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Abstract

Conventional wisdom suggests that marginal damages from particulate matter pollution are high in less-developed countries because they are highly polluted. Using administrative data on the universe of births and deaths, we explore birthweight and mortality effects of gestational particulate matter exposure in high-pollution yet high-income Hong Kong. The marginal effects of particulates on birthweight are large but we fail to detect an effect on neonatal mortality. We interpret our stark mortality results in a comparative analysis of pollution-mortality relationships across studies. We provide early evidence that marginal mortality damages from pollution are high in less-developed countries because they are less developed, not because they are more polluted.

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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 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, 2003b; Pope and Dockery, 2006; Bishop et al., 2019; Deryugina et al., 2019). Particulate matter has been shown to affect earnings, property values, criminal behavior, labor productivity, and educational attainment (Chay and Greenstone, 2005; Graff Zivin and Neidell, 2012; Chang et al., 2016; Ebenstein et al., 2016; Isen et al., 2017; Herrnstadt et al., 2019; Colmer and Voorheis, 2020).

Conventional wisdom, and a growing literature, suggest that marginal health damages from pollutants like particulate matter are high in developing countries.[1] However, the mechanisms are not well understood. High marginal damages in developing countries could be explained by higher levels of pollution if damage functions are convex. 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; Pullabhotla, 2019; Bombardini and Li, 2020). Under this logic, cost-effectively reducing marginal damages involves reducing pollution levels. Another possibility is that high marginal damages in developing countries may be due to the limited ability of exposed populations to manage the consequences of pollution. Differences in institutional and economic conditions could lead to different marginal pollution damages, regardless of the shape of the damage function. Following this logic, efforts to reduce marginal damages by directly reducing pollution may be less cost effective. Incremental investments

[1] See, for example, 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); Chang et al. (2019); Heft-Neal et al. (2019); Pullabhotla (2019); Bombardini and Li (2020); Fan et al. (2020); He et al. (2020); Adhvaryu et al. (Forthcoming).

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 particulate matter damages in developed countries by re-examining the relationship between gestational PM exposure and infant health using administrative data on the universe of births and deaths in high particulate pollution, high income Hong Kong between 2001 and 2014. Existing analyses of marginal PM-health relationships come from high income, low particulate matter settings (i.e. developed nations) or low income, high particulate matter settings (i.e. developing nations). By contrast, 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 yet its per capita income levels compare to the United States. Hong Kong's population has excellent baseline health and universal access to free high-quality health care. Life expectancy ranks in the top-10 worldwide.

We identify the effects of endogenous particulate matter exposure on birthweight, low birthweight, and neonatal mortality by exploiting plausibly exogenous variation in thermal inversions (Arceo et al., 2016; Chen et al., 2017, 2018). We find that higher gestational particulate matter exposure is associated with significant reductions in birthweight and significant increases in low birthweight. These marginal PM-birthweight effects are substantial; a 10 $\mu g/m^3$ increase in particulate matter is associated with the equivalent of the estimated effects of smoking 15 cigarettes per day during pregnancy (Currie et al., 2009). Birthweight outcomes are important indicators of health that have been shown to have persistent effects on later life (Almond et al., 2005; Currie and Moretti, 2007; Almond et al., 2010; Bharadwaj et al., 2013; Isen et al., 2017; Colmer and Voorheis, 2020). By contrast, we fail to detect significant marginal effects of gestational PM on neonatal mortality. The marginal effects of PM on neonatal mortality are noisily estimated but small both in absolute terms and relative to the existing literature. We fail to reject the null hypothesis that there is no marginal effect on neonatal mortality in Hong Kong in all specifications.

One interpretation of our Hong Kong results is that the location's wealth and health may not strongly influence marginal PM-birthweight relationships but may play an important role in mitigating marginal PM-mortality relationships. Hong Kong has excellent healthcare yet marginal changes in particulate matter have large effects on birthweight. Since interventions targeting child health are challenging prior to birth, birthweight effects may be less responsive to health and other institutions. Hong Kong has high particulate matter concentrations yet marginal changes in PM have small effects on neonatal mortality.[2] When PM concentrations change, Hong Kong's wealth, health, and institutions may facilitate post-natal health interventions that buffer against effects on neonatal mortality.

To explore this latter conjecture, we combine data from multiple internally valid studies - including our own - to examine and contextualize marginal PM-mortality damage estimates across the literature. This comparative exercise sacrifices precise identification in order to inform unanswered broad economic questions (Akerlof, 2020). We standardize and transparently quantify marginal PM-mortality effects across contexts. We focus on policy-relevant absolute marginal PM-mortality effects. We directly examine macro-level correlates of marginal PM-mortality damages. We find that marginal PM-mortality effects across locations are sharply decreasing in baseline health and decreasing in GDP per capita. By contrast, we find that marginal mortality effects are largely unrelated to a location's baseline mean particulate matter concentrations. Although conventional wisdom suggests that marginal particulate matter damages are high in less-developed countries because they are highly polluted, we provide early evidence that marginal PM-mortality damages are high in less-developed countries because they are less developed.

We make three contributions. First, we provide new evidence on the marginal damages of particulate matter pollution in a unique context. The existing literature provides limited evidence on the marginal effects of pollution on *birthweight and low birthweight* in a

[2] An alternative interpretation is that the dose-response function is concave (Goodkind et al., 2014; Pope et al., 2016). However, as described further elsewhere, neither our large birthweight effects in Hong Kong nor the evidence from our cross-institutional comparative analysis are consistent with a concave dose-response function.

high-particulate setting, as related studies typically focus on the low-pollution settings of the developed world for data availability reasons. Although an emerging literature does explore the effects of pollution on neonatal *mortality* in *high-particulate* settings, most other studies do so using data from low-income developing countries. Second, our comparative exercise allows us to explore correlates of the marginal particulate-mortality relationship across diverse settings. Existing studies take disparate approaches to quantifying estimates, make relatively few cross-institutional comparisons and provide limited detail on how comparisons are constructed, and frequently interpret results in relative percentage terms. Our standardized approach focusing on absolute marginal PM-mortality effects addresses the concern that a given percentage reduction in neonatal mortality means something different in countries with vastly different baseline conditions. Third, the free, high quality healthcare system in Hong Kong facilitates access to excellent data for an entire population. Free universal healthcare offers particularly compelling data on vulnerable populations often undersampled in other contexts.

Our paper is most closely related to recent work by Cheung et al. (2020). Cheung et al. (2020) exploit transboundary pollution to examine the effects of pollution on mortality across the age distribution in Hong Kong, with a focus on cardio-respiratory mortality. Cheung et al. (2020) explain trends in the effects of air pollution over time in Hong Kong, noting significant declines in marginal mortality damages after the SARS epidemic and in neighborhoods that do not have a hospital with accident and emergency services. Our paper differs in several ways. First, outcomes differ. We focus on birthweight, low birthweight, and neonatal mortality. Infant health and mortality are of interest because policy makers focus on protecting vulnerable populations and because infant outcomes have implications for long-term health and human capital development (Currie, 2011). Second, identification strategies differ. Our paper exploits thermal inversions for identification. Studying infants also has advantages for identification since causal relationships between pollution exposure and outcomes occur over short time horizons (Currie et al., 2009). Third, economic questions

differ. Our broad aim is to develop insights for the economic question of explaining differences in marginal pollution damages across economic circumstances.

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.[3] 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 damages.

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 limiting cases. Differences in marginal damages may be lower in developed countries because levels of pollution are different. If, for example, the dose response function is convex 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:

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

Alternatively, differences in marginal damages may arise from differences in the factors that translate pollution exposure into marginal damages. For example, two populations may

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

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.

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

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.[4] 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.[5]

Our mortality records data set includes date of death, age of death, and cause of death. In our final analysis sample, we focus on 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 match the death record to 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 common unique identifiers 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

[4]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, or roughly a circle with a 1.1km radius.

[5]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.

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).[6]

Panel A of Table 1 presents descriptive statistics of our main outcome variables of interest. We observe that the neonatal mortality rate is very low in Hong Kong (1.150 deaths/1,000 live births), around a quarter of that in the US during the same period. Average birthweight (3,130g) is slightly lower than the US average, but similar to the birthweights of individuals born of Asian or Chinese heritage in the US. 6.4% of the births in our sample are low birthweight ($<$ 2,500g).

In Panels (a), (b), and (c) of Figure 1 we see that there is some seasonality in our birthweight and mortality outcomes. On average, children born in the summer are lower birthweight, more likely to be classified as low birthweight, and less likely to survive the first 28 days. In our empirical specification, discussed below, we control for this seasonality using TPU by month-of-year fixed effects.

In Panels (a), (b), and (c) of Figure 2 we plot outcomes of interest throughout the sample, starting in January 2001 and ending in December 2014. We observe that average birthweight/low birthweight are decreasing/increasing over time.[7] We do not see an obvious trend in the neonatal mortality rate. In our empirical specification, discussed below, we control for aggregate and local trends using TPU by year fixed effects.

Pollution Data We collect hourly pollution concentrations for several criteria pollutants from the Environmental Protection Department of Hong Kong. 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

[6] 54% of neonatal deaths are assigned a probability of 1. As long as any measurement error is orthogonal to pollution exposure our estimate of the treatment effect will be unbiased and consistent. Our results are not sensitive to dropping probabilistic deaths (Table B5).

[7] In appendix A we provide suggestive evidence that these trends are associated with increasing maternal age and more frequent multiple births.

buffer.[8] Second, we construct 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 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.[9]

In our main analysis, we focus on PM_{10} exposure due to very high particulate levels for a high-income setting (Table 1).[10] During our study period, average gestational exposure to PM_{10} levels in Hong Kong was $54\mu g/m^3$/day. The minimum average gestational exposure to PM_{10} in our sample was $30\mu g/m^3$/day and the maximum average gestational exposure was $94\mu g/m^3$/day. For perspective, World Health Organization guidelines suggest that the annual average of PM_{10} should not exceed $20\mu g/m^3$/day.

In Panel (d) of Figure 1 we see that there is some seasonality in PM_{10} exposure. Children born in the summer are, on average, exposed to higher levels of PM_{10} during gestation. In Panel (d) of Figure 2 we plot longer-run trends throughout the sample, starting in January 2001 and ending in December 2014. We observe that average gestational exposure to PM_{10} has fallen over time.

Weather Data We collect weather data from two sources. First, we observe daily data on surface-level temperature, precipitation, humidity, and air pressure from Hong Kong Observatory weather monitors. Second, we collect data on atmospheric air temperatures 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

[8] 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 B8).

[9] 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.

[10] 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 traffic and electricity generation. Although PM_{10} is consistently reported over the sample period and facilitates comparisons with an existing literature, we later consider robustness to $PM_{2.5}$.

are provided every 6 hours on a 0.12° × 0.12° geographic grid (13 × 13 km) at two fixed atmospheric levels (111 and 766 meters). 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. Descriptive statistics are presented in Panel D of Table 1.

Final Sample Combining all data sources, we obtain a final analysis sample of 532,726 births between 2001 and 2014. The unit of observation is a birth. Each birth is matched to weather exposure, individual mortality information, and gestational pollution exposure.[11]

4 Research Design

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

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

To identify the marginal PM-mortality and marginal PM-birthweight effects, β, it must be the case that particulate matter is orthogonal to ϵ_{ijt}. The main threat to identification is omitted variable bias. For example, exposure to PM_{10} and birth outcomes may be correlated via changes over time across years and seasonal variation within years. Gestational PM_{10} exposure and birth outcomes (conditional on PM_{10} exposure) may be seasonal. Gestational PM_{10} and birth outcomes (conditional on PM_{10} exposure) may be correlated with local economic activity. Additionally, exposure to PM_{10} and birth outcomes may be correlated with avoidance behavior. In the long run, people choose where to live and so exposure to

[11] Appendix C considers contemporaneous exposure.

PM_{10} and birth outcomes may be associated with residential sorting. Opportunities to avoid PM_{10} damages through investments in air purifiers and HEPA filters, or information about PM_{10} damages, may be correlated with birth outcomes.

One approach to address omitted variable bias is to control for covariates and/or fixed effects. We estimate the relationship between birth outcomes and PM_{10} exposure using the following specification,

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

δ_{jt} is a vector of TPU-by-year fixed effects that control for time-invariant characteristics at the local level as well as annual shocks that are common across everyone within a TPU such as changes in economic conditions. TPU-by-year fixed effects address location-specific equilibrium endogenous exposure due to residential sorting or defensive expenditures on the basis of longer-term pollution averages or year-on-year changes in pollution over time. We also include TPU-by-month-of-year, ϕ_{jm}, fixed effects that control for TPU-specific seasonality in births, deaths, and disease transmission that may also be correlated with PM_{10} exposure. X_{ijmt} is a vector of controls that includes gestational surface weather conditions, parental characteristics, and birth characteristics.

An Instrumental Variables Approach The approach in equation 2 addresses several threats to identification but cannot fully address other concerns, such as measurement error and some forms of short-run individual-level avoidance behavior.[12] We employ an instrumental variable strategy. Our 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 recent papers (Arceo et al., 2016; Hicks et al., 2016; Chen et al., 2017, 2018). Under normal conditions air temperature

[12]It is likely that the population is at least loosely aware of pollution levels (Moretti and Neidell, 2011; Chang et al., 2018; Zhang and Mu, 2018; Barwick et al., 2019). In Appendix D we provide evidence 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.

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 an onset of a thermal inversion if the difference in average daily temperature between the two atmospheric levels measured in the reanalysis data is negative, i.e, $temp_{111m} - temp_{766m} < 0$. 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 (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. We control for a vector of season fixed effects (month-of-year × TPU in our main specification), so we address the seasonality of thermal inversions. 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 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}. 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 (Table B1). 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.[13]

Exclusion Restriction An assumption for identification is that thermal inversions 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. Consequently, after controlling for seasonality and surface-level weather conditions that could be correlated with thermal inversions, pollution, and health, we argue that the exclusion restriction is likely satisfied.

An issue is that thermal inversions could directly affect short-run avoidance behavior like staying indoors, running air purifiers, or wearing masks. One possibility is that individuals are aware of inversions and their consequences for particulate pollution. The existing literature argues that most people are unaware when thermal inversions occur and even fewer are aware that thermal inversions concentrate particulates (Arceo et al., 2016; Hicks et al.,

[13]Thermal inversions are unlikely to have large effects 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) argue that inversions may directly inhibit the formation of these pollutants as the chemical reactions required to form them require warmth and sunlight. We do find statistically significant relationships between inversions and carbon monoxide, consistent with the literature (Table B1).

2016; Chen et al., 2017, 2018).[14]

Another possibility is that thermal inversions are forecastable and that the average individual responds to forecast communication with avoidance behaviors. Although technology has improved over the past few years, thermal inversions have been historically very challenging to forecast at the local level. This is especially true in hilly and mountainous terrain like the Hong Kong landscape (Steyn et al., 2013; Chan and Hon, 2016).

A third possibility is that short-run avoidance behavior is induced by real-time observations of particulate pollution levels. Surveys suggest people are visually insensitive to changes in particulate matter at relatively high concentrations (Hyslop, 2009). Although mobile apps that provide real-time pollution levels and pollution alerts are now widely available in Hong Kong, they were uncommon for the bulk of our sample period. When the Hong Kong Environmental Protection Department offered a free mobile app in late 2013, the app was met with considerable skepticism (Kao, 2013).

Taken as a whole, although we believe it is unlikely that individuals fully observe instrument-induced changes in pollution, we acknowledge that we cannot rule out the possibility. In the case where inversion-induced changes in particulate concentrations are salient, our IV estimate is the short-run *behavior adjusted* effect of pollution on health. In the case where individuals are not able to fully observe instrument-induced changes in pollution, the IV addresses short-run avoidance behavior and may be interpreted as the biological effects of pollution on health.[15]

[14]During an October 2017 seminar presentation of this paper at Hong Kong University of Science and Technology (HKUST), we conducted an informal poll 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, and/or 3) knew that thermal inversions had large effects on local pollution levels. Only one 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.

[15]Behavior adjusted estimates may be more relevant for some policy purposes and biological estimates may be more relevant for other policy purposes (Graff Zivin and Neidell, 2013; Beatty and Shimshack, 2014). Although behavior adjusted estimates will not reflect the full costs of avoidance behavior for benefit-cost analysis, those effects may be more appropriately estimated separately, in the spirit of Zhang and Mu (2018) for air pollution and Graff Zivin et al. (2011) for water pollution.

Multiple Pollutants We assume that thermal inversions in Hong Kong affect infant health through particulate matter rather than through other pollutants like carbon monoxide (CO), ozone (O3), sulfur dioxide (SO_2), and nitrogen dioxide (NO_2). We believe this is a reasonable assumption. In Hong Kong, PM concentrations are high yet concentrations of many other pollutants are low, even for high-income settings.[16] PM_{10}, O3, CO, SO_2, and NO_2 are not strongly correlated, conditional on fixed effects. Our first-stage results indicate that thermal inversions in Hong Kong are a stronger instrument for PM_{10} than for other pollutants, as measured by F-statistics or standardized marginal effects (Table B1).

Our results are robust to estimating reduced form relationships between infant health outcomes and thermal inversions (Table B2). Using estimates from these reduced form relationships, we find that the increase in thermal inversions necessary to generate a 1 $\mu g/m^3$ increase in PM_{10} generates marginal PM-birthweight and marginal PM-mortality estimates that are statistically indistinguishable from the main marginal results discussed in the next section. Our second-stage results are also robust to including, or omitting, controls for ozone, carbon monoxide, sulfur dioxide, and nitrogen dioxide (Table B3). Although it seems reasonable to us to assume that inversions affect infant health in Hong Kong through PM we can't rule out the influence of other pollutants. At a minimum our approach is sufficient to identify the effects of air pollution, broadly defined, on birthweight and mortality (Chay and Greenstone, 2003b; Currie and Neidell, 2005; Arceo et al., 2016; Knittel et al., 2016; Deryugina et al., 2019).

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 PM_{10} for individual i, in TPU j, born in month m of year t. We control for TPU-by-year fixed effects

[16] An exception is Nitrogen dioxide, which also exceeds the WHO standard.

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.

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.[17]

5 Results

We present key results in Table 2. 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 the likelihood of low birthweight (<2,500g) for OLS and IV specifications. Columns (5) and (6) present estimates for the effects of gestational PM_{10} exposure on the likelihood of neonatal death for OLS and IV specifications.

Birthweight We find large effects of gestational PM_{10} exposure on birthweight in a high-PM 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 70 gram reduction in birthweight (Table 2, column (2)).[18] This effect is equivalent to Currie et al. (2009)'s estimated effects of smoking 15 cigarettes per day during gestation. This effect is an order of magnitude larger than estimates in low-PM settings (Chay and Greenstone, 2003b; Currie et al., 2009).

Low Birthweight Consistent with our estimates on continuous measures of birthweight, we estimate large effects of gestational PM_{10} exposure on the likelihood of low birthweight (LBW). Using our preferred IV specification, we estimate that a 10 $\mu g/m^3$ increase in PM_{10}

[17] Standard errors are similar if use one-way clustering by TPU, or by date-of-birth.

[18] In specifications without instruments we fail to reject a null of no relationship between gestational PM_{10} exposure and birthweight (Table 2, column (1)).

is associated with a 3.6 percentage point increase in the likelihood of low birthweight (Table 2, column (4)). Again, the effect is very large relative to the existing literature in high-income, low-particulate matter settings. We know of no other well-identified estimates of the PM-LBW relationship in other high-particulate matter contexts.

Neonatal Mortality We find limited effects of gestational PM_{10} exposure on neonatal mortality in Hong Kong, despite high levels of PM_{10}. Using our preferred IV specification, point estimates 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 high income settings like the US and high pollution settings like mainland China (Chay and Greenstone, 2003a,b; Knittel et al., 2016; Bombardini and Li, 2020; Tanaka, 2015; Chen et al., 2013; He et al., 2016; Fan et al., 2020).

Although our PM-mortality point estimates are small and we fail to reject the null of no marginal effects, our preferred IV estimate is noisy. Our 95% confidence interval doesn't rule out practically meaningful increases in neonatal mortality. We note: (1) neonatal deaths are a rare event in Hong Kong; (2) we estimate more precise null effects on cause-specific neonatal mortality, like respiratory and cardiovascular deaths (Table B4); (3) dropping probabilistic deaths reduces standard errors by 30% (Table B5); (4) we reject a null using the same data for the effects of PM_{10} on birthweight (a continuous measure) and LBW (a binary measure). Failure to reject a null hypothesis can be particularly informative in settings with large datasets and in contexts where existing published results reject the null (Abadie, 2020). More importantly, we note that even our upper confidence limits are small relative to the bulk of the existing literature in high-PM settings.[19]

[19]For example, our upper confidence limit is similar in magnitude to the central estimate of PM_{10} on neonatal mortality in Mexico (Arceo et al., 2016) and smaller than the implied central estimates for West Africa and sub-Saharan Africa (Heft-Neal et al., 2019; Adhvaryu et al., Forthcoming). See Appendix E for further discussion.

Additional Results and Robustness Tests In Appendix B we provide supporting evidence. As noted previously, our results are robust to estimating reduced form relationships between thermal inversions and infant health (Table B2) or estimating relationships between PM and infant health controlling for other pollutants (Table B3). Results are also robust to alternative measures of particulate matter like $PM_{2.5}$ and average daily maximum PM_{10} exposure during gestation (Table B6 and Table B7). We confirm that our results are not driven by a specific choice of distance radii (Table B8).

Table B9 presents IV estimates for the effects of gestational particulate matter exposure by trimester. IV estimation by trimester generates small first stage F-statistics, suggesting a 'many weak' instruments problem. Nevertheless, combining trimester-specific estimates produces results similar to preferred main estimates. Reduced form relationships between infant health by trimester and thermal inversions suggest large relationships between birthweight / low birthweight and particulate exposure in the second and third trimesters (Table B10). In all approaches, we estimate small and significantly insignificant trimester-specific effects on neonatal mortality.

We do not find any effect of contemporaneous exposure to particulate matter on neonatal mortality (Table C1). 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.

Heterogeneity We explore several margins of heterogeneity. Table B11 explores heterogeneity by sex. We continue to find significant and large average effects of gestational PM exposure on birthweight and low birthweight. Coefficient magnitudes suggest girls experience somewhat lower marginal damages than boys. However, we find no statistical difference in marginal damages between girls and boys. We find no effects of gestational PM exposure on neonatal mortality for either girls or boys.

Tables B12 and B13 explore heterogeneity by education and employment. In both cases,

we continue to find significant and large average effects of gestational PM exposure on birthweight and low birthweight. The magnitude of the coefficients in Table B12 suggest that children born to at least one parent with college experience have somewhat smaller marginal birthweight damages than children with parents that have no college experience. The magnitude of the coefficients in Table B13 suggest that children born into households where at least one parent is working experience somewhat smaller marginal birthweight damages than households where no parent is working. However, we find no statistical difference in marginal damages by education or employment status.

We note caution for interpreting heterogeneity results. First, interaction terms involving socio-demographics should not be interpreted as causal. Any given socio-economic indicator may be correlated with other factors and so coefficients represent sub-population differences rather than causal differences. Second, although interaction point estimates are relatively small, effects may be underpowered statistically. Back of the envelope calculations suggest we would need to have uncovered large interaction magnitudes for them to be statistically significant.

Conditional on the above caveats, heterogeneity results suggest two lessons. First, limited detected heterogeneity is generally consistent with free access to health care moderating PM-birthweight disparities across socio-economic status within Hong Kong. Second, limited detected heterogeneity may suggest that pollution information or short-run avoidance behaviors are unlikely to be first-order channels driving our main results. After instrumenting, we detect similar marginal damages for educated vs. uneducated households and for economically active households vs. households without a working parent. The health information and environmental avoidance behavior literatures emphasize that these channels are typically associated with statistical relationships between marginal damages and proxies for education and/or socio-economic status (Grossman, 1972; Ippolito and Mathios, 1990; Neidell, 2004; Shimshack and Ward, 2010; Graff Zivin et al., 2011).

6 Discussion

In Hong Kong, marginal changes in particulate matter exposure are associated with large changes in birthweight but small changes in neonatal mortality. These findings contrast sharply with existing evidence from high-PM, low-income settings and low-PM, high-income settings.

Our large birthweight results are consistent with convex dose response relationships between gestational PM_{10} and birthweight, where marginal damages are increasing in exposure. Hong Kong has high PM levels and birthweight is highly responsive to changes in PM. Our birthweight results are also consistent with marginal PM-birthweight damages that are not particularly moderated by the economic and policy environment. Hong Kong has high per capita income, has free access to excellent health care, and its baseline health indicators are among the best in the world. Yet, we find very large effects of particulate matter on both continuous and discrete birthweight outcomes.

In contrast, our small neonatal mortality results are consistent with marginal damages that are moderated by the economic and policy environment, regardless of the shape of the dose-response function. Hong Kong's free access to high-quality health care, good economic conditions, and other factors that come with economic development may overwhelm typical drivers of marginal PM-mortality relationships. Despite high particulate matter levels in Hong Kong, the marginal effects of PM on neonatal mortality are small.

A natural question is why the economic and policy environment might matter less for birthweight and more for neonatal mortality. One explanation is that anticipating problems and intervening is challenging when the baby is still in the womb. As such, birthweight effects may be less responsive to health and other institutions. By contrast, neonatal mortality in a post-birth environment may be more easily averted through healthcare and 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). Modern medical practice in high-income settings aggressively targets interventions towards at-risk newborns, and technology options to reduce neonatal deaths have expanded dramatically in recent decades (Almond et al., 2010).[20]

Cross-Institutional Comparisons To contextualize our results, and to explore whether macro-level wealth, health, and institutions are more generally associated with smaller marginal pollution-mortality damages, we combine our stark neonatal mortality results with estimates from published studies. We perform cross-institutional comparisons and explore cross-institutional correlates of PM-mortality damages.[21] Absent a credible cross-country identification strategy, the goal of the exercise is to use micro-level evidence from institutions explored in the literature to illuminate broader macro-level economic phenomena (Oster et al., 2016; Meager, 2019).

The closely related literature establishes important empirical regularities and patterns, but its widely varying approaches to quantification make systematic cross-institutional comparisons challenging. For our comparisons, we focus on papers that allow us to identify: (1) a source of exogenous variation for identification; (2) measurable changes in particulate matter air pollution; and (3) outcomes definable by infant or neonatal deaths per 1000 live births. Studies must also provide sufficient detail to put analysis in context. This requires summary statistics like mean pollution and mean neonatal and/or infant mortality rates.

To be more precise, we first consider the set of studies that exploit exogenous sources of variation for identification. We omit studies from the broader related literature that aim to document descriptive associations between air pollution and health outcomes. We then focus on papers that allow us to identify the effects of marginal changes in particulate matter air pollution. We consider studies defining exposure measures with total suspended particulates (TSP), particulate matter less than 10 microns (PM_{10}), and particulate matter less than

[20] We do not observe antepartum mortality. It is possible that our neonatal mortality effects are influenced by selection from antepartum mortality. This has limited implications for any of this paper's conclusions.

[21] This exercise is hard to replicate and interpret for PM-birthweight relationships because there are few internally valid estimates of marginal PM-birthweight effects, and nearly all come from high-income, low-PM settings. This reiterates a contribution of this study, providing birthweight estimates in a high-PM setting.

2.5 microns ($PM_{2.5}$). We convert TSP and $PM_{2.5}$ exposure measures into units of PM_{10} using a PM_{10} to TSP ratio of 0.55 and a $PM_{2.5}$ to PM_{10} ratio of 0.70. We do not explore studies exploring treatments defined by other pollutants like sulfur dioxide or nitrogen dioxide (Leuchinger, 2014; Barrows et al., 2019). We do not examine studies exploring the impacts of exogenous shocks (like wildfires) that cannot be readily converted into measurable changes in particulate matter without ad hoc assumptions (Jayachandran, 2009).

We next consider the subset of papers with outcomes definable in terms of neonatal or infant mortality per 1000 births. Where only estimates of the relationship between particulate matter and infant mortality are available, we assume a constant treatment effect and convert the effect on infant mortality to a neonatal mortality estimate by multiplying the estimate by the context-specific (i.e. paper-specific) ratio of neonatal mortality rates to infant mortality rates. We do not examine papers from the distinguished literature exploring the effects of pollution on adult mortality (Dockery et al., 1993; Chay et al., 2003; Pope et al., 2002; He et al., 2016; Deryugina et al., 2019; He et al., 2020; Cheung et al., 2020; Fan et al., 2020) We do not consider studies that focus on mortality for other age groups (Janke et al., 2009; Cheung et al., 2020). We also do not explore papers that make standardized comparisons unusually difficult, like those with results expressed as risk ratios across percentile bins.

Using our criteria, we standardize 14 estimates from the literature to represent the marginal effect of a nine month change in PM_{10} on neonatal mortality. We also convert relative marginal PM-mortality effects to absolute marginal PM-mortality effects, since a given percentage reduction in neonatal mortality means something different in settings with vastly different baseline conditions. Details on our final sample of comparison estimates are available in Appendix E . Chay and Greenstone (2003a), Chay and Greenstone (2003b), and Knittel et al. (2016) investigate the effects of TSP and PM_{10} on neonatal and infant deaths in the United States. Chen et al. (2013), Tanaka (2015), and Bombardini and Li (2020) explore the effect of TSP and PM_{10} on infant and neonatal mortality in mainland China. Arceo et al. (2016), Cesur et al. (2016), and Pullabhotla (2019) investigate the effects of

PM_{10} on neonatal and infant deaths in Mexico, Turkey, and India. Heft-Neal et al. (2018), Heft-Neal et al. (2019), and Adhvaryu et al. (Forthcoming) estimate the impacts of $PM_{2.5}$ on neonatal and infant mortality for multiple countries in sub-Saharan Africa and West Africa. Our study explores the effect of PM_{10} on neonatal mortality in Hong Kong.

We acknowledge that we have surely not captured every possible study. We also admit that, regardless of the number of studies, our comparative interpretation exercise sacrifices precise identification. Nevertheless, this interpretation exercise grapples directly with issues of external validity and helps to inform broader economic questions like the macro-level correlates of marginal pollution damages highlighted in the conceptual framework (and Hsiang et al. (2019)).

Figure 1 summarizes the results of our comparative exercise. We find that marginal PM-mortality damages are sharply decreasing in baseline health across institutions, as measured by baseline neonatal mortality rates. We find that marginal PM-mortality damages are decreasing in GDP per capita across institutions. By contrast, we find only a weak relationship between average particulate matter concentrations and marginal mortality damages. In light of our conceptual framework, these comparative results suggest that vulnerability to particulate matter exposure may be more important than particulate matter exposure itself in explaining differences in marginal mortality damages across countries. To be clear, we do not assert that baseline particulate matter exposure does not matter but rather that marginal mortality damages may be linear in exposure.

Implications Taken as a whole, our birthweight and neonatal mortality analyses suggest that cost effectively improving environmental health entails investments in both pollution abatement and economic development. Our marginal particulate matter-birthweight 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, may require investments in pollution abatement irrespective of a population's wealth and health.

However, our marginal PM-mortality results suggest that pollution abatement alone may not represent cost-effective policy either. A population's wealth and health appears to play an important role in reducing marginal mortality damages from particulate matter exposure.

Conventional wisdom suggests that marginal pollution damages are high in less-developed countries because they are highly polluted. We provide early evidence that marginal particulate matter damages are high in less-developed countries because they are more polluted *and* because they are less developed. We do not intend to be the final word on these issues, and we hope to inform further research. Nevertheless, with respect to birthweight, we provide suggestive evidence that marginal pollution damages are high in less developed countries because they are highly polluted. With respect to neonatal mortality, we provide suggestive evidence that marginal pollution damages are high in less-developed countries because they are less developed.

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Tables and Figures

Table 1: Descriptive Statistics

	(1) Mean	(2) Std. Dev.	(3) Observations
Panel A: Outcome Variables			
Neonatal Death (× 1,000)	1.150	29.113	532,726
Birthweight (grams)	3,130	482	532,726
LBW (<2,500g)	0.064	0.246	532,726
Panel B: Pollution Variables			
PM_{10} ($\mu g/m^3$)	54.307	8.636	532,726
Carbon Monoxide (ppm)	0.849	0.187	532,726
Ozone ($\mu g/m^3$)	33.068	5.661	532,726
Sulfur Dioxide ($\mu g/m^3$)	16.842	5.5.424	532,726
Nitrogen Dioxide ($\mu g/m^3$)	69.786	11.716	532,726
Panel C: Instrumental Variables			
Thermal Inversions	1.8	2.295	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

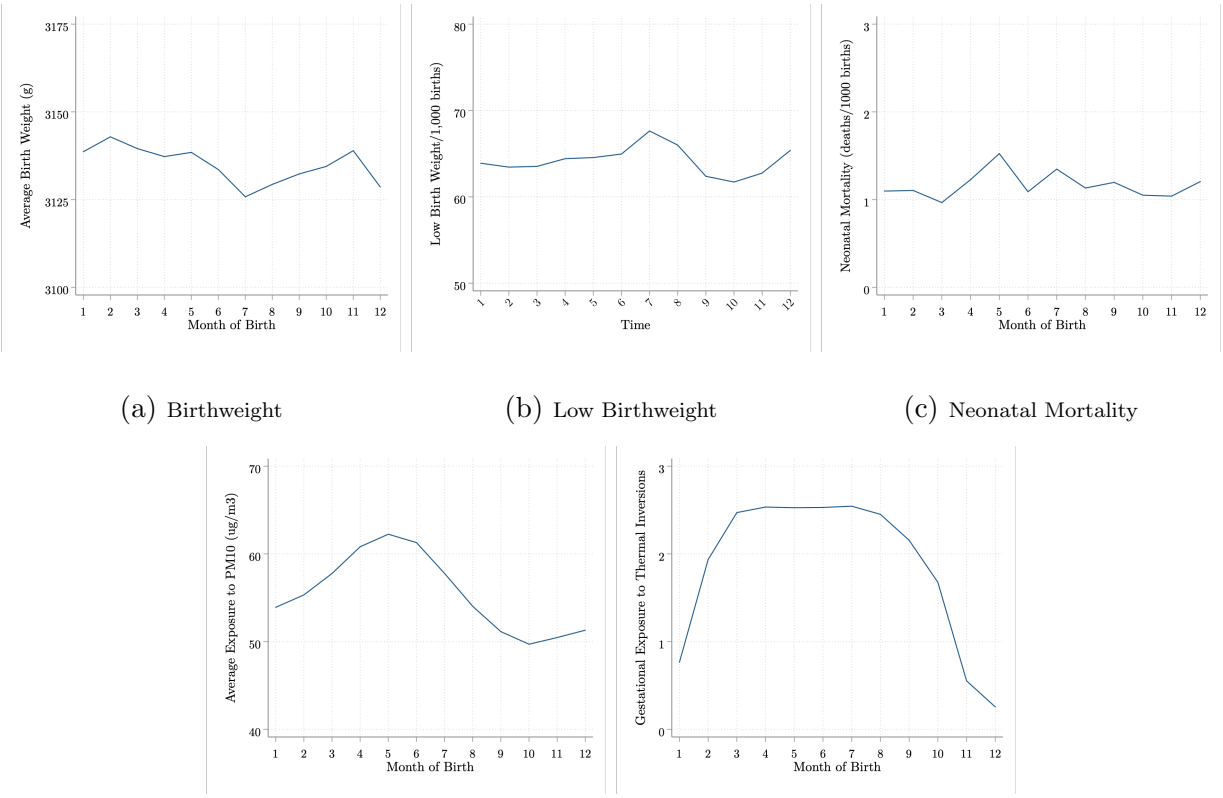
Notes: Outcome variables are measured at birth (birthweight, LBW) or up to the first 28 days after birth (neonatal mortality). All other variables are measured at the individual level during the 270 days of gestation. The implied neonatal mortality rate in Hong Kong is very low. It is the lowest NMR in the world during between 2001 and 2014 (author's caluclations). PM_{10} levels are very high. The World Health Organization standard for annual average PM_{10} concentrations is 20 $\mu g/m^3$. By contrast, Carbon Monoxide (CO), Ozone (O3), and Sulfur Dioxide (SO_2) concentrations are low in Hong Kong, both in absolute terms and relative to other high income-countries. In the United States the National Ambient Air Quality Standard (NAAQS) for CO is an annual mean of 9ppm. In Hong Kong, average gestational exposure during our study period was 0.9ppm. The WHO standard for Ozone is an annual mean of $100\mu g/m^3$. In Hong Kong, average gestational exposure during our study period was $33\mu g/m^3$. The WHO standard for SO_2 is an annual mean of $20\mu g/m^3$. The WHO standard for NO_2 is an annual mean of $40\mu g/m^3$. Hong Kong's climate is sub-tropical. There is less seasonal variability in weather conditions than in other high-income settings.

Table 2: The Effect of Gestational PM_{10} Exposure on Birthweight, LBW, and Neonatal Mortality in Hong Kong

	(1) Birthweight (g)	(2) Birthweight (g)	(3) Low Birthweight (<2,500g)	(4) Low Birthweight (<2,500g)	(5) Neonatal Mortality	(6) Neonatal Mortality
Gestational PM_{10} Exposure	-0.00794	-7.001**	-0.000102	0.00360**	-0.0462	0.00299
	(0.554)	(3.237)	(0.000215)	(0.00145)	(0.0298)	(0.186)
Dependent Variable Mean	3,130	3,130	0.064	0.064	1.150	1.150
TPU × Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes	Yes	Yes	Yes
TPU × Month of Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes	Yes
Specification	OLS	IV	OLS	IV	OLS	IV
First Stage F-Stat	–	59.838	–	59.838	–	59.838
Observations	532,726	532,726	532,726	532,726	532,726	532,726

Notes: This Table presents estimates of the relationship between gestational PM_{10} exposure and our outcomes of interest: birthweight; low birthweight; neonatal mortality. Columns (1), (3), and (5) present OLS estimates. Columns (2), (4), and (6) present 2SLS estimates. 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, Triplet), Number of Previous Births. Pollution controls in columns (1), (3), and (5): prenatal avg. Carbon Monoxide exposure, prenatal avg. Ozone exposure, prenatal avg. Sulfur Dioxide exposure, and prenatal avg. Nitrogen Dioxide exposure.. Standard errors are two-way clustered at the TPU and date-of-birth level.

Figure 1: Within-Year Variation in Pollution, Thermal Inversions, Birthweight, and Neonatal Mortality.

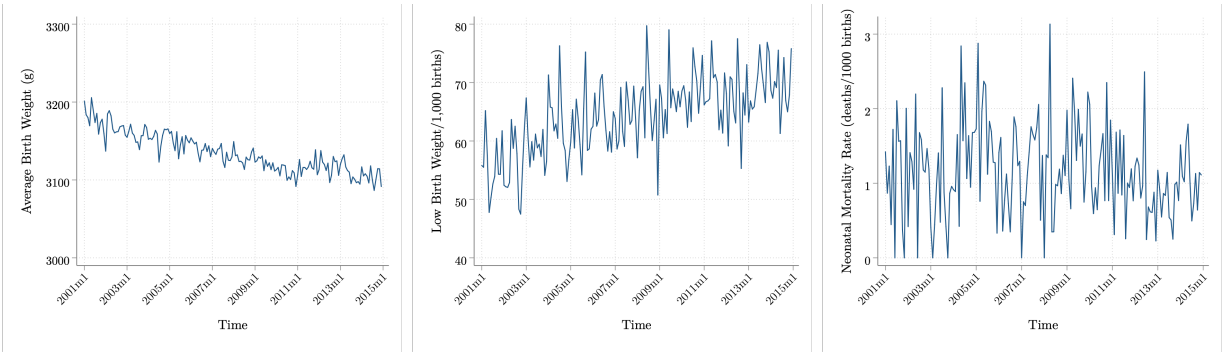


(a) Birthweight (b) Low Birthweight (c) Neonatal Mortality

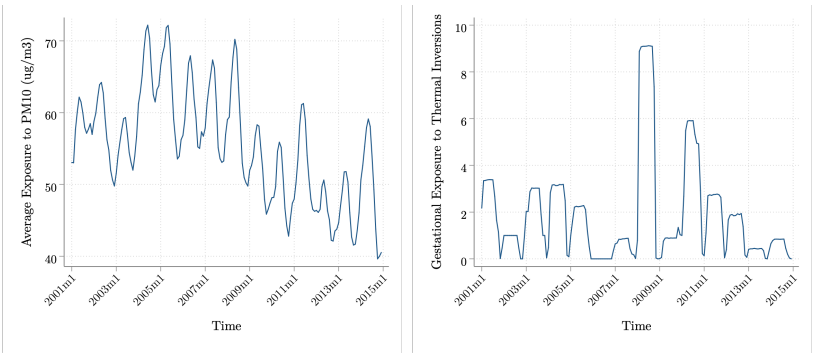
(d) PM_{10} (e) Thermal Inversions

Notes: These figures plot: (a) average birthweight, (b) low birthweight/1,000 live births, (c) the average neonatal mortality rate/1,000 live births, (d) average PM_{10} exposure during gestation, and (e) average thermal inversion exposure during gestation by month of birth.

Figure 2: Full Sample Time-Series Variation in Pollution, Thermal Inversions, Birthweight, and Neonatal Mortality.



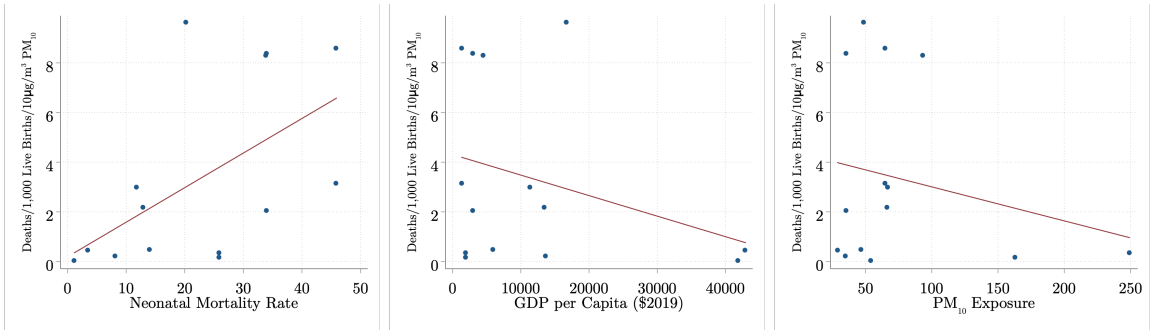
(a) Birthweight (b) Low Birthweight (c) Neonatal Mortality



(d) PM_{10} (e) Thermal Inversions

Notes: These figures plot: (a) average birthweight, (b) low birthweight/1,000 live births, (c) the average neonatal mortality rate/1,000 live births, (d) average PM_{10} exposure during gestation, and (e) average thermal inversion exposure during gestation for each month of our sample period (2001-2014).

Figure 3: The Relationship between Marginal Neonatal Mortality Damages and GDP per Capita, Baseline Health (NMR), and Baseline PM_{10} Exposure



(a) Neonatal Mortality Rate (b) GDP per Capita (c) PM_{10} Exposure

Notes: These results explore the broader relevance of our neonatal mortality results. The data points used in these figures are based on 14 estimates from the literature, including our own analysis of the relationship between gestational PM_{10} exposure and neonatal mortality in Hong Kong. Further details on how comparable estimates were constructed can be found in Appendix E. The figures plot the relationship between the marginal neonatal mortality damages associated with 9-month PM_{10} exposure and three population characteristics. These are: (a) the Neonatal Mortality Rate (a proxy for baseline health); (b) GDP per Capita; (c) average PM_{10} concentrations. In panels (a) we see that there is a positive association between marginal mortality damages and the neonatal mortality rate of a given population – marginal damages are lower in populations with better health. In panel (b) we see that marginal mortality damages are negatively associated with GDP per capita – higher income locations are estimated to have lower marginal mortality damages. In panel (c) we see that there is a weak negative association between marginal mortality damages and baseline particulate matter concentrations – higher pollution locations are not associated with higher marginal damages.

Online Appendices – Not for Publication

Contents

A Additional Descriptive Statistics

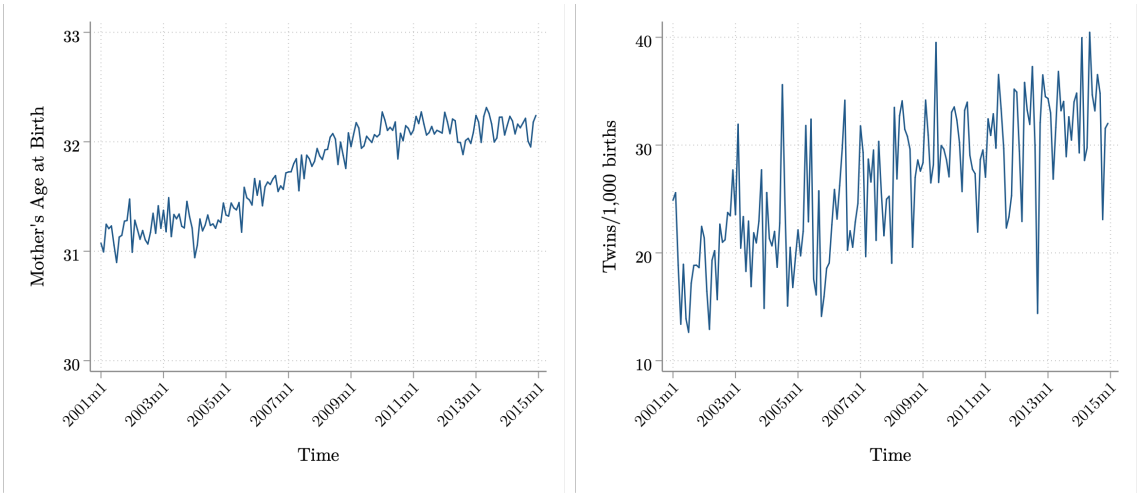
In Figures 2a and 2b we document that average birthweight is decreasing over time and the number of low birthweights (LBW) are increasing over time. In this appendix we present additional descriptive result to provide more insight into these intriguing descriptive facts.

The medical literature suggests that secular trends in birthweight are driven by many factors including demographic composition, maternal age, multiple births, and environmental and health conditions. Declines in birthweight and increases in LBW similar to those that we document for Hong Kong have been observed in other high-income settings such as Japan and Norway (Daltveit et al. 1999; Hokama and Binns 2009). Daltveit et al. (1999) suggests that increasing LBW in Norway was attributable to obstetric procedures like induction or Caesarian procedures that affect gestational age and obstetric procedures like assisted fertilization that increase the incidence of multiple births.

We explored whether descriptive statistics might support similar explanations in our context. Figure A1a documents that average maternal age is sharply increasing for much of our sample period. In 2001, average maternal age was approximately 31. In 2014, average maternal age exceeded 32. Figure A1b documents that multiple births also trend upwards over time. In 2001, the average number of twins per 1000 births was approximately 20. In 2014, the average number of twins per 1000 births exceeded 30.

Although this evidence does definitively establish the causal mechanism driving the secular trends in birthweight/low birthweight, our descriptive evidence is consistent with mechanisms highlighted in the epidemiology literature.

Figure A1: Full Sample Time-Series Variation in Mother's Age and the Number of Twins per 1,000 live births.



(a) Mother's Age

(b) Twins/1,000 live births

Notes: These figures plot: (a) the average age of the Mother and (b) the number of twins/1,000 live births for each month of our sample period (2001-2014).

B Additional Results and Robustness Tests

Table B1: The First Stage Relationships between Gestational Exposure to Thermal Inversions and Gestational Pollution Exposure in Hong Kong

	(1) PM_{10}	(2) CO	(3) O3	(4) SO_2	(5) NO_2
Gestational Exposure to Thermal Inversions	0.220***	0.00301***	-0.0332*	0.0288**	0.0465**
	(0.0284)	(0.000678)	(0.0201)	(0.0131)	(0.0191)
First Stage F-Stat	59.838	19.672	2.735	4.795	5.897
Fixed Efffects	TPU × Year and Month × TPU				
Weather Controls	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes
Dependent Variable Mean	54.307	0.849	33.069	16.842	11.761
Observations	532,726	532,726	532,726	532,726	532,726

Notes: This Table presents first-stage estimates of the relationship between gestational exposure to thermal inversions and gestational exposure to: PM_{10}; Carbon Monoxide (CO); Ozone (O3); Sulfur Dioxide (SO_2); Nitrogen Dioxide (NO_2). Gestational exposure to thermal inversions is defined as the number of thermal inversions that occur during the 270 days of gestation. 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, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

Table B2: The Reduced Form Effect of Gestational Exposure to Thermal Inversions on Birthweight, LBW, and Neonatal Mortality in Hong Kong

	(1) Birthweight (g)	(2) Low Birthweight (<2,500g)	(3) Neonatal Mortality
Gestational Exposure to Thermal Inversions	-1.540**	0.000793**	0.000657
	(0.674)	(0.000305)	(0.0410)
Dependent Variable Mean	3,130	0.064	1.150
TPU × Year FE	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes
TPU × Month FE	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes
Observations	532,726	532,726	532,726

Notes: This Table presents estimates of the reduced form relationship between our outcomes of interest and gestational thermal inversion exposure . We find that an increase in the number of thermal inversions sufficient to deliver a 1 $\mu g/m^3$ increase in average gestational PM_{10} results in almost identical coefficient estimates to those provided by our 2SLS coefficients. 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, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

Table B3: The Effect of Gestational PM_{10} Exposure on Birthweight, LBW, and Neonatal Mortality in Hong Kong (including Pollution Controls)

	(1) Birthweight (g)	(2) Low Birthweight (<2,500g)	(3) Neonatal Mortality
Gestational PM_{10} Exposure	-7.920**	0.00410**	0.0112
	(3.678)	(0.00164)	(0.207)
Dependent Variable Mean	3,130	0.064	1.150
TPU × Year FE	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes
TPU × Month FE	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes
First Stage F-Stat	67.774	67.774	67.774
Observations	532,726	532,726	532,726

Notes: This Table presents estimates from an analysis that explores the robustness of our results to controlling for other criteria pollutants (Carbon Monoxide, Ozone, Sulfur Dioxide, and Nitrogen Dioxide). We find that our estimates are largely unchanged. Pollution controls: prenatal avg. Carbon Monoxide exposure, prenatal avg. Ozone exposure, prenatal avg. Sulfur Dioxide exposure, and prenatal avg. Nitrogen Dioxide exposure. 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, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

Table B4: The Effect of Gestational PM_{10} Exposure on Neonatal Mortality in Hong Kong, by Cause of Death

	(1) Neonatal Mortality (Respiratory)	(2) Neonatal Mortality (Cardiovascular)	(3) Neonatal Mortality (Other)
Gestational PM_{10} Exposure	0.0545	-0.00837	-0.0444
	(0.0608)	(0.0481)	(0.164)
Dependent Variable Mean	0.176	0.162	0.813
Fixed Effects	TPU × Year, TPU × Month		
Controls	Weather Controls and Individual Controls		
First Stage F-Stat	59.622	59.625	59.822
Observations	531,807	531,806	532,447

Notes: This Table presents estimates of the relationship between gestational PM_{10} exposure and neonatal mortality, by cause of death. We explore respiratory deaths, cardiovascular deaths, and deaths from all other causes. We fail to reject the null hypothesis for all categories. 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, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

Table B5: The Effect of Gestational PM_{10} Exposure on Birthweight, LBW, and Neonatal Mortality in Hong Kong (No Probabilistic Deaths)

	(1) Birthweight (g)	(2) Low Birthweight (<2,500g)	(3) Neonatal Mortality
Gestational PM_{10} Exposure	-6.995**	0.00360**	0.0130
	(3.221)	(0.00146)	(0.129)
Dependent Variable Mean	3,131	0.064	0.622
Fixed Effects	TPU × Year, TPU × Month		
Controls	Weather Controls and Individual Controls		
First Stage F-Stat	59.662	59.662	59.662
Observations	531,999	531,999	531,999

Notes: This Table presents estimates of the relationship between gestational PM_{10} exposure and our outcomes of interest, after dropping probabilistic deaths from the sample. 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, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

Table B6: The Effect of Gestational Average Daily Maximum PM_{10} Exposure on Birthweight, LBW, and Neonatal Mortality in Hong Kong

	(1) Birthweight (g)	(2) Low Birthweight (<2,500g)	(3) Neonatal Mortality
Gestational Max PM_{10} Exposure	-5.905**	0.00304**	0.00252
	(2.723)	(0.00123)	(0.157)
Dependent Variable Mean	3,130	0.064	1.150
TPU × Year FE	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes
TPU × Month FE	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes
First Stage F-Stat	54.861	54.861	54.861
Observations	532,719	532,719	532,719

Notes: This Table presents estimates from an analysis of whether our results are affected by a different measure of gestational pollution exposure (the average of daily maximum PM_{10} concentrations measured over the gestational period.). We estimate similar effects. 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, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

Table B7: The Effect of Gestational $PM_{2.5}$ Exposure on Birthweight, LBW, and Neonatal Mortality in Hong Kong

	(1) Birthweight (g)	(2) Low Birthweight (<2,500g)	(3) Neonatal Mortality
Gestational $PM_{2.5}$ Exposure	-6.535**	0.00304**	0.0212
	(2.571)	(0.00123)	(0.169)
Dependent Variable Mean	3,130	0.064	1.150
TPU × Year FE	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes
TPU × Month FE	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes
First Stage F-Stat	71.828	71.828	71.828
Observations	502,812	502,812	502,812

Notes: This Table presents estimates exploring the robustness of our results to using $PM_{2.5}$ concentrations, instead of PM_{10}. This results in a different sample because $PM_{2.5}$ is not as well monitored over our sample period. Our results remain robust. 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, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

Table B8: The Effect of Gestational PM_{10} Exposure on Birthweight, LBW, and Neonatal Mortality in Hong Kong (Different Maximum Distance-to-Monitor Restrictions)

	(1) Birthweight (g)	(2) Low Birthweight (<2,500g)	(3) Neonatal Mortality
Panel A: 10km			
Gestational PM_{10} Exposure	-7.001**	0.00360**	0.00299
	(3.237)	(0.00145)	(0.186)
First Stage F-Stat	59.838	59.838	59.838
Observations	532,726	532,726	532,726
Panel B: 8km			
Gestational PM_{10} Exposure	-8.292**	0.00290*	0.00790
	(3.766)	(0.00168)	(0.215)
First Stage F-Stat	44.730	44.730	44.730
Observations	432,071	432,071	432,071
Panel C: 12km			
	-7.039**	0.00393**	-0.0272
	(3.100)	(0.00152)	(0.176)
First Stage F-Stat	62.712	62.712	62.712
Observations	556,211	556,211	556,211
TPU × Year FE	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes
TPU × Month FE	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes

Notes: This Table presents estimates from an analysis that explores how robust our results are to adjusting the maximum distance to pollution monitors. Our main results (restricting the maximum distance to 10km) are presented in Panel A. Panel B restricts the maximum distance to 8km. Panel C expands the maximum distance to 12km. Our results remain robust across the different samples. 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, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

Table B9: The Effect of Gestational PM_{10} Exposure on Birthweight, LBW, and Neonatal Mortality in Hong Kong (Trimester-specific effects)

	(1) Birthweight (g)	(2) Low Birthweight (<2,500g)	(3) Neonatal Mortality
First Trimester PM_{10} Exposure	-2.672	0.000883	-0.219
	(5.332)	(0.00253)	(0.323)
Second Trimester PM_{10} Exposure	-5.942	0.00182	-0.200
	(7.097)	(0.00330)	(0.427)
Third Trimester PM_{10} Exposure	-1.996	0.00149	0.0329
	(2.564)	(0.00111)	(0.148)
H0: T1 + T2 + T3 = 0	-10.610	0.004	-0.386
	(10.456)	(0.0049)	(0.624)
Dependent Variable Mean	3,130	0.064	1.150
TPU × Year FE	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes
TPU × Month FE	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes
First Stage F-Stat	1.059	1.059	1.059
Observations	532,525	532,525	532,525

Notes: This Table presents estimates from an analysis of trimester-specific PM_{10} exposure on birth weight and neonatal mortality. Trimester-specific weather controls: Avg. trimester max temperature, avg. trimester min temperature, avg. trimester daily rainfall, avg. trimester humidity, avg. trimester surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

Table B10: The Effect of Thermal Inversion Exposure on Birthweight, LBW, and Neonatal Mortality in Hong Kong (Trimester-specific effects)

	(1) Birthweight (g)	(2) Low Birthweight (<2,500g)	(3) Neonatal Mortality
First Trimester Thermal Inversion Exposure	-1.534*	0.000574	-0.0121
	(0.872)	(0.000422)	(0.0529)
Second Trimester Thermal Inversion Exposure	-2.287**	0.00130***	-0.0432
	(0.956)	(0.000460)	(0.0551)
Third Trimester Thermal Inversion Exposure	-2.280***	0.000960**	0.0283
	(0.839)	(0.000396)	(0.0590)
Dependent Variable Mean	3,130	0.064	1.150
TPU × Year FE	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes
TPU × Month FE	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes
Observations	532,525	532,525	532,525

Notes: This Table presents estimates from an analysis of trimester-specific thermal inversion exposure on birth weight and neonatal mortality. Trimester-specific weather controls: Avg. trimester max temperature, avg. trimester min temperature, avg. trimester daily rainfall, avg. trimester humidity, avg. trimester surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

Table B11: The Effect of Gestational PM_{10} Exposure on Birthweight, LBW, and Neonatal Mortality in Hong Kong (by Sex)

	(1) Birthweight (g)	(2) Low Birthweight (<2,500g)	(3) Neonatal Mortality
Gestational PM_{10} Exposure	-7.300**	0.00372**	0.00368
	(3.246)	(0.00148)	(0.187)
PM_{10} Exposure × Female	0.597	-0.000231	-0.00139
	(0.719)	(0.000397)	(0.0510)
H0: PM_{10} + PM_{10} × Female = 0	-6.703**	0.0035**	0.0023
	(3.268)	(0.0015)	(0.189)
Dependent Variable Mean	3,130	0.064	1.150
TPU × Year FE	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes
TPU × Month FE	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes
First Stage F-Stat	29.914	29.914	29.914
Observations	532,726	532,726	532,726

Notes: This Table presents explores heterogeneity in the effects of gestational PM_{10} exposure by sex. Weather controls: Avg. max temperature, avg. min temperature, avg. daily rainfall, avg. humidity, avg. surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

Table B12: The Effect of Gestational PM_{10} Exposure on Birthweight, LBW, and Neonatal Mortality in Hong Kong (Educational Heterogeneity)

	(1) Birthweight (g)	(2) Low Birthweight (<2,500g)	(3) Neonatal Mortality
Gestational PM_{10} Exposure	-6.575**	0.00340**	0.0228
	(3.227)	(0.00144)	(0.188)
PM_{10} Exposure × No College	-1.106	0.000462	-0.0536
	(0.826)	(0.000424)	(0.0502)
H0: PM_{10} + PM_{10} × No College = 0	-7.680**	0.0038**	-0.030
	(3.330)	(0.0015)	(0.186)
Dependent Variable Mean	3,130	0.064	1.150
TPU × Year FE	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes
TPU × Month FE	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes
First Stage F-Stat	29.992	29.992	29.992
Observations	532,726	532,726	532,726

Notes: This Table presents explores heterogeneity in the effects of gestational PM_{10} exposure by education. We define a binary variable to be equal to one if neither parent is economically active. Weather controls: Avg. max temperature, avg. min temperature, avg. daily rainfall, avg. humidity, avg. surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

Table B13: The Effect of Gestational PM_{10} Exposure on Birthweight, LBW, and Neonatal Mortality in Hong Kong (Economic Activity Heterogeneity)

	(1) Birthweight (g)	(2) Low Birthweight (<2,500g)	(3) Neonatal Mortality
Gestational PM_{10} Exposure	-6.851**	0.00354**	0.0228
	(3.262)	(0.00145)	(0.188)
PM_{10} Exposure × Not Economically Active	-3.629	0.00178	-0.0544
	(3.709)	(0.00167)	(0.163)
H0: PM_{10} + PM_{10} × Not Economically Active = 0	-10.480**	0.0053**	-0.0049
	(4.410)	(0.0021)	(0.230)
Dependent Variable Mean	3,130	0.064	1.150
TPU × Year FE	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes
TPU × Month FE	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes
First Stage F-Stat	29.992	29.992	29.992
Observations	532,726	532,726	532,726

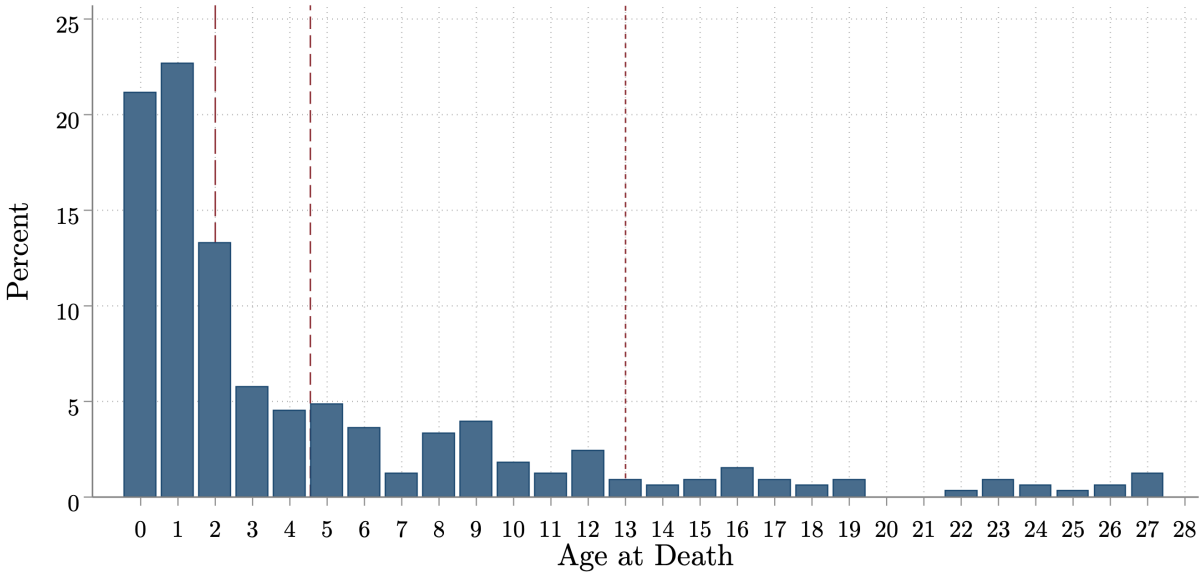
Notes: This Table presents explores heterogeneity in the effects of gestational PM_{10} exposure by education. We define a binary variable to be equal to one if neither parent has at least some college experience. Weather controls: Avg. max temperature, avg. min temperature, avg. daily rainfall, avg. humidity, avg. surface pressure. Individual controls, (included as dummy variables): Mother's age, Sex of the Baby, Type of Birth (Single, Twin, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

C Contemporaneous Exposure

In Table C1 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). 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.[22] 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 C1: 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).

[22]The distribution of age at death is presented in Figure C1.

Table C1: The Association Between Contemporanoeus PM_{10} Exposure and Neonatal Mortality in Hong Kong

	(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: This Table presents estimates from an analysis of the relationship between post-natal average PM_{10} exposure (up to the first 28 days) and neonatal mortality. The results are estimated using OLS. The first stage of our instrumental variable approach is not relevant when exploring contemporaneous exposure to PM_{10}. 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, Triplet), Number of Previous Births. Standard errors are two-way clustered at the TPU and date-of-birth level.

D Awareness of Pollution Exposure

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-year fixed effects, 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.

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

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. We posit that if awareness of pollution is empirically relevant then we would expect larger effects for particulate matter, which is visible, and smaller effects for Ozone and Carbon Monoxide, which are not visible.

The results of our analysis can be found in table D1. 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 D1: The Effect of Daily Average Air Pollution Exposure on Football Game Attendance in Hong Kong

	(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: This Table presents estimates from an analysis of the relationship between daily pollution exposure (PM_{10}, CO, and O3) and football game attendance. We find that higher PM_{10} exposure is associated with reductions in attendance. We do not find that higher exposure to CO or O3 is associated with reduced attendance. Weather controls include maximum and minimum temperature, total rainfall, surface pressure, and average humidity. Standard errors are clustered at the TPU level.

E Comparison Studies and Calculations

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

Chay and Greenstone (2003a)

Sample period: 1969-1974
Context: USA
GDP per capita: $5,987
Average PM_{10} Exposure: $41.58\mu g/m^3$-$51.81\mu g/m^3$ (annual average)
Infant mortality rate: 18.7 deaths per 1,000 births
Neonatal mortality rate: 14 deaths per 1,000 births
Measure of Exposure: Pre-Natal.

Using an IV strategy they estimate that a 10 $\mu g/m^3$ increase in average pre-natal TSP is associated with 0.7-1.3 more infant deaths per 1,000 live births.[23] Following Knittel et al. (2016) we apply a commonly used conversion metric of 0.55TSP = PM_{10}. This translates a 10 $\mu g/m^3$ increase in PM_{10} into 0.39 - 0.72 more infant deaths per 1,000 live births. Using the ratio between the neonatal mortality rate and infant mortality rate we attribute 74% of the estimated deaths to neonatal mortality, resulting in 0.29 - 0.53 more neonatal deaths per 1,000 live births.

Chay and Greenstone (2003b)

Sample period: 1978-1984
Context: USA
GDP per capita: $13,698
Average PM_{10} Exposure: $31.02\mu g/m^3$-$39.11\mu g/m^3$ (annual average)
Infant mortality rate: 12.2 deaths per 1,000 births
Neonatal mortality rate: 8.2 deaths per 1,000 births
Measure of Exposure: Pre-Natal.

Using an IV strategy they estimate that a 10 $\mu g/m^3$ increase in average pre-natal TSP is associated with 0.4 more neonatal deaths per 1,000 live births. Following Knittel et al. (2016) we apply a commonly used conversion metric of 0.55TSP = PM_{10}. This translates a 10 $\mu g/m^3$ increase in PM_{10} into 0.22 more neonatal deaths per 1,000 live births.

[23] The coefficients from columns 2 and 5 in Table 6.

Arceo et al. (2016)

Sample period: 1997-2006
Context: Mexico City
GDP per capita: $11,378
Average PM_{10} Exposure: $66.94 \mu g/m^3$
Infant mortality rate: 20.9 deaths per 1,000 births
Neonatal mortality rate: 11.8 deaths per 1,000 births
Measure of Exposure: Weekly Post-Natal.

Using the estimates from their IV strategy we calculate that a 10 $\mu g/m^3$ increase in PM_{10} is associated with an additional 2.97 neonatal deaths per 1,000 live births (NMR).[24] We note that their IV estimate on neonatal deaths is statistically insignificant, but much larger than our own estimate. The authors also estimate statistically significant effects of a smaller magnitude using a fixed effects specification. If we use their fixed effect estimate, which is smaller but statistically significant we calculate that a a 10 $\mu g/m^3$ increase in PM_{10} is associated with an additional 1.402 neonatal deaths per 1,000 live births (NMR).[25] If we use their estimate on infant mortality (Table 3, Column 4) and adjust by the ratio between the neonatal mortality rate and the infant mortality rate, attributing 56% of the estimated deaths to neonatal mortality we calculate that a 10-unit increase in PM_{10} is associated with an additional 0.5 deaths per 1,000 live births.[26] The inferences drawn from our comparative analysis are not sensitive to these choices.

Cesur et al. (2016)

Sample period: 2001-2011
Context: Turkey
GDP per capita: $13,454
Average PM_{10} Exposure: $66.19 \mu g/m^3$
Infant mortality rate: 20.4 deaths per 1,000 births
Neonatal mortality rate: 12.9 deaths per 1,000 births
Measure of Exposure: Annual Post-Natal.

Using an IV strategy they estimate that a 10 $\mu g/m^3$ increase in PM_{10} is associated with an additional 3.017 infant deaths per 1,000 live births.[27] Using the ratio between the neonatal mortality rate and infant mortality rate we attribute 63% of the estimated deaths to neonatal mortality resulting in an additional 1.9 neonatal deaths per 1,000 births.

[24] 0.007625×39 weeks $\times 10\ \mu g/m^3 = 2.97$ (Table 3, Column 3).

[25] 0.003595×39 weeks $\times 10\ \mu g/m^3 = 1.402$ (Table 3, Column 1).

[26] 0.0023×39 weeks $\times$ 10 units = 0.83. $0.83 \times 0.59 = 0.50$.

[27] 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.

Knittel et al. (2016)

Sample period: 2002-2007
Context: California, USA
GDP per capita: $42,937
Average PM_{10} Exposure: $28.94 \mu g/m^3$
Infant mortality rate: 5.77 deaths per 1,000 births
Neonatal mortality rate: 3.52 deaths per 1,000 births
Measure of Exposure: Weekly Post-Natal.

Using the estimates from their IV strategy we calculate that a 10 $\mu g/m^3$ increase in PM_{10} is associated with an additional 0.74 deaths per 1,000 live births (IMR).[28] Using the ratio between the neonatal mortality rate and infant mortality rate we attribute 61% of the estimated deaths to neonatal mortality, resulting in an additional 0.45 deaths per 1,000 live births (NMR).

Heft-Neal et al. (2018)

Sample period: 2001-2015
Context: Sub-Saharan Africa
GDP per capita: $2,965
Average PM_{10} Exposure: 35.71 $\mu g/m^3$
Infant mortality rate: 71.2 deaths per 1,000 births
Neonatal mortality rate: 34 deaths per 1,000 births
Measure of Exposure: Pre-natal.

These numbers are calculated using the replication files provided by Heft-Neal (2018). Using a fixed effects strategy we estimate that a $10 \mu g/m^3$ increase in gestational $PM_{2.5}$ is associated with an additional 1.435 deaths per 1,000 live births (NMR). To convert $PM_{2.5}$ exposure to PM_{10} exposure we use the ratio $PM_{2.5} = 0.7PM_{10}$. The effect of a 10-unit increase in PM_{10} would be associated with an additional 2.05 deaths per 1,000 live births (NMR, gestational exposure).

[28] 0.0019×39 weeks $\times 10 \ \mu g/m^3 = 0.74$ (Table 8, Column 4).

Heft-Neal et al. (2019)

Sample period: 2001-2015
Context: Sub-Saharan Africa
GDP per capita: $2,965
Average PM_{10} Exposure: 35.71 $\mu g/m^3$ (mean exposure)
Infant mortality rate: 71.2 deaths per 1,000 births
Neonatal mortality rate: 34 deaths per 1,000 births
Measure of Exposure: Annual post-natal.

Using an IV strategy the authors estimate that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ in the year following birth is associated with a 22% increase in infant mortality. Relative to the mean this is an additional 15.66 deaths per 1,000 live births (IMR). To convert $PM_{2.5}$ exposure to PM_{10} exposure we use the ratio $PM_{2.5} = 0.7PM_{10}$. We calculate that a 10 $\mu g/m^3$ increase in PM_{10} 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 10.51 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 7.88 neonatal deaths per 1,000 live births.

Heft-Neal et al. (2019)

Sample period: 2001-2015
Context: West Africa
GDP per capita: $1,333
Average PM_{10} Exposure: 65.2$\mu g/m^3$[29]
Infant mortality rate: 102.8 deaths per 1,000 births
Neonatal mortality rate: 45.9 deaths per 1,000 births
Measure of Exposure: Annual post-natal.

Heft-Neal et al. (2019) estimate an IV effect explicitly for West Africa finding that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ is associated with a 17.47% increase in infant mortality. Relative to the mean this is an additional 17.95 deaths per 1,000 live births. To convert $PM_{2.5}$ exposure into PM_{10} exposure we use the ratio $PM_{2.5}$=$0.7PM_{10}$. We calculate that a 10 $\mu g/m^3$ increase in PM_{10} would be associated with an additional 25.63 deaths per 1,000 live births. Using the average ratio between the neonatal mortality rate and infant mortality rate for West Africa we attribute 44% of the estimated deaths to neonatal mortality, resulting in an additional 11.28 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 8.46 deaths per 1,000 live births.

[29]Heft-Neal et al. (2019) do not report summary statistics for the West Africa analysis and so we take average PM_{10} exposure from Advharyu et al. (forthcoming)

Advharyu et al. (forthcoming)

Sample period: 1986-2005
Context: West Africa
GDP per capita: $1,333
Average PM_{10} Exposure: $65.2\mu g/m^3$
Infant mortality rate: 102.8 deaths per 1,000 births
Neonatal mortality rate: 45.9 deaths per 1,000 births
Measure of Exposure: Pre-Natal.

Using a fixed effects strategy the authors estimate that a cumulative 100 $\mu g/m^3$ increase in $PM_{2.5}$ during gestation is associated with 2.455 deaths per 1,000 (Table 2, column 3). An average increase of 10 $\mu g/m^3$ during gestation is equal to 90 $\mu g/m3$. As such, we calculate that a 10 $\mu g/m^3$ increase in average gestational $PM_{2.5}$ would be associated with an additional 2.20 neonatal deaths per 1,000 live births. To convert $PM_{2.5}$ exposure into PM_{10} exposure we use the ratio $PM_{2.5}$=0.7PM_{10}. We calculate that a 10 $\mu g/m^3$ increase in average gestational PM_{10} would be associated with an additional 3.14 neonatal deaths per 1,000 births. We note that the estimate presented in Advharyu et al. is statistically insignificant. If we use their estimate on infant mortality and weight by the share of neonatal deaths we would estimate that a 10 $\mu g/m^3$ increase in average gestational PM_{10} would be associated with an additional 4.42 neonatal deaths per 1,000 births.[30]

Tanaka (2015)

Sample period: 1991-2000
Context: China
GDP per capita: $1,980
Average PM_{10} Exposure: $163.2\mu g/m^3$
Infant mortality rate: 36.61 deaths per 1,000 births
Neonatal mortality rate: 25.85 deaths per 1,000 births
Measure of Exposure: Annual

Tanaka (2015) estimates the effect of the "Two Control Zones" policy in China on infant mortality, finding that it reduced neonatal deaths by 2.08 neonatal deaths per 1,000 live births.[31] In unpublished appendix material, the author also estimates the effect of the policy on TSP, using a different sample (1991-2004). The policy was associated with a 67 $\mu g/m^3$ increase in TSP.[32] Attributing all of the neonatal mortality effect to reductions in TSP would result in 0.39 fewer neonatal deaths per 1,000 live births per 10 $\mu g/m^3$ reduction in TSP. Translating this into PM_{10} using the conversion factor TSP = 0.55PM_{10} this provides an estimate of 0.22 deaths per 1,000 live births for each 10 $\mu g/m^3$ reduction in PM_{10}. We then weight this annual estimate to be 9-months, providing an estimate of 0.16 deaths per 1,000

[30](7.170 infant deaths per 100 $\mu g/m^3$ × 0.9)/0.7 × 0.44 = 4.05 deaths per 1,000 births.
[31]Table 4, column 6.
[32]Table 3.2A, column 3.

live births for each 10 $\mu g/m^3$ reduction in PM_{10}.

Chen et al. (2013)

Sample period: 1991-2000
Context: China
GDP per capita: $1,980
Average PM_{10} Exposure: 249.23 $\mu g/m^3$
Infant mortality rate: 36.61 deaths per 1,000 births
Neonatal mortality rate: 25.85 deaths per 1,000 births
Measure of Exposure: Annual

Chen et al. (2013) estimates the effect of the Huai river policy in China on TSP and infant mortality. Using a regression discontinuity design they estimate that a 100 $\mu g/m^3$ increase in TSP is associated with a 55% increase in infant mortality.[33] First, we convert this into infant deaths. Chen et al. (2013) do not provide descriptive statistics on the infant mortality rate in their sample so we rely on aggregate statistics. Using the aggregate infant mortality rate in China between 1991-2000 we calculate that a 55% increase in infant mortality corresponds to an additional 20.14 deaths per 1,000 live births. Second, we convert this to PM_{10} using the conversion factor 0.55TSP = PM_{10}. A 100 $\mu g/m^3$ increase in PM_{10} is associated with an additional 11.07 deaths per 1,000 live births. We then assign 70% of these deaths as neonatal deaths using the ratio of neonatal deaths to infant deaths during this period (7.75 deaths per 1,000 live births). Finally, we weight this annual estimate to be 9-months, and divide by 10 to get the estimate in 10 $\mu g/m^3$. This provides a final estimate of 0.581 neonatal deaths per 1,000 live births for each 10 $\mu g/m^3$ reduction in PM_{10}.

Bombardini et al. (2020)

Sample period: 1990-2010
Context: China
GDP per capita: $16,679
Average PM_{10} Exposure: 48.79$\mu g/m^3$
Infant mortality rate: 29 deaths per 1,000 births
Neonatal mortality rate: 20.185 deaths per 1,000 births
Measure of Exposure: Annual

Bombardini et al. (2020) estimate that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ is associated with 13.08 deaths per 1,000 live births.[34] We first convert this to PM_{10} using the conversion factor $PM_{2.5}$=0.7PM_{10}. A 10 $\mu g/m^3$ increase in PM_{10} is associated with an additional 18.67 infant deaths/1,000 live births. We then impute the implied number of neonatal deaths using the ratio of neonatal deaths over infant deaths in this context (0.69), implying 12.88 neonatal deaths/1000 live births for each 10 $\mu g/m^3$ increase in PM_{10}. We then weight the annual

[33] Table S4, column 1.
[34] Table 8, Panel B, column 2.

measure by 0.75 to provide a 9 month estimate. This results in 9.66 neonatal deaths/1,000 for for each 10 $\mu g/m^3$ increase in PM_{10}.

Pullabholtra (2020)

Sample period: 2007-2016
Context: India
GDP per capita: $4,501
Average PM_{10} Exposure: $99.3\mu g/m^3$
Infant mortality rate: NA
Neonatal mortality rate: 33.93 deaths per 1,000 births
Measure of Exposure: Pre-Natal

Pullabholtra (2020) estimates that greater exposure to agricultural fires (defined as being upwind of more than 5 fires within 75k) is associated with a 3.325 $\mu g/m^3$ increase in prenatal exposure to PM_{10}.[35] This exposure is associated with an additional 2.757 neonatal deaths per 1,000 live births.[36] Using these numbers we calculate that a 10 $\mu g/m^3$ increase in PM_{10} is associated with 8.291 neonatal deaths/1,000.

Our study

Sample period: 2001-2014
Context: Hong Kong
GDP per capita: $41,814
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: Pre-Natal.

We estimate that a 10 $\mu g/m^3$ increase in average prenatal PM_{10} is associated with an additional 0.02 neonatal deaths per 1,000 live births.

[35] Table 2, column 1.
[36] Table 4, column 1.