Congestion Pricing, Air Pollution and Children's Health

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February, 2017

Abstract

This study examines the effects of implementing a congestion tax in central Stockholm on both ambient air pollution and the population health of local children. We demonstrate that the tax reduced ambient air pollution by 5 to 10 percent, and this reduction in air pollution was associated with a significant decrease in the rate of acute asthma attacks among young children. The change in health was more gradual than the change in pollution suggesting that it may take time for the full health effects of changes in pollution to be felt. Given the sluggish adjustment of health to pollution changes, short-run estimates of the pollution reduction programs may understate the long-run health benefits.

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Economists have long recognized the negative externalities associated with traffic congestion and have suggested congestion pricing as a possible solution (Vickery, 1969). Typically, research and policy making surrounding congestion pricing have been concerned with the effects on time lost commuting, fuel costs, and business activity (Leape, 2006).[2] However, recent research suggests that congestion also significantly contributes to local air pollution levels, and this pollution has the ability to impact the health and well-being of nearby residents (Currie and Walker, 2011; Knittel, Miller, and Sanders, 2016).

As traffic congestion in cities has increased, rates of respiratory illness 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 low socio-economic status individuals who disproportionately live in densely populated areas characterized by frequent and often severe traffic congestion. Currently, the highest rates of asthma incidence in the United States are found in the Bronx, New York (Garg et al. 2003). This area of northern New York City is bisected by 5 major highways that rank among the most congested in the United States (Bruner 2009). With both traffic congestion and children's asthma rates trending upwards in recent years, some have speculated that the two may be causally related.

This study is the first to examine the link between congestion pricing, ambient air pollution, and inpatient and outpatient visits for asthma in the context of a congestion pricing program in Stockholm, Sweden. Since August 2007, Stockholm has levied charges on most vehicles entering the city center. Permanent implementation of the charges followed a seven-month trial period between January and July, 2006. We combine this program variation in congestion fees with data on ambient air pollution and administrative data on all inpatient and outpatient health visits. We

[2] Travel delays and extra fuel consumption cost the United States an estimated $70 Billion each year (Schrank and Lomax 2007). The average time per year an urban motorist loses to congestion during peak hours was estimated to be 62 hours in 2000.

compare outcomes within the Stockholm city center to outcomes in other city centers within Sweden that did not have a congestion pricing program, in an effort to form a counterfactual for what would have happened in the absence of the program.

The findings suggest that congestion pricing in Stockholm reduced nitrogen dioxide (NO2) and particulate matter (PM10) levels by 5 and 10 percent, respectively. 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 went into place: Visits for urgent asthma care among children under six fell by 12.3 percent during the trial, but eventually fell by 46.5 percent when the congestion pricing program was made permanent. This slow adjustment of individual health stocks to pollution changes suggests that short-run estimates of the effects of pollution reduction programs may significantly understate the long-run health benefits.

A rigorous investigation of the link between congestion pricing, pollution, and children's health is important for several reasons. First, congestion pricing has been evaluated largely in the context of its effects on traffic and commuting times, but may also have significant benefits in terms of pollution reduction and health, and it is important to quantify those benefits.

Second, 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. Lung development continues post-natally until the adolescent years and is susceptible to negative environmental shocks (Pinkerton et al, 2000; Dietert et al, 2000). For all these reasons, current standards for air pollution may not adequately protect children. Recent recommendations of the American Academy of Pediatrics' Committe on Environmental Health include a revision of the current U.S. Environmental Protection Agency (EPA) standards for common traffic-related pollutants and strongly encourage traffic-reducing state- and city-level policies such as car-pooling and increased access to mass public transport (Kim et al, 2004).

Third, because the implementation of the Stockholm congestion pricing program was preceded by a trial, it offers a unique opportunity to assess both the immediate short-run effects of a drop in pollution, and the somewhat longer-term effects. Since health is a stock, it may not be surprising that the longer-term effects, which allow for adjustment of the underlying health stock, are larger than the shorter-term effects. However, the existing empirical evidence documenting how short run changes in pollution manifest themselves in the form of longer run changes in population health is limited.

Fourth, 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 the question of whether pollution levels well below current U.S. regulatory standards can nevertheless have negative effects on chldren's health.

The rest of the paper is laid out 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 congestion pricing zone (CPZ) was to reduce traffic entering the central city in order to reduce congestion. The tax varies between 0 and 2.6 USD per vehicle, depending on the time of the day. There are no charges at night, on weekends and public holidays, or during July. The toll is automatically collected using license plate scanning technology as cars cross the perimeter of the congestion zone.

The implementation of the tax started with a seven-month trial period, the Stockholm Congestion Trials (*Stockholmsförsöket).* The trial period lasted from the 3rd of January 2006

through the end of July 2006. The trial period was considered a success by the government, with government estimates suggesting reductions in inner city traffic counts during the trial of around 20 to 25 percent (Stockholmforsoket, 2006b). Based on the success of the trial program, the Swedish government decided to make the program permanent. The toll charges were adopted again starting August 1st, 2007 and have been in place 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 suggests 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). Exposure to nitrogen dioxide (NO2) also worsens asthma symptoms and is associated with inferior respiratory health (Lipsett et al, 1997; Shima et al, 2000).

According to the U.S. National Asthma Education and Prevention Program Third Expert Panel on the Diagnosis and Management of Asthma (2007), 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. Reversibility of airflow limitation after an initial negative stimulus is removed may be incomplete, which suggests that once asthma is induced, it may not immediately improve to baseline after triggers are removed. In fact, onset of asthma in childhood can be associated with significant deficits in lung growth, which are permanent. Conversely, if the initiation of asthma is avoided, the child's lung growth and health will be better than it would have been in the presence of asthma.

Previous research asking how air pollution affects children's respiratory health has generally focused on the impacts of short-term variations in air pollution (Friedman et al. 2001;

Neidell, 2004; Neidell and Moretti, 2011; Schlenker and Walker, 2015; Jans et al, 2016). For example, Schlenker and Walker (2012) utilize variation in daily aiport congestion rates as a cause of increased carbon monoxide emissions that are then linked to hospital admissions for respiratory conditions. Bauernschuster and co-authors (2015) show that during days in which the German public transport workers strike, there is increased pollution due to heavier car traffic into major cities as well as increased rates of hospitalizations for respiratory conditions among children younger than 5 and the elderly. Both studies rely 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 short-term reduction in air pollution (associated with the trial) and the effects of the later longer-term reduction in pollution that accompanied permanent implementation of the congestion tax.[3]

There is considerable evidence in the medical and epidemiology literatures that the severity of asthma is affected by both the level of pollution and the length of exposure to it. 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 3 to 5 years ago compared to those who moved within the past 1 to 2 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). These studies suggest then that it may take some time after an initial change in pollution levels for a new equilibrium level of asthma to be reached:

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

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.

III. Data

We use detailed data on the residential location and timing of all hospital inpatient and acute (unplanned) outpatient visits among residents in inner city in Stockholm and 102 other Swedish central cities between 2004 and 2010. The analysis focuses on all children up to and including five years of age who resided in major Swedish municipalities that monitor ambient air pollution. We restrict our analysis to children whose mothers were born in Sweden. The focus on children under the age of six is motivated by the fact that these children are the most likely to experience acute asthma episodes perhaps because they and their parents are less likely to have learned how to prevent and control attacks. For example, Moorman, Person, and Zahran (2013) estimate that in the U.S., children 0 to 4 are 1.9 times more likely to have an asthma attack other things being equal than children 12 to 17.

We use GIS to determine whether the residence of the parents is located inside or outside the congestion pricing zone in Stockholm. We drop observations for children who live in the Stockholm region but outside the CPZ. We omit these children for two main reasons. First, it is not clear whether and how they were affected by the CPZ. For example, children who live outside the CPZ, but close to a major traffic artery that leads into the inner city may have experienced a decrease in ambient pollution levels. Alternatively, they may have experienced increases if traffic was diverted from the inner city to surrounding areas. Second, there are very few pollution monitors located in the Stockholm region outside of the CPZ, which impedes our ability to assess how air pollution changed outside central Stockholm.

Data on ambient air pollution were collected from cities' Environmental Agencies (Miljöförvaltningen) from the period 2004-2010 from ambient air monitors. 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 (NO_2) and PM10. Our choice of these pollutants is motivated by the facts that automobiles account for a disproportionate share of both particulate and nitrogen emissions, and that the monitor data is sufficiently detailed to examine these pollutants.

We aggregate the daily monitor data for each city to the monthly level, weighting by the number of monitor observations within each month. The first two rows of Table 1 show the average levels and growth rates in NO_2 and PM10 levels for central Stockholm ("inside CPZ") and all other central cities in the 2 years preceding the CPZ trial.[4] The levels of 33.32 and 33.28 micrograms per cubic meter for these two pollutants respectively, can be compared to current EPA standards for annual average levels of 66.25 micrograms per cubic meter for NO_2 and 50 micrograms per cubic meter for PM10. Hence Table 1 shows that the levels of pollution in Stockholm were below current U.S. EPA standards for these pollutants even prior to the implementation of congestion pricing. Our results should therefore be interpreted as illustrating the health benefits of reducing pollution levels below already relatively low levels of ambient air pollution.

Column (5) of Table 1 presents p-values from a test of the null hypothesis that the levels are the same inside Stockholm and in other central cities. Column (6) shows p-values from a test of the null hypothesis that the pre-trends 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

[4] 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

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. In order to conduct the test, we assign treatment status to different Swedish cities and then re-calculate the differences in mean levels and in pre-trends 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 0. 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 2 years preceding the CPZ trial.

Figure 1 shows the differences in PM10 and NO_2 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 points show the differences in monthly averages representing data from the entire set of available monitors, and the horizontal lines show the mean differences in pollution levels within each time period. In total, we have 72 months of data. For both PM10 and NO_2, one sees a relative reduction in pollution during the trial period, a rebound to original levels in the "in-between" period, and a larger relative reduction in Stockholm compared to other central cities once the CPZ is made permanent.

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 9th and 10th versions of International Classification of

Diseases (ICD). The National Patient Register records all hospital admission 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 that was 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 that are 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 which 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 0 to 5 in the municipality and divide by the total number of resident children.

Table 1 shows that at 18.7 cases per 10,000 children 0 to 5, 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 injuries (accidents) among children 0 to 5. 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 though of course injuries from car accidents could be reduced by the absence of cars from the inner city. 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 pre-trends.

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

In all of 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. The weather data is linked to each city using the inverse distance weighted average of all weather monitors within 100km of the municipal center. 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:

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

where *Trial*, *InBetween*, and *Permanent* are dummy variables equal to one during the relevant periods. Pollution at monitor *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 congestion trial is in place and the pollution monitor is in the CPZ zone. Equation (1) also includes monitor fixed effects υ_i, weather controls, W_{it}, year by month fixed effects, and monitor-specific time trends, Z_{it}.

The coefficients β_1 and β_3 measure the shorter and longer-run effects of implementing congestion pricing, while β_2 measures whether the dependent variable returned to "baseline" during the "in-between" period. Monitor fixed effects ensure that the identifying variation comes from within-monitor changes in air pollution in periods with congestion pricing versus periods without. The main identifying assumption is that even if the levels of pollution 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. Since the issue of differential trends is potentially important, we also include both region-specific time trends and interactions between municipality 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.

We examine asthma rates using models that take much the same form as equation (1) except that they use measures of asthma rates constructed at the municipality, month, and year level as the dependent variable. Now Z_{jt} is a vector of time-varying controls including the average family income in municipality j in month t, the proportion of the population on social support, and the proportion with a college degree, as well as a vector of year by month fixed effects. Once again we include controls for monthly weather conditions. Instead of fixed effects for pollution monitors, we include fixed effects for each municipality, so that our models are identified using within-municipality variation in asthma rates. The identifying assumption is that there would have been parallel trends in asthma in the absence of the CPZ. We provided some evidence in support of this assumption by examining pre-trends in Table 1.

We also use all of the data, aside from that collected in central Stockholm while congestion pricing was in effect, in order to model asthma rates in the rest of the sample, and then ask whether the predicted asthma rates from this model change with the implementation of the CPZ. This test assesses the degree to which the 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 childhood injuries.

All regressions with asthma or injury as a dependent variable are estimated by weighted least squares using the number of children aged 0 to 5 in the municipality as weights.

V. Results

Table 2 presents estimates for effects of the CPZ on levels of PM10 and NO_2. We show analytical standard errors in parentheses, and we present two sets of p-values: p-values that correspond to the analytical standard errors which are shown in curvy brackets and permutation-based p-values which are shown in square brackets. The permutation-based values are based on 300 simulations in which the CPZ "treatment" (trial, in between, or permanent) is randomly assigned across municipality-quarter (of the year) cells (see e.g., Cesarini et al (2016)). For each outcome and each permuted sample we estimate equation (1). We then examine the fraction of the time that the coefficient estimate exceeds the estimated value when the CPZ is correctly assigned to Stockholm.

The estimates and analytic p-values in column (1) and (2) of Table 2 are consistent with Figure 1 in that they suggest that both PM10 and NO_2 declined during the trial, rebounded during the in-between period, and settled at a new lower level similar to that seen during the trial when the CPZ became permanent. The permutation based p-values are more conservative but also support this story. The 3.36 unit decline in PM10 and the 1.58 unit decline in NO_2 after the CPZ

is made permanent correspond to a 10% and a 5% reduction in these two pollutants relative to the mean levels of pre-CPZ pollution in Stockholm shown in Table 1. The estimates in column (3) shows that if one did not allow for the "rebound" effect during the in-between period, one would get a lower a slightly lower estimate of the impact of congestion pricing on pollution.

Appendix Table 1 shows an alternative specification which uses only data from monitors that were continuously operating. Permanently operating monitors might be expected to more accurately capture changes over time, and concerns pertaining to bias introduced by monitor entry and exit are held fixed. On the other hand, there are many fewer continuously operating monitors. The point estimates in Appendix Table 1 are remarkably similar to those discussed above, and the analytic p-values suggest significant negative effects on pollution in both the trial and the permanent CPZ periods. The permutation based tests confirm a negative effect of congestion pricing on PM10, but do not allow rejections of the null in the case of NO_2. Overall Appendix Table 1 re-affirms that the Stockholm CPZ was associated with significant pollution reductions.

The estimated effects of the CPZ on asthma rates are shown in columns (1), (2), and (3) of Table 3. A comparison of columns (1) and (2) shows that the estimates are quite robust to interaction month fixed effects with region (in order to allow for different regional seasonal effects) and interactions of weather and municipality (in order to allow weather conditions to have differential effects in different cities).

However, unlike the pollution estimates which shows 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 2.3 asthma visits per 10,000 children (on a baseline of 18.7 visits per 10,000) during the trial. The "in-between" period saw a reduction of 4.8 cases per 10,000, while the permanent CPZ reduced asthma visits by about 8.7 per 10,000. 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 almost half among children 0 to 5. 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. Column (3) indicates that overall, congestion pricing was associated with a reduction of 5.83 asthma visits per 10,000 children.

Figure 2 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 vs. other cities continued to decline in the "in between" period, and fell to their lowest levels after the CPZ became permanent.

Column (4) of Table 3 shows estimates from a model using predicted asthma rates as the dependent variable. The prediction equation itself is shown in Appendix Table 2. Because we saw in Table 1 that rates of social assistance use 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 model which includes municipality fixed effects, interactions between month and year, interactions between region and month, and interactions between municipality and weather are included in the model. However, with an R-squared of .544, we do a reasonably good job explaining variation in asthma rates.

The estimated models using predicted asthma as the dependent variable suggest that changes the congestion pricing program led to changes in observable characteristics that are correlated with small but significant *increases* in the asthma rate in the short run, followed by an equally small decrease in asthma rates when the CPZ became permanent. While there may be other

changes in unobservables that are correlated with both asthma and the congestion program, the evidence here suggests there changes in observable characteristics have limited scope for explaining the baseline asthma results.

Column (5) of Table 3 presents estimates where the dependent variable is visits due to injuries. This model takes the same form as the model of asthma. The analytical standard errors suggest small but significant effects: An initial decline during the trial followed by a rebounding during the in between and CPZ permanent periods so that by the end of the period there was no overall change. The permutation-based p-values are more conservative suggest that none of the estimated effects on injuries are statistically significant. This "placebo" like test suggests then that the CPZ implementation had limited or no effects on non-respiratory health outcomes in this age group.

VI. Discussion and Conclusions

This paper estimates the impact of a congestion pricing program in a large urban center on ambient pollution and children's health as proxied by childhood asthma rates. Our findings indicate that the congestion tax in central Stockholm reduced ambient pollution levels by between 5 to 10 percent. The evidence regarding pollution control is particularly compelling since the 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 during the period in between the trial and permanent adoption, and then showing a sustained decline following permanent adoption of congestion pricing.

This policy-induced change in congestion pricing is associated with a significant reduction in the rate of acute asthma attacks among children 0 to 5 years of age in the years after the program.

In contrast to the pattern for pollution, although there is an immediate impact of congestion pricing, the impact grows over time and actually continued to decline during what proved to be a temporary hiatus in congestion pricing before the permanent adoption of the policy. 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 health evolves to a new lower-pollution equilibrium level. 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 resulted in a significant decrease in ambient air pollution. Our results also show that permanent reductions in air pollution due to automobiles, even in locations which have average pollution levels well below the current U.S. EPA standards, can have significant positive effects on children's respiratory health.

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Table 1: Means and Pre-trends of Outcome and Key Control Variables in the Pre-CPZ Period

	(1)	(2)	(3)	(4)	(5)	(6)
	Levels		Difference Jan. 2006-Jan. 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
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, January 2006. Columns (3) and (4) present the average 2 year change in characteristics in the years preceding the trial. Column (5) presents p-values from a 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)	(5)	(6)
Dependent Variable:	PM10	PM10	PM10	NO2	NO2	NO2
CPZ*Trial	-3.390	-3.961		-3.457	-1.327	
	(0.692)	(0.767)		(0.937)	(0.493)	
	{0.000}	{0.000}		{0.000}	{0.110}	
	[0.325]	[0.100]		[0.172]	[0.170]	
CPZ*in between	0.602	-1.766		-1.492	-0.498	
	(0.524)	(0.664)		(1.331)	(0.679)	
	{0.250}	{0.009}		{0.267}	{0.470}	
	[0.006]	[0.007]		[0.564]	[0.000]	
CPZ*Perm	-4.916	-3.361		-4.387	-1.580	
	(0.622)	(0.838)		(1.393)	(0.473)	
	{0.000}	{0.001}		{.0026}	{0.002}	
	[0.186]	[0.007]		[0.471]	[0.110]	
CPZ*Congest			-2.826			-1.262
(trial+permanent)			(0.527)			(0.247)
			{0.000}			{0.000}
			[0.120]			[0.072]
Monitor FE	*	*	*	*	*	*
Month * year FE	*	*	*	*	*	*
Region * month FE		*	*		*	*
Municipality*weather		*	*		*	*
Observations	2,879	2,879	2,879	2,669	2,669	2,669
Adjusted R-squared	0.66	0.67	0.67	0.44	0.90	0.90
Mean	20.28	20.28	20.28	22.64	22.64	22.64
Clusters	99	99	99	58	58	58

***Notes*:** Columns (1)-(6) present regression results from six separate regressions, one per column. The regressions relate pollution monitor readings at the monitor-month level to indicator variables describing the various phases of the CPZ implementation and whether or not the monitor is in the CPZ region. For each regression coefficient, we present the coefficient estimate, the standard error (in parentheses) and two sets of p-values. Analytical p-values are in curvy brackets and clustered at the monitor level. Permutation-based P-values appear in square brackets and are estimated using 300 permutations of treatment assignments across monitors and time (in quarters).

Table 3: Acute illness episodes per 10,000 children 0-5 per month

	(1) Asthma	(2) Asthma	(3) Asthma	(4) Predicted Asthma	(5) Accidents
CPZ*Trial	-3.043	-2.272		0.457	-1.614
	(1.174)	(1.145)		(0.118)	(0.376)
	{0.010}	{0.050}		{0.000}	{0.000}
	[0.010]	[0.007]		[0.000]	[0.677]
CPZ*in between	-6.399	-4.849		0.165	1.018
	(1.175)	(1.194)		(0.149)	(0.298)
	{0.000}	{0.000}		{0.271}	{0.000}
	[0.007]	[0.007]		[0.000]	[0.458]
CPZ*Perm	-8.735	-8.731		-0.127	0.966
	(1.898)	(1.860)		(0.257)	(0.337)
	{0.000}	{0.000}		{0.621}	{0.005}
	[0.007]	[0.007]		[0.000]	[0.737]
CPZ*Congest			-5.83		
(Trial+Perm)			(1.358)		
			{0.000}		
			[0.070]		
Month * year FE	*	*	*	*	*
Municipal FE	*	*	*	*	*
Region * month FE		*	*	*	*
Municipality*weather		*	*	*	*
Observations	7,416	7,416	7,416	7,416	7,416
R-squared	0.447	0.544	0.543	0.999	0.211
Mean	19.06	19.06	19.06	19.06	8.711
Clusters	103	103	103	103	103

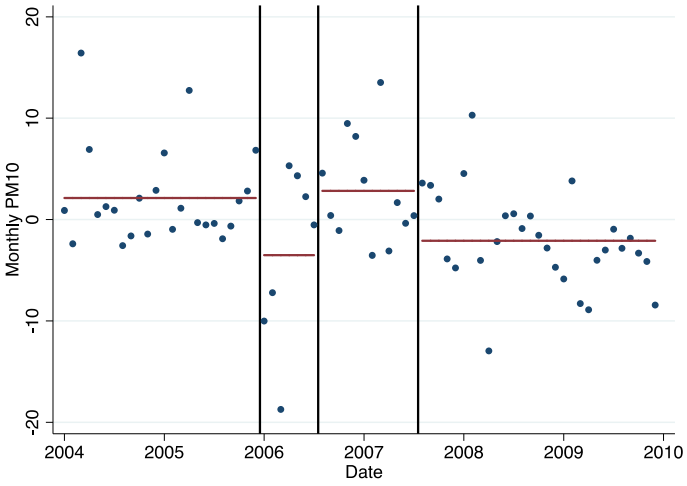
Notes: Each column presents results from a separate regression, relating the variable indicated in the column heading to indicator variables describing the various phases of the CPZ implementation and whether or not the municipality is in the CPZ region. For each regression coefficient, we present the coefficient estimate, the standard error (in parentheses) and two sets of p-values. Analytical p-values are in curvy brackets and clustered at the monitor level. Permutation-based P-values appear in square brackets and are estimated using 300 permutations of treatment assignment across municipalities and time (in quarters). 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 (4).

Table 4: Demographic controls as the dependent variables.

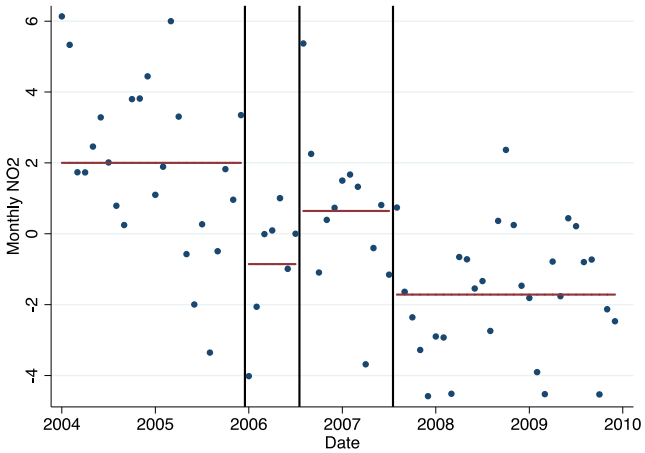
Dependent Variable:	(1) Log N children	(2) Social Assistance	(3) College	(4) Log income
CPZ*Trial	0.030	-0.005	-0.004	0.016
	(0.003)	(0.001)	(0.001)	(0.006)
	{0.000}	{0.000}	{0.000}	{0.006}
	[0.000]	[0.000]	[0.000]	[0.000]
CPZ*in between	0.051	-0.008	-0.002	0.009
	(0.004)	(0.001)	(0.001)	(0.006)
	{0.000}	{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	7.275	0.038	0.41	7.236
Clusters	103	103	103	103
Municipality FE	*	*	*	*
Month * year FE	*	*	*	*
Region * month FE	*	*	*	*
Municipality*weather	*	*	*	*

Notes: Columns (1)-(4) present regression results from four separate models, one per column. Analytical p-values in curvy brackets under the SEs. They are clustered at the municipality level. Permutation-based P-values in square brackets under the analytical p-value estimates. Permutation-based P-values estimated using 300 permutations of treatment assignment across municipalities and time (in quarters).

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



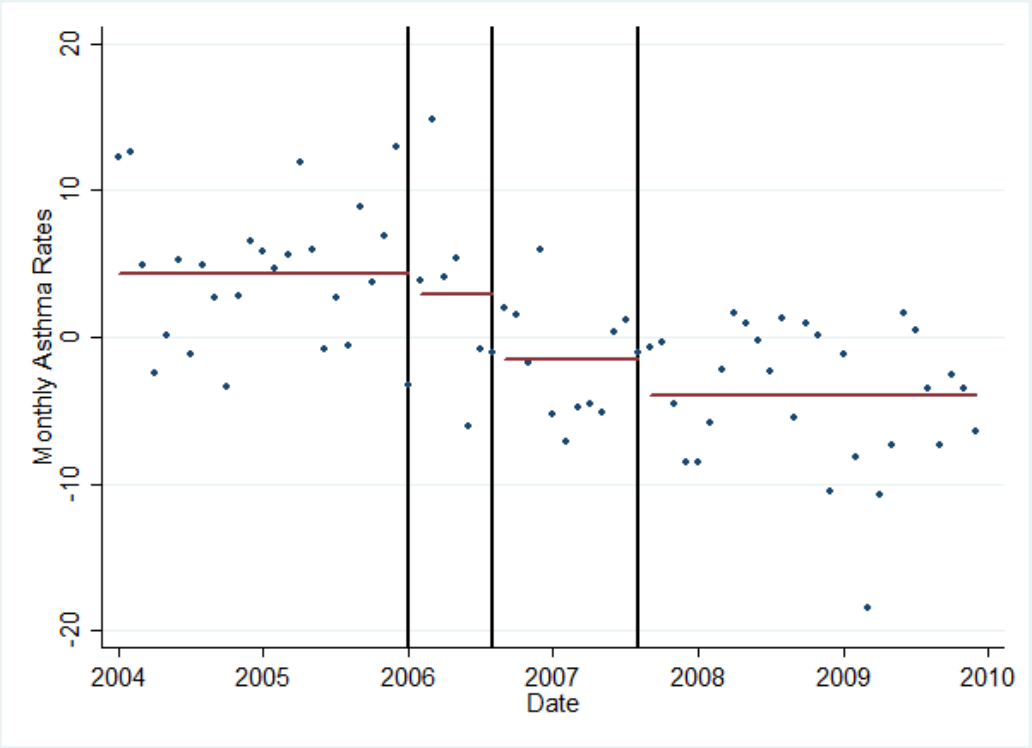
Notes: This figure plots the differences in the unadjusted monthly mean PM10 levels inside versus outside the CPZ. The vertical lines indicate the beginning and end of the trial period and the beginning of the permanent CPZ implementation. Red horizontal lines represent the mean value within each period.



Notes: This figure plots the differences in the unadjusted monthly mean NO2 levels inside versus outside the CPZ. The vertical lines indicate the beginning and 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: Asthma differences inside CPZ vs. other central cities

a) Asthma



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

Appendix Tables

Appendix Table 1: Effects of congestion pricing on ambient air pollution – Continuously operating monitors only

	(1)	(2)	(3)	(4)	(5)	(6)
	PM10	PM10	PM10	NO2	NO2	NO2
CPZ*Trial	-3.703	-3.811		-2.260	-1.804	
	(1.087)	(1.390)		(1.084)	(0.522)	
	{0.003}	{0.013}		{0.050}	{0.030}	
	[0.132]	[0.007]		[0.511]	[0.220]	
CPZ*in between	1.019	-2.318		-0.570	-0.968	
	(0.504)	(0.634)		(1.059)	(0.672)	
	{0.058}	{0.002}		{0.596}	{0.160}	
	[0.272]	[0.007]		[0.760]	[0.620]	
CPZ*Perm	-3.586	-3.073		-2.351	-1.071	
	(0.705)	(0.606)		(1.484)	(0.467)	
	{0.000}	{0.000}		{0.129}	{0.033}	
	[0.657]	[0.007]		[0.764]	[0.680]	
CPZ*Congest			-2.405			-0.922
(trial+permanent)			(0.464)			(0.271)
			{0.000}			{0.003}
			[0.020]			[0.370]
Monitor FE	*	*	*	*	*	*
Month * year FE	*	*	*	*	*	*
Region * month FE		*	*		*	*
Municipality*weather		*	*		*	*
Observations	1,379	2,669	2,669	1,525	1,525	1,525
Adjusted R-squared	0.52	0.90	0.90	0.91	0.92	0.92
Mean	21.63	22.64	22.64	22.57	22.57	22.57
Clusters	20	20	20	20	20	20

Notes: Each column presents results from a separate regression, relating the variable indicated in the column heading to indicator variables describing the various phases of the CPZ implementation and whether or not the monitor is in the CPZ region. For each regression coefficient, we present the coefficient estimate, the standard error (in parentheses) and two sets of p-values. Analytical p-values are in curvy brackets and clustered at the monitor level. Permutation-based P-values appear in square brackets and are estimated using 300 permutations of treatment assignment across municipalities and time (in quarters).

Appendix Table 2: Demographic determinants of Asthma.

	(1)
	Asthma rate
Social assistance	47.951
	(91.294)
	{0.600}
	[0.578]
Ln(Income)	23.089
	(14.63)
	{0.117}
	[0.119]
College	-47.948
	(47.562)
	{0.316}
	[0.179]
Observations	7,368
R-squared	0.544
Mean	19.34
Clusters	103
Municipality FE	*
Month * year FE	*
Region * month FE	*
Municipality*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 these demographics, which we use as an index measure of demographic change in Column (4) of Table 3. For each regression coefficient, we present the coefficient estimate, the standard error (in parentheses) and two sets of p-values. Analytical p-values are in curvy brackets and clustered at the monitor level. Permutation-based P-values appear in square brackets and are estimated using 300 permutations of treatment assignment across municipalities and time (in quarters).