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Why are Pollution Damages Lower in Developed Countries? Insights from High Income, High-Particulate Matter Hong Kong

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Abstract
Conventional wisdom suggests that pollution damages are high in less-developed countries because they are highly polluted. Using administrative data on the universe of births and deaths, we explore the morbidity and mortality effects of gestational particulate matter exposure in high-pollution yet highly-developed Hong Kong. The effects of particulates on birthweight are large. We estimate no effect of particulates on neonatal mortality. We interpret our stark mortality results in a comparative analysis of pollution-mortality relationships across well-known studies. We provide evidence that mortality damages may be high in less-developed countries because they are less developed, not because they are more polluted.

Key words: Particulate Matter, Marginal Damages, Infant Health
JEL Codes: Q53; Q56; I15

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1 Introduction

Between 4 and 9 million premature deaths are attributed to pollution each year, twice as many as those from war, all other forms of violence, HIV/AIDS, tuberculosis, and malaria combined (Ladrigan et al., 2018). Particulate matter air pollution is associated with especially high economic damages, having deleterious effects on infant mortality, life expectancy, physical health, mental health, and health costs (Dockery et al., 1993; Pope et al., 2002; Chay and Greenstone, 2003; Pope and Dockery, 2006; Bishop et al., 2019; Deryugina et al., 2019). Particulate matter has been shown to affect property values, criminal behavior, labor productivity, and educational attainment (Chay and Greenstone, 2005; Graff Zivin, J. and Neidell, M., 2012; Chang et al., 2016; Ebenstein et al., 2016; Herrnstadt et al., 2019).

The best available evidence suggests that marginal damages from pollutants like particulate matter are higher in developing countries (Bharadwaj and Eberhard, 2008; Jayachandran, 2009; Chen et al., 2013; Greenstone and Hanna, 2014; Arceo et al., 2016; Cesur et al., 2016; He et al., 2016; Barwick et al., 2018; Heft-Neal et al., 2018; Adhvaryu et al., Forthcoming; Chang et al., 2019; Heft-Neal et al., 2019). However, the mechanisms are not well understood. Existing scholarship has largely focused on establishing empirical regularities while remaining agnostic on the mechanisms. On the one hand, greater marginal damages in developing countries could be explained by higher levels of pollution. It is well-documented that current particulate concentrations are many times higher in urban areas of India, China, Pakistan, Ghana, etc. than in urban areas of North America and Europe (Currie and Vogl, 2013; Greenstone and Hanna, 2014). Under this logic, cost-effectively reducing marginal damages involves reducing pollution levels. On the other hand, higher marginal damages in developing countries may instead be due to the limited ability of exposed populations to manage the consequences of pollution exposure. If differences in marginal damages across populations are due to institutional and economic differences rather than differences in baseline pollution levels, then efforts to improve environmental quality by directly reducing pollution may have a limited impact. Following this logic, incremental investments in

population health and human capital, health care, and other infrastructure may be more cost-effective.

Here, we inform the debate over the channels explaining lower marginal pollution damages in developed countries by re-examining the relationship between particulate matter and infant health in Hong Kong. Hong Kong offers the unusual combination of both high pollution and high income. Hong Kong's particulate matter levels are close to those in mainland China, India, and Pakistan, and exceed levels in many other developing countries. Hong Kong's per capita income levels compare to the United States. The population has excellent baseline health and universal access to free high-quality health care. Life expectancy ranks in the top-10 worldwide.

We combine administrative data on the universe of births and deaths in Hong Kong between 2001 and 2014 with detailed location-specific daily particulate matter (PM_{10}) levels. We construct individual-level gestational exposure to PM_{10} using monitor-by-day concentrations. To identify the effects of endogenous pollution exposure on birthweight and neonatal mortality, we exploit plausibly exogenous variation in thermal inversions (Arceo et al., 2016; Chen et al., 2017, 2018). We also explore robustness to alternative sources of variation. We focus on birthweight because it is an important indicator of infant health that has been shown to have persistent effects on later life outcomes (Almond et al., 2005; Currie and Moretti, 2007; Almond et al., 2010; Bharadwaj et al., 2013; Isen et al., 2017). We focus on mortality because death is the dominant contributor to the benefits of pollution policy. We focus on neonatal mortality, in particular, because the vast majority of infant deaths arise within the first few weeks of birth, both in our data and more generally (WHO, 2019). We explore mortality and morbidity together in the same study because these outcomes may be differentially affected by the economic and policy environment.

We find that higher gestational particulate matter exposure is associated with significant reductions in birthweight yet has no effect on neonatal mortality.[1] Effects on birthweight

[1]Cheung et al. (2019) explore the effect of contemporaneous pollution exposure on mortality across the age distribution in Hong Kong.

are substantial – a 10 $\mu g/m^3$ increase in particulate matter is associated with an 76-gram reduction in birthweight. This is equivalent to the estimated effects of smoking 15 cigarettes per day during pregnancy (Currie et al., 2009). Despite very high levels of particulate matter, pollution effects on neonatal mortality are statistically insignificant and small both in absolute terms and relative to the existing literature. We fail to reject the null hypothesis that there is no effect of gestational pollution exposure on neonatal mortality in all specifications.

One interpretation of our morbidity results is that marginal pollution-birthweight relationships are not strongly affected by the economic and policy environment. Hong Kong is rich, with excellent healthcare and baseline health, and yet marginal changes in pollution have large effects on morbidity. By contrast, our mortality results suggest that marginal pollution-neonatal death effects may be moderated by the economic and policy environment. Despite very high pollution levels, the marginal effects of pollution on neonatal death are minimal, suggesting that Hong Kong's wealth, health, and institutions swamp marginal pollution-mortality damages. To explore this latter conjecture, we perform an interpretation exercise where we compare marginal pollution-mortality damages from several studies in the literature, including our own. We find that marginal mortality effects are sharply decreasing in GDP per capita and baseline health. As such, investments in economic development may be important for reducing pollution-induced infant deaths. Conventional wisdom suggests that pollution damages are high in less-developed countries because they are highly polluted. Here we provide suggestive evidence that mortality damages are high in less-developed countries because they are less developed.

We make three contributions. First, our unique context helps illuminate the potential mechanisms that drive differences in pollution-mortality relationships across contexts. Our results from high-income, high-pollution Hong Kong are novel on their own and as part of a broader cross-institutional exercise. Second, we provide early evidence on the effects of pollution on birthweight in a high-pollution setting. Existing pollution-morbidity studies focus on low-pollution settings in the developed world, largely due to data limitations in

other contexts. Third, the free, high quality healthcare system in Hong Kong affords access to excellent data for an entire population. Free universal healthcare offers compelling data on vulnerable populations, who may be especially served by the institutions that come with economic development.

2 Conceptual Framework

To fix ideas, following Hsiang et al. (2019) , we conceptualize marginal damages as a function of exposure, e, and a vector of attributes, x.[2] Attributes can be considered a measure of an individual's vulnerability to damages from pollution. Vulnerability is defined as the rate at which exposure to pollution generates damages given economic and environmental conditions. For example, population differences in access to health care, housing quality, baseline health, education, etc. could all lead to differences in the translation of exposure, e, into economic costs.

The key feature of this framework is that exposure is only converted into economic costs through a function that describes the vulnerability of an individual or population. Consequently, higher marginal damages in developing countries – as observed in the existing literature – may be attributable to higher e, different x's, or some combination.

Even in a stylized model there are competing explanations for the same empirical observation. For illustration, we describe the limiting cases. On the one hand, differences in marginal damages may be lower in developed countries because levels of pollution are lower. If the dose response function is non-linear with respect to exposure, e, then two populations facing different levels of pollution will experience different marginal damages, even if they are identical with regards to all other factors that could influence vulnerability, x:

$$\frac{\partial^2 Damages}{\partial e^2} = \frac{\partial^2 f(e, \mathrm{x})}{\partial e^2} > 0$$

[2]We do not claim new modeling contributions. This section simply serves to highlight potential mechanisms influencing marginal pollution damages.

On the other hand, differences in marginal damages may arise from differences in the factors that translate pollution exposure into marginal damages. For example, two populations may be exposed to the same level of pollution but have differential access to high quality health care, x_j. Assuming that access to high quality health care mitigates the marginal damages associated with pollution, populations with restricted access to health care will experience greater marginal damages, holding all else constant.

$$\frac{\partial^2 Damages}{\partial e \partial \mathrm{x}_j} = \frac{\partial^2 f(e, \mathrm{x}_j)}{\partial e \partial \mathrm{x}_j} < 0$$

3 Data

Administrative Birth and Death Records We obtain birth and death records between January 1st 2001 and December 31st 2014 from the Census and Statistics department of Hong Kong. The birth records data set provides detailed information on 942,687 births, including data on birth characteristics (e.g., date of birth, sex, type of birth, hospital, etc.), parental characteristics (e.g., mother's and father's age, occupation, education, etc.), as well as the location of the mother's residence at the Tertiary-Planning-Unit (TPU) level.[3] We retain all birth records from mothers who report Hong Kong as their residence. We retain observations with complete information on sex, birthweight, exact date of birth, and location of birth.[4]

Our mortality records data set includes date of death, age of death, and cause of death. For analysis we focus on the 1,058 neonatal deaths, deaths that occur in the first 28 days. 90% of infant deaths in our sample occur in the first 15 days of life. Moreover, age measured in days is not available beyond 28 days. For each neonatal death, we locate and match an individual birth record using the date of birth, date of death, age at death, sex, and TPU of residence. A practical challenge is that our datasets do not provide unique identifiers

[3]In 2011 there were 289 TPUs in Hong Kong and 7.072 million residents, resulting in an average population density of 24,470 people per TPU. The average area each TPU is 3.83km^2.

[4]Transient births represent roughly one-third of births. Most of these are babies born to parents from mainland China. Only 0.05% of birth records are dropped due to missing data.

across birth and death records. For cases in which there are multiple births and deaths within a given date of birth, date of death, sex and TPU cell, we have to use probabilistic matching. For example, if there are 2 deaths matched to 2 birth cells then a probability of 1 is assigned. However, if there is only 1 death matched to 2 birth cells then a probability of 0.5 is assigned. Probabilistic matching is common when working with administrative data when unique identifiers are not available (Wagner and Layne, 2014).[5]

Pollution Data We collect hourly pollution concentrations for several criteria pollutants from the Environmental Protection Department of Hong Kong. Our pollutant of interest, PM_{10}, includes all suspended solids and liquids that are 10 micrometers in diameter or less. The largest sources of PM_{10} in Hong Kong are boat traffic, road transport, and electricity generation. We focus on particulate matter (PM_{10}) due to its importance for human health and economic damages, its consistency in reporting over the sample period, and its comparability to the existing literature (Dockery et al., 1993; Pope et al., 2002; Chay and Greenstone, 2003, 2005; Pope and Dockery, 2006; Graff Zivin, J. and Neidell, M., 2012; Census and Statistics Department, 2014a,b; Chang et al., 2016; Ebenstein et al., 2016; Bishop et al., 2019; Deryugina et al., 2019; Herrnstadt et al., 2019).

We construct measures of individual-level PM_{10} exposure. First, we assign daily pollution levels to each TPU, interpolating the station-level data to population-weighted TPU-centroids using inverse distance weighting applied to all pollution monitors within a 10km buffer.[6] Second, we reconstruct individual level gestational exposure by mapping pollution at the TPU-level to each individual birth based on mother's TPU of residence and the child's

[5]Of the 1,058 neonatal deaths in the final sample, 331 are assigned a probability of 1. In the analysis data set the mean number of births per cell is 1.74, the median number of births is 1, and the maximum number of births is 12. As long as any measurement error is orthogonal to pollution exposure our estimate of the treatment effect will be unbiased and consistent. The impact on inference is harder to predict but likely small because the occurrence of infant deaths are rare in this population. An alternative approach is to construct a selected sample using unique birth-death pairs. Our results are not sensitive to using this sample (Table A10).

[6]If during gestation a mother lives further than 10km from a monitoring station, we do not construct a measure of pollution exposure for that pregnancy. Results are robust to using alternative distance radii (Table A7).

date-of-birth. We define gestational exposure to be the average of the daily mean of pollution exposure over the 270 days prior to the date-of-birth.[7]

Our data document high PM_{10} concentrations in Hong Kong between 2001 and 2014. The World Health Organization guidelines suggest that the annual average of PM_{10} should not exceed $20\mu g/m^3$/day. During our study period, average gestational exposure to PM_{10} levels in Hong Kong was $53\mu g/m^3$/day. The minimum gestational exposure to PM_{10} in our sample is $30\mu g/m^3$/day. The maximum is $94\mu g/m^3$/day.

Weather Data We collect weather data from two sources. First, we observe daily data on surface-level temperature, precipitation, humidity, and air pressure from 43 Hong Kong Observatory weather stations. Second, we collect data on air temperature at two atmospheric levels from the ERA-Interim Reanalysis archive. Reanalysis data combines observations from ground stations and remote-sensing products with global climate models to provide a consistent best estimate of atmospheric parameters over time and space (Auffhammer et al., 2013). The data are provided every 6 hours and are measured on a $0.12^\circ \times 0.12^\circ$ grid (13×13 km). We aggregate reanalysis data to the daily level. We construct measures of individual exposure to weather and thermal inversions using the same procedures applied to pollution. Our final weather data contain measures of temperature, precipitation, humidity, air pressure, and thermal inversion exposure.

Final Sample Combining all data sources, we obtain a final analysis sample of 532,726 births between 2001 and 2014. Each birth is matched to weather exposure, individual mortality information, and gestational pollution exposure. Appendix A considers contemporaneous exposure. The unit of observation is a birth.

[7]It is possible that, during a mother's gestation period, there are days without valid readings for some pollutants. In our analysis sample 96% of births have valid readings for all days, and 99.83% of births have valid readings for at least 266 days.

4 Research Design

Our objective is to estimate the effect of gestational pollution exposure, P_{ijt}, on birthweight and the probability of mortality and for individual i born in location j at time t. For birthweight and mortality outcomes, Y_{ijt},

$$Y_{ijt} = \alpha + \beta P_{ijt} + \epsilon_{ijt}. \tag{1}$$

To identify the effects of pollution on our outcome of interest, β, it must be the case that pollution is orthogonal to ϵ_{ijt}. The main threat to identification is omitted variable bias.

First, exposure to pollution and birth outcomes may be correlated via changes over time across years and seasonal variation within years. If birth outcomes are worse during the winter and gestational pollution exposure is also higher for babies born in the winter, then mortality estimates are biased upward and morbidity estimates are biased downward. Deterministic trends may also arise due to changes in economic activity. If PM_{10} is higher when economic activity is higher and birth outcomes are positively correlated with economic activity (conditional on pollution) then the pollution-mortality relationship will be biased upwards and the pollution-birthweight will be biased downward. Second, exposure to pollution and birth outcomes may be correlated with avoidance behavior. In the long run people choose where to live and so exposure to pollution and birth outcomes may be associated with residential sorting. In the short run, individual decision-making may affect exposure to pollution, e.g., staying indoors on high pollution days or wearing face masks. If information about, or opportunities to avoid, pollution damages are correlated with other health and human capital investments then the relationship between pollution exposure and birth outcomes may be confounded. Other omitted variable concersn are also possible.

One approach to address omitted variable bias is to use covariates and/or fixed effects. We control for differences in socio-economic characteristics, changes in economic conditions, equilibrium avoidance behaviors such as residential sorting, and seasonality. We estimate the

relationship between birth outcomes and pollution exposure using the following specification,

$$Y_{ijmt} = \alpha + \beta P_{ijmt} + \gamma X_{ijmt} + \delta_{jt} + \phi_{jm} + \epsilon_{ijmt}. \quad (2)$$

γ_{jt} is a vector of TPU-by-year fixed effects that control for time-invariant characteristics at the local level, addressing location-specific equilibrium endogenous exposure, such as residential sorting, as well as controlling for annual shocks that are common across everyone within a TPU, such as changes in economic conditions. We include TPU-by-month, ϕ_{jm}, fixed effects that control for TPU-specific seasonality in births, deaths, and disease transmission that may also be correlated with pollution exposure. X_{ijmt} includes surface gestational weather conditions, parental characteristics, and birth characteristics.

The approach in equation 2 addresses several threats to identification but cannot help us to address time-varying omitted variables like those associated with short-run individual-level avoidance behavior. It is likely that the population is aware of pollution levels (Moretti and Neidell, 2011; Chang et al., 2018; Barwick et al., 2019). Pollution forecasts are widely disseminated to the public during our sample period. In Appendix B we provide evidence that this is the case, showing that a 10 $\mu g/m^3$ increase in PM_{10} on the day of professional football (soccer) matches in Hong Kong is associated with a 17-20% reduction in attendance. Another concern is measurement error in the assignment of ambient pollution to individual level exposure.

To address these residual concerns we also employ instrumental variable strategies. The primary instrumental variable approach exploits a meteorological phenomenon known as a thermal inversion, which arises when a mass of hot air settles above a mass of cooler air. This instrument has been popularized in several papers by Paulina Oliva and co-authors (Arceo et al., 2016; Hicks et al., 2016; Chen et al., 2017, 2018). Under normal conditions air temperature in the troposphere (the lowest region of the atmosphere) falls with altitude at a rate of 6.5°C per 1,000 meters. Under these conditions emissions are released into the atmosphere, then naturally rise and dissipate. However, in the case of a thermal inversion air

temperature in the troposphere can rise with altitude. Under these circumstances a ceiling effect can trap pollution at the ground level. It is only after the sun's energy eventually equates the non-standard arrangement of hot and cold air masses, that the thermal inversion dissipates and the concentrated pollutants are able to rise out of the lower atmosphere as normal.

To construct our instrument for a given day and location, we codify the onset of a thermal inversion if the difference in average daily temperature between 111 meters and 766 meters is negative, i.e, $temp_{111} - temp_{766} < 0$. We then calculate the number of thermal inversions during the 270 days of gestation.

Exogeneity Thermal inversions are not caused by pollution or economic factors. They are more likely to occur on clear nights when the ground and the air in touch with the ground are cooled faster than the air layers above, resulting in a relative inversion (Arceo et al., 2016). As such, they are more frequent in winter – confirmed in our setting – as cold ground temperatures cause the air that is close to the ground to remain at a lower temperature than the air above ground that is warmed by earth's infrared emissions. To address the seasonality of thermal inversions we control for a vector of season fixed effects (month-of-year $\times$ TPU in our main specification). Conditional on seasonality controls, the incidence of thermal inversions is plausibly exogenous.

Relevance Other studies have shown that thermal inversions have substantial effects on particulate matter, which tends to be released during morning rush hours when inversions typically occur (Kukkonen et al., 2005; Malek et al., 2006; Arceo et al., 2016). In the context of Hong Kong, Lee and Hills (2003) study seven serious pollution episodes between 1996 and 2002, finding that daily average PM_{10} levels exceeding $150\mu g/m^3$ were all associated with the existence of thermal inversions.

We directly test the relevance of thermal inversions as a driver of pollution in Hong Kong. We regress gestational exposure, P_{ijmt}, on the instrument. We control for a vector

of individual level controls, including gestational exposure to surface weather conditions, a vector of TPU-by-year fixed effects, and TPU-by-month-of-year fixed effects:

$$P_{ijmt} = \alpha + \delta Inversion_{ijmt} + \gamma X_{ijmt} + \delta_{jt} + \phi_{jm} + \nu_{ijmt} \tag{3}$$

Consistent with previous studies we find that there is a strong correlation between the incidence of thermal inversions and gestational exposure to PM_{10}.[8] A one standard deviation increase in exposure to thermal inversions during gestation (an additional 2.3 inversions) is associated with a 0.506 $\mu g/m^3$ increase in average gestational particulate matter exposure. For context, the extreme case where an individual was exposed to a thermal inversion every day would more than double gestational exposure to particulate matter relative to the mean.

Exclusion Restriction A key assumption for identification is that thermal inversions only affect health outcomes through pollution. It is not possible to test this assumption directly. However, we argue that it is plausible. Thermal inversions do not present a direct health risk to individuals themselves. Consequently, after controlling for seasonality and surface-level weather conditions that could be correlated with thermal inversions, pollution, and health outcomes, we argue that the exclusion restriction is likely satisfied. One argument against this concern is that thermal inversions could directly affect short-run avoidance behavior if, on average, Hong Kong residents are aware of thermal inversions when they occur and are aware that thermal inversions trap pollution. Although we cannot rule out this possibility, it seems less likely. The existing literature argues that most people are unaware when thermal inversions occur and even fewer are aware that thermal inversions concentrate particulates

[8]Thermal inversions are unlikely to have an effect on secondary pollutants such as Ozone. Ozone requires time to form from the mixture of primary pollutants and so may only appear later in the day when it is likely that inversions have already dissipated (Jacobson, 2002). Arceo et al. (2016) further argue that inversions may directly inhibit the formation of these pollutants as the chemical reactions required to form them require warmth and sunlight. We also regress ozone and carbon monoxide on thermal inversions. We detect no correlation between gestational exposure to Ozone and thermal inversions as expected. We estimate a relationship between thermal inversions and carbon monoxide, consistent with the existing literature. A more detailed discussion of these results, alongside regression results, can be found in Appendix A.2.1.

(Arceo et al., 2016; Hicks et al., 2016; Chen et al., 2017, 2018). [9]

One final caveat is that when we evaluate the effects of thermal inversions on health through particulate matter (PM_{10}) we are assuming that thermal inversions only affect health through PM_{10} and not through other pollutants. This is a common issue associated with any instrumental variable that affects multiple pollutants. We argue, as others have done before us, that our identification strategy is sufficient to identify the effects of pollution on health, rather than a particular pollutant, and that this is sufficient for the exercise at hand (Chay and Greenstone, 2003; Currie and Neidell, 2005; Arceo et al., 2016; Knittel et al., 2016; Deryugina et al., 2019). Note also that results are robust to including, or omitting, controls for ozone and carbon monoxide (Appendix A.2.3).

Estimation Details Our preferred specification is equation 4,

$$Y_{ijmt} = \alpha + \beta \hat{P}_{ijmt} + \gamma X_{ijmt} + \delta_{jt} + \phi_{jm} + \varepsilon_{ijmt} \tag{4}$$

where $\hat{P}_{ijmt}$ is the two-stage least squares prediction of gestational exposure to pollution for individual i, in TPU j, born in month m of year t. We continue to control for TPU-by-year fixed effects and TPU-by-month-of-year fixed effects, as well as a vector of individual-level controls such as gestational exposure to surface weather conditions, parental characteristics, and birth characteristics.

For robustness, we also consider a second instrumental variable strategy that exploits variation in air pollution induced by port traffic. Following Moretti and Neidell (2011), we exploit variation in the log of net registered tonnage of marine traffic to Hong Kong in a

[9] During an October 2017 seminar presentation of this paper at Hong Kong University of Science and Technology (HKUST), we conducted an informal poll on the selected sample of seminar participants (economists, social scientists, and environmental scientists). We asked whether they 1) knew about thermal inversions (even by a different name) 2) knew thermal inversions were common during Hong Kong winters 3) knew thermal inversions had large effects on local pollution levels. Only one seminar participant expressed full awareness of these issues. While clearly this sample is not representative of the population and the poll was crudely implemented it suggests that even highly educated individuals living in Hong Kong are not necessarily aware of thermal inversions or their consequences.

month, interacted with the distance from port to each population-weighted TPU centroid. The relevance of this instrument is weaker than the thermal inversion instrument.

Across all specifications, standard errors are two-way clustered at the TPU-level to account for serial correlation over time and at the date-of-birth level to account for spatial dependence in pollution exposure across all births on a given day.[10]

5 Results

We present key results in Table 1. Columns (1) and (2) report the effects of gestational PM_{10} exposure on birthweight for OLS and IV specifications. Columns (3) and (4) present the effects of gestational PM_{10} exposure on neonatal death for OLS and IV specifications.

Birthweight We find large effects of gestational PM_{10} exposure on birthweight in a high-pollution context. Using our preferred specification from an instrumental variables approach we estimate that a 10 $\mu g/m^3$ increase in PM_{10} is associated with a 76 gram reduction in birthweight (Table 1, column (2)).[11] This effect is equivalent to Currie et al. (2009)'s estimated effects of smoking 15 cigarettes per day during gestation. This effect is larger than estimates in low-pollution settings. The existing literature offers few examinations of the pollution-birthweight relationship in high-pollution contexts due to data limitations, so the context and empirical magnitude are new to the literature.

Neonatal Mortality We provide early evidence that gestational PM_{10} exposure has limited effects on neonatal mortality in Hong Kong, despite high levels of pollution. For both OLS and IV specifications we estimate small and statistically insignificant relationship between gestational PM_{10} exposure and neonatal mortality. Point estimates from our preferred

[10] Results are unchanged if we only cluster along one of the dimensions. Standard errors are similar across all three combinations: one-way clustering by TPU, one-way clustering by date-of-birth, and two-way clustering by TPU and date-of-birth.

[11] In specifications without instruments we fail to reject a null of no relationship between gestational PM_{10} exposure and birthweight (Table 1, column (1)).

specification using an instrumental variable approach suggest that a 10 $\mu g/m^3$ increase in PM_{10} is associated with 0.02 additional deaths per 1,000 births. This marginal impact is among the smallest in the literature, including those from the United States (Knittel et al., 2016). We defer interpretation until the next section, although we reiterate here the novelty of evidence from a high-pollution setting with high average incomes and excellent health care.

We acknowledge that pollution-mortality relationships are not precisely estimated. Although our central estimates are extremely small our 95% confidence interval doesn't rule out meaningful increases in neonatal mortality. Nevertheless, our point estimates and upper confidence limit are small relative to the existing literature in high-pollution settings.[12] Moreover, we reject a null using the same data for the effects of PM_{10} on birthweight.

Additional Results and Robustness Tests In Appendix A we provide supporting evidence. Our results are robust to using an alternative instrument based on port traffic (Table A3). This instrument is weaker than the thermal inversion instrument, yet we estimate qualitatively similar results. Our results are also robust to estimating reduced form relationships between thermal inversions, birthweight and neonatal mortality (Table A8). We estimate a statistically significant effect of gestational thermal inversion exposure on birthweight and a statistically insignificant effect of gestational thermal inversion exposure on neonatal mortality. To address concerns that our estimates of particulate matter are confounded by other pollutants we show that our results are robust to controlling for carbon monoxide and ozone (Table A4). We also show our results are robust to alternative measures of particulate matter like $PM_{2.5}$ and average daily maximum exposure during gestation (Table A5 and Table A6). We also explore outcomes defined by specific causes of death (Table A9). We find no effects on any cause of death. We also confirm that our results are not driven by our specific choice of distance radii (Table A7).

[12]For example, our upper confidence limit is similar in magnitude to the central estimate in Arceo et al. (2016), Heft-Neal et al. (2018), Cesur et al. (2016) and smaller than the central estimate in Heft-Neal et al. (2019).

Finally, we do not find any effect of contemporaneous exposure to particulate matter on neonatal mortality (Table A11). We caution that we do not have a strong instrument largely due to limited opportunity for post-natal exposure. 50% of neonatal deaths occur within 2 days. 90% of neonatal deaths occur within 2 weeks.

6 Discussion and Conclusion

We find that marginal changes in pollution exposure in high-income, high-pollution Hong Kong are associated with large changes in birthweight but no change in neonatal mortality. The analyses and results are novel in two regards. First, we use high quality administrative data to provide early evidence on the effects of gestational PM_{10} exposure on morbidity in a high-pollution context. Morbidity estimates in our setting are considerably larger than existing evidence from lower pollution settings. Second, we fail to reject a null hypothesis that gestational PM_{10} exposure has no effect on neonatal mortality in a high-pollution yet high-income context. This finding contrasts sharply with existing evidence from high-pollution, low-income settings.

Extrapolating results in the context of the literature suggests two implications for economics and policy. With respect to birthweight, marginal damages may not be particularly moderated by the economic and policy environment. Hong Kong has free access to excellent health care and baseline health indicators are among the best in the world. Yet we find very large effects of particulate matter on birthweight in Hong Kong. Given the magnitude of the birthweight effects, our results that marginal morbidity damages are high when PM_{10} is high suggest that marginal morbidity damages are increasing with pollution. With respect to neonatal mortality, marginal pollution damages appear to be swamped by the economic and policy environment. Free access to high-quality health care, good economic conditions, and other factors that come with economic development appear to overwhelm any convexity in marginal mortality damages that might arise from exposure to higher levels of particulates.

A natural question is why the economic and policy environment might matter more for mortality and less for morbidity. One explanation is that the relationship between pollution and mortality, a severe and discrete outcome, can be more easily renegotiated through technology. Evidence from the clinical literature suggests that neonatal mortality is highly correlated with the quality of health care provision (through neonatal care units, obstetrics, and delivery room care) (Richardson et al., 1998; Horbar et al., 2001; Noble, 2003; te Pas, 2017; WHO, 2019). Less severe margins such as those associated with morbidity may remain sensitive to pollution. Anticipating and preventing reductions in birthweight is also difficult and costly prior to birth.[13]

To explore the broader relevance of our stark mortality results, we perform back-of-the-envelope calculations using internally valid estimates from several studies in the literature.[14] The literature to date has focused on maximizing internal validity and has established important empirical regularities. However, the constraints of micro-level identification impede cross-country and cross-institutional comparisons that may illuminate a broader understanding of economic phenomena (Oster et al., 2016; Meager, 2019). In this spirit, we interpret our own empirical results by comparing marginal mortality damage estimates from 7 prominent studies. These studies provide different population characteristics that allow us to explore the correlates of marginal mortality damages across contexts, providing insight into the drivers of heterogeneity. Figure 1 summarizes the results of our interpretation exercises. We find that marginal mortality damages are decreasing in GDP per capita and baseline health. For perspective, and to highlight the magnitude of economic and institutional differences across countries, we conduct an extreme non-marginal comparison. Holding levels of pollution constant, increasing GDP per capita in India ($1,939) to the level of the United States

[13] We do not measure the effects of PM_{10} on healthcare costs. Our finding of limited mortality effects may reflect large changes in healthcare spending. This represents an interesting area for future research. This reinforces the point that the economic and policy environment is important in affecting the translation of pollution exposure into damages.

[14] This exercise is essentially impossible to do for morbidity because there are fewer internally valid estimates of the pollution-morbidity relationship and all have been estimated in high-income, low-pollution settings. This reiterates the contribution of this study, providing morbidity estimates in a high-pollution setting.

($59,531) would be associated with 5.98 fewer deaths from pollution per 1,000 live births, a roughly one-quarter reduction in the total neonatal mortality rate.[15] By contrast, we find no relationship between average particulate matter concentrations and marginal mortality damages. This suggests that vulnerability to pollution exposure may be more important than pollution exposure itself in explaining differences in marginal mortality damages. We do not assert that pollution exposure does not matter but rather that marginal mortality damages may be linear (rather than increasing) in pollution exposure.

Combining insights from our birthweight and neonatal death analyses suggests that improving environmental health entails investments in both pollution abatement and economic development. Our morbidity results suggest that economic development alone is not sufficient. Reducing health effects such as low birthweight, which has been shown to have long-run economic consequences, requires investments in pollution abatement irrespective of a populations wealth and health. However, our mortality results suggest that pollution abatement alone is not sufficient. A population's wealth and health can dramatically reduce mortality from pollution. Conventional wisdom suggests that pollution damages are high in less-developed countries because they are highly polluted. We provide early evidence that pollution damages are high in less-developed countries because they are more polluted and less developed. With respect to mortality, we provide suggestive evidence that damages are high in less-developed countries because the are less developed.

[15]The estimated change in mortality is the predicted effect from a multivariate regression of marginal mortality damages on GDP per capita, the baseline neonatal mortality rate, and average particulate matter concentrations, multiplied by the difference in GDP per capita between India and the United States.

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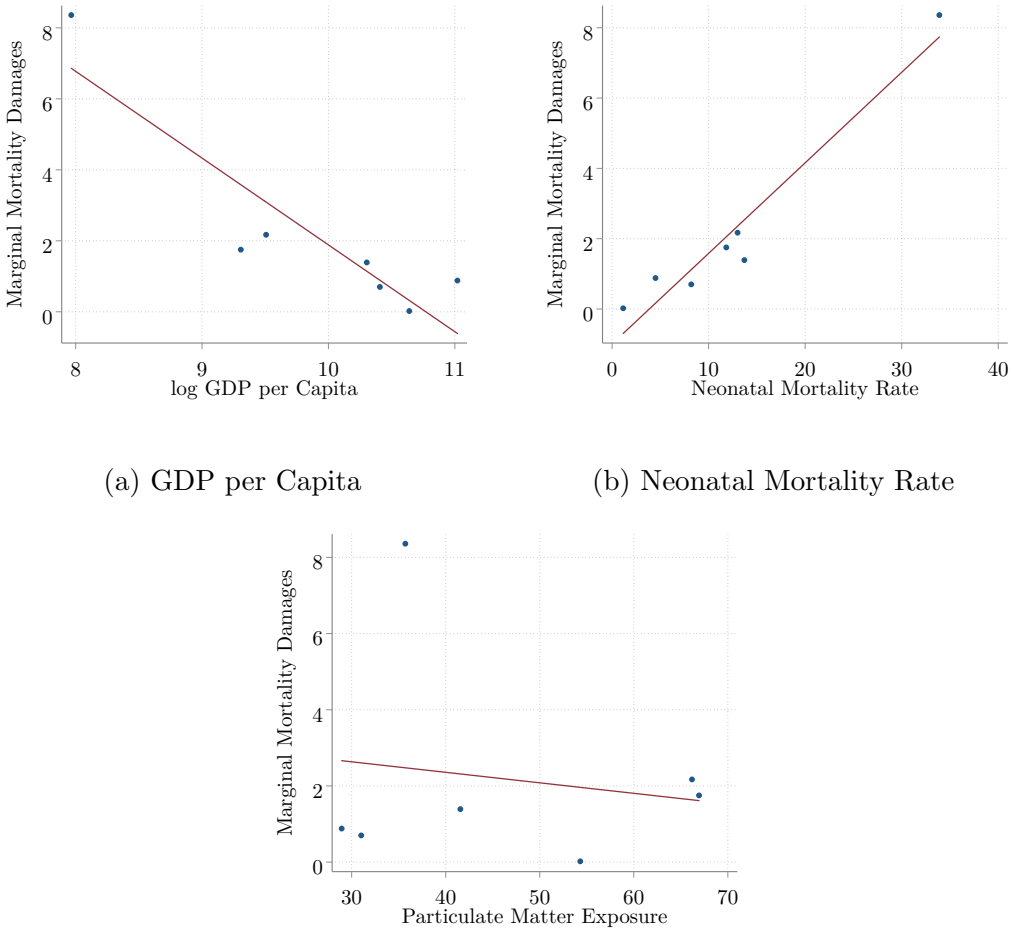
Tables and Figures

Table 1: Gestational PM_{10} Exposure, Birthweight, and Neonatal Mortality in Hong Kong

	(1) Birthweight (g)	(2) Birthweight (g)	(3) Neonatal Mortality	(4) Neonatal Mortality
Gestational PM_{10} Exposure	0.754	-7.001	-0.0377	0.00299
	(0.450)	(3.237)	(0.0268)	(0.186)
Dependent Variable Mean	3,130	3,130	1.150	1.150
TPU × Year FE	Yes	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes	Yes
TPU × Month FE	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes
Specification	OLS	IV	OLS	IV
First Stage F-Stat	–	59.838	–	59.838
Observations	532,726	532,726	532,726	532,726

Notes: Weather controls: Avg. gestational max temperature, avg. gestational min temperature, avg. gestational daily rainfall, avg. gestational humidity, avg. gestational surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triple), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

Figure 1: The Relationship between Marginal Damages, GDP per Capita, and Pollution Exposure



(a) GDP per Capita

(b) Neonatal Mortality Rate

(c) Particulate Matter Exposure

Notes: The purpose of this exercise is to explore the broader relevance of our mortality results. The data points used in these figures are taken from 7 frontier studies in the literature, including our own analysis of the relationship in Hong Kong. Details on how comparable estimates were constructed can be found in Appendix C. The figures plot the relationship between the marginal mortality damages associated with particulate matter exposure and three population characteristics. We focus on (a) GDP per Capita and (b) the Neonatal Mortality Rate and (c) average particulate matter concentrations. In panel (a) we estimate that marginal damages are negatively associated with GDP per capita – higher income locations are estimated to have lower marginal damages. In panel (b) we estimate that there is a positive association between marginal damages and the neonatal mortality rate of a given population – marginal damages are lower in populations with better health. In panel (c) we estimate that there is no association between marginal damages and baseline particulate matter concentrations – higher pollution locations are not associated with higher marginal damages.

Online Appendices – Not for Publication

Contents

A Tables and Figures

This appendix presents descriptive statistics, as well as the first-stage estimates and a wide range of robustness tests.

A.1 Descriptive Statistics

Table A1 presents descriptive statistics of our main variables of interest. We observe that the neonatal mortality rate is very low in Hong Kong, around a quarter of that in the US during the same period. In addition, average birthweight is slightly lower than the US average, but similar to the birthweights of individuals born of Asian or Chinese heritage in the US.

Particulate matter levels in Hong Kong are very high. On average, mothers were exposed to average daily levels of $54.307 \mu g/m^3$ during gestation. There is also quite a lot of variation in PM_{10} across births with a standard deviation in gestational exposure of $8.636 \mu g/m^3$. By contrast, levels of carbon monoxide and ozone tend to be lower than levels in the United States.

In terms of our main instrumental variable, we observe that mothers were exposed to approximately two thermal inversions, on average, during gestation. There is a reasonable amount of variation in exposure to thermal inversions across births with a standard deviation of 2.304 thermal inversions.

Figure A1 plots the average exposure to PM_{10} and thermal inversions by month of birth during our sample period, as well as average birthweight and the average neonatal mortality rate. In the raw data we observe that there is a relationship between PM_{10} levels, thermal inversion exposure and birthweight. Seasonality in neonatal mortality appears to mirror the pattern observed for birthweight.

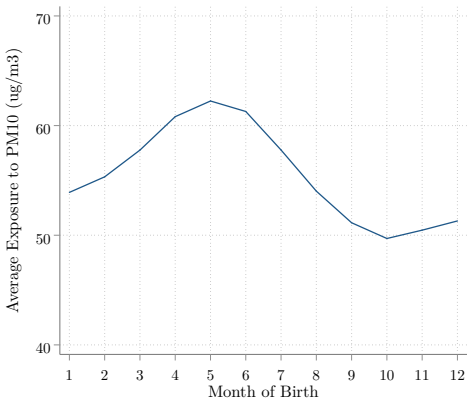
Figure A2 plots average exposure to PM_{10} and thermal inversions over our full sample period, as well as average birthweights, and the average neonatal mortality rate. We see that PM_{10} levels fell moderately over the period, that the number of thermal inversions over time doesn't follow a particular pattern, and that the average birthweight fell during this period. We do not see any obvious trends in neonatal mortality during this period; however, the variance in neonatal mortality rates appears to decline in the second half of the sample period.

Table A1: Summary Statistics

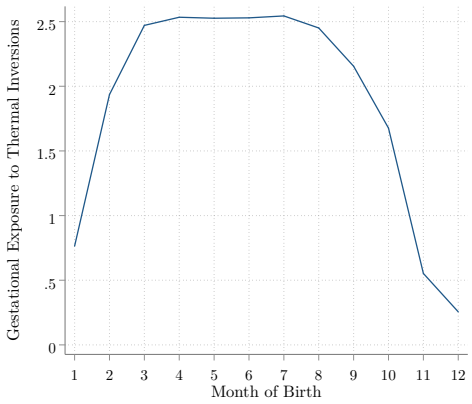
	(1) Mean	(2) Std. Dev.	(3) Observations
Panel A: Outcome Variables			
Neonatal Mortality Rate (per 1,000)	1.150	29.113	532,726
Birthweight (grams)	3,130	482	532,726
Panel B: Pollution Variables			
PM_{10} ($\mu g/m^3$)	54.307	8.636	532,726
Carbon Monoxide ($10\mu g/m^3$)	97.220	21.426	532,726
Ozone ($\mu g/m^3$)	33.068	5.661	532,726
Panel C: Instrumental Variables			
Thermal Inversions	1.8	2.295	532,726
Net Registered Tonnage	30,486	4,233	532,726
Distance to Port	8.516	6.251	532,726
Panel D: Weather Variables			532,726
Avg. Daily Max Temperature (°C)	25.637	1.673	532,726
Avg. Daily Min Temperature (°C)	20.017	1.698	532,726
Avg. Daily Rainfall (mm)	5.619	2.090	532,726
Pressure (hPa)	1,012.819	1.810	532,726
Average Humidity (%)	79.028	3.870	532,726

Notes: Outcome variables are measured at birth (birthweight), and up to the first 28 days after birth (neonatal mortality). All other variables are measured at the individual level during the 270 days of gestation.

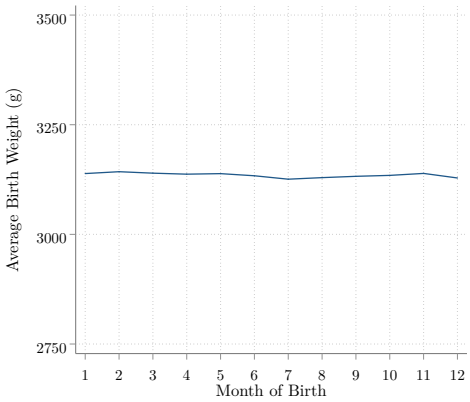
Figure A1: Within-Year Variation in Pollution, Thermal Inversions, Port Traffic, Neonatal Mortality and Birthweight



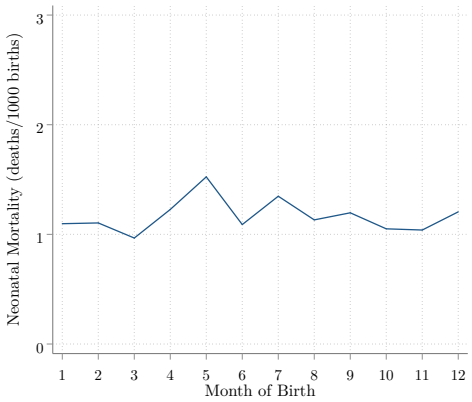
(a) PM_{10}



(b) Thermal Inversions



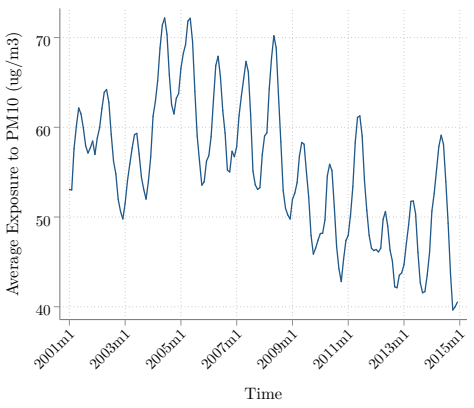
(c) Birthweight



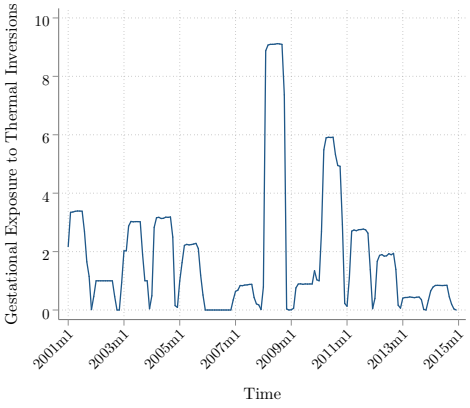
(d) Neonatal Mortality

Notes: These figures plot: (a) average PM_{10} exposure during gestation (b), average thermal inversion exposure during gestation (c), average birthweight (d) and the average neonatal mortality rate by month of birth.

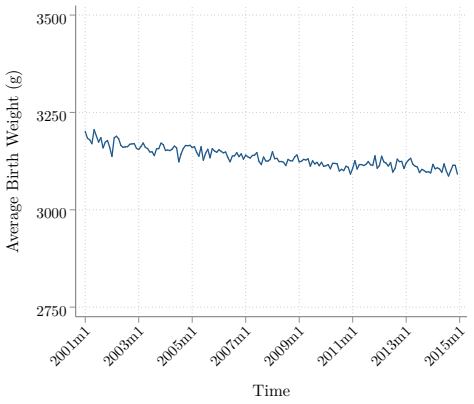
Figure A2: Full Sample Time-Series Variation in Pollution, Thermal Inversions, Port Traffic, Neonatal Mortality and Birthweight



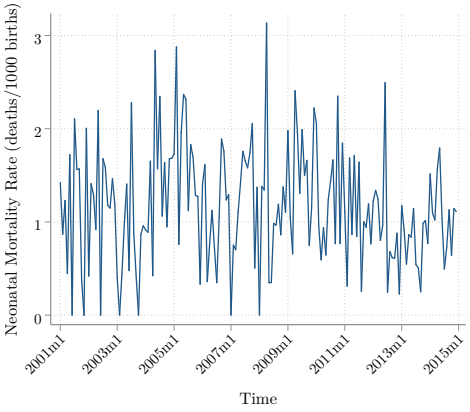
(a) PM_{10}



(b) Thermal Inversions



(c) Birthweight



(d) Neonatal Mortality

Notes: These figures plot: (a) average PM_{10} exposure during gestation (b), average thermal inversion exposure during gestation (c), average birthweight (d) and the average neonatal mortality rate for each month of our sample period (2001-2014).

A.2 Additional Results and Robustness Tests

In this section we present a series of supporting results and robustness tests.

A.2.1 First Stage Relationships for all Pollutants

In Table A2 we present the first stage relationship between our instrumental variables and the three main pollutants in our data. We estimate strong positive relationships between thermal inversions and both PM_{10} and carbon monoxide. We estimate a less significant relationship between thermal inversions and ozone, consistent with our understanding of atmospheric science and the existing literature. The first-stage F-stats for thermal inversion is highest for PM_{10} and well above 10, a common heuristic. The first-stage F-stat is also large for carbon monoxide, but very small for Ozone, as expected.

The relationship between port traffic and particulate matter is also significant. We find that an increase in net registered tonnage is associated with large increases particulate matter, but that this effect is decreasing in distance from the port. A 1% increase in port traffic is associated with a 0.09 $\mu g/m^3$ increase in PM_{10}. For each 1km shift away from the port this effect diminishes by $0.009 \mu g/m^3$, (roughly 10%). The same relationship is estimated for carbon monoxide; however, the interaction term on distance is statistically insignificant. We also find large effects on ozone; however, the sign of the main coefficient is inconsistent with previous applications of this instrumental variable (Moretti and Neidell (2011). For this reason we have less confidence in this secondary IV. Furthermore the first-stage F-stat between port traffic and PM_{10} is below 10, indicating that the instrument may be weak.

Table A2: First Stages of Instrumental Variables (All Pollutants)

	(1) PM_{10}	(2) PM_{10}	(3) CO	(4) CO	(5) O3	(6) O3
Gestational Exposure to Thermal Inversions	0.220***		0.344***		-0.0332*	
	(0.0284)		(0.0777)		(0.0201)	
Gestational Exposure to Port Traffic		9.606***		37.80***		-14.17***
		(2.410)		(6.219)		(2.276)
Distance to Port × Exposure		-0.923***		-0.475		-0.966***
		(0.230)		(0.770)		(0.140)
First Stage F-Stat	59.838	9.681	19.672	27.241	2.735	81.355
Fixed Efffects	TPU × Year and Month × TPU					
Weather Controls	Yes	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes	Yes
Dependent Variable Mean	54.307	54.307	97.218	97.218	33.069	33.069
Observations	532,726	532,726	532,726	532,726	532,726	532,726

Notes: Gestational exposure to thermal inversions is defined as the number of thermal inversions that occur during the 9 months of gestation. Gestational exposure to port traffic is defined as the log of Net Registered Tonnage that passed through Hong Kong Port during the 9 months of gestation. This measure of exposure is interacted with the distance (in km) between the port and the TPU of residence. Weather controls: Avg. gestational max temperature, avg. gestational min temperature, avg. gestational daily rainfall, avg. gestational humidity, avg. gestational surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triple), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

A.2.2 Second Stage Effects (Port Traffic IV)

In Table A3 we use our port traffic instrumental variable to explore the robustness of our findings derived from the use of the thermal inversion instrument. Similar to the thermal inversion instrument we estimate no statistically significant effect of gestational PM_{10} on neonatal mortality. The magnitude of the effect is larger than the estimate derived from the thermal inversion instrument; however, even the largest effect implying 0.6 additional deaths per 1,000 live births for a 10 $\mu g/m^3$ is still one of the smallest in the literature, comparable in magnitude to recent estimates for the United States (Knittel et al., 2016).

The effect on birthweight is statistically insignificant and around half the size of the estimate derived from the thermal inversion instrument. However, the magnitude of the central estimate is still meaningful. Taken at face value a 10 $\mu g/m^3$ increase in PM_{10} is associated with a 31.61 gram reduction in birthweight, equivalent to the estimated effects of smoking 6.2 cigarettes per day during gestation (Currie et al., 2009).

Table A3: The Relationship between Gestational PM_{10} Exposure Birthweight and Neonatal Mortality (Port Traffic IV)

	(1) Birthweight Weight (g)	(2) Neonatal Mortality
Gestational PM_{10} Exposure	-3.161	0.0610
	(5.271)	(0.402)
Dependent Variable Mean	3,130	1.150
TPU × Year FE	Yes	Yes
Weather Controls	Yes	Yes
TPU × Month FE	Yes	Yes
Individual Controls	Yes	Yes
First Stage F-Stat	9.681	9.681
Observations	532,726	532,726

Notes: Weather controls: Avg. gestational max temperature, avg. gestational min temperature, avg. gestational daily rainfall, avg. gestational humidity, avg. gestational surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triple), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

A.2.3 Controlling for Other Pollutants

In Table A4 we explore whether our IV results are affected by controlling for other criteria pollutants (Carbon Monoxide and Ozone). We find that our estimates are largely unchanged when controlling for these variables.

Table A4: The Relationship between PM_{10} Exposure, Birthweight and Neonatal Mortality (including Pollution Controls)

	(1) Birthweight (g)	(2) Neonatal Mortality
Gestational Max PM_{10} Exposure	-6.694**	0.00969
	(3.236)	(0.183)
Dependent Variable Mean	3,130	1.150
TPU × Year FE	Yes	Yes
Weather Controls	Yes	Yes
TPU × Month FE	Yes	Yes
Individual Controls	Yes	Yes
First Stage F-Stat	67.774	67.774
Observations	532,726	532,726

Notes: Weather controls: Avg. gestational max temperature, avg. gestational min temperature, avg. gestational daily rainfall, avg. gestational humidity, avg. gestational surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triple), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

A.2.4 Maximum Pollution Exposure

In Table A5 we explore whether there are birthweight or mortality responses arising from differences in gestational exposure to daily maximum levels of PM_{10}. We find similar effects to our main estimates that exploit gestational exposure to daily average levels of PM_{10}.

Table A5: The Relationship between Maximum PM_{10} Exposure, Birthweight and Neonatal Mortality (Gestational Average Daily Maximum PM_{10} Exposure)

	(1) Birthweight (g)	(2) Neonatal Mortality
Gestational Max PM_{10} Exposure	-5.905**	0.00252
	(2.723)	(0.157)
Dependent Variable Mean	3,130	1.150
TPU × Year FE	Yes	Yes
Weather Controls	Yes	Yes
TPU × Month FE	Yes	Yes
Individual Controls	Yes	Yes
First Stage F-Stat	54.861	54.861
Observations	532,719	532,719

Notes: Weather controls: Avg. gestational max temperature, avg. gestational min temperature, avg. gestational daily rainfall, avg. gestational humidity, avg. gestational surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triple), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

A.2.5 The Relationship Between Gestational $PM_{2.5}$ Exposure, Birthweight, and Neonatal Mortality

The focus of our analysis is PM_{10} because it is more consistently monitored over our sample period. However, we do have $PM_{2.5}$ measures for a sub-sample of the data. In Table A6 we show that our findings are robust to using $PM_{2.5}$ in place of PM_{10}. The estimated effects on neonatal mortality remain very small. A 10 $\mu g/m^3$ increase in $PM_{2.5}$ is associated with 0.26 additional deaths per 1,000 live births. The effects on birthweight remain meaningful. A 10 $\mu g/m^3$ increase in $PM_{2.5}$ is associated with a 65.35 gram reduction in birthweight.

Table A6: The Relationship between Gestational $PM_{2.5}$ Exposure, Birthweight, and Neonatal Mortality

	(1) Birthweight (g)	(2) Neonatal Mortality
Gestational $PM_{2.5}$ Exposure	-6.535**	0.0212
	(2.571)	(0.169)
Dependent Variable Mean	3,130	1.150
TPU × Year FE	Yes	Yes
Weather Controls	Yes	Yes
TPU × Month FE	Yes	Yes
Individual Controls	Yes	Yes
First Stage F-Stat	71.828	71.828
Observations	502,812	502,812

Notes: Weather controls: Avg. gestational max temperature, avg. gestational min temperature, avg. gestational daily rainfall, avg. gestational humidity, avg. gestational surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triple), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

A.2.6 Adjusting the Sample by Distance to Pollution Monitors

In our main analysis we restrict our sample to locations that are within 10km of a pollution monitor. A choice about distance has to be made out of necessity due to the fact that there are fewer monitors than neighborhoods in our context. However, one may be concerned that distance to monitors introduces non-classical measurement error if monitors are endogenously placed (Knittel et al., 2016; Grainger et al., 2018; Zou, 2018). We explore the potential for this by using alternative distance restrictions. We restrict our sample to 8km and 12km respectively. Across all samples we fail to reject a null that PM_{10} has no effect on neonatal mortality (Table A7). Our estimated effects of PM_{10} on birthweight are also robust across the different samples (Table A7).

Table A7: The Relationship between PM_{10} and Neonatal Mortality Using Different Distance Restrictions (IV)

	(1) Birthweight (g)	(2) Neonatal Mortality
Panel A: 10km		
Gestational PM_{10} Exposure	-7.001**	0.00299
	(3.237)	(0.186)
First Stage F-Stat	59.838	59.838
Observations	532,726	532,726
Panel B: 8km		
Gestational PM_{10} Exposure	-8.292**	0.00790
	(3.766)	(0.215)
First Stage F-Stat	44.730	44.730
Observations	432,071	432,071
Panel C: 12km		
	-7.039**	-0.0272
	(3.100)	(0.176)
First Stage F-Stat	62.712	62.712
Observations	556,211	556,211
TPU × Year FE	Yes	Yes
Weather Controls	Yes	Yes
TPU × Month FE	Yes	Yes
Individual Controls	Yes	Yes

Notes: Weather controls: Avg. gestational max temperature, avg. gestational min temperature, avg. gestational daily rainfall, avg. gestational humidity, avg. gestational surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triple), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

A.2.7 Reduced Form Relationships

In Table A8 we estimate the reduced form relationship between gestational exposure to thermal inversions and neonatal mortality. We fail to reject a null that the number of gestational thermal inversions has an effect on neonatal mortality.

We do estimate a strong reduced form relationship between thermal inversions and birthweight. We find that exposure to an additional thermal inversion during gestation is associated with a 1.54 gram reduction in birthweight.

Table A8: The Relationship between Thermal Inversions, Birthweight, and Neonatal Mortality (Reduced Form)

	(1) Birthweight (g)	(2) Neonatal Mortality
Gestational Exposure to Thermal Inversions	-1.540**	0.000657
	(0.674)	(0.0410)
Dependent Variable Mean	3,130	1.150
TPU × Year FE	Yes	Yes
Weather Controls	Yes	Yes
TPU × Month FE	Yes	Yes
Individual Controls	Yes	Yes
Observations	532,726	532,726

Notes: Weather controls: Avg. gestational max temperature, avg. gestational min temperature, avg. gestational daily rainfall, avg. gestational humidity, avg. gestational surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triple), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

A.2.8 The Relationship Between Gestational PM_{10} Exposure and Neonatal Mortality, by Cause of Death

In Table A9 we estimate the relationship between PM_{10} and neonatal mortality, by cause of death. We look at deaths due to respiratory causes, cardiovascular causes, prenatal causes and all other causes. A priori one should expect that any mortality response to pollution should be in the first three categories. We fail to reject a null hypothesis for all categories.

Table A9: The Relationship between Gestational PM_{10} Exposure and Neonatal Mortality (Cause of Death)

	(1) Neonatal Mortality (Respiratory)	(2) Neonatal Mortality (Cardiovascular)	(3) Neonatal Mortality (Prenatal)	(4) Neonatal Mortality (Other)
Gestational PM_{10} Exposure	0.0519	-0.0109	0.138	0.0930
	(0.0593)	(0.0479)	(0.134)	(0.473)
Dependent Variable Mean	0.176	0.160	0.642	1.539
Fixed Effects	TPU × Year, TPU × Month			
Controls	Weather Controls and Individual Controls			
First Stage F-Stat	59.622	59.628	59.609	59.830
Observations	531,807	531,805	532,245	532,487

Notes: Weather controls: Avg. gestational max temperature, avg. gestational min temperature, avg. gestational daily rainfall, avg. gestational humidity, avg. gestational surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triple), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

A.2.9 Unique Mortality Sample

In Table A10 we explore the relationship between PM_{10} and neonatal mortality using the unique mortality sample. This sample is restricted to only include unique deaths within each TPU, date of birth, sex cell. This sample reduces measurement error in the dependent variable but is unlikely to be representative of the Hong Kong population as the data is based on locations with smaller populations. We estimate marginally larger effects of PM_{10} on mortality; however, the effects remain statistically insignificant. Taken at face value the results suggest that a 10 $\mu g/m^3$ increase in PM_{10} is associated with an additional 0.6 neonatal deaths per 1,000 births, comparable to recent estimates in the United States where pollution levels are markedly lower (Knittel et al., 2016).

We also estimate birthweight effects that are similar to the main analysis sample. However, the estimates are no longer statistically significant, likely due to a substantial reduction in sample size.

Table A10: The Relationship Between Gestational PM_{10}, Birthweight, and Neonatal Mortality (Unique Mortality Sample)

	(1) Birthweight (g)	(2) Neonatal Mortality
Gestational PM_{10} Exposure	-6.784	0.0605
	(4.682)	(0.258)
Dependent Variable Mean	3,148	1.093
Fixed Effects	TPU × Year, TPU × Month	
Controls	Weather Controls and Individual Controls	
First Stage F-Stat	57.169	57.169
Observations	287,816	287,816

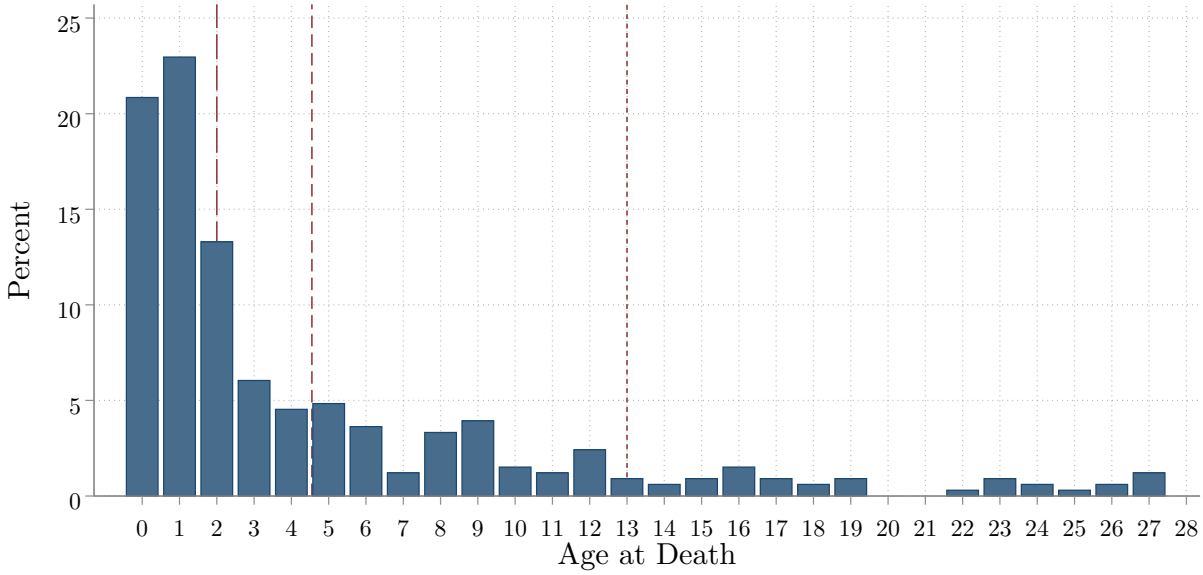
Notes: Weather controls: Avg. gestational max temperature, avg. gestational min temperature, avg. gestational daily rainfall, avg. gestational humidity, avg. gestational surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triple), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

A.2.10 Contemporaneous Exposure

In Table A11 we explore the relationship between post-gestational PM_{10} exposure and neonatal mortality. We measure exposure as the average daily PM_{10} exposure from the date of birth until death, or until 28 days after birth. We do this given that the existing literature has highlighted the relevance of contemporaneous exposure for infant mortality (Currie and Neidell, 2005; Arceo et al., 2016; Knittel et al., 2016). However, once again we find no effects of pollution on neonatal mortality. A caveat to this exercise is that we have to depend on a fixed effect approach because the first stages of our IV strategy are not relevant over this short time period (a maximum of 28 days).

Across a broad range of fixed effect specifications we find no meaningful relationship between PM_{10} and neonatal mortality. Conditional on a neonatal death occurring the median age of death is 2 days old.[16] Consequently, there does not appear to be sufficient opportunity for post-natal exposure to have a meaningful effect on neonatal mortality in this context. For this reason, as well as to estimate the effects of comparable measure of exposure for both neonatal mortality and birthweight, our main analysis focuses on pre-natal exposure.

Figure A3: Age of Death in Days



Notes: The histogram presents the age of death, conditional on death in the first 28 days. The long dashed line represents the median (2 days). The medium dashed line represents the mean (4.55 days). The short dashed line represents the 90th percentile (13 days).

[16]The distribution of age at death is presented in Figure A3.

Table A11: The Effects of Air Pollution on Neonatal Mortality – Contemporaneous Exposure (OLS)

	(1) Neonatal Mortality	(2) Neonatal Mortality
PM_{10} Exposure	-0.0119*	-0.0124*
	(0.00708)	(0.00723)
Dependent Variable Mean	1.150	1.150
TPU × Year FE	Yes	Yes
Weather Controls	Yes	Yes
TPU × Month FE	Yes	Yes
Individual Controls	Yes	Yes
Prenatal Pollution Controls	No	Yes
Observations	528,440	528,440

Notes: Weather controls: In column 1 we include avg. max temperature, avg. min temperature, avg. daily rainfall, avg. humidity, avg. surface pressure between birth and death or birth and 28 days. In column 2 we include the same controls as column 1 but also include weather controls for the prenatal period. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triple), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

B Awareness of Pollution Exposure

The purpose of this exercise is to explore the relevance of individual-level avoidance paper in our empirical context. To do this we estimate the effects of daily pollution exposure on the attendance of football matches in the stadiums in Hong Kong.

We collected data on the attendance of 1,002 professional football games played at 15 stadiums in Hong Kong between the 2008/09 and 2013/14 seasons. We combined these data with pollution exposure at the stadium on the day of the match to explore the degree to which spectators are aware of pollution levels. The combination of these data sets results in 943 matches. After including date fixed effects we are left with a panel of 560 matches due to singleton observations.

We regress the log of attendance on pollution exposure for that day, controlling for stadium (TPU)-year fixed effects, and date fixed effects, and match-type fixed effects, which control for level differences in the types of match that are played, e.g. international matches, regular season matches, charity games, etc.

$$logAttendance_{ijdt} = \beta Pollution_{idt} + \alpha_{it} + \alpha_{dt} + \alpha_j + \epsilon_{ijdt}$$

where α_{it} is the stadium-year fixed effect, α_{dt} is the date of match fixed effect, and α_j is the match type fixed effect. We explore the effects of three types of pollution: Particulate Matter, Ozone and Carbon Monoxide. If awareness of pollution is empirically relevant then we may find effects for particulate matter, but not for Ozone or Carbon Monoxide, which are not visible.

The results of our analysis can be found in table B1. We estimate that a 10 $\mu g/m^3$ increase in PM_{10} is associated with an 17-20% reduction in match attendance. By contrast, there is no meaningful effect of Carbon Monoxide (CO) or Ozone (O3) on match attendance. This findings suggest that awareness about particulate matter appears to be a relevant consideration in Hong Kong.

Table B1: The Effects of Air Pollution on Football Game Attendance

	(1) log Attendance	(2) log Attendance	(3) log Attendance
PM_{10} Exposure	-0.0194***	-0.0184***	-0.0169***
	(0.00466)	(0.00472)	(0.00453)
CO Exposure	0.00750**	0.00681*	0.00587*
	(0.00321)	(0.00320)	(0.00284)
O3 Exposure	0.00405	0.00301	0.000823
	(0.00711)	(0.00760)	(0.00593)
Stadium TPU × Year FE	Yes	Yes	Yes
Date of Match FE	Yes	Yes	Yes
Match Type FE	No	Yes	Yes
Weather Controls	No	No	Yes
Observations	560	556	556

Notes: Standard errors are two-way clustered at the TPU level. Weather controls include maximum and minimum temperature, total rainfall, surface pressure, and average humidity.

C Comparison Studies and Calculations

In this appendix we document the results and characteristics from the studies used in our cross-institution analysis, as well as the calculations we made to construct comparable estimates.

Heft-Neal et al. (2019)

Sample period: 2001-2015
Context: Sub-Saharan Africa
GDP per capita: $2,880*
Average PM_{10} Exposure: 35.71 $\mu g/m^3$ (mean exposure)
Infant mortality rate: 71 deaths per 1,000 births
Neonatal mortality rate: 33.9 deaths per 1,000 births*[17]
Measure of Exposure: Annual post-natal.

Using an IV strategy the authors estimate that a 10-unit increase in PM2.5 in the year following birth is associated with an additional 15.62 deaths per 1,000 live births (IMR). To convert PM2.5 exposure to PM10 exposure we use the ratio PM2.5 = 0.7PM10. Consequently, the effect of a 10-unit increase in PM10 would be associated with an additional 22.3 deaths per 1,000 live births (IMR). Using the average ratio between the neonatal mortality rate and infant mortality rate for sub-Saharan Africa we attribute 47% of the estimated deaths to neonatal mortality, resulting in an additional 11.15 deaths per 1,000 live births (NMR). We then multiply this effect by 0.75 to provide a comparable 9-month measure of exposure, giving an estimate of 8.36 neonatal deaths per 1,000 live births.

Knittel et al. (2016)

Sample period: 2002-2007
Context: California, USA
GDP per capita: $61,124*
Average PM_{10} Exposure: 28.94$\mu g/m^3$
Infant mortality rate: 2.81 deaths per 1,000 births
Neonatal mortality rate: 4.5 deaths per 1,000 births (the U.S. average)*
Measure of Exposure: Weekly Post-Natal.

Using the estimates from their IV strategy we calculate that a 10-unit increase in PM10 is associated with an additional 0.74 deaths per 1,000 live births (IMR).[18] Using the US average ratio between the neonatal mortality rate and infant mortality rate we attribute 66% of the estimated deaths to neonatal mortality, resulting in an additional 0.48 deaths

[17] Both the infant mortality rates and the neonatal mortality rates in this section come from the original papers. When such information is not available, we cite numbers from the United Nations Children's Fund (UNICEF) and mark the numbers using *.

[18] 0.0019×39 weeks×10 units = 0.74 (Table 8, Column 4).

per 1,000 live births (NMR).

Arceo et al. (2016)

Sample period: 1997-2006
Context: Mexico City
GDP per capita: $11,015 (Mexico)*
Average PM_{10} Exposure: $66.94 \mu g/m^3$
Infant mortality rate: 19.87 deaths per 1,000 births
Neonatal mortality rate: 11.83 deaths per 1,000 births
Measure of Exposure: Weekly Post-Natal.

Using an IV strategy they find that a 10-unit increase in PM10 is associated with an additional 2.97 neonatal deaths per 1,000 live births (NMR).[19]

Chay and Greenstone (2003a)

Sample period:1969-1974
Context: USA
GDP per capita: $29,843
Average PM_{10} Exposure: $41.58 \mu g/m^3$-$51.81 \mu g/m^3$ (annual average)
Infant mortality rate: 17.9 deaths per 1,000 births
Neonatal mortality rate: 13.7 deaths per 1,000 births
Measure of Exposure: Gestational.

Using an IV strategy they find that a 10-unit drop in TSP is associated with 0.7-1.3 fewer infant deaths per 1,000 live births.[20]. Following Knittel et al. (2016) we apply a commonly used conversion metric of 0.55TSP = PM10, a 10-unit increase in PM10 would be associated with 1.29 - 2.37 more infant deaths per 1,000 live births. Using the ratio between the neonatal mortality rate and infant mortality rate we attribute 76% of the estimated deaths to neonatal mortality, resulting in 0.98 - 1.80 fewer neonatal deaths per 1,000 live births.

Chay and Greenstone (2003b)

Sample period:1978-1984
Context: USA
GDP per capita: $33,102
Average PM_{10} Exposure: $31.02 \mu g/m^3$-$39.11 \mu g/m^3$ (annual average)
Infant mortality rate: 11.8 deaths per 1,000 births
Neonatal mortality rate: 8.2 deaths per 1,000 births

[19] 0.007625×39 weeks$\times 10$ units = 2.97 (Table 3, Column 3).
[20] The coefficients from columns 2 and 5 in Table 6

Measure of Exposure: Gestational.

Using an IV strategy they find that a 10-unit drop in TSP is associated with 0.4 fewer neonatal deaths per 1,000 live births. Using the TSP conversion metric this translates into 0.7 fewer neonatal deaths per 1,000 live births per 10 unit reduction in PM10.

Cesur et al. (2016)

Sample period: 2001-2011
Context: Turkey
GDP per capita: \$13,455*
Average PM_{10} Exposure: $66.19\mu g/m^3$
Infant mortality rate: 9.2 death per 1,000 births[21]
Neonatal mortality rate: 13.0 deaths per 1,000 births*
Measure of Exposure: Annual Post-Natal.

Using an IV strategy they estimate that a 10 unit increase in PM10 is associated with an additional 3.017 infant deaths per 1,000 live births.[22] Using the Turkey-wide ratio between the neonatal mortality rate and infant mortality rate we attribute 72% of the estimated deaths to neonatal mortality resulting in an additional 2.172 neonatal deaths per 1,000 births.

Our study

Sample period: 2001-2014
Context: Hong Kong
GDP per capita: \$41,764
Average PM_{10} Exposure: $54.31\mu g/m^3$
Infant mortality rate: 1.5 deaths per 1,000 births
Neonatal mortality rate: 1.15 deaths per 1,000 births
Measure of Exposure: Gestational.

We find that a 10-unit increase in PM10 is associated with an additional 0.02 neonatal deaths per 1,000 live births.

[21] As discussed in Cesur et al. (2016), official statistics on deaths, especially for infants, are incomplete in most developing countries, including turkey, due to under-reporting. The paper cites the infant mortality rates from other sources as well. According to WHO, the average mortality rate in the study period is 18.0 deaths per 1,000 births.

[22] This estimate is calculated as a 1.15% increase in PM_{10} relative to the mean, resulting in a 1.45% increase in infant deaths per 1,000 live births. Using the Infant Mortality Rate this corresponds to 4.023 additional deaths. We then multiply this by 0.75 to correspond to 9 months of exposure, resulting in an estimate of 3.017.

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