THE ECONOMIC JOURNAL

The Economic Journal, **126** (*March*), 257–280. Doi: 10.1111/ecoj.12273 © 2015 Royal Economic Society. Published by John Wiley & Sons, 9600 Garsington Road, Oxford OX4 2DQ, UK and 350 Main Street, Malden, MA 02148, USA.

DOES THE EFFECT OF POLLUTION ON INFANT MORTALITY DIFFER BETWEEN DEVELOPING AND DEVELOPED COUNTRIES? EVIDENCE FROM MEXICO CITY

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Much of what we know about the marginal effect of pollution on infant mortality is derived from developed country data. However, given the lower levels of air pollution in developed countries, these estimates may not be externally valid to the developing country context if there is a non-linear dose relationship between pollution and mortality or if the costs of avoidance behaviour differ considerably between the two contexts. In this article, we estimate the relationship between pollution and infant mortality using data from Mexico. Our estimates for PM_{10} tend to be similar (or even smaller) than the US estimates, while our findings on CO tend to be larger than those derived from the US context.

Pollution is a grave concern in much of the developing world, with levels that are often orders of magnitude higher than in developed countries. Using comparable data, Greenstone and Hanna (2011) document air pollution levels that are five to seven times higher in India and China than in the US. This may translate into many lost lives: the OECD estimates that almost 1.5 million individuals die from exposure to particulates each year, many more than who die from malaria or unclean water. With pollution levels predicted to rise, the OECD claims that this figure may exceed 3.5 million people per year by 2050, with most of these deaths occurring in rapidly industrialising countries, such as India and China (OECD, 2012).

In contrast with these concerns, Mexico, another rapidly industrialising country, has experienced important gains in air quality during the last 20 years. Between 1997 and 2006, an array of policies aimed at cutting down pollution in Mexico City resulted in pollutant concentration reductions of between 23% (ozone) and 48% (carbon monoxide). The policies implemented in this period include centralised fuel improvement, driving bans, more stringent vehicle and industry emission standards among others.[1] During the same period, the infant mortality rate dropped by 30% and the neonatal mortality rate dropped by 20%. Whether or not all – or part of – the time

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We thank Jon Hill and Katherine Kimble for excellent research assistance. We are grateful to David Card, Janet Currie, Olivier Deschenes, Heather Royer, Heidi Williams, Catherine Wolfram and Lucas Davis for helpful comments, as well as seminar participants at CEPR Development Meetings Pre-Conference, the ARE Berkeley Seminar, the Environment and Human Capital Conference, the UC Santa Barbara Labor Lunch, UC Berkeley Labor Lunch and El Colegio de Mexico Econ Lunch. We also thank the editor and two anonymous referees for their very helpful comments. We especially thank Marta Vicarelli for providing us with the temperature data. This project was funded in part by the Harvard Center for Population and Development Studies and UCMexus.

[1] So far no single policy has been proved to be responsible for the sharp drop in pollution. In fact, Davis (2008) finds no effect of the driving ban and Oliva (2015) finds strong evidence of corruption in the smog check programme that enforces vehicle emission limits.

series relationship between pollution and infant mortality is causal is still an open question.

The challenges with uncovering the causal effect of air pollution on health are well known in the economics and epidemiological literature. One of the biggest concerns is that of attributing to pollution the effect of other factors that may be correlated with health, such as weather, socio-economic status and changes in economic conditions. Another important challenge is that of attributing to pollution deaths of individuals who would have died within a few days due to other causes (harvesting). Recent studies within economics have overcome some of these challenges using rich data from the US and quasi-experimental approaches that are arguably robust to confounding factors and harvesting. These methodologies fit generally in one of two groups: fixed effects (Currie and Neidell, 2005) and instrumental variables (Chay and Greenstone, 2003; Knittel *et al.*, 2011).

Many of the existing studies for the developing world are in the epidemiological literature. These studies have typically relied on time series variation in air pollution while controlling for temperature (Borja-Aburto *et al.*, 1998; Loomis *et al.*, 1999) and sometimes controlling for time-fixed unobservable socio-economic or sub-regional characteristics (Borja-Aburto *et al.*, 1997; O'Neill *et al.*, 2004). These empirical strategies might be subject to omitted variable bias from unobserved shocks that can affect both pollution and mortality and are very sensitive to measurement error. In addition, none of these studies has examined the effect of carbon monoxide on infant health. While one can in principle address omitted variable bias using either instrumental variables or fixed effects, these techniques pose additional challenges in developing countries. For example, a common strategy to find a valid instrument is to use a policy or regulation that can arguably generate exogenous variation in pollution. However, despite the fact that the regulations in developing countries often look similar to those in the US, they are often riddled with implementation and enforcement problems, resulting in a weak first stage.[2] The remaining approach, fixed effects, effectively controls for time-invariant unobserved differences across locations and overall trends (Currie and Neidell, 2005). This type of empirical model is challenging when using developing country data, as the measurement error that may arise from using sparser pollution data may be exacerbated by the inclusion of fixed effects.[3]

Studies in the economic literature that aim to have a more causal interpretation are scarce and often lack actual pollution data, which makes it complicated to estimate the magnitude of the effect of pollution concentrations on health (Jayachandran, 2009; Gutierrez, 2010).[4] This comes from the fact that the availability and quality of the air

[2] For example, Greenstone and Hanna (2011) experience this problem when using environmental regulations in India as an instrument for pollution and Davis (2010) finds no effect of driving restrictions on air pollution.

[3] As Currie and Neidell (2005) discuss, measurement error has also been noted in the US context as well. Schlenker and Walker (2011) and Knittel *et al.* (2011) find larger impacts of pollution on health when using an instrumental variables strategy as compared to fixed effects methods using US data, which both claim is consistent with classical measurement error being exacerbated with the fixed effects methodology.

[4] Other studies include Greenstone and Hanna (2011) who estimate the effect of mandated catalytic converters on infant mortality rates in India, but have a noisy estimate of the policy impact due to limited data; and Tanaka (2015), who measures the effect of more stringent environmental regulation in China.

pollution and mortality data are often more limited in developing countries. Quite frequently, disaggregated data on infant births and deaths are not accurately recorded or computerised. Even when the data are available, the validity of the data may be questionable as there is substantial selection as to which births and deaths are registered. Moreover, there are fewer stations systematically measuring pollution levels in developing countries, and so there is potentially less variation in pollution to exploit. From this standpoint, relying on estimates from developed countries to estimate the costs of air pollution in developing countries on health might be attractive option.

There are, however, two important reasons why estimates from developed countries may have limited external validity to the developing country context. First, they may be limited if there is a non-linear dose–response relationship between pollution and infant mortality. If we expect, for example that marginal changes in pollution are more damaging at higher levels of air pollution, using developed country estimates would cause us to grossly underestimate the effect in many developing countries. On the other hand, if there is an inflection point which pollution needs to fall beneath before health gains can be realised, using developed country estimates could alternatively lead us to overestimate the effect.

Second, the effect of pollution on health may be highly dependent on behaviour (Zivin and Neidell, 2009; Moretti and Neidell, 2011; Deschenes *et al.*, 2011). Avoidance behaviour may be costlier in the developing world, given less access to health care and lower quality housing stock, which would imply that a marginal decrease in pollution may have a larger overall health impact in the developing world. Alternatively, the effect could be smaller if, for example individuals have permanently adapted to bad pollution by keeping infants indoors or wearing breathing masks regularly.[5] Given these two potential factors, applying estimates of the marginal effect of pollution that are derived from the US to developing countries may be highly misleading for policy.

In this study, we aim to address these problems and estimate the impact of pollution on infant mortality in a developing country context. To do so, we construct weekly, municipality-level measures of pollution and mortality for 48 municipalities across Mexico City between the years 1997 and 2006. Mexico City is a highly relevant context in which to study this relationship. On average, it experiences both the high levels of pollution and mortality that are common in many developing countries. However, given the high variance in pollution levels, the range of pollution also encompasses a range similar to that observed in the US. These two facts will allow us to estimate the marginal effect of pollution at a range that is typical for developing countries and then to compare this estimate to the marginal effect at the ranges used in the previous estimates for the US.

We first employ a fixed effects technique, controlling for time-invariant characteristics of municipalities, bimonthly $\times$ municipality fixed effects, weather and municipality-specific week trends. Using this method, we find a small effect of pollution on mortality. However, as we discuss below, even with fixed effects, there may be remaining endogeneity concerns. Moreover, despite access to very high quality pollution measures, station coverage is sparse: depending on the pollutant and year,

[5] As higher pollution is more visible, avoidance behaviour may be more likely since the costs of learning about pollution levels may be lower.

our pollution measures are derived from between 10 and 26 stations. Given that fixed effects models are particularly sensitive to classical measurement error, our estimates may be severely biased downward.

Instead, we exploit the meteorological phenomenon of thermal inversions. An inversion occurs when a mass of hot air gets caught above a mass of cold air, trapping pollutants. Conditional on temperature, inversions themselves do not represent a health risk *per se* other than the accumulation of pollutants. As such, we can use the number of inversions in a given week to instrument for pollution levels that week. We find that each additional inversion leads to a 5.7% increase in particulate matter measuring 10 μm or less (PM_{10}) and a 6.3% increase in carbon monoxide (CO), conditional on municipality fixed effects, bimonthly × municipality fixed effects, municipality-specific time trends, polynomials in temperature and weather controls.

With the instrumental variables strategy, we find robust evidence of pollution on infant mortality. Our estimates imply that 1 $\mu g/m^3$ increase in 24-hour PM_{10} results in 0.23 weekly infant deaths per 100,000 births. Similarly, 1 ppb increase in the 8-hour maximum for CO results in 0.0046 weekly deaths per 100,000 births.[6] We find no significant effect on neonatal (children 28 days and younger) deaths overall. As a test of the causal pathway, we then separate deaths into those that are likely to be pollution related (i.e. respiratory and cardiovascular disease) *versus* those that are less likely to be pollution related (i.e. digestive, congenital, accidents, homicides etc.). We find statistically and policy significant effects of pollution on both neonatal and infant deaths from respiratory and cardiovascular disease. As we would expect if we had indeed isolated the effect of pollution from other factors (i.e. income, health preferences), we find no effect of pollution on deaths from other causes.

Finally, we compare our estimates to those derived in the US setting. Specifically, we compare our estimates to Chay and Greenstone (2003), Currie and Neidell (2005), Currie *et al.* (2009) and Knittel *et al.* (2011).[7] We find larger marginal effects of CO on infant mortality than Currie and Neidell and Currie *et al.*; we also find larger point estimates that Knittel *et al.*, but they do not observe a significant effect of CO on infant mortality. For PM_{10}, our results are near identical to Chay and Greenstone's results, despite the fact that the mean level of pollution in their setting is roughly half of that in Mexico City.

The article proceeds as follows. In Section 1, we describe our empirical methods and data, while we provide our findings in Section 2. Section 3 provides a discussion of our estimates with those from the US context. Section 4 concludes.

1. Empirical Method, Data and Summary Statistics

In this Section, we first discuss some of the existing empirical methods for estimating the relationship between air pollution and infant mortality. We then detail our empirical strategy in subsection 1.2. Finally, we describe the data that we collected for this project.

[6] As we illustrate below, these results are robust to different definitions of mortality, different ways to control for seasonality, the inclusion of outliers and different weather and temperature controls.

[7] Note that other papers in the US context explore the effect of pollution on child and infant health (Lleras-Muney, 2010). We only include papers that study comparable infant mortality outcomes.

1.1. *Existing Empirical Methodologies*

Our objective is to estimate the relationship between pollution (P_{mw}) in a municipality (m) in a given week (w) and mortality per 100,000 live births (Y_{mw}), or the parameter β_1:

$$Y_{mw} = \beta_0 + \beta_1 P_{mw} + \varepsilon_{mw} \tag{1}$$

where ε_{mw} captures all unobserved determinants of mortality. There are many reasons for believing the identification assumption, $\mathrm{E}(P_{mw}, \varepsilon_{mw}) = 0$, does not hold in this case. For example, areas with low levels of pollution may be richer and thus have lower levels of mortality regardless of pollution. One method to solve the endogeneity problem would be to estimate a fixed effects model:

$$Y_{mw} = \beta_0 + \beta_1 P_{mw} + \alpha_m + \sigma_{mj} + \varepsilon_{mw} \tag{2}$$

where α_m is a set of municipality fixed effects that control for permanent differences across municipalities, such as time-invariant socio-economic characteristics. Similarly, σ_{mj} is a set of bimonthly $\times$ municipality fixed effects, which control for common factors in a given two month block that could affect both pollution levels and infant mortality within a municipality.[8] The fixed effects model represents a substantial improvement over the standard cross-sectional regression. However, two concerns remain. First, β_1 may still be subject to bias if there are unobservable, time-varying differences across municipalities. One way to account for this is to include municipality-specific, linear time trends. However, this may not capture sharp or non-monotonic changes in omitted pollution and infant mortality determinants, such as road improvements that could result in fewer traffic jams and faster access for emergency vehicles, or similarly, protests and demonstrations that results in disrupted travel patterns. Second, classical measurement error in the pollution variable will bias $\hat{\beta}_1$ downwards. Fixed effects estimators exacerbate measurement error, biasing $\hat{\beta}_1$ further towards zero. As compared to developed country settings, this may be particularly problematic in developing countries, where pollution-monitoring stations are sparse: for example, as we discuss below, we exploit data from 10 to 26 stations.

1.2. *Exploiting Thermal Inversions in an Instrumental Variables Framework*

We consider an instrumental variables strategy, which is likely to minimise bias from both endogeneity and classical measurement error. Specifically, we exploit a meteorological phenomenon: the existence of thermal inversions. Inversions are a common occurrence in many cities around the world, ranging from Mumbai, Los Angeles, San Paulo, Salt Lake City, Santiago, Vancouver, Prague etc.[9] Air temperature in the

[8] We experimented with different ways of modelling the fixed effects of time and location. One natural way would be to include week fixed effects. However, to be consistent with the IV model below, we include the bimonthly $\times$ municipality fixed effects (note that we drop the first month pair so that it is not co-linear with municipality). The results (both in the fixed effects and IV) look almost identical if we just include bimonthly fixed effects that are not interacted by municipality, so we decided to include the more restrictive set of fixed effects. Note that the results are also robust to dropping the time trends and instead including a year fixed effect.

[9] The great smog of 1952 in the UK was caused by an inversion episode and was blamed for upwards of 12,000 deaths (Bell and Davis, 2001). This incident sparked greater interest in environmental regulation in the UK.

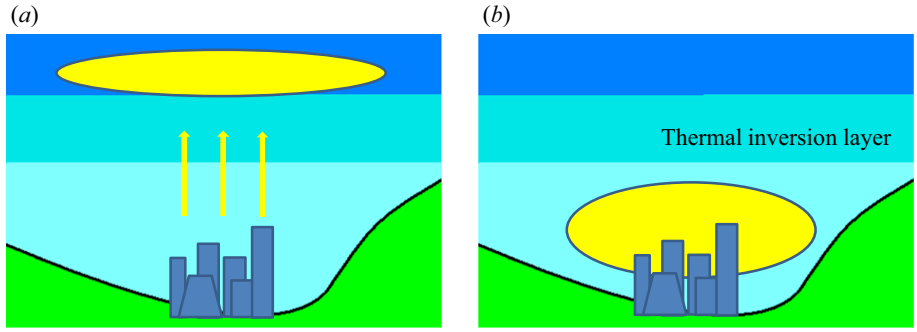

Fig. 1. *Thermal Inversions. (a) Without Inversions, Pollutants Rise and Disburse. (b) Pollutants are Trapped Beneath the Inversion Layer*

troposphere usually falls with altitude at about 6.5°C per 1,000 metres. However, sometimes there is a mass of hot air on top of a mass of cold air; this is called a thermal inversion. There are typically three reasons why this can occur: first, radiation inversions are generated on clear nights when the ground and the air in touch with the ground are cooled faster than higher air layers. The conditions for radiation inversions are more frequent in the winter: under clear conditions, the earth's infrared emissions warm the higher layers of air. The cold ground temperatures cool causing the air that is close to the ground to remain at a lower temperature than the air above. Second, inversions by subsidence occur from vertical air movements when a layer of cold air descends through a layer of hot air. Third, inversions can also be produced when layers of air at different temperatures move horizontally and a layer of cold air develops below a layer of hot air (Jacobson, 2002).

The thermal inversion does not represent a health risk in itself but when it occurs in conjunction with high levels of vehicle and industrial emissions, it may result in the temporary accumulation of pollutants (Secretaría del Medio Ambiente, 2005). Specifically when emissions are released in the atmosphere, they rise and can get trapped in the inversion (see Figure 1). As the sun's energy equates the temperatures of the cold and hot air masses, the 'lid' effect disappears (the inversion 'pops') and the pollutants rise again. Inversions may have substantial effects on the concentration levels of certain types of pollutants, particularly primary pollutants (CO, particulate matter, NO, NO_x and SO_x, VOC) that may be released in the morning rush hours when the inversions typically occur (Jacobson, 2002). Out of the primary pollutants, we would, therefore, expect the largest effects for pollutants in which vehicles comprise a large share of their emissions. For example, in Mexico City, 98% of CO emissions came from vehicles in 1998, and therefore, we expect that a large share is released in the morning commute hours. In contrast, we may expect weaker effects for pollutants like particulate matter, in which 36% is released by vehicles, or SO_2, in which only 21% is.

Inversions may have muted effects on secondary pollutants (O_3, NO_2, sulphuric acid), which require time to mix from the primary pollutants, and therefore, may only appear later in the day when it is likely that the inversions have already 'popped' (Jacobson, 2002). Moreover, inversions may inhibit the formation of these pollutants in other ways. For example, in the particular case of O_3, given that the chemical

reactions that result in O_3 require warmth and sunlight, the thick layers of pollution associated with thermal inversions may interfere with O_3 formation.[10]

We can, therefore, formally test whether inversions increase the concentrations of different types of pollutants (3) and, if so, we can use the number of thermal inversions in a given week (TI_w) to instrument for pollution in (4):[11]

$$P_{mw} = \pi_0 + \pi_1 TI_w + \sum \pi_{2m} w + h(W_{mw}) + \alpha_m + \sigma_{mj} + \mu_{mw}; \quad (3)$$

$$Y_{mw} = \beta_0 + \beta_1 P_{mw} + \sum \beta_{2m} w + h(W_{mw}) + \alpha_m + \sigma_{mj} + \varepsilon_{mw}. \quad (4)$$

Note that TI_w varies at the week level and, therefore, week by year fixed effects are not identified.[12] We, therefore, control for municipality-specific week by year trends (w). We also include municipality fixed effects (α_m) to control for time-invariant characteristics across municipalities and bimonthly × municipality fixed effects (σ_{mj}) to account for seasonal effects within each municipality.[13]

Importantly, we include a flexible set of controls for temperature and weather conditions $h(W_{mw})$ that includes a fourth polynomial in mean temperature, a third degree polynomial in minimum and maximum temperatures during the week, a second degree polynomial in precipitation, cloud cover and humidity measures. Controlling for temperature is important for the exclusion restriction to hold, since inversions have a clear seasonal pattern and temperature may independently affect infant mortality (Deschenes and Greenstone, 2011).[14] Figure 2, panel (a) shows the average number of thermal inversions per week for each month of the year (bars), as well as the average temperatures for each month of the year (spikes) measured by the right axis. As expected, given the conditions necessary for a radiation inversion, a large share of the inversions occurs in the winter (November–March). However, inversions also occur in months with relatively high temperatures (April, May and October), which will allows us to disentangle the effects of temperature on infant mortality from that of air pollution.

Note four additional specification details. First, all regressions are clustered at the week level, which is the level of variation in our instrument. However, our estimates are robust to alternative modelling assumptions for the error term; for example, our reduced form results remain unchanged if we employ Conley standard errors to adjust for geospatial correlation (online Appendix Table A1) and our IV results look nearly identical if we cluster by both week and municipality (online Appendix Table A2). Second, all regressions are weighted by the number of births in the respective cohort

[10] Ozone Formation, EPA, http://www.epa.gov/oar/oaqps/gooduphigh/bad.html#6.

[11] We have experimented with different ways to model the instrument. For example, interacting inversions with municipality to allow for differential effects across municipality yields very similar results.

[12] Note that as we exploit week-to-week variation within municipalities in this setting, sorting across different municipalities due to differential pollution should not be a large concern. Moreover, Hanna and Oliva (2015) show that sorting is not a large concern within Mexico City as very few households move across census blocks, which are an even smaller geographic unit than municipalities.

[13] As we illustrate below, our results are robust to different configurations of the control variables, such as omitting controls for minimum and maximum temperatures during the week, omitting municipality-specific time trends and including different types of seasonal effects.

[14] In addition, including precipitation, cloud cover and humidity is also essential as it is possible that an inversion can lead to a thunderstorm if moisture is trapped in the inversion.

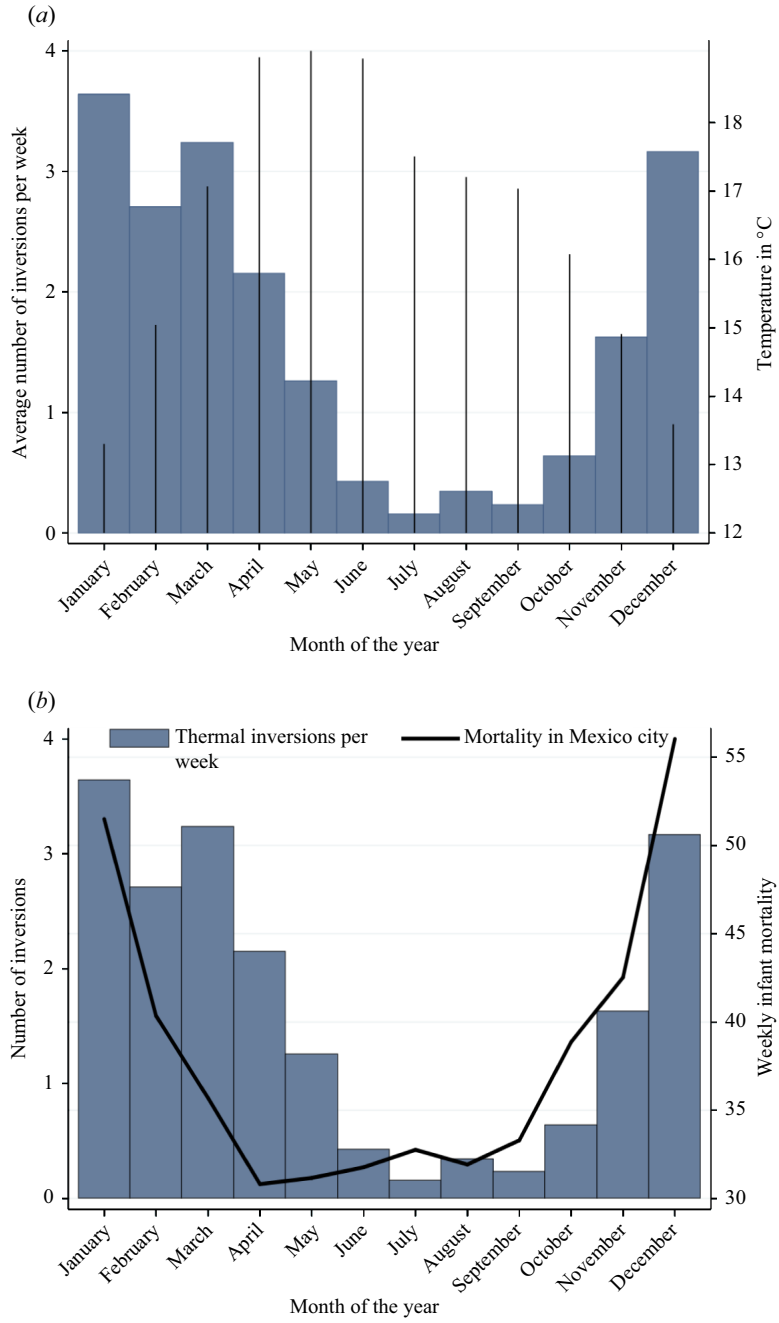

Fig. 2. *Thermal Inversions, Temperatures and Infant Mortality, by Month of the Year. (a) Inversions and Temperature. (b) Inversions and Infant Mortality*

Notes. Panel (*a*) of this Figure compares the average number of inversions per week (bars) with the monthly average temperature in Celsius (spikes) for each month of the year. Panel (*b*) compares the average number of inversions per week (bars) against the infant mortality rate in Mexico City (line) for each month of the year.

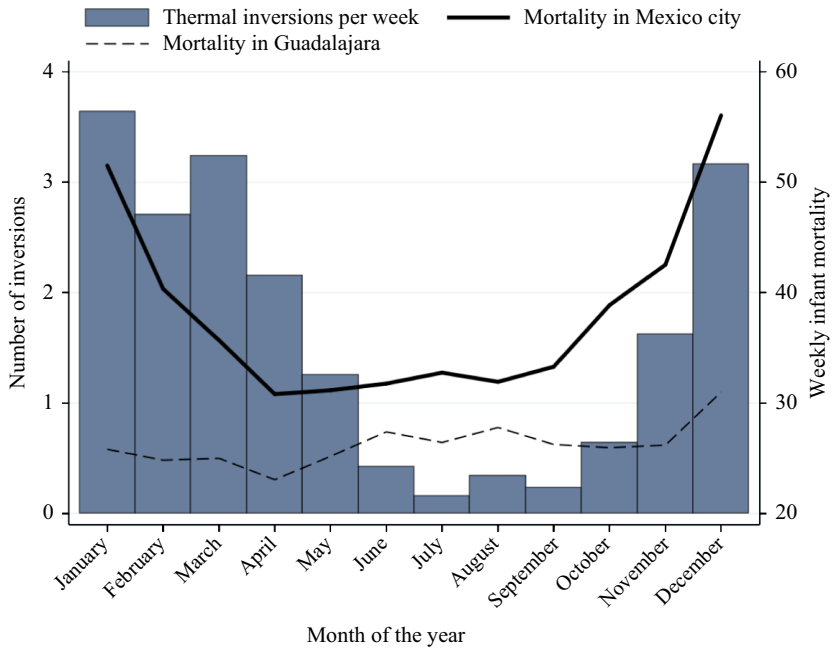

Fig. 3. *Comparing Mexico City and Guadalajara*

Notes. This Figure compares the average number of inversions per week (bars) against the infant mortality rate in Mexico City (bold line) and the infant mortality rate in Guadalajara (dashed line) for each month of the year. Guadalajara's infant mortality rate appears to be lower and nearly constant across the different months of the year, while Mexico's City infant mortality appears to have strong seasonal patterns that coincide with thermal inversion patterns. Thermal inversions are absent in Guadalajara.

(online Appendix Table A1 also shows that the results are not sensitive to these weights). Third, we can also try to disentangle the effects of different pollutants on infant health by taking advantage of the fact that the inversion effect may vary based on the geographical features of a location, such as its altitude. Specifically, this will allow us to create multiple instruments for the pollution variables.

Finally, we can also estimate models that control for mortality in the second largest city of Mexico, Guadalajara, which shares similar weather patterns to Mexico City but does not experience inversions. As Figure 3 illustrates, Guadalajara experiences similar seasonal patterns in mortality to Mexico City. This provides us with an additional method to control for seasonal patterns in mortality that may be due to weather or seasons.

1.3. *Data*

We compiled a comprehensive data set on pollution measures, weather conditions and mortality for Mexico City for the years 1997–2006. Each data source is described in detail below.

1.3.1. *Neonatal and infant mortality*

We constructed the mortality measures from data from the Ministry of Health (Secretaría de Salud Pública). These data include mortality measures for both the Mexico City Metropolitan Area (MCMA) and Guadalajara. We utilise two sources. First, we compiled data from death certificates, including information on day of death, gender of the child, municipality of residence, age of the child at death and cause of death. Second, to compute mortality rates, we gained access to the birth certificate registry, which contains information on date of birth and municipality of residence.

We then computed weekly, municipality-level neonatal mortality rates (those that are 28 days of age and younger) and infant mortality rates (those that are one year old and younger). To do so, for each week-municipality observation, we calculate the number of births in the last 28 days and in the last year.[15] Mortality rates are then calculated by dividing the total number of deaths in each week-municipality by the total number of live births in the corresponding age group and then multiplying by 100,000. Thus, our coefficients can be interpreted as the number of deaths in a week per 100,000 children born alive in the respective age cohort.

1.3.2. *Pollution*

Pollution data are notoriously absent in many developing countries. When available, they are often only cross-sectional, or of mixed quality. In this article, we are able to take advantage of a relatively rich, panel data set that is available for Mexico City, namely the Automatic Network of Atmospheric Monitoring (RAMA). Measures are available for particulate matter under 10 micrometres (PM_{10}), sulphur dioxide (SO_2), carbon monoxide (CO) and ozone (O_3). These data are considered to be of high quality and, as Davis (2008) points out, '[t]hese measures are widely used in scientific publications' (p. 41). However, it is important to note that they are drawn from relatively few stations: PM_{10} is available for 10 stations from 1997 to 1999, and from 16 stations starting in 2000, SO_2 is drawn from 26 stations, CO is drawn from 24 stations and O_3 is drawn from 21 stations.

From these data, we construct weekly measures of pollution for each of the 56 municipalities in Mexico City using the inverse of the distance to nearby stations as weights (see Currie and Neidell, (2005) for description of the methodology). Out of the 56 municipalities in Mexico City for which we have infant mortality data, we include the 48 that are within 15 kilometres of a station. There is a trade-off between constraining the sample to municipalities that are even closer to at least one station for greater precision of the pollution measure and increasing the distance cut-off to include more municipalities. As shown in online Appendix Table A3, the key infant mortality results are fairly robust to alternative definitions of this cutoff.

We use the hourly measures of pollution to calculate the maximum daily 8-hour average for CO and average this over the week, the maximum daily 24-hour average for PM_{10} and average this over the week, and then weekly averages for SO_2 and for O_3.[16]

[15] Since 0.03% of the births certificates have missing month and day of birth, we adjust weekly estimates of births by dividing un-dated births equally among all weeks of the year.

[16] We use these measures for ease of comparison with Currie and Neidell (2005) and Knittel *et al.* (2011). However, our results are robust to alternative measures of pollution.

1.3.3. *Thermal inversions*

Thermal inversions are recorded by the Meteorological Unit of the local Ministry of Environment. They conduct screenings almost every day, which consist of measuring hourly temperatures at different altitudes using an aerostatic balloon.[17] The existence of an inversion is determined upon finding non-monotonic temperature gradients. Records are kept on the time and temperature of the inversion rupture, so that one can also compute the number of hours an inversion lasted, as well as the thickness of its layer. We aggregate the data to the weekly level by computing the number of thermal inversions in a given week.

1.3.4. *Temperature and weather*

We obtained temperature and weather variables as additional controls in our specification. Hourly temperature measures are available from 24 stations in the RAMA network, and daily level measures of humidity, precipitation and cloud measures are available daily from 219 local weather stations. Using the same methodology to compute weekly, municipality-level measures of pollution, we use this information to compute the temperature and weather controls.

1.4. *Data Description*

We describe the data in Table 1. In panels (*a*) and (*b*), we provide information on neonatal mortality rates (for children that are 28 days and younger) and infant mortality rates (those that are one year and younger) respectively. In addition to the means for the weekly measures used in the regression analysis (column (1)), we additionally include the mean across municipalities in a given year per 100,000 births for ease of comparison with the US Figures (column (4)).[18] Over the period of study, yearly mortality rates are more than double in Mexico City than in the US: the neonatal mortality rate is 1,183, while the US rate is 460. Similarly, the infant mortality rate in Mexico City was 1,986, while the comparable US Figure was 698.

Note that we also provide mortality estimates by cause of death. We can test whether the effect of pollution on mortality is driven by deaths that we expect to be related to pollution. We define this comparison in two ways. First, we can compare all internal deaths with all external deaths (i.e. accident, homicides). This is a very strict definition, in that it assumes that pollution affects all internal deaths, including, for example those from digestive diseases, which may or may not be affected by pollution. Moreover, there are relatively fewer deaths from external sources (61.37 per 100,000), with relatively less variation, and so we may not capture an effect with the same sample size due to power concerns.

[17] Of the 3,652 days within our sample period, we have data on whether an inversion occurred for 95% of these days. We drop the weeks in which we are missing inversion data.

[18] Note that the weekly measure for infant deaths in column (1) appears smaller than that of neonatal mortality, despite the fact that the latter also includes neonatal deaths. The difference in magnitudes is mainly due to scaling: neonatal mortality rates are computed by dividing the number of deaths occurred in a single week within the 28-day cohort by the number of live births corresponding to that cohort. Hence, the denominator for the neonatal mortality figure is necessarily smaller than the denominator for the infant mortality figure. Column (4) shows mortality rates on a yearly basis. This column is computed by multiplying column (1) by (4) (or 52/13) in the case of neonatal mortality and by 12 in the case of infant mortality.

Table 1

Sample Statistics

	Mean of deaths in a week/municipality per 100,000 births	Standard deviation	Observations	Mean of deaths in a year/municipality per 100,000 births
	(1)	(2)	(3)	(4)
Panel (a): Neonatal mortality rates (28 days and younger)				
All causes	295.83	520.49	24,691	1,183.34
Non-external causes	287.73	513.74	24,691	1,150.93
External causes	3.27	49.09	24,691	13.07
Respiratory causes	7.85	96.17	24,691	31.39
Non-respiratory causes	283.15	507.73	24,691	1,132.61
Panel (b): Infant mortality rates (one year and younger)				
All causes	38.21	56.93	24,691	1,986.82
Non-external causes	36.52	55.14	24,691	1,898.85
External causes	1.18	9.61	24,691	61.37
Respiratory causes	6.91	29.35	24,691	359.14
Non-respiratory causes	30.79	46.65	24,691	1,601.08
Panel (c): Pollution and thermal inversions				
Particulate matter 24-hour PM_{10}	66.94	23.85	18,017	
Carbon monoxide 8-hour avg (CO)	2,707.56	797.70	18,167	
Sulphur dioxide avg (SO_2)	13.30	5.21	18,173	
Ozone avg (O_3)	32.33	7.47	18,167	
Number of inversions in a week	1.68	1.88	18,538	
Number of inversions in a week, conditional on an inversion	2.77	1.68	11,257	

Notes. This Table provides descriptive statistics for the key variables in the regression analysis. Panel (*a*) provides information on neonatal mortality, while panel (*b*) provides information on infant mortality. Panel (*c*) reports information on each pollutant and the thermal inversions. External cause is defined as deaths from accidents and homicides; internal cause encompasses all causes not including accidents or homicides. Respiratory causes (RC) includes respiratory and cardiovascular disease, while non-respiratory includes digestive, congenital, accidents, homicides etc. Mortality data come from death certificates and were provided by Secretaría de Salud Pública. Pollution data come from the Sistema de Monitoreo Atmosferico de la Ciudad de Mexico (www.sma.df.gob.mx). Inversion data were provided by the Meteorological Unit at the Secretaría de Medio Ambiente.

Therefore, we also compare diseases that are more likely to be directly attributed to pollution (i.e. respiratory and cardiovascular disease) *versus* those that are less likely to be directly attributed to pollution (digestive, congenital, accidents, homicides etc.).

In panel (*c*), we provide means for particulate matter of 10 μm or less (PM_{10}), carbon monoxide (CO), ozone (O_3), and sulphur dioxide (SO_2), as well as summary information on inversions.[19] Despite falling pollution levels in Mexico City, the average is still quite high. For example, the mean level of PM_{10} is about 67 μm as compared to 39.45 μm observed in California, as documented by Currie and Neidell (2005). Inversions are fairly frequent: on average, there are 1.7 inversions in a municipality-week. Conditional on an inversion occurring that week, there is an average of 2.77 inversions in a municipality-week.

[19] Given the presence of outliers in the data, we trim the top and bottom 1% of values. The primary results remain largely unchanged if we do not trim the outliers as shown in columns (5) and (6) of online Appendix Table A6.

Finally, we describe the evolution of pollution and infant mortality over time. In Figure 4, we graph average weekly neonatal (panel (*a*)) and infant mortality rates (panel (*b*)) against each of the four air pollutants over time. Mexico City has been successful in reducing pollution city-wide over the 1997–2006 time period, with all four air pollutants

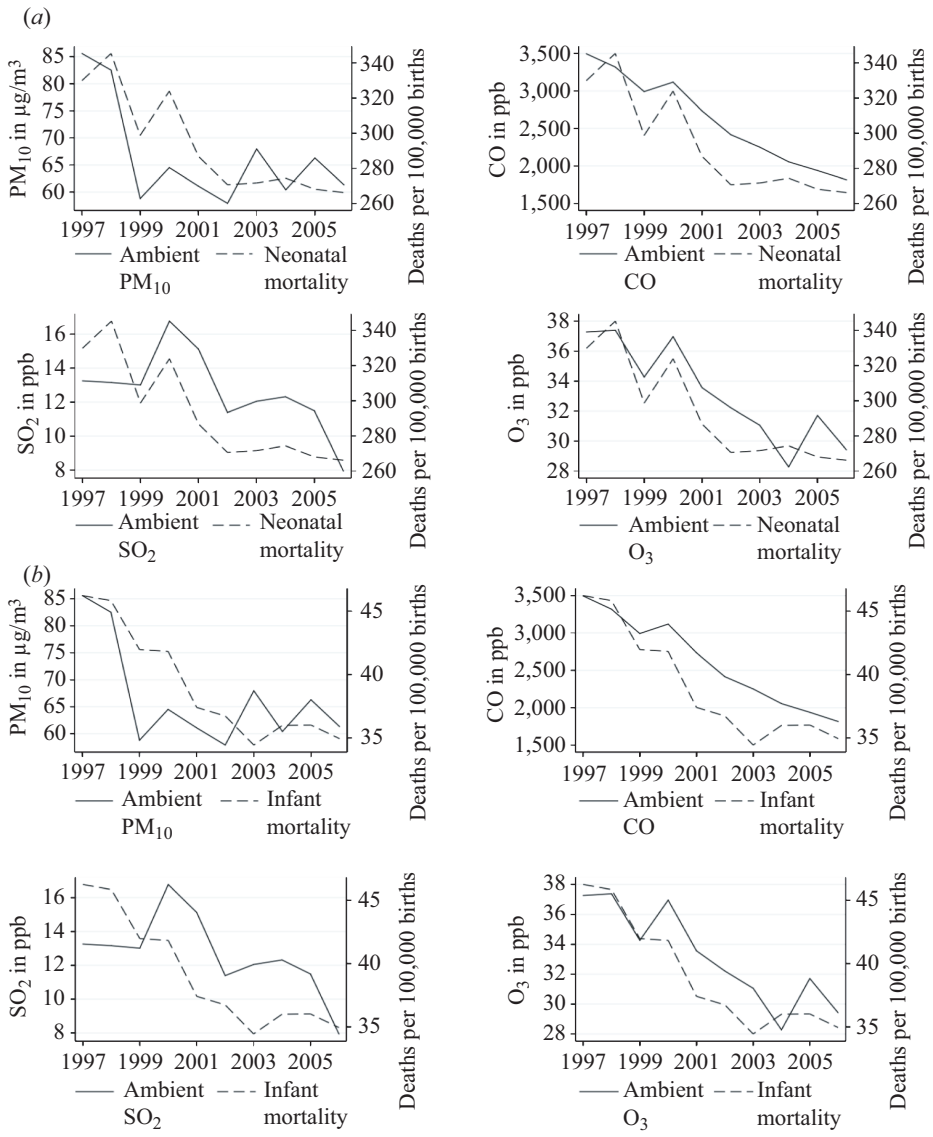

Fig. 4. *Mortality and Pollution Trends in MCMA. (a) Pollution and Neonatal Mortality. (b) Pollution and Infant Mortality*

Notes. This Figure plots the average annual pollution concentrations over time for the maximum daily 24-hour average of PM_{10}, the maximum daily 8-hour average of CO, the average concentration of SO_2 and the average concentration of O_3 (solid lines). It also plots average weekly neonatal (panel (*a*)) and infant mortality rates (panel (*b*)) for Mexico City (dashed lines).

falling sharply over this period (Molina and Molina, 2002). As the Figures illustrate, mortality rates are also falling and, in some cases, the rates closely track pollution changes.

2. Results

2.1. *First Stage Estimates*

We begin by examining the relationship between the occurrence of an inversion and each of the four pollutants (PM_{10}, CO, O_3 and SO_2), which comprises the first stage of our instrumental variables strategy. In Figure 5, we graph the average pollutant level by number of inversions. As the Figure illustrates, we observe a strong, and fairly linear, relationship between the number of inversions in the last week and PM_{10} and CO levels. In contrast, there does not appear to be an obvious relationship between the number of inversions and either O_3 or SO_2.

We provide the corresponding regression analysis in Table 2. Specifically, we present coefficient estimates from (3). As suggested by the Figure, inversions have a large and significant effect on PM_{10} and CO. One additional inversion in the last week results in a 3 $\mu g/m^3$, or 3.4%, increase in PM_{10} (column (1)). Similarly, one additional inversion

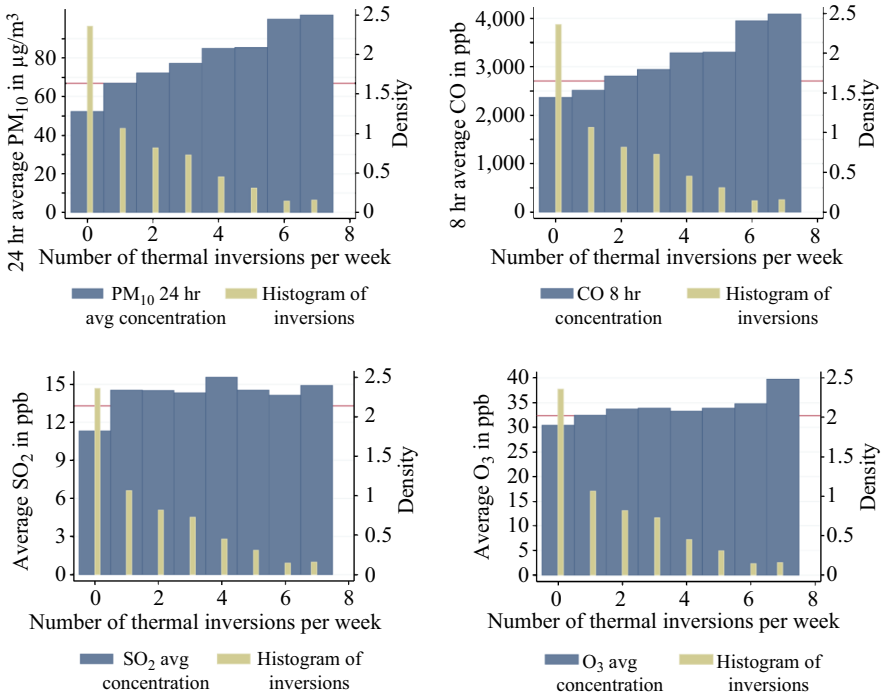

Fig. 5. *The Relationship Between the Number of Thermal Inversion Per Week and Pollution*
Notes. This figure plots average pollutant concentrations (maximum daily 24-hour average of PM10, the maximum daily 8-hour average of CO, the average concentration of SO_2 and the average concentration of O_3) by the number of thermal inversions per week (thick bars) and frequency of thermal inversions per week (thin bars). The horizontal line corresponds to the average concentration of the corresponding pollutant in the period of study.

Table 2

The Effect of Thermal Inversions on Pollution (First Stage)

	PM_{10}	CO 8-hour	SO_2	O_3
	(1)	(2)	(3)	(4)
Inversions	3.311***	169.884***	−0.257*	0.424**
	(0.503)	(13.451)	(0.145)	(0.172)
F test	43.37	159.5	3.146	6.060
Mean of outcome variable	57.67	2,707.56	13.30	32.33
Two-month of year × municipality FE	X	X	X	X
Municipality fixed effects	X	X	X	X
Weather controls	X	X	X	X
Municipality-week trends	X	X	X	X
N	18,017	18,167	18,173	18,167

Notes. This table provides the coefficient estimates of the effect of the number of thermal inversions per week on pollution concentrations, controlling for two-month-of-the-year by municipality fixed effects, municipality fixed effects, municipality-specific week trends, a fourth degree polynomial in average temperature during the week, a third degree polynomial in maximum and minimum temperatures during the week, a second degree polynomial in precipitation and cloud and humidity measures. Standard errors (listed below each estimate in parenthesis) are clustered at the week level. Statistical significance is denoted by: ***$p < 0.01$, **$p < 0.05$, *$p < 0.10$.

results in a 170 ppb, or 5.6%, increase in CO (column (2)). The effects on PM_{10} and CO are both significant at the 1% level and both would pass a weak instruments test. We find less precisely estimated effects of thermal inversions on SO_2 and O_3 (columns (3) and (4)); with F-statistics of 3.146 and 6.060 respectively neither of which pass a weak instruments test. These overall findings are consistent with the theoretical predictions discussed in Section 1 that we would expect the largest effects on PM_{10} and CO.

2.2. *Causal Estimates of Pollution on Infant Mortality and Health*

In columns (1) and (2) of Table 3, we provide the coefficient estimates of the effect of each pollutant on neonatal and infant mortality respectively from estimating (2) (the fixed effects model). Note that we additionally include municipality-specific week trends in this model.[20] In columns (3) and (4), we provide our IV estimates for the individual effects of PM_{10} and CO on mortality; here, we report the coefficient estimates from (4).[21] As there is no first stage result for SO_2 or O_3, we do not estimate an IV estimate for these pollutants.

[20] As online Appendix Table A4 shows, the results are qualitatively smaller if we drop the municipality-week trends (columns (1) and (2)) and look similar if include fewer temperature controls (columns (3) and (4)). We find much smaller effects with Currie and Neidell (2005)'s specification (columns (5) and (6)).

[21] Online Appendix Table A5 explores the robustness of the IV estimates to different control variables. As shown in columns (1) and (2), the effects on neonatal and infant mortality are slightly larger in magnitude if we do not include the municipality-week trends. Relaxing the temperature controls (columns (3) and (4)) also leads to slightly larger estimates for infant mortality but the effects are still not significant for neonatal mortality. Including a more flexible time trend (columns (5) and (6)) yields similar results.

Table 3

The Effect of Pollution on Infant Mortality

	Fixed effects		Instrumental variables		IV with mortality in GDL and MTY as controls	
	Neonatal	Infant	Neonatal	Infant	Neonatal	Infant
	(1)	(2)	(3)	(4)	(5)	(6)
Particulate matter 24-hour avg (PM_{10}) in $\mu g/m^3$	0.3595*	0.0649***	0.7625	0.2313***	0.6817	0.2033**
	(0.2063)	(0.0234)	(0.6410)	(0.0821)	(0.6110)	(0.0792)
Carbon monoxide 8-hour avg. (CO) in parts per billion (ppb)	0.0040	0.0014**	0.0162	0.0046***	0.0142	0.0040***
	(0.0060)	(0.0007)	(0.0135)	(0.0016)	(0.0128)	(0.0015)
Sulphur dioxide avg (SO_2) in parts per billion (ppb)	−0.8158	−0.1231*				
	(0.5857)	(0.0742)				
Ozone avg (O_3) in parts per billion (ppb)	0.3174	0.0176				
	(0.5612)	(0.0667)				
Mean of outcome variables	295.83	38.21	295.83	38.21	295.83	38.21

Notes. This Table presents fixed effects and instrumental variable estimates of the effect of pollution on infant mortality. Each co-efficient corresponds to a separate regression. All specifications control for 2-month-of-the-year by municipality fixed effects, municipality fixed effects, municipality-specific week trends and weather controls. In the instrumental variables estimation, the number of thermal inversions per week is the excluded instrument. Weather controls are a fourth degree polynomial in average temperature during the week, a third degree polynomial in maximum and minimum temperatures during the week, a second degree polynomial in precipitation, and cloud and humidity measures. Standard errors (listed below each estimate in parenthesis) are clustered at the week level. Statistical significance is denoted by: ***$p < 0.01$, **$p < 0.05$, *$p < 0.10$.

Using a fixed effects strategy, we find an effect of CO and PM_{10} on mortality (columns (1) and (2)). As compared to a pure cross-sectional analysis, the fixed effects estimates, for the most part, tend to be smaller in magnitude, which is consistent with classical measurement error.

Instead, we turn to our instrumental variables strategy. Here, we find large effects of pollution on infant mortality but smaller and non-significant effects on neonatal mortality. In the case of infant mortality a 1 $\mu g/m^3$ increase in PM_{10} over the week leads to 0.23 deaths per 100,000 births, while a 1 ppb increase in CO leads to 0.0046 deaths.[22] This implies that a 1% increase in PM_{10} over a year leads to a 0.40% increase in infant mortality, while a 1% increase in CO results in a 0.33% increase. These findings are consistent with the fact that deaths by respiratory diseases, which are likely to be affected by pollution exposure, are a higher share of total deaths for infants than for those under 28 days and may reflect underlying differences in ambient air exposure (i.e. infants may be more likely to be taken outside during the day than newborns).

Next, we explore whether any residual seasonal variation – that is after controlling for bimonthly × municipality fixed effects, temperature and weather – is driving our results. First, we can control for mortality in the second and third largest cities of Mexico, Guadalajara and Monterrey, which share similar weather patterns as Mexico City but do not experience inversions. Including the mortality rate in Guadalajara and Monterrey as controls (columns (5) and (6) of Table 3) does not qualitatively affect the results. Second, we can confirm whether the effect of pollution is similar across seasons, particularly whether it differs across the winter and summer months. Online Appendix Table A6 shows these results.[23] Note that we observe a significant first stage for both the summer and winter months.[24] On net, the effects on infant mortality do not appear to be significantly different across the winter and summer months. Thus, taken together, we believe that seasonality does not drive our findings.[25]

Another concern is that models that estimate the effect of pollution on infant death within a short time frame, such as week, overstate the effect. This bias would occur if pollution simply accelerates infant deaths by a short time period rather than causing additional deaths ('harvesting'). We do not find evidence of this. We can see this in two ways. First, the effect on neonatal deaths is much smaller in magnitude than infant

[22] Note that Table 3 reports effects of CO in terms of neonatal/infant deaths associated with one part per billion, which corresponds to an increase in CO concentration of 0.037%.

[23] For completeness, we show two possible definitions of seasons. Specifically, we can define summer from week 13 to week 42 (columns (1) and (2)) or from week 14 to 43 (columns (3) and (4)). The results are qualitatively similar across the two definitions.

[24] The Angrist-Pischke F-statistics are above the Stock-Yogo 10% threshold for weak instruments.

[25] Online Appendix Table A7 explores the effects of non-linearity. Using information on the mean and variance from Currie and Neidell (2005), we define a spline at one standard deviation above their mean (39.45 $\mu g/m^3$ for PM_{10}, and 1998 ppb for CO). This results in two endogenous variables per regression, so we use indicator variables denoting 1, 2–3 and 4–7 inversions per week as the instrument set. We find suggestive evidence of non-linearities in the CO effect (Table A7, panel (*b*)). Importantly, the Angrist-Pischke F-statistics suggest we can separately identify variation in CO above and beyond the 3,167 ppb threshold by breaking thermal inversions into indicator variables denoting 1, 2–3 and 4–7 inversions per week. We then find that the marginal effect of CO is close to zero when CO concentrations are below 3,167 ppb and 0.0092 when concentrations are above this threshold (significant at the 5% level). However, we cannot reject that the coefficients are the same at conventional levels (the p-value for the test of equality of the slopes is 0.256).

deaths. Due to the scaling, we need to multiply the infant mortality estimates to compare the neonatal and infant mortality estimates. When doing this, it becomes apparent that the effects on neonatal mortality are not only insignificant but also 50% smaller in magnitude. The fact that we find smaller effects of air pollution on children below the age of 28 days suggests that air pollution is not accelerating the death of already vulnerable children but is causing deaths of children with otherwise long life expectancies. Second, in online Appendix Tables A8 and A9, we present the results of our analysis when we aggregate the data to the month level rather than at the week level. If harvesting is an issue of concern with our estimates, we would expect much smaller effects when using data aggregated at the month level. However, this is not the case.[26]

It is important to note that our IV results are very robust to changes in the model specification, providing a high level of confidence in this strategy. In online Appendix Table A10, we first test whether our results are driven by changes in the denominator of the left-hand side variable, that is, the number of births. This would be the case if pollution shocks have an impact on the number of live births in the current week, which are included in the denominator. We perform this check by estimating our IV model with the log of deaths as a dependent variable and the log of births as an additional control variable. The results of this specification, reported in columns (1) and (2), can be compared with our main results divided by the average mortality rate. We find that the results of the log specification for infant mortality are slightly smaller but not significantly different than our main estimates, and they are still significant at the 5% level (the effects on neonatal remains insignificant). Thus, it is unlikely that the results are driven by changes in births.[27]

In Table 4, we replicate the IV analysis for mortality that results from different causes. This can be viewed as a placebo test: if we find that our pollution measure is resulting in deaths that are unlikely to be related to pollution, we would conclude that our instrument might be directly linked to unobserved socio-demographic determinants of mortality. We can define pollutant-related deaths in two ways. First, we compare deaths from all types of internal sources and those from purely external sources. We find no observable effect on either internal or external effects for neonatal (columns (1) and (2)). The effect of pollution on infant mortality appears to be driven by internal deaths (column (6)); there is no observable effect on external deaths for infants. However, mortality from external sources is rare compared to that from internal sources, meaning it would be more difficult to detect an effect on external deaths if there indeed is one. Moreover, it is possible that external deaths are simply

[26] To make the magnitudes of infant mortality coefficients comparable across online Appendix Table A9 and our main results (Table 3), it is useful to compute the yearly mortality effects associated with both (i.e. multiplying coefficients in Table 3 (column (2)) by 52 and the coefficients in online Appendix Table A9 (column (2)) by 12). The coefficients from monthly aggregated data appear about three times as large as the coefficients from weekly aggregated data. Note, however, that only the coefficients from the CO IV regression are reliable since the first stage for PM_{10} is lost when aggregating data at such a coarse level (see online Appendix Table A8).

[27] Note that in columns (3) and (4) of online Appendix Table A12, we also show the estimates had we not dropped the top and bottom 1% of values in pollution and show that the results are not sensitive to their inclusion.

Table 4

IV Effect of Pollution on Mortality, by Cause of Death

	Neonatal				Infant			
	External	Internal	Non-RC	RC	External	Internal	Non-RC	RC
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Particulate matter	−0.0283	0.4263	−0.0448	0.4428***	0.0105	0.1902***	0.0773	0.1235***
24-hour avg (PM_{10})	(0.0541)	(0.4742)	(0.4521)	(0.0967)	(0.0088)	(0.0701)	(0.0486)	(0.0341)
Carbon monoxide	−0.0008	0.0085	−0.0016	0.0093***	0.0002	0.0037***	0.0014	0.0025***
8-hour avg (CO)	(0.0011)	(0.0102)	(0.0096)	(0.0021)	(0.0002)	(0.0014)	(0.0010)	(0.0007)
Mean of outcome variable	3.27	287.73	283.15	7.85	1.18	36.52	30.79	6.91

Notes. This Table presents the coefficient estimates from the IV estimation of each pollutant on mortality, by cause of death. Each coefficient corresponds to a separate regression. External cause is defined as deaths from accidents and homicides; internal cause encompasses all causes not including accidents or homicides. Respiratory causes (RC) include respiratory and cardiovascular disease, while non-respiratory (Non-RC) includes digestive, congenital, accidents, homicides etc. All regressions control for two-month-of-the-year by municipality fixed effects, municipality fixed effects, municipality-week trends, a fourth degree polynomial in average temperature during the week, a third degree polynomial in maximum and minimum temperatures during the week, a second degree polynomial in precipitation and cloud and humidity measures. The number of thermal inversions per week is the excluded instrument. Standard errors (listed below each estimate in parenthesis) are clustered at the week level. Statistical significance is denoted by: ***$p < 0.01$, **$p < 0.05$, *$p < 0.10$.

accidental and/or immediate, and thus are altogether uncorrelated with income or health care quality.

Therefore, we can also classify deaths by those that are more likely to be attributed to pollution (i.e. respiratory and cardiovascular disease) *versus* those from sources that are less likely (digestive, congenital, accidents, homicides etc.). This is not a perfect separation, as children who are weakened by high pollution may be more likely to pass away from other sources (such as digestive disorders) and causes of death may be imperfectly diagnosed. However, we should still expect the effect on respiratory diseases to be relatively large if pollution is driving much of the effect, and not other socio-demographic characteristics. Our results suggest that most of the deaths related to pollution are linked to respiratory and cardiovascular causes: we find no effect on non-respiratory deaths for either neonatal (column (3)) or infant (column (7)) deaths. Importantly, if we focus on deaths from respiratory and cardiovascular disease, we find large effects of pollution on both infant (column (8)) and neonatal (column (4)) deaths. The fact that we find that the effect of pollution on mortality is driven mainly by respiratory causes implies that our instrument is capturing exogenous variation in pollution and not just trends in socio-economic characteristics.

Finally, in all the IV specifications above, the effect for any individual pollutant may be capturing its own effect, as well as the effect of the other pollutant affected by thermal inversions (either PM_{10} or CO). This is particularly problematic given that we have used one main instrument to identify the effect of each pollutant. In order to account properly for the total amount of deaths attributed to pollution fluctuations, we adopt two different approaches. First, we include all pollutants affected by thermal inversions in the same specification, so that the estimated effect of each pollutant is purged of potential bias from the others. This results in multiple endogenous variables and, therefore, we need to identify multiple instruments in order to estimate the causal effect of each pollutant, conditional on the others. Second, we construct a pollution index using the principal components method in order to generate a single endogenous variable that captures information on fluctuations of both PM_{10} and CO.

To generate multiple instruments, we exploit the fact that the effect of the inversions on different pollutants may differ based on the altitude of the municipality and the thickness of the inversion. Based on this, we create three instruments: the number of inversions, the number of inversions interacted with altitude and the thickness of inversion interacted with altitude. As online Appendix Table A11 illustrates, PM_{10} concentrations are lower at higher altitudes, presumably because higher areas are likely to be above the thermal inversion layer. However, altitude does not seem to matter as much for CO concentrations, unless the thermal inversion layer is thick. Thickness of the thermal inversion induces higher concentrations of CO at higher altitudes. The Angrist-Pischke F-statistic for both estimated equations is above the Stock-Yogo 10% critical value for single endogenous regressors, which suggests that our instrument combination induces at least some independent variation in both pollutants.

The IV estimates of this multiple pollutant model are presented in panel (*a*) of Table 5; note that given that we have multiple instruments, we estimate the model using LIML. We do not observe a significant effect of either pollutant on neonatal

Table 5

Multiple Pollutant Models

	Neonatal	Infant	Change in pollution between 1997 and 2006	Change in infant death rate per year between 1997 and 2006
	(1)	(2)	(3)	(4)
Panel (a): Multipollutant model				
Particulate matter 24-hour avg (PM_{10})	−0.0598	0.1335	−21.87	−151.8
	(1.9501)	(0.2176)		
Carbon monoxide 8-hour avg (CO)	0.0185	0.0021	−1,581.73	−172.7
	(0.0410)	(0.0046)		
Chi-squared stat joint significance test	1.682	9.834		−324.5
p-value	0.431	0.00732		
Panel (b): Single pollution index				
Pollution index	12.5322	3.4382***	−1.72	−307.8
	(9.8438)	(1.1470)		

Notes. In panel (*a*), each column in this Table presents the results of a single specification that includes CO and PM_{10} simultaneously. We present the results of an IV estimation of the multivariate pollution model, where inversions, inversions × altitude and inversions × thickness are instruments for CO and PM_{10} (the first stage is reported in online Appendix Table A10). Coefficients can be interpreted as the effect of each pollutant, conditional on the other pollutant. In panel (*b*), we use the principal components method to construct an index of pollution. This index summarises information on both PM_{10} and CO. Because this model has a single endogenous variable, we use the number of thermal inversions per week as a single instrument. All regressions control for two-month-of-the-year by municipality fixed effects, municipality fixed effects, municipality-week trends, a fourth degree polynomial in average temperature during the week, a third degree polynomial in maximum and minimum temperatures during the week, a second degree polynomial in precipitation and cloud and humidity measures. Statistical significance is denoted by: ***$p < 0.01$, **$p < 0.05$, *$p < 0.10$. For ease of interpretation, in columns (3) and (4), we calculate the change in infant deaths per year that can be attributed to the change in pollution in Mexico City between 1997 and 2006 (assuming 282,000 births per year).

mortality, and the two pollutant variables are jointly insignificant (column (1)). While each pollutant is not an individually significant predictor for infant mortality, the two pollutants are jointly significant in predicting infant mortality at the 10% level (column (2)). In terms of magnitude, we find that given the overall decline in pollution in Mexico City from 1997 to 2006 (weighted by births), our estimates would predict a change in the infant mortality rate that is due to pollution of 325 deaths per 100,000 (columns (3) and (4)).

Panel (*b*) of Table 5 shows the IV results where a single pollution index appears as the unique endogenous variable. The units of the index can be interpreted in terms of standard deviations. For reference, the average value of the index is 1.24 in 1997 and −0.68 in 2006. Note that when we collapse the two pollutants into a single variable, the effect of pollution is statistically significant. This is not surprising given that the index overcomes the identification problem posed by the high collinearity between PM_{10} and CO (the correlation is 0.48). Note, that the drop in pollution measured by the units of the index results in a very similar reduction in deaths per year as the reduction implied by the separate measures of the two pollutants: 308 compared to 325 deaths per 100,000.

3. Discussion

One of the main goals of this study is to understand better whether pollution estimates derived from the US context are externally valid to the developing world. If we believe that there is a non-linearity in the relationship between pollution and infant mortality, or that the costs of avoidance behaviour differ between the two settings then estimates derived from US settings may not be valid in conducting cost–benefit analysis of environmental regulations in the developing world. As we discussed earlier, the direction of the bias is ambiguous and, therefore, it is hard to benchmark whether we would be over or under-estimating the benefits.

Thus, we compare our estimates to those from Chay and Greenstone (2003), Currie and Neidell (2005), Currie *et al.* (2009) and Knittel *et al.* (2011) in Table 6. Panel (*a*) reports our estimates, while panel (*b*) provides the comparable results for the papers in the US setting. For ease of interpretation, we provide the mean level of infant mortality (column (1)), the mean value of each pollutant (columns (2) and (5)), the point estimates (columns (3) and (6)) and the elasticity (columns (4) and (7)). Note several features regarding the Table. First, we use the estimates from the single pollutant models because our subset of pollutants differs from these papers. Even though the models are not fully comparable, we replicate this Table using the multiple pollutant models in online Appendix Table A12 for completeness. Second, note that Chay and Greenstone study total suspended particulates (TSP) and not PM_{10}. For comparability to our estimates, we follow Knittel *et al.* and convert the TSP estimates using the following formula: $PM_{10} = 0.55$ TSP. Third, Chay and Greenstone and Knittel *et al.* study internal deaths rather than all deaths, so we additionally report the estimates for internal deaths for our sample. Finally, we put all estimates at the year level for ease of comparison.

Table 6

Comparison with Literature in US Setting (Sample for One Year Olds)

		CO			Particulate matter		
	Infant mortality rate	Mean level	Effect size (year)	Elasticity	Mean level	Effect size (year)	Elasticity
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel (a): Mexico City data							
Infant mortality	1,986.82	2.71	239.2***	0.326	66.94	12.3188***	0.405
Infant mortality-internal	1,898.85	2.71	192.4***	0.274	66.94	9.2196***	0.348
Panel (b): Estimates from US setting							
Currie and Neidell (2005)	391	2.00	16.501**	0.084	39.45	0.013	0.001
Currie *et al.* (2009)	688	1.58	17.6***	0.040	29.60	−0.189	−0.008
Chay and Greenstone (2003)	1,179				35.33	9.47**	0.284
Knittel *et al.* (2011)	280	1.01	40.56	0.146	28.94	17.68**	1.827

Notes. In this Table, we compare our primary estimates with estimates derived from the US context. We convert Chay and Greenstone (2003) from total suspended particulars to particulate matter for ease of comparison. We also provide the elasticity of the infant mortality rate to pollution (columns (4) and (7)). Given that Currie *et al.* (2009) does not have a directly comparable group, we report their estimates for the zero to two week age group.

We find that a 1 ppm increase in CO over a year leads to 239.2 infant deaths per 100,000 births (panel (*a*)). This implies that a 1% increase in CO over the year leads to a 0.326% increase in the infant mortality rate. We find a much larger effect on the infant mortality rate than either Currie and Neidell (2005), Currie *et al.* (2009) or Knittel *et al.* (2011) (column (3)). The estimated elasticity using the Mexico City data is larger than Currie and Neidell, who find an elasticity of 0.084, or Currie *et al.*, who find an elasticity of 0.04. Our estimates are larger but not qualitatively different from Knittel *et al.* (0.146); however, it is important to note that Knittel *et al.* cannot statistically distinguish their estimate from zero.

While we again point out that we are cautious about our non-linear estimation, they suggest that the effect of particulates is linear. Thus, we expect that our estimates for PM_{10} should be similar to those from the US. Despite the fact that the overall level of particulates is roughly half (66.94 in Mexico City *versus* 35.33 in the US), both our point estimates for internal deaths and elasticity for PM_{10} are fairly similar to Chay and Greenstone (2003) (columns (6) and (7)). However, our estimates are much smaller than Knittel *et al.*, who find that a one unit increase in PM_{10} in the year leads to 17.68 deaths per 100,000 births, or that a 1% increase in PM_{10} results in a 1.82% increase in the infant mortality rate.

4. Conclusion

There is a growing concern about the effects of pollution on health in the developing world. Especially in their urban areas, high population densities and low quality health services are colliding with high levels of harmful pollutant concentrations. This article sheds light on the importance of air quality improvements in the effort to curtail mortality rates.

Using a novel instrumental variables strategy, we find statistically significant effects of pollution on infant mortality in Mexico City. Our estimates imply that a 1 ppb increase in CO over a week leads to a 0.0046 per 100,000 births increase in the infant mortality rate, while a 1 $\mu g/m^3$ increase in PM_{10} leads to a 0.23 per 100,000 births increase in their mortality rate. This implies that a 1% increase in PM_{10} over a year leads to a 0.40% increase in infant mortality, while a 1% increase in CO results in a 0.33% increase. Our results on CO are generally larger than those estimated with data from the US, while we find comparable results for PM_{10} despite the fact that pollution levels are more than double in Mexico City. Our findings illustrate that there may be differences in estimates for developed and developing countries, suggesting using estimates from the US setting in benefit–cost calculations may understate the benefits from greater environmental regulation in developing countries.

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Accepted: 7 December 2014

Additional Supporting Information may be found in the online version of this article:

Appendix A. Robustness checks.
Data S1.

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