related to reductions in ambient pollution. For example, more time spent outside, more active lifestyles, or reductions in stress levels could have beneficial effects on health. Because we are not able to directly examine the relative contributions of different channels to the overall rate of asthma in the population, we present our analysis as the reduced-form effect of implementing a CPZ on young children's asthma and investigate effects on other health conditions as well.

III. Data

We use detailed data on the residential location and timing of all hospital inpatient and acute (unplanned) outpatient visits among residents of inner-city Stockholm and 102 other Swedish central cities between 2004 and 2010. The analysis focuses on all children aged zero through five who resided in major Swedish municipalities that monitor ambient air pollution. We focus on children aged five and younger because they are much more likely to have acute asthma episodes compared to older children: their rate of acute asthma incidents is 19.060 per 10,000, compared to 3.336 per 10,000 among children aged six to 18.[9] However, for comparison, we also show estimates for children aged six to 18 in the Appendix. We also restrict our analysis to children whose mothers were born in Sweden. We aggregate to the monthly level to avoid small-cell sizes, because acute asthma attacks are relatively rare events.

We use Geographic Information System (GIS) to determine whether the parents' residence is inside or outside the CPZ in Stockholm. We drop observations for children who live in the Stockholm region but outside the CPZ for two reasons. First, it is not clear whether and how they were affected by the CPZ. Children who live outside the CPZ but close to a major traffic artery leading into the inner city may have experienced a decrease in ambient pollution levels. Alternatively, they may have experienced increased pollution levels if traffic was diverted from the inner city to surrounding areas. More important, there is only one pollution monitor in the Stockholm region but outside the CPZ, which impedes our ability to measure how air pollution changed in different areas outside central Stockholm.

We collected data on ambient air pollution from cities' environmental agencies (Miljöförvaltningen) for the period 2004–2010. The data are recorded by ambient air monitors

[9] This may be because asthma in older children is likely to be better controlled and therefore less likely to lead to an acute attack. Moorman, Person, and Zahran (2013) estimate that in the United States, children aged zero to four are 1.9 times more likely to have an asthma attack, all other things being equal, than children aged 12 to 17.

located in these cities. In total, we have air pollution data from 103 Swedish municipalities, including central Stockholm. We focus on how the congestion trial affected levels of nitrogen dioxide (NO2) and PM10.[10] We choose these pollutants because automobiles account for a disproportionate share of both particulate and nitrogen emissions and the monitor data is sufficiently detailed to examine these pollutants.

We aggregate the daily monitor data for each city to the monthly level. The first two rows of Table 1 show the average levels and growth rates in NO2 and PM10 levels for central Stockholm (inside CPZ) and all other central cities in the two years preceding the CPZ trial. The levels of these two pollutants—33.32 and 33.28 micrograms per cubic meter, respectively—are less than current EPA standards of 66.25 micrograms per cubic meter for NO2 and 50 micrograms per cubic meter for PM10. Hence, Table 1 shows that the pollution levels in Stockholm were below current U.S. EPA standards for these pollutants even prior to the implementation of congestion pricing. Our results therefore illustrate the health benefits of reducing pollution levels below levels of ambient air pollution that are already relatively low.

Column (5) of Table 1 presents *p*-values from a test of the null hypothesis that the levels are the same inside Stockholm as in other central cities. Column (6) shows *p*-values from a test of the null hypothesis that the pretrends in these pollutants are the same. These *p*-values are based on permutation tests (Fisher 1935; Good, 2005; Dinardo and Lee 2011). These tests do not depend on distributional assumptions, and in small samples (such as our 103 municipalities), they are likely to yield more reliable *p*-values for differences between treatments and controls. The permutation-based test assumes exchangeability of treatments and controls under the null.

[10] For current EPA standards, see: https://www3.epa.gov/region1/airquality/pm-aq-standards.html and https://www.epa.gov/no2-pollution/fact-sheets-and-additional-information-regarding-2010-revision-primary-national. Note that for NO_2, one part per billion is equal to 1.25 micrograms per cubic meter.

To conduct the test, we assign treatment status to different Swedish cities and then recalculate the differences in mean levels and in pretrends between the index city and all other Swedish cities. The *p*-value corresponds to the percentile of the distribution where the observed difference falls, relative to the other permutations. For example, if none of the permutation differences exceeded the actual difference, then the *p*-value would be zero. In this group of 103 cities, if for 76 permutations, the difference was greater than the difference between Stockholm and all other cities, then the *p*-value would be 0.738 (as it is in the first row of column 5 of Table 1). The results in the first two rows of columns (5) and (6) suggest there are no significant differences in pollution pre-trends in the two years preceding the CPZ trial.

Figure 1 shows the differences in PM10 and NO2 between the Stockholm CPZ and other Swedish central cities in each month of the sample. The vertical lines indicate the "pre," "trial," "in between," and "permanent CPZ" periods. The figure presents the raw data, after taking out month-of-year dummies to adjust for seasonality. We took means by month for both the inside and outside group so as not to plot thousands of points (for example, bin scatter) and then plotted the difference. We overlaid the means (that is, the flat lines) within each period. In total, we have 72 months of data.

For both PM10 and NO2, after the CPZ was made permanent, there is a relative reduction in pollution during the trial period, a rebound in the "in-between" period, and a larger relative reduction in Stockholm compared to other central cities. Despite the finding in Table 1 that there are no statistically significant pre-trends in the pollution levels, Figure 1 suggests that there may be a pre-trend in NO2 levels. Hence, in Appendix Figure 1, we redo this figure using linear and quadratic regression overlays for comparison. Both figures suggest however, that there

is a real fall in NO2 associated with congestion pricing. Figure 1 also shows that there is one large outlier for PM10 during the trial period. However, Appendix Figure 2 shows that excluding this observation does not change our qualitative conclusion that there was a reduction in PM10 during the trial.

Health data were collected from the inpatient (Swedish National Patient Register (Socialstyrelsen, 2009)) and outpatient registries. The inpatient register contains administrative information such as date of admission, number of days in hospital care, as well as discharge diagnoses classified according to the ninth and tenth versions of the International Classification of Diseases (ICD). The National Patient Register records all hospital admissions that included an overnight hospital stay whether or not it originated in the Emergency Room.

The outpatient register contains information on all outpatient visits to primary care providers and specialists including visits to Emergency Rooms that did not result in inpatient admissions. The date of the visit and the primary ICD 9 (or ICD10) code identifying the main reason for the visit are also provided. Importantly, the register records whether the visit was planned (such as a routine yearly physical check-up) or urgent. Urgent outpatient visits are same-day visits initiated at the request of the patient and usually concern an acute health problem that would be treated as an emergency on an outpatient basis.

To construct an acute asthma rate, we add the number of overnight hospital visits and the unscheduled outpatient visits that record asthma as the primary reason for the visit. We then calculate the cumulative number of acute asthma episodes for each calendar month among children aged zero through five in the municipality and divide by the total number of resident children.

Table 1 shows that at 18.7 cases per 10,000 children aged zero through five, the asthma rate was higher in central Stockholm than in other central cities, and the rate was rising during the pre-period in both Stockholm and in other central cities. The *p*-values shown in column (6) show that there were, however, no significant differences in these trends between Stockholm and other central cities.

For comparison, we also examine visits for unintentional injuries (accidents) among children aged zero through five. Unintentional injuries are one of the most common reasons for children to seek medical attention, and they should not be mechanically related to air pollution and/or asthma although the absence of cars from the inner city could reduce injuries from car accidents. The baseline incidence of accidents was lower in Stockholm than in other central cities. However, once again the *p*-values in Table 1 show that there was no difference in pretrends in any of the health conditions considered.

Table 1 also shows pretrends in several measures of the socioeconomic status of parents as well as a measure of city size. Although the Stockholm CPZ is much larger, has higher per capita income, more college graduates, and fewer people on social assistance than other Swedish central cities, differences in the pre-trends in these variables are generally not statistically significant.

In all our regression results, we control for weather conditions that may affect the extent of ambient air pollution independent of the congestion pricing policy. We use data from the Swedish Meteorological Institute that come from weather stations in each municipality and are considered high quality. The weather data from each station is linked to each city centroid using the inverse distance weighted average of all weather monitors within 100 km of the municipal center. This procedure places much more weight on monitors located at the city center than on

monitors located far away so that the means are not sensitive to the exact cutoff for the inclusion of monitors. Daily data on rain (mm), rain squared, mean temperature, temperature squared, maximum temperature, minimum temperature, average wind speed, and maximum wind speed is calculated for each weather monitor and then aggregated to the municipality by month level.

IV. Methods

We first investigate the extent to which both the Stockholm congestion trial and the eventual full implementation of the congestion fee affected ambient air pollution. Formally, we estimate the following equation, which allows the effects of the trial, the "in-between" period, and the period after the charges were made permanent to be distinguished:

$$(1)\quad Poll_{it} = \alpha + \beta_1 CPZ_i * Trial_t + \beta_2 CPZ_i * InBetween_t + \beta_3 CPZ_i * Permanent_t + \upsilon_i + \mu_{ty} Z_{it}\gamma + \omega W_{it} + \varepsilon_{it},$$

where *Trial*, *InBetween*, and *Permanent* are dummy variables equal to one during the relevant periods. Pollution in municipality *i* in month *t* ($Poll_{it}$) is regressed on a set of interactions, where, for example, $CPZ_i * Trial_t$ is an indicator equal to one if the period is one in which the [11]congestion trial is in effect. Equation (1) also includes fixed effects for month*year, μ_{ty}, which allows very flexibly for pollution trends in all central cities. For example, changes in cars or fuel formulation could lead to such changes. All models also include municipal fixed effects, υ_i, to allow for the fact that some municipalities may have higher or lower asthma rates for reasons other than traffic congestion.

[11]

In addition, we estimate versions of Equation (1) with and without weather controls, W_{it}, interactions of weather with municipality, interactions of weather with calendar month, and region-specific time trends, Z_{it}. There are twenty-five provinces in Sweden, ranging from the southern tip, which is about the same geographic latitude as Denmark, to the northern regions close to the Arctic circle. Weather may have independent effects on asthma rates, and it is possible that the same weather conditions could have different effects in different places due to variations in the availability of air conditioning or wood-burning, for example. Included weather controls are total rainfall, mean temperatures, and mean wind speed during the month. In practice, our results are all quite similar with and without these additional controls.

The coefficients β_1 and β_3 measure the shorter- and longer-run effects of implementing congestion pricing, and β_2 measures whether the dependent variable returned to "baseline" during the "in-between" period. Municipality fixed effects ensure that the identifying variation comes from within-municipality changes in air pollution in periods with congestion pricing versus periods without. The main identifying assumption is that even if the pollution levels were different between Stockholm and other municipalities, the trends did not differ systematically for reasons other than the implementation of congestion pricing. This assumption is the motivation for testing for differing pre-trends in pollution, as was discussed above. Because the issue of differential trends is potentially important, our regressions include both region-specific time trends and interactions between municipality and weather controls and calendar month and weather controls. These latter interactions allow for the fact that the same weather patterns could have different impacts on pollution levels in different cities and across different calendar months. Appendix Table 5 demonstrates that the results are not sensitive to including weather interactions with municipality and calendar month.

We examine asthma rates and other health outcomes using the following model:

(2) $Asthma_{it} = \alpha + \beta_1 CPZ_i * Trial_t + \beta_2 CPZ_i * InBetween_t + \beta_3 CPZ_i * Permanent_t + X_{it}\delta + \upsilon_i + \mu_{ty}$

$Z_{it}\gamma + \omega W_{it} + \varepsilon_{it}$,

which takes much the same form as Equation (1) except that it uses measures of asthma rates at the municipality and month level as the dependent variable and includes a vector X_{it} of time-varying municipality-level demographic characteristics. These characteristics include: average family income in municipality j in month t, the proportion of mothers on social support, and the proportion of mothers with a college degree.

The key identifying assumption is that there would have been parallel trends in pollution and in asthma in the absence of the CPZ. We provide some evidence in support of this assumption by examining pretrends in Table 1 and graphically representing the difference in pollution levels in the CPZ and other cities in Figure 1, as discussed above.

As a robustness check, we also model asthma rates using all of the data except that collected in central Stockholm while congestion pricing was in effect, and then ask whether the predicted asthma rates from this model change after the CPZ was implemented. This test assesses the degree to which underlying demographics and other observables changed in Stockholm in a way that would predict changes in asthma rates during the CPZ period. Finally, as a comparison, we also examine the effects of the CPZ on visits for childhood unintentional injuries (accidents), and for all other acute visits (that is, those that are not related to respiratory or unintentional injury diagnoses). We expect that if the effects of the CPZ on asthma are coming mainly through pollution, then the implementation of congestion pricing should have small or nonexistent effects on these other classes of diagnoses.

As discussed above, an important issue is whether the short- and longer-term effects of congestion pricing vary for young children. A child who never develops asthma in the first place will be less likely to have asthma attacks than one who already has developed asthma. Moreover, given the timing of the trial and the permanent implementation of congestion pricing, children who were between ages zero and five at some point during those intervals could have had very different experiences with congestion pricing. For example, a Stockholm child who was three years old in January 2006, might have already developed asthma and might then have experienced mild relief as a result of the January to July 2006 trial period. A Stockholm child who was three years old in August 2010 would have lived his or her entire life under congestion pricing and may have been able to avoid ever developing asthma.

To make an apples-to-apples comparison, we offer an additional analysis that focuses on children six months old and younger and excludes the first six months of the "in-between" period. In this way, we can ensure that children were affected at similar ages by the trial and by the permanent implementation of congestion pricing, and that children's health outcomes measured during the in-between period did not receive the benefit of congestion pricing at all. Unfortunately, given the design of the implementation, it is not possible to do the same experiment (that is, to find an "in-between" group unaffected by congestion pricing) for older children.

All regressions involving health data are estimated by weighted least squares using the number of children of the respective age group residing in the municipality as weights. The results are not sensitive to weighting however.

V. Results

A. Congestion Pricing and Pollution Levels

Table 2 presents estimates for effects of the CPZ on levels of PM10 and NO2. We show analytical standard errors in parentheses, clustered by municipality, and we present two sets of *p*-values: *p*-values that correspond to the analytical standard errors, which are shown in curvy brackets, and our preferred permutation-based *p*-values, which are shown in square brackets for all estimates that are statistically significant at conventional levels. The permutation-based values are based on 500 simulations in which the type of CPZ "treatment" (trial, in between, or permanent) is randomly assigned across municipalities semi-annually, so that the permuted cells represent each municipality in each six-month period during the observation window (see for example, Cesarini et al (2016)). For each outcome and each permuted sample, we estimate Equation (1). We then examine the fraction of the times that the coefficient estimate exceeds the estimated value when the CPZ is correctly assigned to Stockholm.

The estimates are consistent with Figure 1 in that they suggest that both PM10 and NO2 declined during the trial, rebounded somewhat during the in-between period, and settled at a new lower level similar to that seen during the trial when the CPZ became permanent. Specifications with and without controls for location-specific weather and region-specific seasonality are quite similar to each other, in terms of the estimated effects of permanent pollution reductions. The estimated effects of the congestion pricing trial become somewhat larger when controls are added, as do the estimated effect of the in-between period. Although we cannot completely rule out the hypothesis that PM10 and NO2 levels reverted to the pretrial levels during the in-between period, there is some evidence that on average, they remained at somewhat lower levels. This inference is also consistent with the available data on traffic flows. For example, Figure 2 shows a vehicle count reduction of 10 percent relative to baseline levels during the in-between period.

The 4.56 unit decline in PM10 and the 6.2 unit decline in NO2 after the CPZ was made permanent correspond to a 13.7 percent and a 18.7 percent reduction in these two pollutants relative to the mean levels of pre-CPZ pollution in Stockholm shown in Table 1. If one did not allow for the lack of a "rebound" effect during the in-between period, one would get a slightly lower estimate of the impact of congestion pricing on pollution.

Appendix Table 1 shows an alternative specification that uses only cities that had continuously operating monitors. We end up with only 20 cities in this table and the *p*-values (now also calculated using the wild bootstrap to account for the small number of clusters) suggest that the results are not statistically significant but that the point estimates are similar to those discussed above.

B. Congestion Pricing and Asthma

Table 3 shows the estimated effects of the CPZ on asthma rates in children aged zero through five. Column (1) shows the basic specification controlling for month-specific and municipality-specific fixed effects. In column (2) we add interactions of month fixed effects with region (to allow for different regional seasonal effects), weather variables, and interactions of weather and municipality (to allow weather conditions to have differential effects in different cities) and weather and calendar month (to allow weather conditions to have differential effects by month of the year). Appendix Table 5 shows that the estimates are not sensitive to the inclusion of weather interactions. The models also control for the basic demographic characteristics discussed above.

Unlike the pollution estimates, which show a reduction in pollution followed by a rebound and then a permanent reduction, column (2) shows that the congestion pricing trial was

associated with a continuous decline in asthma cases from the trial period onwards. There was a reduction of 2.1 asthma visits per 10,000 children (on a baseline of 19.06 visits per 10,000) during the trial. The in-between period saw a reduction of 5.7 cases per 10,000, and the permanent CPZ reduced asthma visits by about 9.6 per 10,000. These numbers represent the total changes for each period (they are not cumulative). These estimates suggest that the trial brought an immediate reduction in asthma rates, but that the permanent CPZ had a much larger effect, reducing urgent visits and hospitalizations for asthma by half among children aged zero through five. In these models, the analytical and permutation-based *p*-values are very close to each other, and both suggest that the estimated effects are strongly statistically significant.

Figure 3 follows a format similar to Figure 1 and shows the difference in asthma rates by calendar month in Stockholm compared to other central cities before and after the adoption of congestion pricing. This figure shows an initial decline in asthma in Stockholm relative to the other central cities during the trial period. However, instead of rebounding, relative asthma rates in Stockholm versus other cities continued to decline in the in-between period, and fell to their lowest levels after the CPZ became permanent.

Figure 4 provides insight into whether the changes in asthma rates accompanying congestion pricing are reasonable given the overall relationships observed between pollution and asthma in Sweden. The figure is based on data for each January and February from 2004 to 2009 for each month and municipality *except* Stockholm. We use January and February because asthma is highly seasonal, and these are peak months. The line shows the relationship between the monthly asthma rate and the mean monthly PM10 level conditional on municipal fixed effects, mean temperature, mean rainfall, and average wind speed. The shaded area is the 99 percent confidence interval. The blue triangles show estimates for Stockholm in the pre-CPZ

period; the red circles show Stockholm estimates during the trial period; and the black rectangles show the values for Stockholm after the CPZ became permanent. All Stockholm-specific data points are within the 99 percent confidence interval for the estimated association between asthma and mean PM10. Hence, the figure demonstrates that the fall in asthma rates in Stockholm is within the range that one would expect given the relationship between pollution and acute asthma rates in the rest of Sweden.

Figures 5 and 6 are similar to Figure 3, but for the alternative outcomes of accidents and all other acute care visits (that is, visits that are not for respiratory or accident-related causes, respectively). The difference in the rate of accidents increases over time and the difference in the rate of all other acute visits first increases and then decreases during the in-between and the period after congestion pricing became permanent. However, all changes are small relative to those shown for asthma, as illustrated in Figure 3.

Table 4 shows an additional analysis that focuses on children younger than o six- months old and excludes the first six months of the in-between period. In this way, we can ensure that children were affected at similar ages by the trial and by the permanent implementation of congestion pricing, and that children's health outcomes measured during the in-between period did not receive the benefit of congestion pricing at all. Unfortunately, given the design of the implementation, it is not possible to do the same experiment (that is, to find an in-between group unaffected by congestion pricing) for older children.

Table 4 shows that when we chose cohorts of children who are exactly comparable in terms of age, then we do see a pattern that mimics the pattern for pollution. That is, there is a 16.5 percent reduction in asthma during the trial, which falls during the in-between- period after the trial ended and then jumps to a 15.2 percent reduction in asthma after congestion pricing

becomes permanent. Importantly, in this subsample, acute asthma episodes do not continue to decline during the in-between period and thus follow the "fall-increase-fall" pattern of pollution more closely. The estimates lose conventional statistical significance when all fixed effects are included in the model, but on the whole, they support the story that the effects of congestion pricing on young children are cumulative and may act by helping young children to avoid the onset of asthma. Appendix Figure 3 shows these results graphically.

C. Robustness

Table 5 presents results from several robustness checks. The specification in column (1) is the same as our main specification in Table 3, except that it includes municipality-specific linear trends. These trends should absorb any unobserved municipality-specific changes in asthma prevalence that happen during the observation period. The coefficients of interest do not change significantly.

In column (2) we assign the first municipality in which children are observed as their permanent municipality of residence. For example, if a child was born in Stockholm but moved outside the city after a few years, we include this child as part of the Stockholm sample for all years included in the observation window. Similarly, if a child moved into Stockholm, but was first observed as a resident of another city, we assign the original city as that child's residence throughout the observation window. Constructing the analysis sample in this fashion may address concerns about children differentially moving in or out of Stockholm based on their health status. On the other hand, we are introducing noise in the treatment variable, which could bias our estimates downward and increase our standard errors. However, the estimates shown in column (2) are robust to specifying the analysis sample in this way, and the standard errors are

also similar to those in the column 2 of Table 3. This is perhaps unsurprising given that only about 7 percent of children younger than five in Stockholm during the period 2004–2009 were born in a different city, so mobility in and out of central Stockholm was relatively low.

Column (3) of Table 5 shows estimates from a model using predicted asthma rates as the dependent variable. Estimates of the prediction equation itself are shown in Appendix Table 3.[12] The purpose of this specification is to see whether the characteristics of children in Stockholm changed after the introduction of the CPZ in a way that would have reduced asthma rates even in the absence of any change in pollution. By construction, predicted asthma rates have the same mean as the actual asthma rates (as shown in the row marked "Mean of Dep. Var." in Table 5).

The model using predicted asthma as the dependent variable shown in column (3) of Table 5 suggests that changes in the congestion pricing program were associated with changes in the observable characteristics of Stockholm children that were predictive of small but statistically significant *increases* in the asthma rate during the trial. The estimates suggest that observable characteristics of Stockholm children changed in a way that would have predicted about 0.457 more acute asthma episodes per 10,000 children annually during the trial period. There is no significant change in predicted asthma during the in-between or the permanent CPZ periods, and the point estimates are small. Although there may be other changes in unobservables that are correlated with both asthma and congestion pricing, the evidence presented here suggests that changes in observable characteristics have limited scope for explaining the baseline asthma results.

[12] With an R-squared of .544, we do a reasonably good job explaining variation in asthma rates. Because we saw in Table 1 that rates of social assistance and income levels in Stockholm are different than in other central cities, it is reassuring that neither of these variables are predictive of asthma rates in our baseline Appendix Table 3 model, which includes municipality fixed effects, interactions between month and year, interactions between region and month, and interactions between municipality and weather.

Column (4) of Table 5 presents estimates from specifications in which the dependent variable is visits due to unintentional injuries. Injuries are another common reason for acute-care visits, with a mean of 8.711 visits per 10,000 children. This model takes the same form as the baseline model of asthma. The analytical standard errors suggest statistically significant effects: an initial decline of 1.614 injuries during the trial is followed by increases of 1.018 and 0.966 during the in-between period and CPZ permanent periods, respectively. However, our preferred permutation-based *p*-values suggest that none of the estimated effects on unintentional injuries are statistically significant.

Column (5) shows the same specification with all other acute visits (that is, nonrespiratory and non-accident-related visits) as the outcome variable. Because many causes are aggregated, there is a mean of 183 acute visits per 10,000 children. The point estimates are not large at three to four percent of the mean rate over the period (7.325, 5.511, and -7.885 for the trial, in-between, and permanent periods, respectively), and the standard errors are substantial. Hence, this model also suggests that congestion pricing had little effect on nonrespiratory health outcomes in this age group.

We have also estimated a series of models in the same form but using demographic characteristics as the dependent variables. The purpose of these regressions is to assess the extent to which the reductions in asthma rates that accompanied congestion pricing could have been due to changes in demographic characteristics in central Stockholm relative to other cities rather than to congestion pricing *per se*. Appendix Table 2 shows that there were some significant changes over time. More children came to Stockholm than to other central cities and this movement tended to reduce the number of children on social assistance over the entire period. The trial period is associated with a slightly lower proportion of children whose mothers

had a college education and higher household income levels than in the period when congestion pricing became permanent. However, as we show in Appendix Table 3, none of these demographic characteristics have statistically significant effects on asthma rates in Sweden. The variable that comes closest is income, and the decline in household income over the course of the sample would predict an increase rather than a decrease in asthma rates.

VI. Discussion and Conclusions

This paper estimates the impact of congestion pricing in a large urban center city on ambient pollution and children's health. Our findings indicate that the congestion tax in central Stockholm reduced ambient pollution levels. The two pollutants we can measure best, PM10 and NO2, declined between 10–20 percent, relative to precongeston pricing levels. This decline suggests that other pollutants from traffic are also likely to have fallen. Declines in relative ambient pollution levels in Stockholm compared to other central cities show a step-wise pattern, first falling during the trial period, then rebounding somewhat during the period in between the trial and permanent adoption, and then showing a sustained decline following the permanent adoption of congestion pricing.

Turning to health outcomes, we find a significant reduction in visits for acute asthma attacks among children aged zero through five in the years after congestion pricing took effect. Although congestion pricing had an immediate impact on asthma, the impact grew over time and asthma rates continued to decline during what proved to be a temporary hiatus in congestion pricing before its permanent adoption. Our findings therefore suggest that congestion charges in large cities can have significant positive effects on health in the short term, but even larger effects in the longer term as the stock of child health evolves to a new lower-pollution

equilibrium level. In the short run, children with asthma will have fewer attacks when pollution is reduced. In the long run, fewer children will develop asthma to begin with. This finding is consistent with our understanding of health as a stock that often changes relatively slowly over time, as suggested by the medical literature.

Our estimates are among the first to demonstrate that congestion pricing as implemented in Stockholm not only resulted in a significant decrease in ambient air pollution but also had significant impacts in children's respiratory health. These improvements in health occurred even though initial pollution levels were well below the current U.S. EPA standards, suggesting that reductions in air pollution from traffic can have large positive effects on children's respiratory health.

References

Anas, Alex and Robin Lindsey. 2011. "Reducing Urban Road Transportation Externalities: Road Pricing in Theory and in Practice." *Review of Environmental Economics and Policy* Association of Environmental and Resource Economists, 5(1): 66–88.

Avol, Edward, James Gauderman, Sylvia Tan, Stephanie London, and John Peters. 2001. "Respiratory Effects of Relocating to Areas of Differing Air Pollution Levels." *American Journal of Respiratory and Critical Care Medicine* 164 (11):2067-2072

Bauernschuster, Stefan, Timo Hener, and Helmut Rainer. 2015. "When Labor Disputes Bring Cities to a Standstill: The Impact of Public Transit Strikes on Traffic, Accidents, Air Pollution and Health." Ifo Institute Working Paper.

Bruner, Jon. 2009. "America's Worst Intersections." *Forbes*, February 24, 2009.

Cesarini, David, Erik Lindqvist, Robert Ostling, and Björn Wallace. 2016. "Wealth, Health, and Child Development: Evidence from Administrative Data on Swedish Lottery Players." *Quarterly Journal of Economics*, 131: 687-738

Chen, Yuyu, Avraham Ebenstein, Michael Greenstone, and Hongbin Li. 2013. "Evidence on the Impact of Sustained Exposure to Air Pollution on Life Expectancy from China's Huai River Policy." *Proceedings of the National Academy of Sciences* 110(32): 12936–41.

Currie, Janet and Reed Walker. 2011. "Traffic Congestion and Infant Health: Evidence from E-Zpass." *American Economic Journal: Applied Economics* 3: 65–90.

Currie, Janet, Joshua Graff Zivin, Jamie Mullins, and Matthew Neidell. 2014. "What Do We Know about Short and Long Term Effects of Early Life Exposure to Pollution?" *Annual Review of Resource Economics* 6: 217–47.

Currie, Janet, Douglas Almond, and Valentina Duque. 2018. "Childhood Circumstances and Adult Outcomes: Act II." *The Journal of Economic Literature*, 56(4): 1360–1446

Daniel, Joseph and Bekka, Khalid. 2000. "The Environmental Impact of Highway Congestion Pricing." *Journal of Urban Economics* 47: 180–215.

Daunfeldt, Sven-Olov., Niklas Rudholm, and Ulf Rämme. 2009. "Congestion Charges and Retail Revenues: Results from the Stockholm Road Pricing Trial." *Transportation Research Part A: Policy and Practice* 43(3): 306–09.

Dietert Rodney, Ruth Etzel, DavidChen D, Marilyn Halonen, Steven Holladay, Annie Jarabek, Kenneth Landreth, David Peden, Ralph Smialowicz,, Kent Pinkerton, and Tracey Zoetis. 2000. "Workshop to Identify Critical Windows of Exposure for Children's Health: Immune and Respiratory Systems Work Group Summary." *Environmental Health Perspectives* 108(3): 483–490.

Dinardo, John and David Lee. 2011. “Program Evaluation and Research Designs.” In *Handbook of Labor Economics,* eds. Orley Ashenfelter and David E. Card, Volume 4 Part A, 463-536
Eliasson, Jonas. 2017. “Congestion Pricing.” In *The Routledge Handbook of Transport Economics*. Cowie and Ison, eds. London: Routledge.

Fisher, Sir Ronald Aylmer. 1935. *Design of Experiments*. Edinburgh, London: Oliver and Boyd).

Fletcher, Jason, Jeremy Green, and Matthew Neidell. 2010. “Long Term Effects of Childhood Asthma on Adult Health.” *Journal of Health Economics* 29(3): 377–87.

Friedman, Michael S., Kenneth E. Powell, Lori Hutwagner, LeRoy M. Graham, and W. Gerald Teague. 2001. “Impact of Changes in Transportation and Commuting Behaviors During the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma.” *Journal of the American Medical Association* 285(7): 897–905.

Garg, Renu, Adam Karpati, Jessica Leighton, Mary Perrin, and Mona Shah. 2003. “Asthma Facts.” *New York City Department of Health and Mental Hygiene.*

Gauderman W James, Rob McConnell, Frank Gilliland, Stephanie London, Duncan Thomas, Edward Avol, Hita Vora, Kiros Berhane, Edward Rappaport, Fred Lurmann, Helene Margolis, and John Peters (2000. “Association Between Air Pollution and Lung Function Growth in Southern California Children.” *American Journal of Respiratory and Critical Care Medicine* 162: 1383–90.

Gibson, Matthew and Maria Carnovale. 2015. "The Effects of Road Pricing on Driver Behavior and Air Pollution." Department of Economics Working Papers 2015–16, Department of Economics, Williams College.

Good, Phillip. 2005. *Permutation, Parametric, and Bootstrap Tests of Hypotheses*. New York: Springer.

Green, Colin, John Heywood, and Maria Navarro2016. “Traffic Accidents and the London Congestion Charge.” *Journal of Public Economics* 133: 11–22.

Hoek Gerard, Douglas Dockery, Arden Pope, Lucas Neas , Willem Roemer, and Bert Brunekreef 1998. “Association between PM10 and Decrements in Peak Expiratory Flow Rates in Children: Reanalysis of Data from Five Panel Studies.” *European Respiratory Journal*. v11:1307–11.

Jans, Jenny, Per Johansson, and J. Peter Nilsson (2016), "Economic Status, Air Quality, and Child Health: Evidence from Inversion Episodes." Manuscript April 2016, Stockholm University

Johansson, Christer, Lars Burman, and Bertil Forsberg. 2009. “The Effects of Congestions Tax on Air Quality and Health.” *Atmospheric Environment* 43. 4843-54.

Knittel, Miller, and Ryan Sanders .2016. “Caution, Drivers! Children Present: Traffic, Pollution, and Infant Health.” *The Review of Economics and Statistics,* 98(2): 350–66. ,

Leape, Jonathan. 2006. "The London Congestion Charge." *Journal of Economic Perspectives* 20(4): 157–76.

Lipsett Michael, Susan Hurley, and Bart Ostro 1997. "Air Pollution and Emergency Room Visits for Asthma in Santa Clara County, California." *Environmental Health Perspectives* 105:216 –22.

McConnell, Rob, Kiros Berhane, Ling Yao, Michael Jerrett, Fred Lurmann, Frank Gilliland, Nino Kunzli, Jim Gauderman, Ed Avol, Duncan Thomas, and John Peters. 2006. "Traffic, Susceptibility, and Childhood Asthma." *Environmental Health Perspectives* 114(5):, 766–72.

Moorman, Jeanne, Cara Person, and Hatice Zahran. 2013. "Asthma Attacks among Persons with Current Asthma – United States, 2001–2010." *Morbidity and Mortality Weekly Report* Nov. 22, 62(3): 93–8.

Neidell, Matthew. 2004. "Air Pollution, Health, and Socio-Economic Status: The Effect of Outdoor Air Quality on Childhood Asthma." *Journal of Health Economics*, , 23(6): 1209–36.

Enrico Moretti and Matthew Neidell. 2011. "Pollution, Health, and Avoidance Behavior Evidence from the Ports of Los Angeles." *Journal of Human Resources* 46(1): 154–75

Pinkerton Kent and Jesse Joad. 2000.. "The Mammalian Respiratory System and Critical Windows of Exposure for Children's Health." *Environmental Health Perspectives* 108(3):457–62.

Pope, C. Arden, Richard Burnett, Michael Thun, Eugenia Calle, Daniel Krewski, Kazihiko Ito, and George D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution." *JAMA* 287 #9, 1132–41.

Schlenker, Wolfram and Reed Walker. 2016. "Airports, Air Pollution and Contemporaneous Health." *Review of Economic Studies*, 83(2): 768–809.

Shima Masayuki and Motoaki Adachi .2000. "Effect of Outdoor and Indoor Nitrogen Dioxide on Respiratory Symptoms in Schoolchildren," *International Journal of Epidemiology* 29: 862–70.

Schrank, David and Tim Lomax. 2007. "The 2007 Urban Mobility Report." Texas Transportation Institute, Texas A&M University, September 2007 (accessible at http://www.ncga.state.nc.us/documentsites/committees/21stcenturytransportation/prioritization-best%20practices-efficiency/presentations/mobility_report_2007_wappx.pdf)

U.S. Environmental Protection Agency NAAQS table (https://www.epa.gov/criteria-air-pollutants/naaqs-table)

U.S. National Asthma Education and Prevention Program (2007). Third Expert Panel on the Diagnosis and Management of Asthma. Bethesda (MD): National Heart, Lung, and Blood Institute (US); Aug.

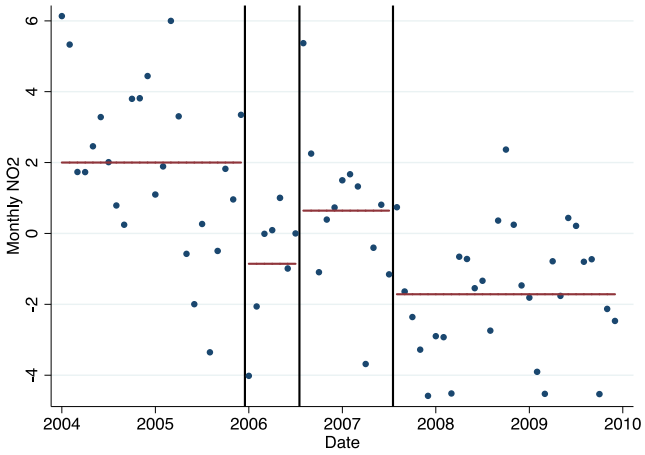
Vickrey, William. 1969. “Congestion Theory and Traffic Investment.” *The American Economic Review*, 59(2), Papers and Proceedings of the Eighty-first Annual Meeting of the American Economic Association (May, 1969), 251–60

Wolff, Henrik. 2014., “Keep Your Clunker in the Suburb: Low-Emission Zones and Adoption of Green Vehicles.” *Economic Journal*, 124: F481-F512.

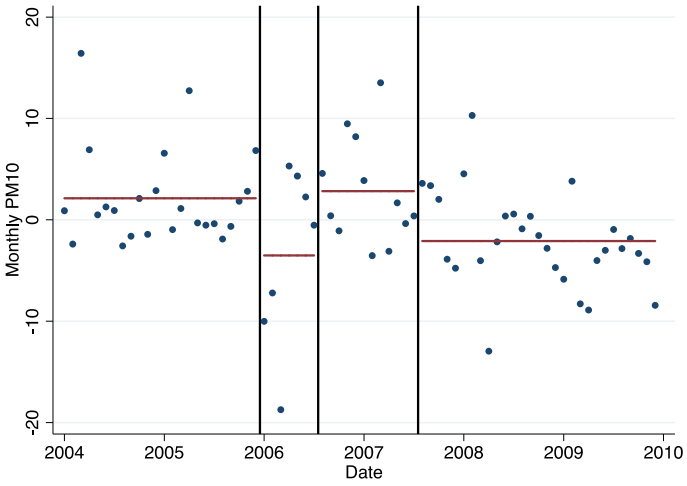
Yu Onchee, Lianne Sheppard , Thomas Lumley , Jane Q Koenig , and Gail Shapiro G 2000. “Effects of Ambient Air Pollution on Symptoms of Asthma in Seattle-Area Children Enrolled in the CAMP Study.” *Environmental Health Perspectives* 108: 1209–14.

Graphs

Figure 1: Scatterplot of differences in measured NO2 and PM10 inside the CPZ and in other central cities by implementation period.

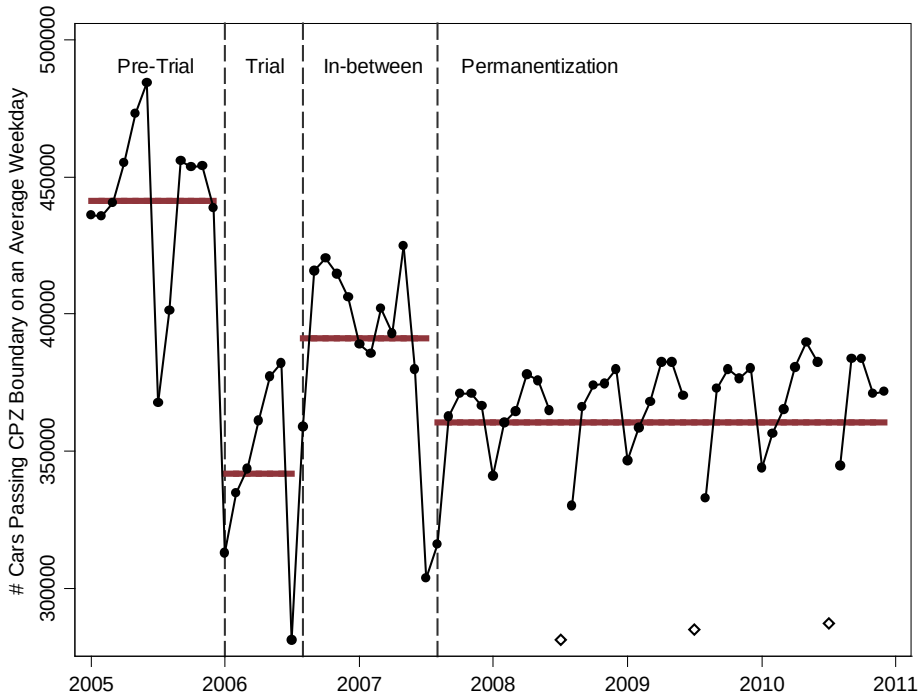


Notes: This figure plots the differences in the unadjusted monthly mean NO2 levels inside versus outside the CPZ. The vertical lines indicate the beginning of the trial period, the end of the trial period, and the beginning of the permanent CPZ implementation. Red horizontal lines represent the mean value within each period.



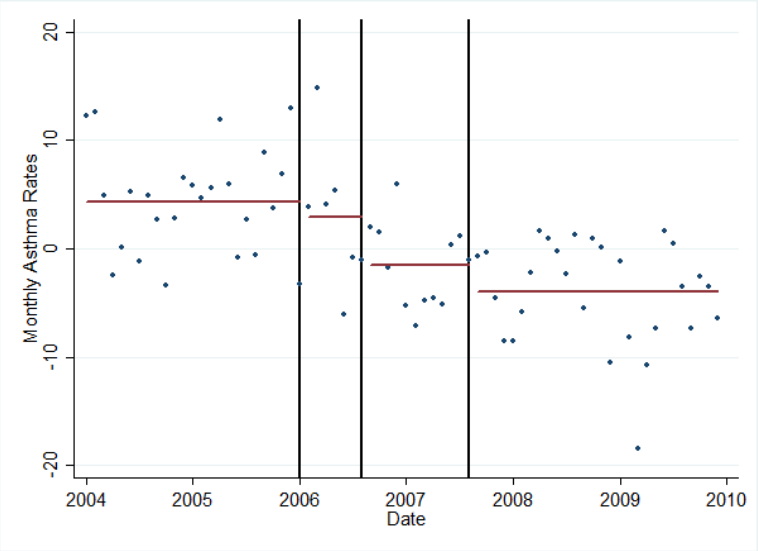
Notes: This figure plots the differences in the unadjusted monthly mean PM10 levels inside versus outside the CPZ. The vertical lines indicate the beginning of the trial period, the end of the trial period, and the beginning of the permanent CPZ implementation. Red horizontal lines represent the mean value within each period.

Figure 2: Number of cars passing the CPZ toll boundary on an average weekday



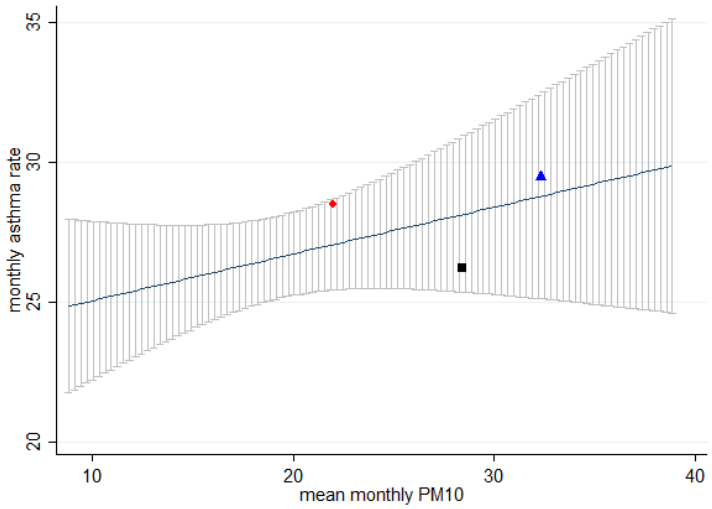
Note: The average number cars per day (by month) passing toll boundary is connected by the black line. Hollow diamonds show predictions in years with missing data on July traffic. Mean traffic levels for the four periods are in solid maroon lines.

Figure 3: Asthma differences inside CPZ versus other central cities



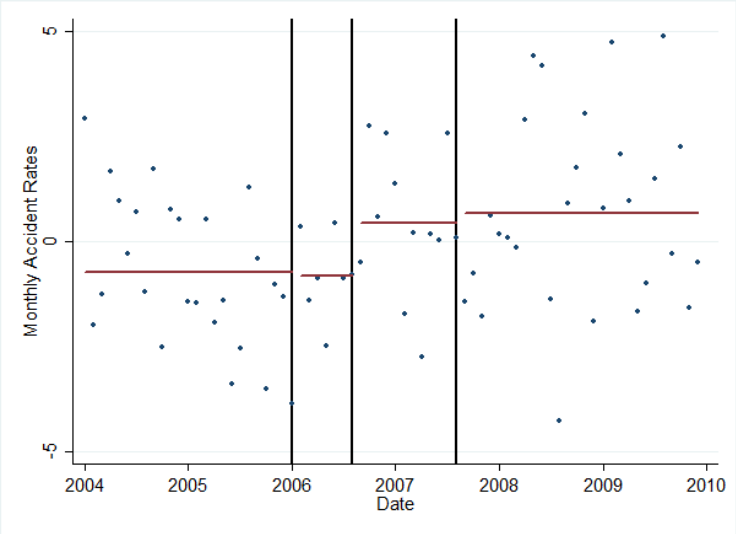
Notes: This figure plots the differences in the unadjusted monthly mean asthma rates inside versus outside the CPZ. The vertical lines indicate the beginning of the trial period, the end of the trial period, and the beginning of the permanent CPZ implementation. Red horizontal lines represent the mean value within each period.

Figure 4: PM10 and the incidence of acute asthma attacks in Swedish municipalities



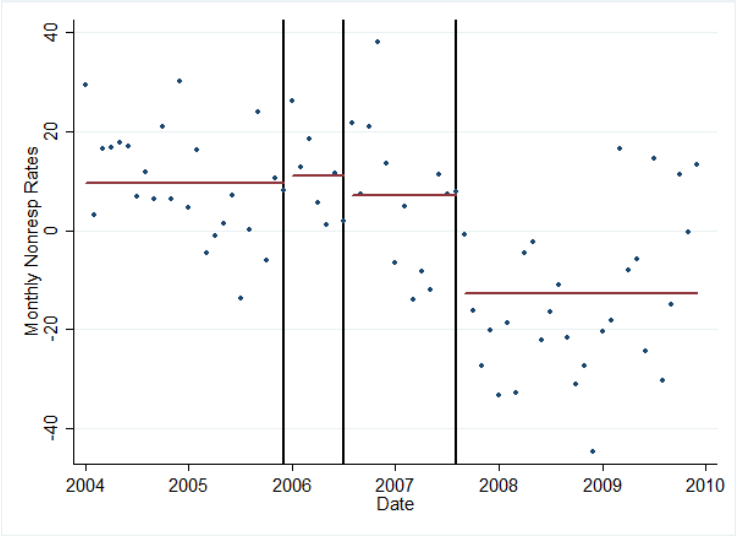
Data for January and February 2004–2009. The shaded area is the 99 percent confidence interval around the estimated relationship between the mean number of acute asthma episodes per month and municipality and the mean PM10 measured in the municipality during the month, net of municipality fixed effects and controlling for weather parameters such as mean temperature, rainfall, and wind. The blue triangle indicates the values for Stockholm in the pre-CPZ period; the red circle indicates the values for Stockholm during the trial period; the black rectangle indicates the values for Stockholm after the CPZ was made permanent.

Figure 5: Accidents difference in CPZ versus other cities



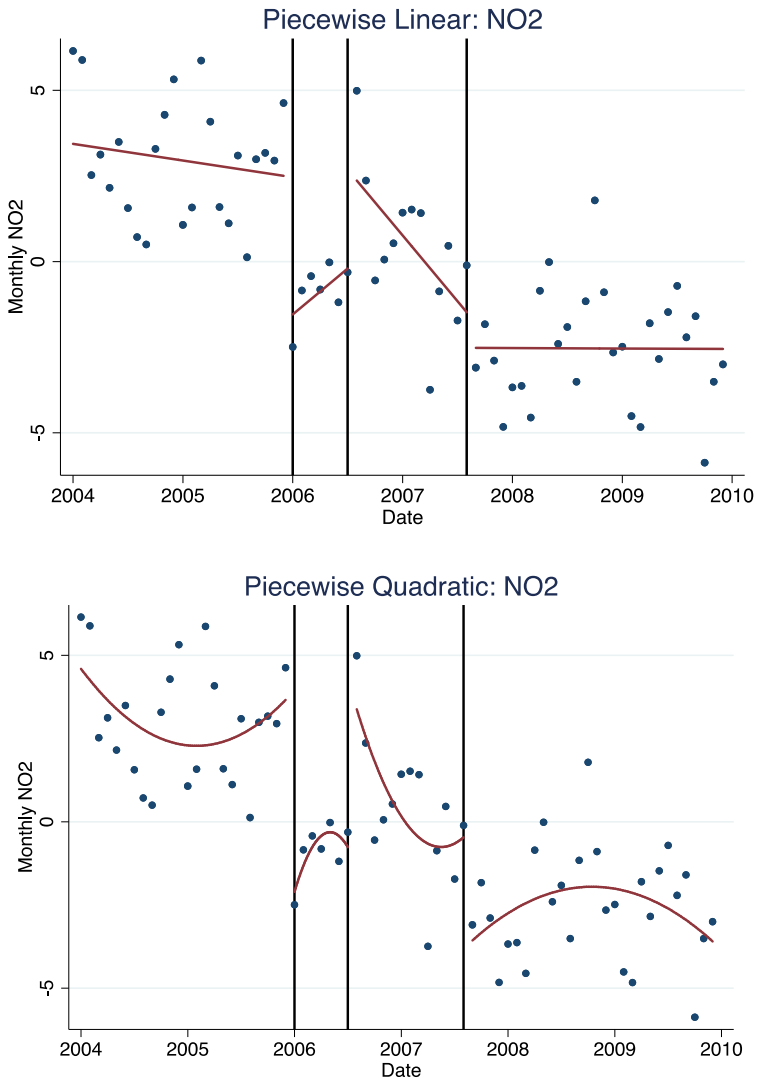
Notes: This figure plots the differences in the unadjusted monthly mean accident rates inside versus outside the CPZ. The vertical lines indicate the beginning of the trial period, the end of the trial period, and the beginning of the permanent CPZ implementation. Red horizontal lines represent the mean value within each period.

Figure 6: All other nonrespiratory diseases, differences between CPZ and other cities



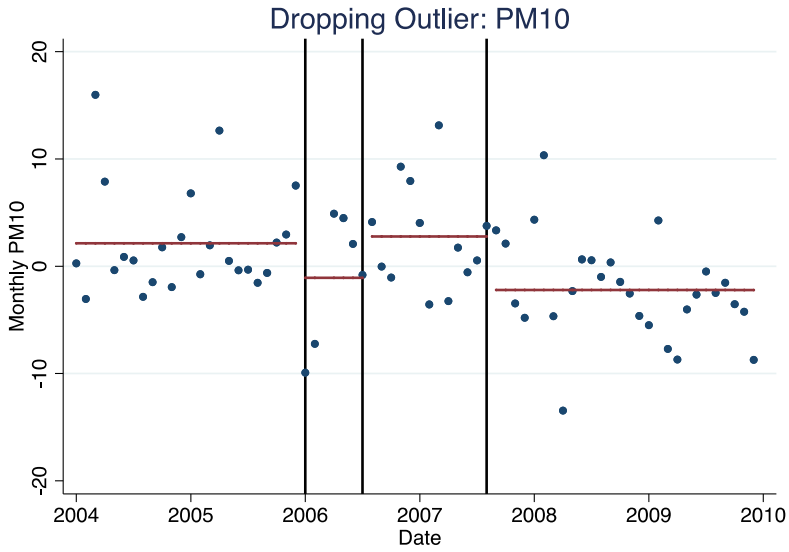
Notes: This figure plots the differences in the unadjusted monthly mean rates for all other diseases (that is, nonrespiratory and nonaccidents) inside versus outside the CPZ. The vertical lines indicate the beginning of the trial period, the end of the trial period, and the beginning of the permanent CPZ implementation. Red horizontal lines represent the mean value within each period.

Appendix Figure 1: Scatterplot of differences in measured NO2 inside the CPZ and in other central cities by implementation period, with piecewise linear and quadratic regression overlays within each period



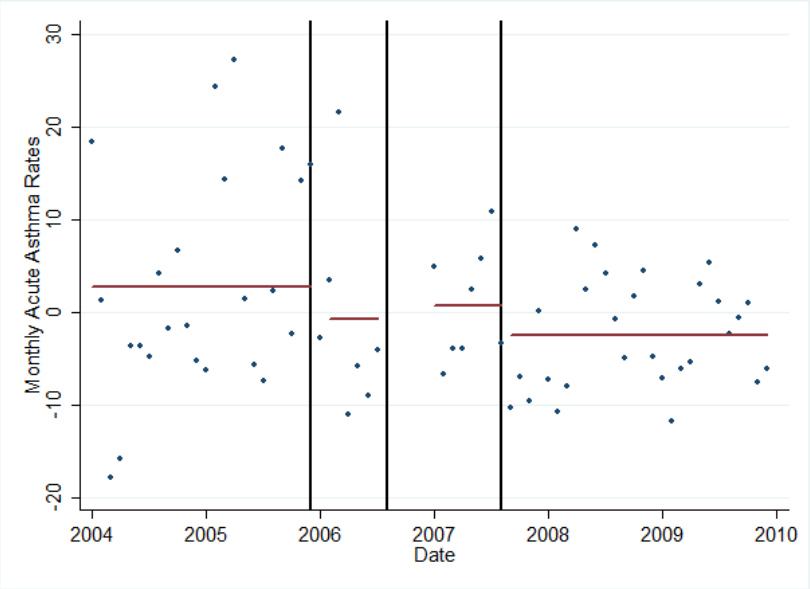
Notes: These figures plot the differences in the unadjusted monthly mean NO2 levels inside versus outside the CPZ. The vertical lines indicate the beginning of the trial period, the end of the trial period, and the beginning of the permanent CPZ implementation. Red lines represent linear and quadratic regression overlays within each time period.

Appendix Figure 2: Scatterplot of differences in measured PM10 inside the CPZ and in other central cities by implementation period, dropping an outlying observation in the preperiod



Notes: This figure plots the differences in the unadjusted monthly mean PM10 levels inside versus outside the CPZ. The vertical lines indicate the beginning of the trial period, the end of the trial period, and the beginning of the permanent CPZ implementation. Red horizontal lines represent the mean value within each period.

Appendix Figure 3: Asthma differences inside CPZ versus other central cities for children younger than six-months old only. First six months of the in-between period are omitted



Notes: This figure plots the differences in the unadjusted monthly mean asthma rates inside versus outside the CPZ for children younger than six-months old only. The vertical lines indicate the beginning of the trial period, the end of the trial period, and the beginning of the permanent CPZ implementation. Red horizontal lines represent the mean value within each period.

Table 1: Means and Pretrends of Outcome and Key Control Variables in the Pre-Congestion Pricing Zone (CPZ) Period

	(1)	(2)	(3)	(4)	(5)	(6)
	Levels		Difference Dec. 2005–Dec. 2004		*p*-value	*p*-value
	inside CPZ	other cities	inside CPZ	other cities	(2)-(1)	(4)-(3)
NO2 μg/m3	33.322	17.555	-0.153	-0.105	0.738	0.981
PM10 μg/m3	33.279	13.958	0.237	-0.067	0.660	0.854
Asthma per 10,000	18.7	15.9	1.2	1.4	0.447	0.835
Accidents per 10,000	5.5	8.3	-2.32	0.0	0.272	0.641
All other diagnoses per 10,000	179	173	-1.6	3	0.94	0.92
Ln Annual Income	7.150	6.838	-0.005	-0.002	0.019	0.913
Ln Ratio College Graduates	-0.834	-0.859	0.084	0.046	0.883	0.320
Ln Ratio on Social Assistance	-2.247	-1.585	-0.129	-0.021	0.019	0.592
Log # children 0–5	10.685	7.774	0.114	0.050	0.000	0.078

Notes: Columns (1) and (2) display the average characteristics inside the Stockholm Congestion Pricing Zone (CPZ) and other Swedish cities that did not have a congestion pricing program. The data in columns (1) and (2) come from the month preceding the trial, December 2005. Columns (3) and (4) present the average two-year change in characteristics in the years preceding the trial. Column (5) presents *p*-values from a two-tailed permutation test of the null hypothesis that the difference between columns (1) and (2) is equal to zero. Column (6) presents *p*-values from a similar test, comparing column (3) to column (4).

Table 2: Effects of Congestion Pricing on Ambient Air Pollution – PM10 and NO2

	(1)	(2)	(3)	(4)
Dependent Variable:	PM10	PM10	NO2	NO2
CPZ*Trial	-2.860	-3.994	-3.580	-3.85
	(0.633)	(0.717)	(1.176)	(1.425)
	{0.000}	{0.000}	{0.000}	{0.009}
	[0.423]	[0.004]	[0.004]	[0.004]
CPZ*in between	0.779	-2.509	-2.927	-3.83
	(0.582)	(0.792)	(1.013)	(0.974)
	{0.184}	{0.002}	{0.005}	{0.002}
	[0.814]	[0.100]	[0.050]	[0.004]
CPZ*Perm	-4.572	-4.568	-6.025	-6.19
	(0.694)	(0.840)	(1.295)	(1.44)
	{0.000}	{0.000}	{0.000}	{0.000}
	[0.430]	[0.004]	[0.004]	[0.004]
Month * year FE	*	*	*	*
Municipal FE	*	*	*	*
Region * month FE		*		*
Weather controls		*		*
Municipality*weather		*		*
Calendar month*weather		*		*
Observations	2,177	2,177	1,431	1,431
Adjusted R-squared	0.56	0.72	0.75	0.79
Mean of Dep Var	18.79	18.79	18.55	18.55
Clusters	99	99	58	58

***Notes*:** Columns (1)–(4) present regression results from four separate regressions, one per column, where the dependent variable is in the column heading. The regressions relate pollution monitor readings aggregated to the municipality-month level to indicator variables describing the various phases of CPZ implementation and whether or not the monitor is in the CPZ region. Weather controls include: total rainfall in the month, mean temperatures, and mean wind speed during the month. For each regression coefficient, we present the coefficient estimate, the standard error (in parentheses), as well as two sets of *p*-values. Standard errors are clustered at the municipality level. Analytical *p*-values are in curvy brackets. Permutation-based two-tailed *p*-values appear in square brackets under the analytical *p*-values. Permutation-based *p*-values are estimated using 500 permutations of treatment assignments across municipalities and time (in six-month cells).

Table 3: Acute Asthma Episodes per 10,000 Children Younger Than Five Years Old, per Month

	(1) Limited Controls	(2) Full Controls
CPZ*Trial	-3.043	-2.125
	(1.174)	(1.173)
	{0.010}	{0.050}
	[0.010]	[0.007]
CPZ*in between	-6.399	-5.680
	(1.175)	(1.176)
	{0.000}	{0.000}
	[0.007]	[0.007]
CPZ*Perm	-8.735	-9.597
	(1.898)	(1.935)
	{0.000}	{0.000}
	[0.007]	[0.007]
Month * year FE	*	*
Municipal FE	*	*
Region * month FE		*
Weather controls		*
Municipality*weather		*
Calendar month*weather		*
Observations	7,416	7,416
R-squared	0.447	0.567
Mean of Dep Var	19.06	19.06
Clusters	103	103

Notes: Each column presents results from a separate regression. Demographic controls are included in all specifications and include: the log of mean income, the proportion of mothers on social support, and the proportion of mothers with a college education. Weather controls include: total rainfall in the month, minimum and maximum temperatures, and mean wind speed during the month. For each regression coefficient, we present the coefficient estimate, the standard error (in parentheses), as well as two sets of *p*-values for estimates that are statistically significant at conventional levels. The SEs are clustered at the municipality level. Analytical *p*-values are in curvy brackets under each estimate. Permutation-based two-tailed *p*-values appear in square brackets and are estimated using 500 permutations of treatment assignment across municipalities and time (in six-month cells). All regressions use the number of children younger than five years old in the municipality as weights.

Table 4: The Effect of the CPZ on Acute Asthma Episodes for Children Younger Than Six-Months Old, Excluding the First Six Months of the In-Between Period

	(1)	(2)
CPZ*Trial	-2.885	-2.719
	(1.574)	(2.227)
	{0.070}	{0.225}
	[0.004]	[0.004]
CPZ*In between	-1.227	0.012
	(1.317)	(1.985)
	{0.350}	{0.995}
	[0.001]	[0.99]
CPZ*Permanent	-4.314	-2.516
	(1.392)	(1.546)
	{0.003}	{0.110}
	[0.004]	[0.004]
Month * year FE	*	*
Municipal FE	*	*
Region * month FE		*
Weather controls		*
Municipality*weather		*
Calendar month*weather		*
Observations	6,901	6,901
R-squared	0.307	0.396
Mean of Dep Var	16.52	16.52
Clusters	103	103

Notes: The sample only includes children younger than six-months old. We drop the first six months of the in-between period to avoid contamination from potential long-term effects of the trial period. Demographic controls are included in all specifications and include: the log of mean income, the proportion of mothers on social support, and the proportion of mothers with a college education. Weather controls include: total rainfall in the month, minimum and maximum temperatures, and mean wind speed during the month. Standard errors are clustered at the municipality level and reported in parentheses under the estimates. Analytic *p*-values in curly brackets appear under all estimates. Permutation based *p*-values appear in square brackets under the standard errors, and are based on the 500 bootstrap replications of treatment assignment across municipalities and time (in six-month cells). All regressions use the number of children younger than six months old in the municipality as weights.

Table 5: Robustness and Placebo Checks

	(1) Asthma	(2) Asthma	(3) Predicted Asthma	(4) Unintentional Injury	(5) All other diagnoses
CPZ*Trial	-4.282	-1.875	0.457	-1.614	7.325
	(1.098)	(1.25)	(0.118)	(0.376)	(11.373)
	{0.000}	{0.138}	{0.000}	{0.000}	{0.950}
	[0.004]	[0.004]	[0.000]	[0.677]	[0.370]
CPZ*in between	-7.923	-5.150	0.165	1.018	5.511
	(1.951)	(1.290)	(0.149)	(0.298)	(13.398)
	{0.000}	{0.000}	{0.271}	{0.000}	{0.670}
	[0.004]	[0.004]	[0.000]	[0.458]	[0.350]
CPZ*Perm	-10.488	-8.770	-0.127	0.966	-7.885
	(3.000)	(1.88)	(0.257)	(0.337)	(23.685)
	{0.000}	{0.000}	{0.621}	{0.005}	{0.740}
	[0.004]	[0.004]	[0.000]	[0.737]	[0.120]
Month * year FE	*	*	*	*	*
Municipal FE	*	*	*	*	*
Region * month FE		*	*	*	*
Weather controls		*	*	*	*
Municipality*weather		*	*	*	*
Calendar month*weather		*	*	*	*
Municipality trends	*				
Observations	7,416	7,416	7,416	7,416	7,416
R-squared	0.544	0.54	0.999	0.211	0.672
Mean of Dep Var	19.06	19.06	19.06	8.711	183
Clusters	103	103	103	103	103

Notes: Each column presents results from a separate regression, relating the variable indicated in the column heading to indicator variables describing the phases of the CPZ implementation and whether the municipality is in the CPZ region. (1) Including municipality trends; (2). Assigning the first municipality observed in the data; (3). Using predicted asthma; (4). Using unintentional injury and (5). Using all other diagnoses (nonrespiratory and non-accident-related) as dependent variables. Demographic controls are included in all specifications and include: the log of mean income, the proportion of mothers on social support, and the proportion of mothers with a college education. Weather controls include: total rainfall in the month, minimum and maximum temperatures, and mean wind speed during the month. For each regression coefficient, we present the coefficient estimate, the standard error (in parentheses), as well as two sets of *p*-values for estimates. Analytical *p*-values are in curvy brackets and clustered at the municipality level. Permutation-based two-tailed *p*-values appear in square brackets and are estimated using 500 permutations of treatment assignment across municipalities and time (in six-month cells). Predicted asthma is constructed using an auxiliary regression based on all observations except those in the CPZ during the periods with congestion pricing. The predicted values from this auxiliary regression are used as the dependent variable in column (3). All regressions are using the number of children aged zero through five in the municipality as analytic weights.

Appendix Tables

Appendix Table 1: Effects of Congestion Pricing on Ambient Air Pollution – Continuously Operating Monitors Only

	(1)	(2)	(3)	(4)
	PM10	PM10	NO2	NO2
CPZ*Trial	-2.721	-3.434	-2.292	-3.149
	(1.03)	(1.29)	(1.394)	(1.458)
	{0.017}	{0.016}	{0.05}	{0.0438}
	[0.004]	[0.79]	[0.004]	[0.007]
	0.566	0.562	0.378	0.294
CPZ*in between	1.486	-2.426	-1.034	-2.203
	(0.598)	(0.665)	(1.141)	(0.942)
	{0.023}	{0.002}	{0.376}	{0.030}
	[0.758]	[0.08]	[0.427]	[0.007]
	0.400	0.356	0.470	0.299
CPZ*Perm	-2.741	-2.772	-3.235	-3.942
	(0.719)	(0.749)	(1.719)	(1.902)
	{0.003}	{0.002}	{0.075}	{0.052}
	[0.805]	[0.834]	[0.69]	[.007]
	0.430	0.503	0.500	0.451
Month * year FE	*	*	*	*
Municipal FE	*	*	*	*
Region * month FE		*		*
Weather controls		*		*
Municipality*weather		*		*
Observations	903	903	1,000	1,000
Adjusted R-squared	0.70	0.86	0.85	0.89
Mean of Dep Var	19.19	19.19	18.54	18.54
Clusters	20	20	20	20

Notes: Each column presents estimates from a separate regression, relating the variable indicated in the column heading to indicator variables describing the phases of the CPZ implementation and whether the monitor is in the CPZ region. Monitor-level data are aggregated at the municipality-month level. Weather controls include: total rainfall in the month, minimum and maximum temperatures, and mean wind speed during the month. Analytic weights are based on the number of observations per municipality and are applied to all regressions. For each regression coefficient, we present the coefficient estimate, the standard error (in parentheses) and three sets of *p*-values. Analytical *p*-values appear in curvy brackets. They are clustered at the municipality level. Permutation-based *p*-values appear in square brackets and are estimated using 500 permutations of treatment assignment across municipalities and time (in six-month cells). To estimate the wild bootstrap *p*-values we used Rademacher weights and 999 replications. Bootstrap clustering is by municipality.

Appendix Table 2: Demographic Controls as the Dependent Variables.

Dependent Variable:	(1) Log N children	(2) Social Assistance	(3) College	(4) Log income
CPZ*Trial	0.030	-0.005	-0.004	0.016
	(0.003)	(0.001)	(0.001)	(0.006)
	{0.000}	{0.000}	{0.000}	{0.006}
	[0.000]	[0.000]	[0.000]	[0.000]
CPZ*in between	0.051	-0.008	-0.002	0.009
	(0.004)	(0.001)	(0.001)	(0.006)
	{0.000}	{0.000}	{0.050}	{0.116}
	[0.000]	[0.000]	[0.006]	[0.000]
CPZ*Permanent	0.123	-0.011	0.001	0.006
	(0.006)	(0.001)	(0.002)	(0.006)
	{0.000}	{0.000}	{0.453}	{0.315}
	[0.000]	[0.000]	[0.640]	[0.000]
Observations	7,416	7,416	7,416	7,416
R-squared	0.981	0.999	0.996	0.997
Mean of Dep Var	7.275	0.038	0.41	7.236
Clusters	103	103	103	103
Month * year FE	*	*	*	*
Municipal FE	*	*	*	*
Region * month FE	*	*	*	*
Weather controls	*	*	*	*
Municipality*weather	*	*	*	*
Calendar month*weather	*	*	*	*

Notes: Columns (1)–(4) present regression results from four separate models, one per column, where the dependent variable is labeled in the column heading. Weather controls include: total rainfall in the month, minimum and maximum temperatures, and mean wind speed during the month. Analytical *p*-values in curvy brackets under the SEs for all estimates. Standard errors under the coefficient are clustered at the municipality level. Permutation-based two-tailed *p*-values appear in square brackets under the analytical *p*-value estimates. Permutation-based *p*-values are estimated using 500 permutations of treatment assignment across municipalities and time (in six-month cells). All regressions use the number of children younger than five years in the municipality as analytic weights.

Appendix Table 3: Demographic Determinants of Asthma.

	(1)
	Asthma rate
Social assistance	47.951
	(91.294)
Ln(Income)	23.089
	(14.63)
College	-47.948
	(47.562)
Observations	7,368
R-squared	0.544
Mean of Dep Var	19.34
Clusters	103
Month * year FE	*
Municipal FE	*
Region * month FE	*
Weather controls	*
Municipality*weather	*
Calendar month*weather	*

Notes: This table presents results relating the asthma rate to observable predictors of asthma using observations outside the CPZ region. We use this predictive model to predict asthma rates as a function of demographics. Weather controls include: total rainfall in the month, minimum and maximum temperatures, and mean wind speed during the month. For each regression coefficient, we present the coefficient estimate and the standard error. Standard errors are clustered at the municipality level.

Appendix Table 4: Asthma Rates in Children Older Than Five

	(1) Ages 6–18
CPZ*Trial	-0.054
	(0.970)
	{0.960}
	[0.010]
CPZ*in between	-0.284
	(1.471)
	{0.850}
	[0.59]0
CPZ*Perm	-1.448
	(2.393)
	{0.550}
	[0.01]
Month * year FE	*
Municipal FE	*
Region * month FE	*
Weather controls	*
Municipality*weather	*
Calendar month*weather	*
Observations	7,416
R-squared	0.496
Mean of Dep Var	3.336
Clusters	103

Notes: The model corresponds to Model 2 in Table 3. Demographic controls are included in all specifications and include: the log of mean income, the proportion of mothers on social support, and the proportion of mothers with a college education. Month-by-year fixed effects and municipality fixed effects are included in all specifications. Weather controls include: total rainfall in the month, minimum and maximum temperatures, mean wind speed during the month. Standard errors are reported in parentheses under the estimate, clustered on the municipality level. Analytical weights based on the number of children of the associated age group residing in the municipality during the months applied.

Appendix Table 5: Asthma Rates in Children Younger Than Five Years Old, Sensitive to Excluding Weather Interactions

	(1) Full Controls	(2) Weather Only Controls
CPZ*Trial	-2.125	-2.920
	(1.173)	(1.163)
CPZ*in between	-5.680	-6.289
	(1.176)	(1.104)
CPZ*Perm	-9.597	-9.292
	(1.935)	(1.883)
Month * year FE	*	*
Municipal FE	*	*
Region * month FE	*	
Weather controls	*	*
Municipality*weather	*	
Calendar month*weather	*	
Observations	7,416	7,416
R-squared	0.567	0.455
Mean of Dep Var	19.06	19.06
Clusters	103	103

Notes: Each column presents results from a separate regression. Demographic controls are included in all specifications and include: the log of mean income, the proportion of mothers on social support, and the proportion of mothers with a college education. Weather controls include: total rainfall in the month, minimum and maximum temperatures, and mean wind speed during the month. The SEs are clustered at the municipality level. All regressions use the number of children younger than age five in the municipality as weights.