

ABSTRACT

Title of Dissertation: **ESSAYS ON THE EFFECTS OF AIR
POLLUTION ON HUMAN HEALTH**

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According to recent reports by The World Bank and the World Health Organization millions of people die every year because of exposure to ambient air pollution—the vast majority of them in developing countries (World Bank 2016; World Health Organization 2016). Policy makers throughout the developing world are starting to seriously address this issue by designing and implementing a battery of policies for reducing ambient air pollution. To weight the cost and benefits of these policies policy makers need estimates of the benefits of reducing ambient air pollution. In this dissertation I provide estimates of the benefits of air pollution reduction in terms of its effects on human health. I use data from Chile, a middle income country that in recent years experienced a period of rapid industrialization and economic growth—similar to the process that many developing economies are experiencing these days. I believe that estimates and methods from this dissertation can provide a valuable tool to aid policy makers in the developing world in their goals to reduce ambient air pollution.

Chapter 1 examines the effects of exposure to ambient air pollution on infant mortality. Using state-of-the art techniques to identify causal effects and reduce possible bias due to measurement error in air pollution exposure, results from this chapter show significant effects of exposure to ambient air pollution on infant mortality. This effect is larger for infant mortality due to respiratory and cardiovascular diseases.

Chapter 2 examines the effect of exposure to ambient air pollution on urgent care visits for different age groups and across different types of urgent care visits. Using a novel strategy to identify causal effects, results from this chapter show a significant effect on respiratory urgent care visits and on cardiovascular and circulatory urgent care visits. This effect is larger for the elderly and for respiratory urgent care visits due to pneumonia and lower respiratory diseases.

Chapter 3 examines the effect of exposure to ambient air pollution on the probability of a pregnancy ending in a stillbirth delivery. Results from this chapter show a significant effect of acute exposure to air pollution on the probability of stillbirth delivery. This effect is larger for those stillbirths that are due to hypoxia.

ESSAYS ON THE EFFECTS OF AIR POLLUTION ON HUMAN HEALTH

by

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Foreword

The first chapter is the product of joint work with Dr. Sebastian Miller, who was my supervisor while I was working at the Research Department of the Inter-American Development Bank. The idea of looking at the effects of air pollution on overall mortality was his, while I contributed by narrowing down the focus to looking only at the effects of air pollution on infant mortality (for reasons explained in the chapter). While Dr. Miller conducted the spatial imputation of air pollutants (using the Kriging method) and built a panel dataset of air pollution, infant mortality rate and weather covariates; I brought in thermal inversion data which allows to instrument for air pollution and thus address potential biases from confounding effects. I conducted the statistical analysis, reviewed the literature and wrote the paper. The second chapter is solely my responsibility. The third chapter is also joint work with Dr. Miller where he contributed with the spatial imputation of air pollutants while I contributed with the data for pregnancy outcomes (stillbirths and livebirths). I reviewed the relevant literature, conducted statistical analysis and wrote the paper. The Dissertation Committee acknowledges that Juan Cristobal Ruiz-Tagle made substantial contributions to the relevant aspects of these chapters.

Dedication

To my wife, for her understanding and unconditional support that made this dissertation possible; and to my parents, for their support in the distance.

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Chapter 1: Ambient Air Pollution and Infant Mortality in Emerging Economies: Evidence from Santiago, Chile

1. Introduction

Exposure to air pollution is known to have damaging effects on human health, particularly to the very young and the elderly (Pope III and Dockery 2006).¹ Estimating the adverse effects of ambient air pollution on human health has important implications for assisting policymakers in weighing the costs and benefits of public policies designed for reducing ambient air pollution concentrations. Moreover, studying ambient air pollution for emerging economies provides some important lessons on the challenges that developing countries will face on their path to development. In this chapter we look at the case of Chile, a country which has experienced a successful growth performance over the past few decades, and that has suffered from the adverse consequences of high concentrations of ambient air pollution during the last 40 years (Instituto de Asuntos Públicos 2003).² We focus on exposure to ambient air pollution in the form of particulate matter of diameter of 10 microns or less (PM₁₀) in Santiago, Chile, and examine the effects on mortality for infants – a particular vulnerable population. We focus on the effects on infant mortality for two reasons. First, since these very young individuals are unlikely to

¹ Exposure to air pollution can cause a constriction of the bronchial system that impairs lung functioning and this in turn can cause respiratory and cardiovascular diseases that may have serious consequences to human health and even cause death (Nadadur and Hollingsworth 2015).

² Chile's per capita GDP increased from US\$9.000 to US\$12.600 between the late 1990s and 2008 (constant 2000, PPP adjusted).

have experienced previous health complications in the past that may affect their health outcomes (particularly, those associated to respiratory and cardiovascular diseases), focusing on the infant population allows to better identify an effect on health outcomes that is actually due to exposure to air pollution. And second, unlike adult mortality or mortality among the elderly, infant mortality represents full life-years lost in terms of life expectancy.

There is a vast epidemiological literature that examines the association between both acute and chronic exposure³ to ambient air pollution and its effects on mortality (Ostro 1993; Dockery et al. 1993; Dockery and Pope 1994; Schwartz 1994).⁴ However, most of the existing epidemiological literature fails to establish a causal relationship between exposure to ambient air pollution and mortality. Whereas this problem is more severe for the case of studies that look at the effects of chronic exposure, it is also an important problem for those studies that look at the effects of acute exposure.

To estimate a causal effect on mortality in this chapter we focus on the effects of acute exposure to ambient air pollution. Therefore, we must address the problem of

³ Whereas acute exposure usually refers to exposure during a 'short' period of time (where short is broadly defined), chronic exposure usually refers to exposure for a long period of time (usually lifetime exposure).

⁴ There have also been epidemiological studies conducted previously for Santiago, Chile (Ostro et al. 1996). Indeed, using data from 1989 to 1991 Ostro et. al. (1996) examine the effects of acute exposure to ambient air pollution on all-age mortality in Santiago, Chile. Ostro et. al. (1996) find that a change of 10 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) in three-day ambient PM_{10} is associated with a 1.1 percent increase in daily all-cause mortality. In addition, Cifuentes et al. (2000) examined the impact of ambient fine particulate matter ($\text{PM}_{2.5}$) on daily mortality in Santiago, Chile. Using a three-day moving average data from 1988 to 1996, Cifuentes et al (2000) estimate that a $10\text{-}\mu\text{g}/\text{m}^3$ change in $\text{PM}_{2.5}$ is associated with a .65 per cent change in all-cause mortality in Santiago, Chile.

possible factors that may affect mortality and that are also likely to be correlated with acute exposure to ambient air pollution (Chay and Greenstone 2003). For example, if not properly accounted for, the following confounding factors may introduce bias in the estimates of the effect of acute exposure to ambient air pollution on infant mortality: (i) differences in income and access to quality health care that allow to cope with the adverse effects of exposure to air pollution; (ii) changes in economic activity that may affect both pollution emissions as well as access to high-quality health care –which allows to cope for the adverse effects of pollution; (iii) sudden widespread health risks (such as influenza or other viral outbreaks) that usually occur contemporaneously to high concentrations of ambient air pollution; and, (iv) geographical sorting due to individuals choosing to live (or work) at specific locations due to their associated exposure to ambient air pollution concentrations. To the best of our knowledge this is the first piece of research that simultaneously addresses all of these potential confounding factors.

In estimating the effects of exposure to ambient particulate pollution on infant mortality only a few studies have successfully addressed the problem of potential confounders. For instance, Chay and Greenstone (2003) address the problem of potential confounders by exploiting variation on total suspended particles (TSP) induced by the 1981-1982 economic recession in the U.S.⁵ Also, Currie and Neidell (2005) address the problem of potential confounders by controlling for location-

⁵ Chay and Greenstone (2003) use U.S. wide data (at the county level) from the early 1980s to examine reductions in Total Suspended Particles (TSP, all particles with diameters less than or equal to 40 microns μm) to estimate the effect of ambient air pollution on infant mortality. They exploit geographic variation in TSP to estimate its effect on infant mortality rates.

specific unobserved characteristics of high ambient air pollution areas in California.⁶ In a more recent study, Arceo, Hanna, and Oliva (2016) examine whether estimates from developed countries (such as the aforementioned studies) can be transferred to developing countries. Using week-level data for Mexico City for years 1997-2006 Arceo, Hanna, and Oliva (2016) use a similar strategy as Currie and Neidell (2005) to estimate the effects of ambient air pollution on infant mortality by introducing municipality-level fixed effects. Moreover, Arceo, Hanna, and Oliva (2016) instrument for ambient air pollution using week counts of thermal inversions in order to address the attenuation bias generated by imperfect imputation of air pollution data for each municipality.

In this chapter, we use data on ambient PM_{10} pollution and infant mortality to construct a municipality-week panel dataset for 34 municipalities in Santiago's Metropolitan Area over the period 1997-2008. In estimating the effect of exposure to ambient PM_{10} pollution on infant mortality the panel structure of our dataset allows for estimating with municipality fixed-effects to control for municipality-specific factors that may have a direct effect on infant mortality—and that may act as confounders if not properly accounted for (e.g., factors such as the overall differential access to quality health care of those infants living in different municipalities). In addition, infant mortality as well as ambient PM_{10} pollution concentrations usually present a strong seasonal pattern, both intensifying during the winter months in

⁶ Using data for years 1989 to 2000 Currie and Neidell (2005) use zip-code level fixed-effects to estimate linear hazard-functions. In this way Currie and Neidell (2005) estimate the effect of week-level ambient air pollution concentrations on infant mortality rates. Notice that although Currie and Neidell (2005) have a detailed dataset at the week-level, they only find significant estimates of the effect of PM_{10} on infant mortality when aggregating PM_{10} pollution at the quarterly level.

Santiago, Chile. Moreover, widespread viral outbreaks and pandemics usually take place during the same period of time as the period of highest concentrations of ambient air pollution (Ruiz-Tagle 2017). These can deteriorate individuals' health and make infants more vulnerable to die. Although we do not directly observe when these viral outbreaks are most severe we indirectly account for these seasonal viral outbreaks by controlling for week-level average temperature and humidity—which act as proxies for viral outbreaks as they capture the weather conditions in which they are more likely to occur.

Furthermore, we take advantage of the meteorological phenomenon of thermal inversions in Santiago's air basin which create city-wide exogenous variation in the concentration of ambient air pollutants. Thermal inversions drive concentration of ambient air pollutant in Santiago by largely determining the overall ventilation of the air basin. Thereby, we use two-stage least squares (2SLS) to estimate the effects of ambient PM₁₀ pollution on infant mortality. Unlike Arceo, Hanna, and Oliva (2016) that use a week count of thermal inversions, we use disaggregated meteorological data that explains both the presence and intensity of thermal inversions for Santiago. Therefore, we use detailed thermal inversion data to instrument for ambient PM₁₀ pollution concentration and obtain 2SLS estimates of the effects of acute exposure to ambient PM₁₀ pollution on infant mortality. Moreover, in this chapter we also address the *indirect* effect that *residential sorting* may have on infant mortality. The problem of residential sorting arises because wealthy individuals can afford to live in municipalities that are consistently exposed to lower levels of ambient air pollution.

This means that wealthy individuals are not only better equipped to cope with the adverse effects of air pollution (by having better access to high-quality health care), but they are also exposed to lower concentrations of ambient air pollution. If not properly accounted for, this indirect effect of residential sorting may yield upward biased estimates of the effect of exposure to ambient air pollution on infant mortality.⁷ To address this indirect effect on infant mortality, due to differential exposure to ambient air pollution because of residential sorting, we use exogenous variation in air pollution that is municipality-specific. We do this by constructing municipality-specific instruments out of the city-wide thermal inversion data; thus, introducing municipality-specific exogenous variation in ambient PM₁₀ pollution concentrations.

On the other hand, the ambient air pollution data usually employed in the existing literature comes from sparse air quality monitoring stations and does not necessarily reflect the ambient air pollution to which individuals are actually exposed to. To have a more accurate measure of the ambient pollution for the relevant geographical areas, both Currie and Neidell (2005) and Arceo, Hanna, and Oliva (2016) impute ambient air pollution data at the relevant geographical area (either zip-code or municipality level) by weighing the imputed zip-code-level or municipality-level air pollution data by the inverse of the distance (inverse distance weighting, or IDW) to the closest air quality monitoring stations. However, although IDW actually improves the precision of imputed ambient pollution exposure, the IDW method yields only an imperfect

⁷ In fact, wealthy families in Santiago tend to live in municipalities in the east side of the city—at a higher elevation and closer to the Andes Mountains—that are exposed to lower concentrations of ambient air pollutants.

imputation of the actual ambient air pollution exposure of the population at the relevant geographical area (zip-code or municipality) and an error component still remains.⁸ This error in the imputation of ambient air pollution at the relevant geographical area introduces a bias in the estimates of the effects of ambient air pollution on infant mortality, producing what is known as attenuation bias.⁹ This attenuation bias is further exacerbated when estimating via fixed effects (Cameron and Trivedi 2005).¹⁰ In this chapter we employ the *Kriging* method to impute ambient air pollution for each municipality in Santiago, Chile. By exploiting the geographical dispersion of the air quality monitoring stations in Santiago the *Kriging* method constructs a full spatial mapping of ambient PM₁₀ pollution for a large number of geographical points within each municipality in Santiago, allowing for a more flexible non-linear geospatial variation in concentrations of ambient air pollutants. Thereby, by using the *Kriging* method we obtain a more accurate ambient air pollution imputation for each municipality than by using the IDW method, and this reduces the attenuation bias.

In sum, this chapter provides new evidence of the effects of exposure to ambient air pollution on infant mortality for the high levels of pollution concentrations observed

⁸ In the applied econometrics literature this problem is commonly known as measurement error.

⁹ This error in air pollution imputation is even more likely to arise in the case of developing countries where, oftentimes, there are few and sparse air pollution monitoring stations, or these monitoring stations fail to report pollution data for certain periods of time.

¹⁰ Arceo, Hanna, and Oliva (2016) use instrumental variables estimation to address the problem of attenuation bias caused by the error in imputation of air pollution at the municipality level. The authors exploit the exogenous variation in air pollution caused by thermal inversions in the air basin of Mexico City. As an instrument for imputed air pollution data Arceo, Hanna, and Oliva (2016) use a weekly count of city-wide thermal inversions. By estimating two-stage least squares (2SLS) they reduce the attenuation bias caused by the error in the imputation of air pollution at the municipality level. As a consequence, they obtain considerable larger estimates of the effects of air pollution on infant mortality. Arceo, Hanna, and Oliva (2016) conclude that their 2SLS estimates are comparable to those results from previous studies in developed economies.

in emerging economies. We find that exposure to ambient PM_{10} pollution has a significant effect on infant mortality and that this effect is more severe for infant mortality that is caused by respiratory and cardiovascular diseases. Furthermore, we find considerable larger effects of exposure to ambient PM_{10} pollution over an extended period of time (several weeks). The rest of the chapter is organized as follows. The next section presents an overview of infant mortality and ambient PM_{10} pollution in Santiago, Chile for the period 1997 to 2008. Section 3 discusses the data and the *Kriging* method for imputing ambient PM_{10} pollution at the municipality level. Section 4 presents descriptive analysis of the data and section 5 presents the framework for econometric analysis. The main results are presented in section 6 and additional results are presented in Section 7. Finally, section 8 provides a policy discussion and section 9 concludes.

2. Background on Infant Mortality and Air Pollution in Santiago, Chile

a. Infant Mortality

Ambient air pollution can cause severe damage to human health. Clinical evidence of the consequences of exposure to particulate matter suggests that air pollutants can cause bronchia pulmonary and cardiovascular diseases such as lung inflammation and blood coagulation that can obstruct blood vessels, leading to angina or even to myocardial infraction, which can eventually result in death (Kampa and Castanas

2008). Meanwhile, the general public in Santiago seems to be aware of the adverse effects of air pollution on health. A recent survey by Chile's Ministry of Environment shows that air pollution is the most important environmental problem for Santiago's population (Dirección de Estudios Sociales 2017). This is in large part due to the adverse health effects on the most vulnerable population, particularly during the winter.

b. Ambient Air pollution Concentrations and Thermal Inversions

Ambient air pollution concentrations in Santiago's Metropolitan Area are caused by emissions as well as by accumulation of pollutants due to the lack of ventilation of the air basin. Emissions of PM₁₀ pollution in Santiago's Metropolitan Area are largely due to motor vehicles (accounting for 40.5 percent of total anthropogenic sources) and the combustion of petroleum derivatives for industrial processes (24.6 percent of total anthropogenic sources). The use of firewood or coal for residential heating and cooking accounts for only 14.8 percent of total anthropogenic sources (De la Maza and Serrano 2013).

In addition to emissions of airborne pollutants, the actual concentrations of these pollutants also depend on the ventilation of ambient pollutants in Santiago's air basin. This, in turn, is largely explained by both Santiago's unique geography and by the presence of a thermal inversion above the city's air basin. Here we briefly describe Santiago's geography and then we further explain the phenomena of thermal

inversion. Santiago is located in Chile's central valley in a basing surrounded by Chile's coastal mountain range to the west and the much taller Andes Mountains to the east and north east.¹¹ Figure 1 shows a terrain map of Santiago's surrounding area. Mild winds blow from the west-south-west (WSW) at the elevation of the city ground-level (around 550 meters above sea level). Conversely, at elevations above Santiago's western coastal mountain range, strong winds blow from the west-north-west (WNW), at elevations starting at around 1,000 to 1,200 meters above sea level. The winds at the city's elevation bring in a constant fresh breeze of clean air to the WSW end of the city (light blue arrows in Figure 1). This breeze compresses the cloud of pollutants in the air basin against the Andes Mountains. That is, at the only gap of the mountains that surround Santiago (at the city's WSW bound), mild winds blow towards the basin, and thus further trapping air pollutants.¹²

¹¹ Whereas the coastal mountain range reaches elevations of 800 to 1,500 meters above sea level, at this latitude the Andes Mountains presents elevations between 4,500 to 6,500 meters above sea level.

¹² During the winter time these winds' speed could be of less than 2 m/s.

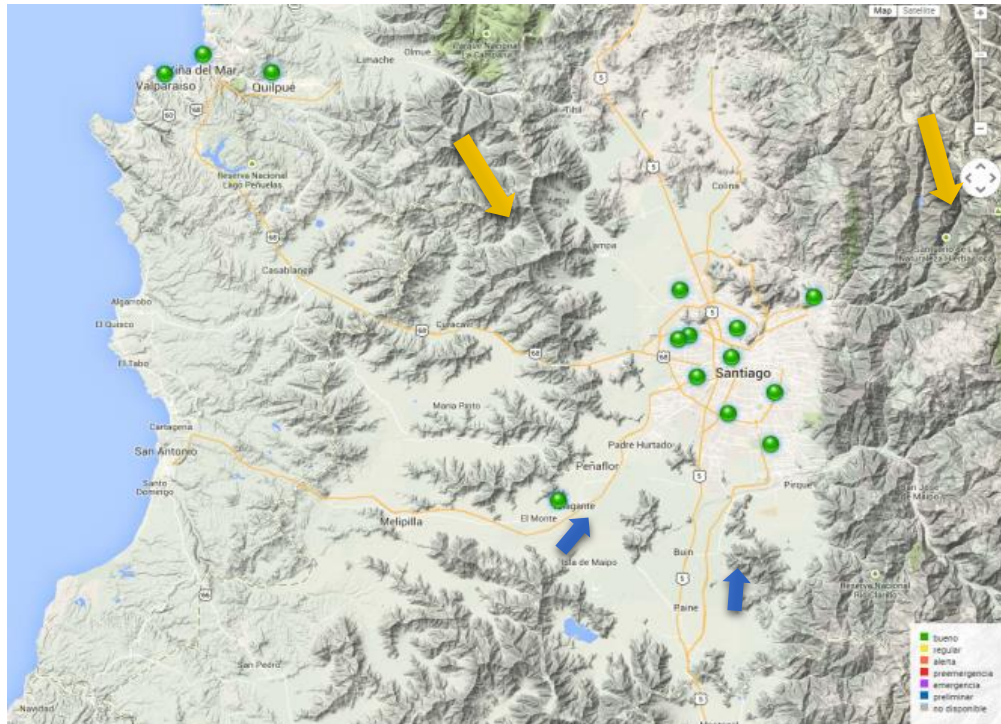


Figure 1: Geographical terrain and direction of winds in Santiago’s air basin.

Usually, fine particles of air pollutants that are emitted as a result of a combustion process are warmer than the air that immediately surrounds them. The difference between the temperature of the particle and the air around them, together with the little weight of these particles, creates buoyancy that makes these particles rise. Once these fine particles reach an altitude above Santiago’s northern and eastern mountain range, strong winds from the WNW blow these air pollutants away (see purple arrows in the map of Figure 1). This phenomena of ventilation of air pollutants in Santiago’s air basin is characterized as *vertical ventilation* (Garreaud and Rutllant 2006) and refers to a vertical movement of the cloud of air pollutants.

Thermal inversions largely explain the vertical ventilation of Santiago’s air basin. Air generally turns cooler as is ascends at higher elevations, so the higher the elevation

the lower the air temperature. However when air at higher elevations is warmer than air at lower elevations this phenomenon is known as a thermal inversion. Thermal inversions trap airborne pollutants and impede the vertical ventilation of Santiago's air basin by breaking with the buoyancy of these small particles that allows them to rise. This results on these small particles resting right above the mass of colder air beneath the warm layer brought in by the thermal inversion. As these small particles cool down and start to slowly descend, at the same time as more pollutants are being emitted at the ground level, these small particles start filling up the space between the thermal inversion layer and the city level, thus creating a cloud of airborne pollutants that fills Santiago's air basin and increases the concentrations of ambient air pollutants to which the population living in Santiago is exposed to.

As consequence of this phenomenon, thermal inversion keep the cloud of ambient air pollutants at an elevation below Santiago's northern and western mountain range, thus obstructing the vertical ventilation of the city's air basin (see Figure 2 and Figure 3). When the thermal inversion breaks down the cloud of pollutants rises above the elevation of the western mountain range, reaching elevations above 1,000 meters above sea level, and thus this cloud of pollution particles is blown away by the strong WNW winds that prevail at these higher elevations. Similarly, during hot (summer) days air pollutants rise above the elevation of this mountain range and, in this way, strong winds clean the pollutants away from the air basin. In sum, thermal inversions determine whether a cloud of air pollutants would rest above the city, and this in turn

drives the overall ventilation of the entire air basin of Santiago's Metropolitan Area, particularly during the late Fall and Winter seasons.¹³

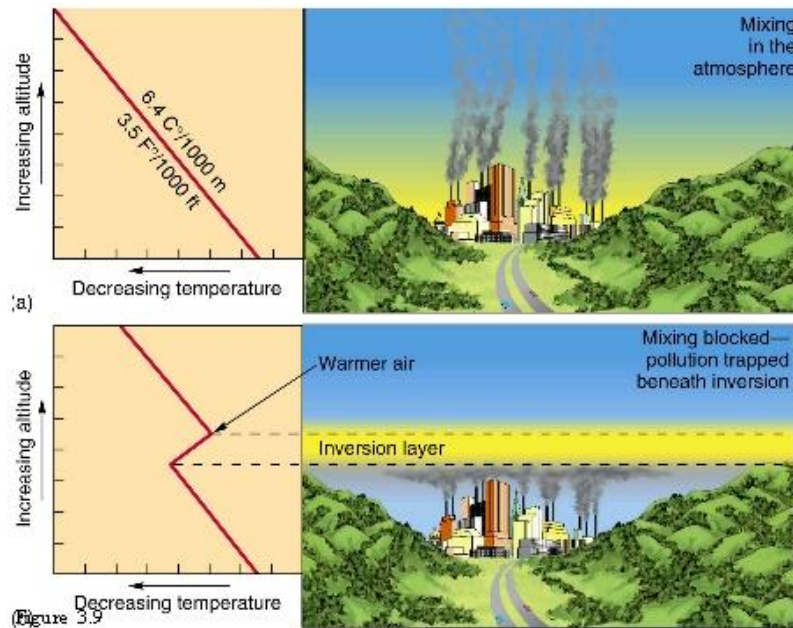


Figure 2: Thermal Inversion - Temperature, elevation and trapping of pollutants.

¹³ Gramsch et al. (2014) examine how thermal inversions drive concentrations of PM pollution in Santiago. They refer to these meteorological phenomena as subsidence inversion and surface inversion. To measure outdoor air temperature at different elevations above Santiago's air basin Gramsch et al. (2014) use data from commercial airplanes departing from the main airport in Santiago (located on the westbound area of the city, at an elevation of 474 meters above sea level). Santiago's main airport is located at about 13 kilometers from the meteorological station at Lo Prado, from where we obtain ambient air temperature data to measure the presence (and intensity of) the thermal inversion. This data allows them to accurately identify both subsidence and surface thermal inversions. On days of thermal inversions, outdoor air temperature in Santiago's air basin initially increases at higher elevations from the ground level. The first stretch of this increase in outdoor temperature occurs up to 600 meters (the surface inversion). Then, at 800 to 1000 meters, there is a sharp increase in outdoor temperature (the subsidence inversion). The surface inversion usually breaks down each day at around noon, particularly on clear sunny days—sunny days are rare during Santiago's winter, so that the breakdown of the surface inversion is much more frequent during the summer time than during the winter time. This allows for the escape of pollutants via vertical ventilation (in the absence of subsidence inversions). However, subsidence inversions may last several days, an up to a week, trapping air pollutants in Santiago's air basin and preventing from vertical ventilation. Gramsch et al. (2014) report that on days when there is a surface inversion PM is 35 percent higher than on days without inversions. Moreover, when there is both surface and subsidence inversion, PM can be up to 84 percent higher than on days without inversions.



Figure 3: Photo of the Thermal Inversion on Santiago, Chile.

Often times, the thermal inversion breaks due to the presence of precipitations, usually in the form of rain. The presence of precipitations usually is accompanied by strong winds at the level of this cloud that breaks the thermal inversion. Furthermore, rainfall facilitates the vertical exchange of air layers at different temperatures and thus breaks the thermal inversion. Furthermore, rain not only breaks down the thermal inversion, but the droplets themselves carry down the pollution particles, and in this way rain brings these pollution particles down to the ground level.¹⁴

¹⁴ Therefore, despite the high concentration of particulate pollution during Santiago's winter, it is not unusual to see a clear clean sky on the immediate days following rain precipitations in Santiago, as the concentrations of air pollutants plunge after a rainy day. Bharadwaj and Eberhard (2008) exploit this characteristic of rainfall on driving air pollution concentrations in Santiago. They use rainfall (as deviations from the month average) as an instrumental variable for air pollution in Santiago to estimate the effects of air pollution on birthweight.

3. *Data and Methodology for Imputation of Ambient Air Pollution Data at the Municipality Level*

We construct a weekly panel dataset of 34 municipalities for the period 1997-2008.

We explain the dataset in detail next.

a. Infant Mortality Data

We use data from the Department of Health Statistics and Information (DEIS, according to the acronym in Spanish) of Chile's Ministry of Public Health to generate municipality-level infant mortality rates. DEIS's provide data on each deceased individual, his or her age and the municipality of residence. Furthermore, we have data on total live births for each municipality (based on the mother's municipality of residence) so that we can compute total live population of infants (those under one year old). Week-level infant mortality rate is defined as number of infant deaths in a given week divided by the total number of live infants up to that given week, per 100,000 inhabitants. In this way, for each of the 34 municipalities in Santiago, we compute municipality-week-level infant mortality rates over the period 1997-2008. In addition, in order to analyze the more direct effect of pollution on infant health, we compute infant mortality rates for those causes that are more directly linked to exposure to air pollution – namely, respiratory and cardiovascular diseases – by considering only those deaths with codes I and J according to the 10th revision of the International Classification of Diseases (ICD-10). Accordingly, we compute

municipality-week-level infant mortality rate due to respiratory and cardiovascular causes for 34 municipalities over the period 1997-2008.

b. Ambient Air Pollution Data and *Kriging* Imputation

We obtain ambient air pollution data from Chile's Air Quality National Information System (SINCA, according to the acronym in Spanish). SINCA has 11 air quality monitoring stations throughout Santiago's Metropolitan Area. The monitoring stations provide daily records of particulate matter less than 10 microns per cubic meter (PM₁₀) for the period 1997-2008.

We use the *Kriging* method to construct a geo-spatial mapping of ambient air pollution and impute air pollution concentrations to each municipality as follows. First, for each of the 11 SINCA's air quality monitoring stations in Santiago we take the week-average PM₁₀ pollution readings. Second, we employ the geographical coordinates of the monitoring stations to impute pollution levels to specific spatial reference-points using the *Kriging* method. This consists of estimating the parameters that describe the spatial correlation among observations of air pollution data from the air quality monitoring stations and then, using these estimates, finding predictions to specific spatial reference-points that minimize the sum of squared errors (Lleras-Muney 2010).¹⁵¹⁶ The specific spatial reference-point we use for *Kriging* imputation

¹⁵ The *Kriging* method has several advantages over the alternative methods previously used by economists, such as the inverse distance weighting method (IDW) (Cressie 2015). First, *Kriging* is the best linear unbiased predictor. Second, measures of fit of standard errors of the predictions can be

of ambient PM₁₀ pollution data is that of public and private schools, as we assume that most people live within a vicinity to schools.¹⁷ That is, we impute air pollution exposure to the geographical location of each school within a municipality and then we average the imputed air pollution data across all (selected) schools for each municipality. Furthermore, we restrict our sample of schools to those schools located (i) at a distance of no more than 5 kilometers from the nearest air quality monitoring station, and, (ii) between 5 to 10 kilometers to the nearest air quality monitoring station but no farther than 20 kilometers to the second-nearest monitoring station.¹⁸ Once we restrict the sample of schools, we averaged the imputed air pollution data across all selected schools for each municipality. In this way we generate a municipality-week panel for air pollution data that we then merge with the individual-

obtained. Third, covariates can be used to improve the predictions. Lastly, *Kriging* allows prediction for a much larger number of locations compared to IDW or municipality-averages. Indeed, using simulation methods Zimmerman et al. (1999) find that “Kriging methods were substantially superior to the inverse distance weighting methods over all levels of surface type, sampling pattern, noise and correlation”.

¹⁶ Lleras-Muney (2010) is one of the first papers in economics to use the *Kriging* method for spatial imputation of pollution data. Using air pollution data from U.S. Environmental Protection Agency, Lleras-Muney (2010) estimates the effects of air pollution on hospital admissions of children of the U.S. military personal.

¹⁷ Notice that the spatial reference-point could be any place, within a given municipality, that readily represents the air that people in that municipality are actually exposed to. Previous research has interpolated to the geographical location of the centroid of each zip-code area in the U.S. However, the surface area of many municipalities in our sample extend far beyond the areas where people actually live, sometimes all the way up to include large parts of rural areas or even large parts of the Andes Mountains. So that, interpolating to the centroid of such a municipality would be equivalent to interpolating pollution data to locations in the middle of the countryside or in the middle of the Andes, where very few people live. Alternatively, we could interpolate to the geographical location of City Hall building for each municipality. However, most city hall buildings have been built long before the expansion of the city, and therefore, tend to be clustered towards the central areas of the city, and do not necessarily represent the geographical location where people actually live.

¹⁸ Imputing pollution data using the *Kriging* methodology is much more reliable for interpolation than for extrapolation. Pollution data from *Kriging* interpolation for a geographical area that has more than one monitoring stations is quite reliable, particularly, when the monitoring stations are located at a short distance from each other and surrounding this particular spatial reference-point in its vicinity. Conversely, pollution data from *Kriging* extrapolation is much less reliable. That is, when there is only one monitoring station near the particular spatial reference-point or, if there is more than one monitoring station, these are located at a far distance from this particular spatial reference-point and are not surrounding it in its vicinity.

level infant mortality data outlined above. The appendix presents technical details of the *Kriging* method used for mapping ambient PM₁₀ pollution data.

c. Thermal Inversion Data

To account for the presence of a thermal inversion in Santiago's air basin we use data from a meteorological station—Lo Prado—located on the top of Santiago's western mountain range, at an elevation of 1,080 meters above sea level, just about 500 meters above the city's elevation. From station Lo Prado we obtain ambient air temperature, barometric air pressure, wind speed and precipitations. Furthermore, we contrast this data from Lo Prado—basically ambient air temperature—with data from a meteorological station located at the city's ground elevation, Torre Entel, at 540 meters above sea level. That is, we compute the difference in ambient air temperature at Lo Prado (at 1080 meters) with ambient air temperature at Torre Entel (at 540 meters), as well as detailed meteorological data at Lo Prado (barometric air pressure, wind speed, precipitations, etc). Furthermore, to obtain a measure of the thermal inversion that is municipality-specific we interact the difference in air temperature with a municipality-specific dummy.

d. Additional Weather Controls

Furthermore, we obtain we obtain municipality-level temperature and precipitation data from the Air Quality Meteorological Network of Chile's Meteorological Service and from the Water Service of Chile's Public Works Ministry.

4. *Descriptive Statistics*

a. Descriptive Statistics for Ambient PM₁₀ Pollution and Infant Mortality

Table 1 presents descriptive statistics for ambient PM₁₀ pollution, overall infant mortality and infant mortality due respiratory and cardiovascular causes (henceforth, R&C), for the period 1997-2008.¹⁹ Table 1 shows that the average ambient PM₁₀ pollution over this period is $77.2 \mu\text{g}/\text{m}^3$.²⁰ Also, the average infant mortality rate for this period is 15.5, per 100,000 live births, and the average infant mortality for respiratory and cardiovascular causes is 1.1, per 100,000 live births. The table also shows total municipality-week standard deviation as well as both standard deviations only across municipalities and standard deviations only across weeks (times series). Thus, Table 1 shows that most of the variation in ambient air pollution and infant mortality occurs across weeks. Whereas the cross-municipality variation represents 30 percent of the total variation of PM₁₀ pollution, the times series variation (across weeks) represents about 90 percent of the total variation of PM₁₀ pollution. Similarly,

¹⁹ Mean infant mortality (Inf Mort R&C) was multiplied by 52 weeks to obtain the column 'Mean deaths in a year (per 100,000)' on the far right of Table 1.

²⁰ Chile's standard for annual concentrations of PM₁₀ is $50 \mu\text{g}/\text{m}^3$ and the standard for 24-hr concentration is $150 \mu\text{g}/\text{m}^3$.

whereas the cross-municipality variation for infant mortality and infant mortality R&C represents less than 10 percent of the total variation, the time series variation (across weeks) represents slightly more than 20 percent of the total variation. Next, we discuss in more detail the patterns and source of these cross-municipality and time series variations.

Table 1: PM₁₀ Pollution, Infant Mortality Rate (all-causes) and Infant Mortality due to Respiratory and Cardiovascular Diseases (Inf. Mort. R&C) for Santiago's Metropolitan Area.

	Mean	Standard Deviation			Observations	Average population (each week)	Mean deaths in a year (per 100,000)
		Municipality-week	Cross-municipality	Time series (week)			
PM10 pollution	77.2	28.4	8.8	26.4	18,800		
Infant Mortality	15.5	28.4	2.7	5.8	18,800	85,006	807.4
Inf Mort (R&C)	1.1	7.2	0.5	1.6	18,800	85,006	58.4

b. Municipality-level Variation

Santiago is a highly segregated city where high-income families tend to cluster together in a few high-income neighborhoods and municipalities. Furthermore, high-income individuals tend to have access to better health care, such as having access to an expensive network of private health care facilities through a private insurance network. Therefore, we should expect that high-income municipalities would present lower infant mortality rates. Table 2 presents mean ambient air pollution and infant mortality for each of the 34 municipalities in Santiago. For ease of analysis, we group these municipalities into 11 geographical sub-regions (City Center, Central East, Central North, Central South, Central West, East, North, South, West, South East and South West). Table 2 also presents other relevant variables that are likely to affect infant mortality—namely, average per-capita income and percentage of population with private/public health insurance for each municipality level. Table 2 also presents ground-level elevation at the urban centroid for each municipality.²¹ Table 2 shows that, municipalities in the East sub-region present low concentrations of ambient air pollution as well as low infant mortality rates. Those municipalities in the East sub-region also present the highest per-capita income as well as the highest percentage of private health insurance. Thereby, it may likely be that better access to quality health care and high per-capita income largely explain low infant mortality rates in those

²¹ Table 2 also shows that effects of pollution on municipality-level infant mortality should be weighed by its relative population size. For instances, whereas municipalities in the South East region represent more than fifteen percent of Santiago's population, municipalities in the Central East and North regions represent less than five percent. As a consequence, when estimating the effect of PM₁₀ pollution on infant mortality we weigh each observation in our dataset by its corresponding municipality-weight.

municipalities in the East sub-region. That is, access to quality health care and high per-capita income may have a large *direct* effect on infant mortality. In our empirical model in the next section we estimate by municipality-level fixed effects to capture those factors across municipalities, such as average per-capita income or percentage of private/public health insurance, that are likely to affect infant mortality rates.²²

²² We also introduce municipality-specific time trends to control for changes over time that might be specific to each municipality. For example, there is evidence that those rich individuals (living in rich municipalities) increased their income by a larger proportion than the rest of the population during the 1997-2008 period, and thereby, they may have increased the access to a better health care at a fastest rate than the rest of the population. These municipality-specific time trends should capture this time patterns.

Table 2: Municipality-level statistics for PM₁₀ pollution, Infant Mortality Rate, Per-Capita Income and Ground-level Elevation.

Municipality	PM10	Elevation at centroid	Per-capita Income (2003 USD)	Private/Public Health Insurance (%)	Infant Mortality Rate	Number of Infants
City Center						
Santiago	76	540	5,249	36/48	16.2	3,374
Central East						
Nuñoa	76	584	5,981	45/43	10.9	2,140
Providencia	73	600	11,275	64/20	12.3	1,649
Central North						
Conchalí	75	499	2,260	15/65	17.9	2,246
Independencia	74	528	3,222	24/62	22.4	1,101
Recoleta	72	522	2,057	12/78	17.7	2,585
Central South						
La Cisterna	84	561	3,414	26/55	14.8	1,203
La Granja	82	591	1,677	11/78	15.6	2,351
Lo Espejo	81	513	1,586	9/80	18.7	1,841
Macul	82	556	4,056	29/54	14.7	1,686
Pedro Aguirre Cerda	80	514	2,058	13/78	16.8	1,681
San Joaquín	83	547	2,126	15/73	14.2	1,451
San Miguel	82	541	4,645	32/52	19.0	1,161
San Ramón	84	580	1,601	10/81	14.1	1,900
Central West						
Estación Central	77	487	2,791	22/63	14.9	2,063
Lo Prado	78	492	2,302	15/75	16.8	1,758
Quinta Normal	76	502	2,384	16/65	16.6	1,656
East						
La Reina	72	640	8,122	59/23	12.2	1,190
Las Condes	64	722	12,571	69/19	10.1	3,580
Lo Barnechea	55	825	12,279	58/36	11.1	1,173
Peñalolén	78	640	2,487	19/70	14.9	4,055
Vitacura	65	695	17,915	74/17	9.7	1,172
North						
Huechuraba	69	520	2,707	18/67	14.3	1,532
Quilicura	81	471	2,272	15/75	16.1	2,615
South						
El Bosque	80	575	1,999	11/81	16.6	2,973
La Pintana	66	623	1,411	5/91	14.5	3,648
San Bernardo	69	556	2,028	11/79	18.3	4,703
South East						
La Florida	77	621	2,706	25/62	13.9	5,490
Puente Alto	60	667	2,560	26/61	16.4	7,946
South West						
Cerrillos	78	493	2,781	19/67	14.3	1,143
Maipú	78	461	2,475	29/58	15.3	7,422
West						
Cerro Navia	80	479	1,805	7/85	16.7	2,519
Pudahuel	81	462	2,090	18/70	16.0	3,382
Renca	79	492	1,995	14/76	15.9	2,279
AVERAGE	76	561.7	4,085	26/62	15.3	2,608

(*) Number of Infants refers to the number of those 365 days old and younger observed in a (calendar) week, averaged for each municipality. Thereby, an infant enters our dataset for a period of no more than fifty two

In addition to a direct effect of access to quality health care due to high percentage of private health insurance and high per-capita income, those individuals in East municipalities are also exposed to low level of air pollution as compared to that of other municipalities. This shows that there is *geographical sorting* in which rich individuals can afford to live in municipalities that are exposed to low levels of air pollution. This geographical sorting will create an indirect effect on mortality via low levels of exposure to air pollutants of those rich individuals. By failing to properly account for geographical sorting one may overestimate the effect of air pollution on infant mortality (yielding upward biased estimates). Indeed, Table 3 shows that municipality-level per-capita income has a strong negative correlation with infant mortality.²³ This could be due to both, the direct effect of per-capita income (and access to better health care) on infant mortality as well as due to the indirect effect of per-capita income via low exposure to air pollutants.²⁴

Table 3: Municipality-level Correlation Matrix for PM₁₀ pollution, Infant Mortality Rate, Per-Capita Income and Ground-level Elevation.

	Infant Mortality Rate	Per-capita Income	PM10	Elevation
Infant Mortality Rate	1			
Per-capita Income	-0.69	1		
PM10	0.36	-0.57	1	
Elevation	-0.61	0.68	-0.70	1

²³ Similarly, the correlation between municipality per-capita and percentage of population with private health insurance is .98 (not shown in Table 3).

²⁴ Table 3 shows that the correlation between ambient PM₁₀ pollution and average municipality-level per-capita income is -.57.

Moreover, those municipalities in the East are also located at the highest ground elevation, and those municipalities at high ground elevations also present low concentrations of PM₁₀ pollution (Table 2). There are at least two reasons that explain this high negative correlation between ground elevation and concentrations of ambient air pollution. First, the chemical formation of fine part of particulate matter is largely influenced by the presence of humidity contained in the air. Regions at lower elevations tend to attract more humidity (so that they are more foggy and often times get swamped) which facilitates the chemical process underlying the formation of fine particulate matter, which in turn increases concentrations of PM₁₀ pollution. For instance, the West sub-region is an area of low ground elevation and former wetlands that is characterized by high content of air humidity. The West sub-region also presents high concentrations of PM₁₀ pollution. Second, in the early hours of the day a large mass of cold air descends from the Andes Mountains through the East mountain canyons blowing mostly on the East side of Santiago and creating a constant breeze of clean air that pushes away air pollutants towards the westbound of the city. This causes that the concentrations of air pollutants in the east side of the city are very low, particularly early in the mornings (Garreaud and Rutllant 2006).

c. Time Variation

In this section we discuss yearly time trends as well as seasonal variation for ambient PM_{10} pollution and infant mortality rate. Furthermore, we discuss how the prevalence of thermal inversions largely drives seasonal variation of air pollutants.

i. Yearly Trends for Ambient PM_{10} Pollution and Infant Mortality

Figure 4 presents yearly concentrations of ambient PM_{10} pollution during the period 1997-2008 as well as annual average infant mortality over the same period for 34 municipalities of Santiago's metropolitan area. Figure 4 show a steady decline in concentrations of ambient PM_{10} pollution over this period, as well as a decline in infant mortality rates. The steady decline in ambient PM_{10} pollution during this period can be (at least partially) explained by a bundle of policies aimed at reducing ambient particulate air pollution enforced by Chile's health and environmental authorities. After three years of consistently exceeding the yearly standard for ambient PM_{10} pollution concentrations during the early nineties (1992-1995), in year 1996 Santiago's Metropolitan Region was officially declared a 'saturated zone' by airborne particulate matter, carbon monoxide and ozone. As a consequence, the Chilean environmental authority designed a bundle of policies to address the problem of air pollution in Santiago's Metropolitan Area, known as the first Air Pollution Control and Prevention Plan (PPDA, according to the acronym in Spanish). The actual implementation and enforcement of the provisions of the PPDA actually started in year 1998. The first PPDA was derogated and substituted by a new updated one in

2003 (and then again in 2010). They all focused on reducing concentrations of PM₁₀ pollution.²⁵ The PPDA includes both long-term and short-term provisions. Among the long-term ones was the total conversion of the city-wide motor vehicle fleet in which all new vehicles must have a catalytic converter that significantly reduces pollution emissions, and taxis and buses for public transportation must adopt this converter in order to be allowed to circulate. Accordingly, the gasoline mix for these motor vehicles was also substituted by a cleaner one. Furthermore, there were similar conversions in the standards for the most polluting stationary emitters (manufacturing facilities) in Santiago's Metropolitan Area. With the exception of provisions that target short-lasting episodes of high concentrations of PM₁₀ pollution, none of these long-term policies have been evaluated thus far, neither as a package nor individually.²⁶

²⁵ Chile's Ministry of Environment is currently revising the latest PPDA that will now focus on reducing concentrations of fine particulate matter (PM_{2.5}).

²⁶ One of the most salient policies of the PPDA was a short-term provision in which the government would identify and announce days in which PM₁₀ pollution was expected to exceed certain dangerous threshold. The government would flag those days and announce a package of 'emergency provisions': mandatory restrictions on driving, the shutdown of some major stationary emitters (factories in the outskirts of the city), the strict prohibition of wood fuel for heating of dwellings, etc. Mullins and Bharadwaj (2015) estimate that these 'emergency provisions under Santiago's PPDA resulted in reductions of PM₁₀ concentrations in the order of 17 percent to 25 percent in the days immediately following these environmental episodes.

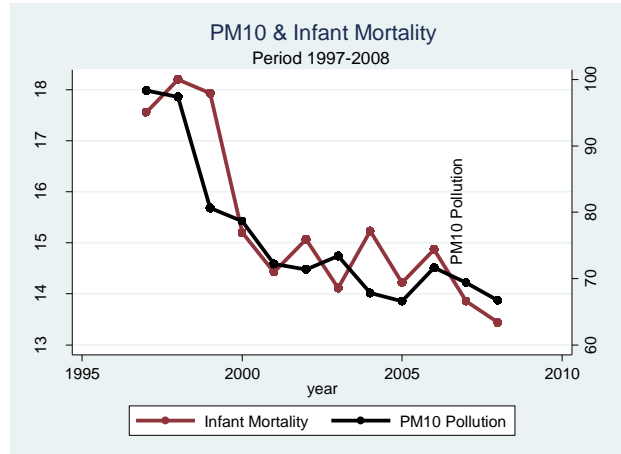


Figure 4 : Year Time Trends of PM₁₀ Pollution and Infant Mortality. Santiago’s Metropolitan Area. Period 1997-2008.

ii. Seasonal Variation of Ambient PM₁₀ Pollution and Infant Mortality

Most of the variation in ambient PM₁₀ pollution in Santiago’s Metropolitan Area occurs across the different seasons within any given year (see Figure 5). In the late summer (January and February) concentrations of PM₁₀ start off low to then increase sharply during the autumn and reach a peak during the late autumn and early winter (May through June). Then these concentrations decline with the first rainfalls in the middle of the winter. They subsequently decline sharply with the continuous precipitations, warmer temperatures and intense winds of early spring (late August, September and October), and remain low throughout the rest of the summer (December through January).

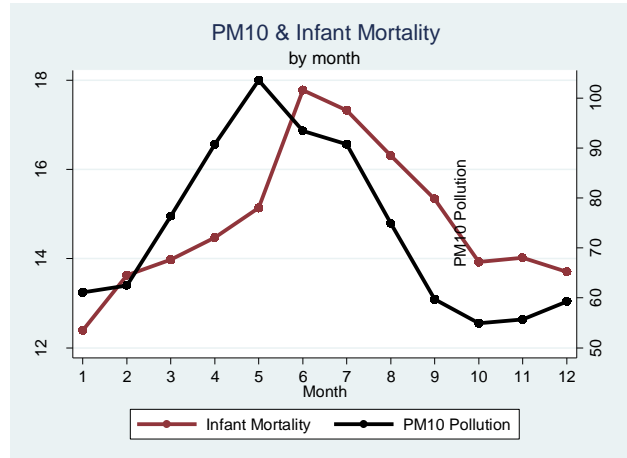


Figure 5: Seasonal trends of PM₁₀ Pollution and Infant Mortality. Santiago’s Metropolitan Area. Period 1997-2008.

The plot presented in Figure 5 show that infant mortality follow a seasonal pattern similar to that of ambient PM₁₀ pollution concentrations, but with some lag. Infant mortality increases steadily during the autumn to then sharply peak in the early winter (June) and remain high during the first months of spring (through September). Although the seasonal pattern presented in Figure 5 suggests strong correlation (with some lag) of ambient PM₁₀ pollution and infant mortality, this does not necessarily imply that there is a causal relationship. There are important factors that present similar seasonal variation, such as seasonal viral outbreaks, that may as well drive infant mortality. In the next section we further discuss these correlations and we discuss our identifying strategy in order to estimate a causal effect.

iii. Seasonal Variation of Thermal Inversions and Ambient PM₁₀ Pollution

Seasonal variation in ambient PM₁₀ pollution concentrations can be explained by changes in pollution emissions as well as by changes in the ventilation conditions of Santiago's air basin. As explained earlier, thermal inversions and precipitations play a major role in the ventilation of air pollutants in Santiago. Also, both thermal inversions as well as precipitations present a clear seasonal pattern; both are prevalent mostly during the winter. Figure 6 and Figure 7 show how the weekly variation in PM₁₀ pollution in an average year closely tracks both thermal inversions as well as precipitations. Figure 6 presents weekly concentrations of ambient PM₁₀ pollution as well as temperature difference (at elevations of 1080 m vs 540m) over the period 1997-2008. Positive temperature difference indicates the presence of a thermal inversion. Thermal inversions are usually prevalent between week 16 (late April) and week 35 (early September) of the calendar year. Furthermore, Figure 7 also presents weekly precipitations and temperature at Santiago's ground level over the same period. The bottom plot shows that precipitations are more prevalent between week 21 (early to mid-June) and week 36 (mid-September), which roughly correspond to Santiago's winter. Furthermore, the bottom plot shows that average temperatures drop rapidly between weeks 12 and 17 (April and early May) to remain low throughout the rest of Santiago's winter and then slowly start to climb back up by week 36 (mid-September). In sum, Figure 6 shows that concentrations of PM₁₀ pollution highly correlate with the presence and intensity of thermal inversions during the year. Indeed, the highest concentrations of ambient PM₁₀ pollution usually occur around the same time of the year as when the thermal inversion is strongest (largest temperature difference at elevations of 1080m and 540m).

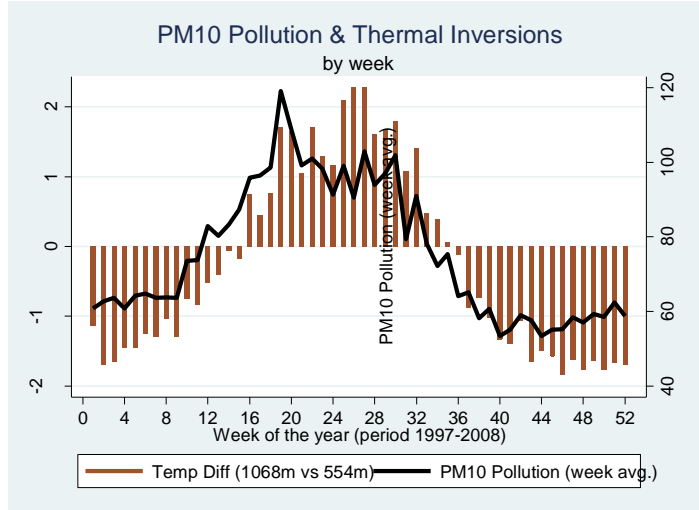


Figure 6: PM₁₀ and Thermal Inversions by Week of the Year. Period 1997-2008.

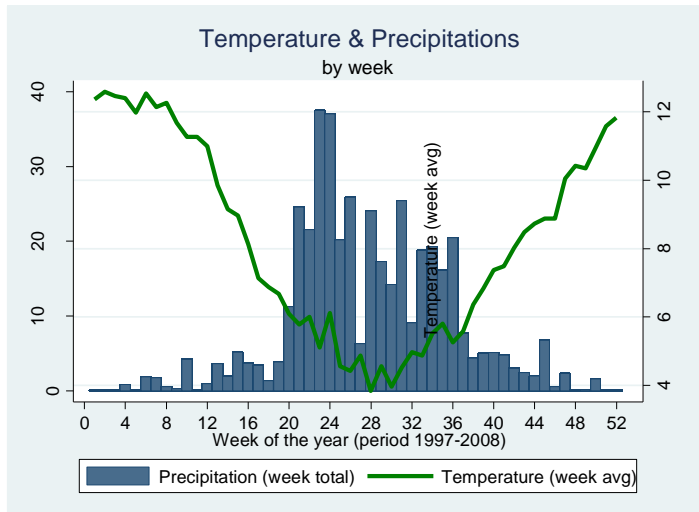


Figure 7: Precipitations and Temperature by Week of the Year. Period 1997-2008.

5. Framework for Regression Analysis

a. Ordinary Least Square (OLS) and Fixed Effects (FE) Estimation for Panel Data

To estimate the effect of PM₁₀ pollution on infant mortality we take advantage of the municipality-week panel structure of our dataset over the period 1997-2008. Equation (1) below lays out the econometric framework.

$$M_{iw} = \beta_0 + \beta_1 P_{iw} + \beta_2 W_{iw} + \beta_3 D_M + \beta_4 D_Y + \varepsilon_{iw} \quad (1)$$

Where M_{iw} denotes mortality rate at municipality i for week w ; P_{iw} denotes ambient PM₁₀ pollution at municipality i during week w ; W_{iw} denotes weather variables for municipality i and week w , such as precipitation, daily minimum and maximum temperature; D_M and D_Y denote dummies for month and year, respectively, and ε_{iw} denotes the unobserved error term. The parameter β_1 captures the effect of exposure to ambient PM₁₀ pollution on infant mortality rate.

However, as we discussed in the previous section, characteristics of the population living in these municipalities (such as per-capita income or access to quality health care) as well as municipality-level health care infrastructure (such as primary care

facilities, hospitals, etc.) are likely to determine the infant mortality outcome.²⁷ In fact, Table 3 shows that per-capita income is highly correlated with infant mortality, and so is the percentage of population with private/public health insurance (not shown in Table 3).²⁸ Furthermore, as also shown in Table 3, municipality-level per-capita income—as well as the percentage of the population with private/public health insurance—is highly correlated with PM₁₀ pollution concentrations at the municipality level. Therefore, leaving out of the model in equation (1) variables that determine infant mortality and that also correlate with the PM₁₀ pollution variable would introduce bias in the estimation of β_1 . However, by estimating by municipality-level fixed effects we can indirectly capture these municipality-specific factors. Thereby, in the fixed-effect estimation we assume that the error term ϵ_{iw} has the form $\epsilon_{iw} = \epsilon_i + \epsilon_{iw}$, so that equation (1) can be re-written as

$$M_{iw} = \beta_0 + \beta_1 P_{iw} + \beta_2 W_{iw} + \beta_3 D_M + \beta_4 D_Y + \beta_5 y_i + \epsilon_i + \epsilon_{iw} \quad (1')$$

Where ϵ_i is a municipality-specific error term that captures all time-invariant differences across municipalities (such as municipality-level socio-economic conditions, access to health-care or municipality-level health care infrastructure). Furthermore, in equation (1') above we add a municipality-specific year trend, y_i , that allows to control for municipality-specific time trends.

²⁷ For instance, public provision of primary health care in Chile is locally managed at the municipality-level.

²⁸ Unfortunately, we do not have data on municipality-level health care infrastructure or data on the quality of public primary care provision at the municipality level.

b. 2SLS Estimation (OLS and FE)

There is recent evidence of a positive relationship between mortality and economic activity (procyclical mortality). Heutel and Rhum (2016) examine this relationship for the U.S. and find that air pollution concentrations explain part of this positive relationship between economic activity and mortality. The rationale for this positive relationship goes as follows. During economic booms there is higher manufacturing production which would trigger higher emissions of air pollutants by the manufacturing plants. Likewise, economic booms allow for both more government revenue and that allow for more funding and provision of public healthcare as well as for higher individual disposable income that allows families to afford high-quality private healthcare—both of these allow for higher capacity to cope with the adverse effects of air pollution. In other words, possible time-varying confounding effects may arise from failure to control for changes in economic activity. Therefore, to identify a causal effect of air pollution on infant mortality in the two-stage least squares regressions (2SLS) we use variation in PM_{10} pollution that is not driven by emissions—which are in turn affected by economic activity—but variation in PM_{10} pollution that is driven by changes in ventilation of Santiago’s air basin (via variation in thermal inversions). Therefore, in this section we use the meteorological variables that explain thermal inversions as instrumental variables for air pollution and estimate equations (1) and (1’) by 2SLS. In the first stage we regress P_{iw} on the instruments, conditional on the rest of the variables in the model, and predict values for P_{iw} to obtain \hat{P}_{iw} . Then, in the second stage we regress M_{iw} on \hat{P}_{iw} and the rest of the variables in the model. We use as instruments a vector of meteorological variables

that drive thermal inversions and ventilation of Santiago's air basin, which includes: (i) a three-degree polynomial of week-average temperature difference at 1068 vs. 540 meters, (ii) a two-degree polynomial of week-average wind speed at 1068 meters, and (iii) a two-degree polynomial of week-average atmospheric air pressure at 1068 meters. All these meteorological variables create city-wide variation in the concentrations of air pollution in Santiago's air basin and we argue that this source of variation is exogenous to infant mortality. That is, the only effect that these variables have on infant mortality is via their effect on concentrations of ambient PM pollution, but they are not correlated with the error term. Furthermore, to create municipality-specific exogenous variation in the concentrations of air pollution, we interact the meteorological variables that drive thermal inversions (specifically, a two-degree polynomial of week-average temperature difference, at 1068 vs. 540 meters) with municipality-specific dummies. In this way, we estimate the equation below to obtain the vector of parameters $\hat{\gamma}$ s and obtain predicted values for P_{iw} .²⁹

$$P_{iw} = \gamma_1 + \gamma_2 TI_{iw} + \gamma_3 W_{iw} + \gamma_4 D_M + \gamma_5 D_Y + \gamma_6 y_i + u_{iw}$$

Where TI_{iw} denotes the set of thermal inversion instruments outlined above.

In the second stage we substitute \hat{P}_{iw} for P_{iw} in equations (1) and (1') above, and regress M_{iw} on \hat{P}_{iw} and the rest of the variables in the model (W_{iw} , D_M , D_Y and y_i).

$$M_{iw} = \beta_0 + \beta_1 \hat{P}_{iw} + \beta_2 W_{iw} + \beta_3 D_M + \beta_4 D_Y + \beta_5 y_i + \varepsilon_{iw} \quad (1'')$$

²⁹ $\hat{P}_{iw} = \hat{\gamma}_1 + \hat{\gamma}_2 TI_{iw} + \hat{\gamma}_3 W_{iw} + \hat{\gamma}_4 D_M + \hat{\gamma}_5 D_Y + \hat{\gamma}_6 y_i$

where, in the FE 2SLS model it is assumed that $\varepsilon_{iw} = \varepsilon_i + \varepsilon_{iw}$.

Therefore, if confounding effects may bias our OLS (or FE) estimates of β_1 , the 2SLS estimation yields unbiased estimates for β_1 if the underlying assumptions for the instruments TI_{iw} holds true (that is, that they are correlated with air pollution but uncorrelated with the error term). Moreover, even in the presence of measurement error in the imputation of air pollution at the municipality-level—which would introduce attenuation bias in the estimation of β_1 —the 2SLS estimation also yields unbiased estimates for β_1 .

6. Results

We present results of the first stage regression in Table 4, and results for the effect of exposure to ambient PM_{10} pollution on infant mortality in Table 5 (for OLS, FE, 2SLS and FE 2SLS). Furthermore, we present results for the effect of ambient PM_{10} pollution on infant mortality due to respiratory and cardiovascular diseases in Table 6. The parameter estimates associated to PM_{10} correspond to a $10\text{-}\mu\text{g}/\text{m}^3$ change in PM_{10} concentrations. Columns 1 and 2 in tables 5 and 6 present results of OLS estimations and columns 3 and 4 present results for municipality-level Fixed Effects (FE) estimations. In addition, columns 5 through 7 present results for 2SLS estimations and columns 8 through 10 present results for FE 2SLS estimations. However, before discussing the results from the second stage, in the next section we examine the first-stage estimation and discuss the validity of the instruments.

a. First-stage Regressions

As explained earlier, in this section we look at the geographical and meteorological drivers of ventilation of ambient PM_{10} pollution in Santiago's Metropolitan Area. Moreover, for the second stage we use the meteorological variables that explain thermal inversions for Santiago's air basin as source of exogenous variation of ambient PM_{10} pollution. More precisely, we look at the difference of ambient air temperature between the meteorological station Lo Prado (located at 1068 meters above sea level) and Torre Entel (located at the city's elevation, at 540 meters above sea level). Furthermore, we use data on atmospheric air pressure and wind speed at station Lo Prado as well as municipality-week level data on precipitations, high and low temperatures. We assume that none of the meteorological variables measured at Lo Prado should have a direct effect on infant mortality other than via their indirect effect on ambient air pollution concentrations. That is, we impose the exclusion restriction that, in the infant mortality regression (equation 1'), the parameters associated to temperature, air pressure and wind speed at Lo Prado are all equal to zero. Hence, using this meteorological data we instrument for the municipality-level PM_{10} pollution data to obtain estimates of the effects of exposure to ambient PM_{10} pollution on both infant mortality and infant mortality due to cardiovascular and respiratory causes.

Table 4 presents results of a FE regression of ambient PM_{10} pollution on meteorological variables. Explanatory variables are as follows: (i) a third degree polynomial of week-average temperature difference (540m vs. 1068m); (ii) a two degree polynomial of week-average wind speed at 1068 meters above sea level; (iii) a two degree polynomial of week-average atmospheric air pressure at 1068 meters above sea level; (iv) municipality-level week-average low daily temperature; (v) municipality-level week-average low daily temperature; (vi) municipality-level week-average precipitations; (vii) month and year dummies; and, (viii) a municipality-specific year trend. As shown in Table 4, all coefficients are highly statistically significant. Furthermore, the set of regressors (i) through (iii) pass Staiger and Stock (1997)'s test of weak instruments at the 95 percent confidence level. We use these estimates presented in Table 4 to generate predicted values for ambient PM_{10} pollution. Thereby, in the results presented in tables 5 and 6 we use these predicted values to substitute for the ambient PM_{10} pollution, and then run 2SLS and FE 2SLS regressions.

Table 4: First Stage Regression for PM₁₀ Pollution

VARIABLES	2SLS			FE 2SLS		
	(1)	(2)	(3)	(4)	(5)	(6)
Temperature Difference (Temp. at 1068 vs. 550 mts.)	0.799*** (0.026)	0.794*** (0.021)	1.797*** (0.103)	0.775*** (0.032)	0.795*** (0.028)	1.862*** (0.127)
TempDiff (squared)	-0.037*** (0.006)	-0.048*** (0.004)	-0.082*** (0.024)	-0.034*** (0.003)	-0.048*** (0.003)	-0.104*** (0.008)
TempDif (cubic)	-0.014*** (0.002)	0.006*** (0.001)	0.006*** (0.001)	-0.014*** (0.001)	0.006*** (0.000)	0.006*** (0.000)
WindSpeed at 1068m	-0.581*** (0.214)	-0.842*** (0.108)	-0.832*** (0.101)	-0.753*** (0.088)	-0.843*** (0.052)	-0.833*** (0.054)
WindSpeed (squared)	-0.024 (0.022)	0.080*** (0.012)	0.079*** (0.011)	0.002 (0.009)	0.080*** (0.007)	0.079*** (0.007)
Atmospheric Air Pressure at 1068 mts.	27.531** (10.931)	-32.551*** (8.322)	-32.898*** (8.200)	23.355*** (3.481)	-32.114*** (3.311)	-32.312*** (3.477)
Atmospheric Air Pressure (squared)	-0.015** (0.006)	0.018*** (0.005)	0.018*** (0.005)	-0.013*** (0.002)	0.018*** (0.002)	0.018*** (0.002)
MunicipalityGroundElevation*TempDiff			-1.777*** (0.168)			-1.889*** (0.215)
MuniGroundElev*TempDiff (squared)			0.059 (0.040)			0.099*** (0.013)
Low temp (weekly avg., by municipality)	-0.045*** (0.016)	0.006 (0.012)	0.004 (0.012)	-0.030*** (0.009)	0.010 (0.008)	0.009 (0.007)
High temp (weekly avg., by municipality)	0.071*** (0.009)	0.010 (0.007)	0.011 (0.007)	0.045*** (0.005)	0.009*** (0.003)	0.009*** (0.003)
Precipitations (weekly avg., by municipality)	-0.122*** (0.007)	-0.185*** (0.007)	-0.184*** (0.007)	-0.125*** (0.005)	-0.185*** (0.010)	-0.184*** (0.010)
Month & year dummies	No	Yes	Yes	No	Yes	Yes
Municipality-specific year trend	No	No	No	Yes	Yes	Yes
Wu-Hausman p-value	0.917	0.00248	0.00644	0.933	0.00357	0.00655
F test on instruments	748.6	756.9	616.3	659.7	659.7	564.4
UnderID p-value	0	0	0	0	0	0
R-squared	0.366	0.743	0.753	0.380	0.722	0.735
Observations	18,800	18,800	18,800	18,800	18,800	18,800

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

b. Effect of Ambient PM₁₀ Air Pollution on Overall Infant Mortality

Table 5 presents parameter estimates for the effect of PM₁₀ pollution on overall infant mortality. Column 1 shows that the OLS estimate for the specification that does not include any additional controls yields statistically significant estimate for β_1 . Adding controls for temperature, precipitation and month and year dummies (column 2) results in a much better fit of the infant mortality data (which is reflected in a higher R² statistic) but the OLS estimate for β_1 turns statistically non-significant. Recall that

PM₁₀ pollution presents a strong seasonal pattern (see Figure 5), so that adding these seasonal controls captures most of its seasonal variation, which may explain why this specification results in a non-significant estimate of β_1 .³⁰

On the other hand, controlling for municipality-specific fixed-effects allows to account for the different access to quality healthcare of those families living in different municipalities, which in turns allows to control for the differential capacity to cope with the adverse effects of exposure to PM₁₀ pollution.³¹ The FE regressions allow to account for this effect. In turn, the parameter estimates for β_1 in the FE regressions (columns 3 and 4) turn smaller than those of the OLS regressions (columns 1 and 2). Furthermore, the inclusion of seasonal variables (temperature, precipitation) as well as month and year dummies, in column 4, results in a non-significant estimate for β_1 —while also resulting in a better fit of the data (larger R² statistic).

Columns 5 through 7 of Table 5 present 2SLS estimates for β_1 . These estimates are robust to the possible presence of time-varying confounding effects. In particular, by instrumenting for PM₁₀ pollution using variation driven by thermal inversions, the

³⁰ On the other hand, low temperature presents a negative (seasonal) correlation with PM₁₀ pollution and a negative (seasonal) correlation with infant mortality (as shown in Figure 6 and Figure 7); thereby ignoring low temperature (as in column 1) would overestimate the parameter β_1 . Conversely, precipitations present a strong negative correlation with PM₁₀ pollution and positive correlation with infant mortality (Figure 6 and Figure 7); thus ignoring precipitations (as in column 1) would underestimate the parameter β_1 .

³¹ In fact, Table 2 shows that high-income municipalities also present high percentage of families with private health insurance. Thus, families living in different municipalities have different access to quality health care, and are better/worse equipped to cope with the adverse effects of exposure to PM₁₀ pollution.

2SLS regression exploits the variation in PM₁₀ pollution concentrations that is due solely to variation in ventilation of the air basin, and not due to variation in emissions of PM₁₀ pollutants. Possible time-varying confounding effects may arise from failure to control for changes in economic activity. That is, economic activity should be positively correlated with pollution emissions, as well as it is likely to be negatively correlated with infant mortality (via its effect on access to, or supply of, high-quality healthcare). Thereby, failing to account for changes in economic activity would result in downward biased estimates of β_1 . This may explain the larger parameter estimates for β_1 from the 2SLS regressions (columns 5 through 7 of Table 5) as compared to those of OLS regressions (columns 1 and 2 of Table 5).³² Moreover, the 2SLS estimates for β_1 turn significant even after adding controls for temperature, precipitation, month and year dummies (columns 5 and 6 of Table 5). However, as in the OLS and FE estimations outlined above, the 2SLS estimate for β_1 turns smaller after adding these controls.

Finally, controlling for municipality-specific fixed effects, in columns 8 through 10 of Table 5, the FE 2SLS regression yields parameter estimates that are robust to the presence of possible time-varying confounders and that also account for municipality-specific fixed-effects. Results presented in columns 8 through 10 of Table 5 show that the FE 2SLS estimates turn statistically significant, even after adding seasonal controls (temperature, precipitation), month and year dummies, and municipality-specific year trends.

³² On the other hand, if the main source of bias in the OLS and FE estimates had been seasonal viral outbreaks, and if PM₁₀ and seasonal viral outbreaks are positively correlated (seasonal correlation), then we should have found larger estimates for β_1 under OLS and FE than under 2SLS and FE.

The parameter estimates in columns 7 and 10 are of particular interest. They are not just robust to the presence of possible unobserved time-varying confounders, but also—by introducing municipality-specific exogenous variation in air pollution—they are robust to possible presence of residential sorting that may overestimate the effect of air pollution on infant mortality.³³ In fact, a comparison of the parameter estimates presented columns 9 and 10 of Table 5 (as well as those presented in columns 6 and 7) suggests that residential sorting may indeed overestimate the effect of air pollution on infant mortality rate. For example, the parameter estimate for β_1 in column 9 suggests that a $10\text{-}\mu\text{g}/\text{m}^3$ reduction in ambient PM_{10} concentrations decreases infant mortality rate by .424 points. However, as well-off individuals can afford to live in expensive municipalities, in the East bound of Santiago, they are thus exposed to lower levels of pollution. Thereby, according to our estimate presented in column 10, the effect of a $10\text{-}\mu\text{g}/\text{m}^3$ reduction in ambient PM_{10} concentrations is to decrease infant mortality rate by .346 points, which is about 18 percent smaller than the one presented in column 9.³⁴

³³ That is, certain groups of people (say, those well-off living in municipalities with high per-capita income) are not just better equipped to cope with the adverse effects of PM_{10} pollution (due to, say, better access to quality health care) but they are also exposed to lower concentrations of PM_{10} pollution because they can afford to live in relatively expensive municipalities (that is, municipalities in the East bound of the city, which are exposed to lower concentrations of air pollution). Whereas the former presents a *direct* effect on infant mortality (which is controlled for by the municipality-specific fixed effect), the latter has an *indirect* effect on infant mortality, via lower exposure to air pollution (which is controlled for by introducing municipality-specific exogenous variation in air pollution).

³⁴ We also ran this specification of municipality-specific instruments by interacting thermal inversions with ground elevation at the urban centroid of each municipality. Results from this specification (not shown) remain qualitatively the same.

In terms of the overall effect of air pollution on infant mortality, the parameter estimates presented in column 10 of Table 5 should be interpreted as follows. The estimate for the effect of ambient PM₁₀ pollution on infant mortality presented in column 10 suggests that a 10- $\mu\text{g}/\text{m}^3$ reduction in average exposure to ambient PM₁₀ yields 17.8 fewer all-cause infant deaths per 100,000 infants, per year [$52 \times .346 = 18.0$]. This represents a 2.2 percent decrease with respect to mean all-cause infant mortality (presented in the last column of Table 1). The average number of infants a year in Santiago for the period 1997-2008 is 85,006 (see Table 1), so that our estimate suggests there would be about 15.3 fewer infant deaths a year as a result of a 10- $\mu\text{g}/\text{m}^3$ reduction in average ambient PM₁₀ pollution. Moreover, Figure 4 shows that average ambient PM₁₀ pollution in Santiago decreased by about 30- $\mu\text{g}/\text{m}^3$ from the late 1990s to 2008. Thereby our estimate suggests that such a reduction in average ambient PM₁₀ pollution prevented about 45.9 infant deaths over the period 1997-2008.

Table 5: Estimates of the Effects of Exposure to PM₁₀ Pollution on Overall Infant Mortality Rate.

VARIABLES	Infant Mortality Rate									
	OLS		FE		2SLS			FE 2SLS		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
PM10	0.396*** (0.065)	0.085 (0.096)	0.375*** (0.075)	-0.039 (0.109)	0.698*** (0.112)	0.469*** (0.180)	0.445*** (0.166)	0.711*** (0.115)	0.424** (0.184)	0.346** (0.174)
- Controls for temperature, precipitations, month & year dummies	No	Yes	No	Yes	No	Yes	Yes	No	Yes	Yes
- Controls for municipality-specific year-trends	No	No	Yes	Yes	No	No	No	Yes	Yes	Yes
- Municipality-specific instruments					No	No	Yes	No	No	Yes
Observations	18,800	18,800	18,800	18,800	18,800	18,800	18,800	18,800	18,800	18,800
R-squared	0.002	0.008	0.002	0.008	0.001	0.007	0.007	0.000	0.009	0.009

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

Table 6: Estimates of the Effects of Exposure to PM₁₀ Pollution on Infant Mortality Rate Due to Respiratory and Cardiovascular Diseases.

VARIABLES	Infant Mortality Rate Due to Respiratory and Cardiovascular Diseases									
	OLS		FE		2SLS			FE 2SLS		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
PM10	0.131*** (0.020)	0.026 (0.029)	0.140*** (0.026)	0.024 (0.031)	0.202*** (0.031)	0.109** (0.053)	0.066 (0.048)	0.206*** (0.032)	0.104* (0.054)	0.094* (0.052)
- Controls for temperature, precipitations, month & year dummies	No	Yes	No	Yes	No	Yes	Yes	No	Yes	Yes
- Controls for municipality-specific year-trends	No	No	Yes	Yes	No	No	No	Yes	Yes	Yes
- Municipality-specific instruments					No	No	Yes	No	No	Yes
Observations	18,800	18,800	18,800	18,800	18,800	18,800	18,800	18,800	18,800	18,800
R-squared	0.003	0.015	0.003	0.015	0.002	0.015	0.015	0.003	0.019	0.019

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

c. Effect of Ambient PM₁₀ Pollution on Infant Mortality Due to
Respiratory and Cardiovascular Diseases

Table 6 presents results for the effect of ambient PM₁₀ pollution on infant mortality due to respiratory and cardiovascular diseases. The results mimic those presented in the previous section. Whereas both the OLS and FE estimates turn statistically significant (columns 1 and 3), once we introduce seasonal controls, time dummies (and municipality-specific year trends, in column 4 only), these estimates turn non-significant (columns 2 and 4). On the other hand, the 2SLS estimates presented in column 5 and the FE 2SLS estimates presented in column 8 both turn statistically significant, even after introduction of seasonal controls, month and year dummies and time trends (columns 6 and 9) both the 2SLS as well as FE 2SLS yield (weakly) significant estimates. Furthermore, when exploiting municipality-specific exogenous variation in air pollution both 2SLS and FE 2SLS estimates turn slightly smaller (columns 7 and 10) and only weakly significant in the FE 2SLS regression.

Our estimate for the effect of ambient PM₁₀ pollution on infant mortality due to respiratory and cardiovascular diseases presented in column 10 suggests that a 10- $\mu\text{g}/\text{m}^3$ reduction in average exposure to ambient PM₁₀ concentrations prevents, each year, 4.8 infant deaths due to respiratory and cardiovascular diseases, per 100,000 infants. This figure represents an 8.3 percent decrease with respect to mean deaths due to respiratory and cardiovascular disease a year.

7. Additional Results – Effect of Cumulative Exposure to Ambient PM₁₀

Pollution

The epidemiological and medical literatures have studied both acute and chronic effects of exposure to air pollutants. Whereas chronic effects usually refer to exposure during the entire life span, acute effects have usually been only vaguely defined as short term exposure. Due to limitations for identification, most of the recent literature has focused largely on acute exposure to air pollution.³⁵ The medical literature suggests that exposure to air pollutant should have a detrimental effect on human health, and that it should continuously deteriorate when exposed to a (highly) polluted environment. Indeed, in the case of mortality as an extreme health outcome, it is likely that those deceased during episodes of high pollution are individuals whose health has been continuously deteriorating up to a point that they became very vulnerable to die if exposed to a (highly) polluted environment, even for a relatively short period of time.

In this section we use the same identification strategy as in section 6 to explore the effects of *cumulative exposure* to ambient PM₁₀ pollution over a longer period of time, that is, over several consecutive weeks. To instrument for average ambient PM₁₀ pollution over n ($n > 1$) weeks we use n lags for the meteorological variables described in section 5 (thermal inversions). Figure 8 presents FE 2SLS results for the effect of cumulative exposure to PM₁₀ pollution for a period of up to two twenty six

³⁵ An exception is the work of is the recent work of Anderson (2015) that looks at mortality among elderly individuals living in the proximity of U.S. highways in Los Angeles Metropolitan Area and compares those living downwind to those living upwind from major highways.

weeks (half a year). In the figure each estimate for β_1 is plotted alongside its corresponding 95 percent confidence interval. The figure shows that the effect of cumulative exposure to ambient PM₁₀ pollution is increasing in the number of weeks of exposure up to 13 weeks (one quarter), and it can be more than three times as large as the acute effect (effect of exposure during the current week). This result suggests that our estimates for acute exposure presented in Table 5 should be taken as a conservative (lower bound) estimate of the overall effect of exposure to ambient PM₁₀ pollution on infant mortality.

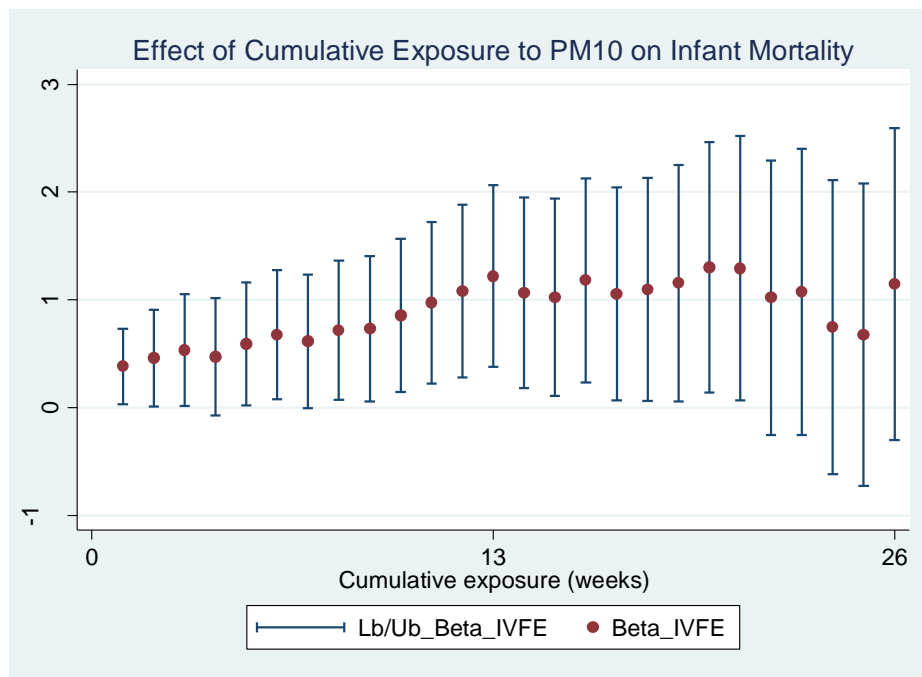


Figure 8: Effect of Cumulative Exposure to PM₁₀ Pollution on Infant Mortality Rate

8. *Discussion*

Our results for the effect of ambient PM₁₀ pollution on infant mortality fall somewhere in between those results from previous studies, such as for Mexico City and for California. Whereas our estimate of the effect of a 10- $\mu\text{g}/\text{m}^3$ reduction in exposure to ambient PM₁₀ on infant mortality is 17.8 fewer infant deaths per 100,000 infants, per year, Arceo, Hanna, and Oliva (2016)'s estimate is 123.2 fewer infant deaths and Currie and Neidell (2005)'s estimate is 16.4 fewer infant deaths. Furthermore, these fewer infant deaths represent a 2.2 percent decrease of mean annual infant mortality for Santiago, a 6.2 percent decrease for Mexico City and a 4.2 percent decrease for California.³⁶

This suggests that the estimates of the concentration-response function for the effects of exposure to ambient PM₁₀ pollution on infant mortality vary according to different countries and geographical locations.³⁷ This is important for the design of environmental health policy for different countries. In Chile, for example, the environmental authority has developed Air Pollution Control and Prevention Plans (PPDA) for Santiago since the late 1990s, and more recently also for other smaller cities. As explained earlier, PPDA consist of a battery of provisions for reducing air

³⁶ Recall that the average infant mortality rate for Santiago over the period 1997-2008 is 1.55 percent. Similarly, the average infant mortality rate in Mexico City over the period considered in Arceo, Hanna, and Oliva (2016)'s paper (1997-2006) is 1.987 percent. On the other hand, the average infant mortality rate in California for the period considered in Currie and Neidell (2005)'s paper (1989-2000) is .391 percent. That is, the average infant mortality rate in Santiago is about eighty percent as large as that of Mexico City and about four times that of California.

³⁷ There is a variety of reasons for these differences. It could be because of differences in the chemical composition of the pollutants, levels of concentrations of pollutants, characteristics of the population such as their health and capacity to cope with low air quality, government environmental and health programs, etc. (Arceo, Hanna, and Oliva (2016).

pollution. These PPDAs are evaluated ex-ante estimating social costs and benefits of expected improvements in air quality using concentration-response estimates from different studies.

For Chile's first PPDA in 1998, the concentration-response estimate that was used was that from Ostro et. al. (1996), which estimates that 10- $\mu\text{g}/\text{m}^3$ decrease in average ambient PM_{10} pollution was associated with a 1.1 percent decrease in mortality across all age groups. Our concentration-response estimate yields that 10- $\mu\text{g}/\text{m}^3$ change in average ambient PM_{10} pollution reduces mean all-cause infant mortality by 2.2 percent, a figure that is twice as large as that of Ostro et. al. (1996). This suggests that the estimated benefits from reducing ambient PM_{10} pollution concentrations in Santiago were likely to be largely underestimated in Santiago's 1998 PPDA.³⁸ Overall, using the estimates presented in this chapter would increase the estimated social benefits of air pollution reduction, thus suggesting that a more ambitious PPDA may have been worthwhile.

In fact, whereas we estimate that a 30- $\mu\text{g}/\text{m}^3$ reduction in ambient PM_{10} pollution (from the late 1990s to 2008) saved 45.9 infants lives, using Ostro et. al. (1996)'s estimates such a reduction in ambient PM_{10} pollution would have saved only 22.5 infant lives. Chile's Ministry of Social development recently set Chile's official value

³⁸ Reductions in the risk of premature mortality (across all age groups) represent 68 percent of the total benefits valued in Santiago's 1998 PPDA.

of a statistical life at US\$ 0.431 million (MINDES 2017). Thereby, those additional 23.4 saved infant lives can be valued today at US\$ 10.085 million.³⁹

9. Concluding Remarks

Air pollution is a growing concern in emerging economies. Estimating the effects of the adverse effects of ambient air pollution on human health is of great importance for the design of environmental policies that aim to reduce emissions of air pollutants and mitigates its adverse effects. In this chapter we analyzed the case of Santiago, Chile, a major city in an emerging economy that has suffered from the high levels of ambient air pollutants since the late 1980s.

We focused on the effects of exposure to ambient PM₁₀ pollution on infant mortality as particularly sensitive population (which can also be used as an index of overall human health). To accurately estimate the effects of air pollution on infant mortality we used a large municipality-week level dataset—spanning a period of 12 years—to obtain FE estimates that control for municipality-specific unobservable effects. Furthermore, we used detailed meteorological data that produces municipality-week exogenous variation on ambient PM₁₀ pollution concentrations (via *thermal inversions*) allowing us to control for possible time-varying municipality-week confounding effects. We found statistically significant effects on infant mortality both

³⁹ Chile's official figure for value of statistical life varies according to whether it is for deaths associated to traffic accidents or for deaths associated to cardiovascular and respiratory due to air pollution. In the calculation above we used the latter.

for all-cause infant mortality as well as infant mortality due to respiratory and cardiovascular diseases. We find that the effect of a $10\text{-}\mu\text{g}/\text{m}^3$ reduction in exposure to ambient PM_{10} pollution on infant mortality results in 18 fewer infant deaths a year, per 100,000 live births. Furthermore, we contrasted our estimates to those from recent studies in California and Mexico City. Our results suggests that extrapolation of concentration-response estimates from industrialized countries to emerging economies should be conducted with caution.

Chapter 2: Air Pollution and Urgent Care Visits: Estimation of a Causal Relationship Using Exogenous Variation of PM_{2.5} Concentrations in Santiago, Chile

1. Introduction

There is a large literature that looks at the effects of real world exposure to air pollution on human health (Pope III and Dockery 2006). Most of the literature that focuses on the effects of acute exposure to air pollution looks at the effects of exposure to particulate matter (PM) on emergency respiratory visits and on hospital admissions due to respiratory diseases (Zheng et al. 2015; Wilson et al. 2004; Lim et al. 2016). For example, early studies in the U.S. (Seattle, WA) examine the effect of PM_{2.5} pollution on hospital emergency department visits among children (Norris et al. 1999) and the non-elderly population (Sheppard et al. 1999). Around the same time, studies in Santiago, Chile look at the effects of PM pollution on urgent care visits to primary health facilities (Ostro et al. 1999) and on visits to a Children's Hospital (Ilabaca et al. 1999).⁴⁰

In this chapter I look the health effects of acute exposure to fine particulate matter (PM_{2.5}) on urgent care visits in Santiago, Chile. I look at both the effects of same-day

⁴⁰ Since then, there is a large literature of studies that looks at the acute health effects of air pollution in industrialized countries such as the U.S., Australia and Europe, as well in middle income and developing countries such as Chile, Australia, Korea, Taiwan and China (Prieto-Parra et al. 2017; Fusco et al. 2001; Hwang, Hu, and Chan 2004; Tsai et al. 2006; Ko et al. 2007; Park et al. 2013; Jalaludin et al. 2008).

exposure as well as the effect of exposure over a period of up to fourteen consecutive days. The existing literature examines the effect of particulate air pollution across different age groups as well as across different cardiopulmonary diseases such as asthma, pneumonia, upper and lower respiratory diseases, cardiovascular and circulatory diseases, etc. (Pope 3rd 2000). Likewise, I estimate effects for different age groups (infants, toddlers, children, adults and elderly) and focus on those urgent care visits associated to respiratory diseases (upper respiratory, lower respiratory, pneumonia and respiratory associated to influenza) and those urgent care visits associated to cardiovascular and circulatory diseases.

However, most of the studies in the existing epidemiological literature fail to identify a causal effect of air pollution on emergency respiratory visits because of failing to control for potential confounding factors. When important factors that affect health outcomes, and that also correlate with air pollution, are not properly accounted for the effect of such unobserved factor is captured by the air pollution variable. This introduces bias on the estimate of the effect of air pollution on the health outcome and, thereby, it prevents from identifying a causal effect of air pollution on the health outcome. Successfully controlling for possible confounding effects remains a major challenge for most of the epidemiological literature (Touloumi et al. 2006) and there is skepticism on whether the effects identified by this literature are actually causal or mere statistical associations (Gamble and Lewis 1996).

With the exception of Braga et al. (2000), who control for respiratory epidemics, most epidemiological studies fail to account for the effect of seasonal respiratory viruses. Outbreaks of seasonal respiratory viruses usually occur during the winter time, around the same time of the highest levels of PM pollution concentrations. Therefore, failing to account for seasonal respiratory viruses overestimates the damaging effects PM pollution on health. In this chapter I explicitly account for seasonal respiratory viruses by using data on the prevalence of seasonal respiratory viruses during Santiago's winter period (syncytial virus, type A flu, type B flu, parainfluenza, adenovirus and metavirus). However, I do not account for other non-viral diseases that may also drive respiratory or cardiovascular and circulatory urgent care visits.

Yet, failing to account for seasonal respiratory viruses (or non-viral diseases) is not the only reason that estimates of the effects of exposure to air pollutants on health outcomes may be biased. For example, avoidance behavior may also act as a confounding effect. The problem of avoidance behavior arises when, at times of high pollution concentrations, those individuals most affected by air pollution take special provisions to avoid exposure to harmful pollutants (such as using a breathing mask or restricting time spent outdoors). Then failing to account for this avoidance behavior will underestimate the effects of pollution on health. In this chapter I provide indirect evidence of the absence of such avoidance behavior in this data from Santiago.

Furthermore, to estimate a causal effect of exposure to air pollution on health outcomes, in this chapter I use exogenous variation in $PM_{2.5}$ pollution concentrations

driven by thermal inversions in Santiago and by FIFA World Cup (WC) games. Whereas thermal inversions trap air pollutants at the city level and prevents from proper ventilation, on days of WC games (particularly weekend games) there are major peaks of $PM_{2.5}$ pollutants emissions as concentrations increase by 63 percent, presumably due to massive grill-outs on occasions of those games. I show that both thermal inversions as well as WC games largely drive $PM_{2.5}$ pollution concentrations during the winter time in Santiago and I argue that these meteorological phenomena and sport events do not affect respiratory health outcomes other than via their effect on air pollution concentrations. However, massive grill-outs on occasions of WC games may affect cardiovascular and circulatory diseases via fatigue due to overeating and/or stress from watching the games. Therefore, when estimating the effects of $PM_{2.5}$ pollution on cardiovascular and circulatory diseases I only use variation in $PM_{2.5}$ pollution concentrations from thermal inversions.

Whereas the existing literature does not exploit the occurrence of massive grill-outs on occasions of WC games as source of exogenous variation in air pollution concentrations, a couple of papers use thermal inversions as source of exogenous variation in air pollution to estimate effects on health outcomes. Arceo, Hanna and Oliva (2016) use a weekly count of thermal inversions as an instrument for weekly PM_{10} and CO concentrations for municipalities within Mexico City to estimate the effect of these pollutants on infant and neonatal mortality. Similarly, Jans, Johansson and Nilsson (2014) use a dichotomic variable for the presence of overnight thermal inversions to instrument for next-day PM_{10} concentrations and estimate its effects on

children's acute respiratory diseases (asthma, pneumonia and bronchitis).⁴¹ However, Jans, Johansson and Nilsson (2014)'s data does not actually guarantee measuring the thermal inversion at the relevant elevation that creates the trapping of air pollutants. To generate data on the presence of thermal inversions they impute temperature data at different elevations based on inferring the associated elevation of air masses at a given level of atmospheric pressure. Furthermore, they have missing data on cloudy days (which represents 13.5 percent of the days in their data), introducing an important selection problem that may bias their results.⁴²

Unlike these previous studies, however, to instrument for PM_{2.5} concentrations I use a continuous variable that measures not just the presence but also the intensity of the thermal inversion (through the difference of air temperature at different elevations). Furthermore, I directly observe the air temperature throughout the entire period considered for this chapter and at the relevant elevation of occurrence of the thermal inversion in Santiago. This, together with the exogenous variation from WC games, provides a much stronger instrument for air pollution concentrations, and yields more accurate health effects estimates. Furthermore, I provide estimates not only for

⁴¹ Beard et al. (2012) look at the 'reduced-form' direct effect of thermal inversions on respiratory visits for asthma in Utah. However, they do not use thermal inversions to instrument for air pollution and, thereby, do not provide estimates of the effect of air pollution on respiratory visits for asthma. Similarly, two papers in the biomedical literature look at the effect of wildfires on increasing respiratory emergency department visits in southern California (Dohrenwend et al. 2013) and Australia (Haikerwal et al. 2016). If wildfires affect respiratory emergency visits only via increasing air pollution, then this could bring in exogenous variation in air pollution that could potentially establish a causal relationship on respiratory emergency department visits. However, these papers look only at the 'reduced-form' direct effect of such wildfires on increasing respiratory emergency department visits, and fail to use statistical tools (such as instrumental variables estimation) that would allow them to estimate a causal effect.

⁴² As we will explain in detail in the next section, thermal inversions, and the trapping of pollutants because of their occurrence, are more likely to occur on cloudy days than on not cloudy days. This means that Jans, Johansson and Nilsson (2014) use a non-random sample of days for which they can readily instrument for air pollution. This may likely introduce bias in their reported estimates.

children but for a wide range of the population according to different age groups (infants, toddlers, children, adults and the elderly).

The rest of the chapter is organized as follows. In the next section I present background information on urgent care visits as well as determinants of $PM_{2.5}$ pollution concentrations in Santiago. Then, in section 3 I present the data on urgent care visits (particularly, respiratory visits and cardiovascular and circulatory visits) as well as data on $PM_{2.5}$ pollution in Santiago, and I show its correlation with both WC games as well as thermal inversions in section 4. In section 5 I present the empirical strategy to estimate the effects of air pollution on urgent care visits using two-stage least squares. In section 6 I present results for the effects of exposure to $PM_{2.5}$ pollution on respiratory urgent care visits (upper respiratory, lower respiratory, pneumonia and respiratory due to influenza) and on cardiovascular and circulatory visits, for different age groups (infants, toddlers, children, adults and the elderly). I present effects of both contemporaneous (same-day) exposure (section 6.a.) as well as cumulative exposure (over consecutive days) (section 6.b.). I present robustness checks in section 7. I discuss policy implications in section 8 and section 9 concludes.

2. Background on Urgent Care Visits and $PM_{2.5}$ Pollution in Santiago

a. Urgent Care Visits in Santiago

The first system in the human body to get in contact with air pollutants is the respiratory tract. Whereas the bigger fraction of particles usually gets trapped on the upper respiratory tracts, the smaller fraction of particles can reach down to the lower respiratory tract and even enter the blood stream through the alveoli in the lungs (Nadadur and Hollingsworth 2015). Furthermore, exposure to air pollution may decrease immune functions and increase the risk of acute respiratory infections. In Santiago, acute respiratory illnesses have become the most common cause of medical visits in the cold winter months and are considered a priority by Chile's health authorities (Ilabaca et al. 1999). Urgent care visits reflect adverse events of clear clinical significance and may feasibly relate to recent exposure to air pollution. The general public in Santiago seems to be aware of the adverse effects of air pollution on health. A recent survey by Chile's Ministry of Environment shows that air pollution is the main environmental problem for Santiago's population (Dirección de Estudios Sociales 2017). This is in large part due to the adverse health effects on the most vulnerable population, particularly during the winter period.

b. PM_{2.5} Pollution Concentrations

Airborne particulate matter less than 2.5 micrometers (PM_{2.5}) is a heterogeneous mixture of chemical and particle sizes. In Santiago it is largely composed of organic

and elemental carbon (52 percent and 10 percent, respectively), nitrates (15 percent), ammonium (14 percent), and sulphates (15 percent) (Ministerio del Medio Ambiente 2016). Concentrations of PM_{2.5} pollution in Santiago's Metropolitan Area are caused by both pollution emissions as well as by accumulation of pollutants due to the lack of proper ventilation of the city's air basin. Emissions of PM_{2.5} in Santiago's Metropolitan Area are largely due to motor vehicles (accounting for 39.6 percent of total anthropogenic sources), the use of firewood or coal for residential heating and cooking (38.5 percent of total anthropogenic sources), and the combustion of petroleum derivatives for industrial processes (15.4 percent of total anthropogenic sources) (Ministerio del Medio Ambiente 2016). On the other hand, as explained in section 2.b. of Chapter 1, the process of ventilation of Santiago's air basin is largely determined by both the unique geography around the city and by the presence of thermal inversions above the city's air basin. In section 2.b. of Chapter 1 I thoroughly explain how thermal inversions drive overall ventilation of air pollutants in Santiago's air basin. Those same concepts also apply for ventilation of PM_{2.5} pollution. However, in addition to ventilation of air pollutants driven by the presence of thermal inversion, in the next subsection I look at short-lasting peaks in emissions of PM_{2.5} pollution.

- i. Peak Emissions of PM_{2.5} Pollutants on Occasions of FIFA World Cup Weekend Games

In Chile there is a long tradition of grilling for social gatherings, particularly when most of the attendees are expected to be men. In a country where FIFA rules' football largely dominates all other sports, there is also great enthusiasm for the Chilean national team, particularly among men.⁴³ During the FIFA 2014 World Cup Chilean football supporters had probably many gatherings on occasion of national team's games. Most of these gatherings, particularly those on occasion of games on weekends or Friday evenings, were probably accompanied by many grill-outs and long hours of emitting high amounts of particulate air pollutants.

At the time of the FIFA 2014 World Cup Santiago's highest official (equivalent to the city mayor), as well as the environmental authority, made public announcement on TV and media warning about the effects of such massive grill-outs on spiking air pollution concentrations and its associated health risks, particularly for the most vulnerable population. Despite the authorities' public calls, on the day of every national team's weekend game, as well as the day after a national team's weekend game, the concentrations of PM_{2.5} pollution reached its highest levels during the winter of 2014 in Santiago, largely exceeding Chile's standards.⁴⁴

⁴³ This enthusiasm is further exacerbated by Chile's national team currently enjoying what is arguably the best generation of players in its one hundred years of history, and the national team currently achieving its highest performance ever. Proof of it is that, after not ever winning a single major tournament during almost a hundred years of history, Chile's team has recently won two consecutive major tournaments (Copa America) in years 2015 and 2016. The buildup of this team started long before winning these two tournaments though, and by the time of the FIFA 2014 World Cup the team's performance was near its peak performance. Enthusiasm among supporters was probably near its peak as well.

⁴⁴ Chile's standards for PM_{2.5} pollution are 20 $\mu\text{g}/\text{m}^3$ (as annual mean), and 50 $\mu\text{g}/\text{m}^3$ (as 24-hour mean).

3. *Data*

I obtain air pollution data from the Air Quality Monitoring Network (AQMN) of Chile's Ministry of Environment. AQMN data comes from 10 monitoring stations located throughout Santiago's Metropolitan Area (green dots in Figure 1). These monitoring stations employ a USEPA-approved air quality monitoring system (Met One BAM-1020) that uses beta ray attenuation to record hourly concentrations, in micrograms per cubic meter ($\mu\text{g}/\text{m}^3$), of particles with diameter less than 2.5 micrometers ($\text{PM}_{2.5}$)⁴⁵. I average out the hourly $\text{PM}_{2.5}$ pollution data across these 10 monitoring stations and construct daily mean $\text{PM}_{2.5}$ pollution for the period May 1st through August 15th.⁴⁶

I obtain meteorological data from Chile's Meteorological Service. I obtain hourly data of temperature, precipitations and relative humidity from a meteorological station located at the city's elevation (La Platina, at an elevation of 556 meters above sea level) and construct daily meteorological data covering the same period of time. In order to account for the presence of a thermal inversion in Santiago's air basin I use data from a meteorological station (Lo Prado, at an elevation of 1,080 meters above sea level) located on the top of Santiago's western mountain range, just about 500 meters above the city's ground elevation. From the meteorological station Lo Prado I obtain ambient air temperature and I contrast this temperature data with that

⁴⁵ This is about 3 percent of the diameter of a human hair.

⁴⁶ I aggregate $\text{PM}_{2.5}$ pollution at the day-level because data on urgent care visits is also available also at the day level. This allows us to examine how daily variation on exposure to $\text{PM}_{2.5}$ pollution affects urgent care visits in Santiago. Later on, I also look at the effect of cumulative exposure to $\text{PM}_{2.5}$ pollution by aggregating throughout consecutive days.

of La Platina. I take the difference in ambient air temperature from these two stations to measure the presence and intensity of a thermal inversion on top of Santiago's air basin.

For the health data I obtain official daily registry data on health statistics from the Department of Statistics and Health Information of Chile's Ministry of Health. The data comes from both public hospitals as well as public primary urgent care facilities ("*Servicios de Atención Pública de Urgencia*", also known as SAPUs). I restricted the data to include only those municipalities that make the Metropolitan Area of Santiago. These public health facilities provide primary health care to more than 70 percent of Santiago's population, primary for the bottom four quintiles of the income distribution. I aggregate this daily data across all Santiago's public primary care facilities for the period of May 1st through August 15th, roughly covering Santiago's full winter season. Furthermore, I also collect weekly data on the prevalence of seasonal respiratory viruses from the Public Health Institute (PHI) of Chile's Ministry of Health. PHI collects weekly lab samples from public hospitals' outpatients (infants and children less than 5 years old) and tests for the presence of respiratory viruses. These respiratory viruses include respiratory Syncytial Virus (RSV), Type A Flu, Type B Flu, Parainfluenza, Adenovirus and Metavirus.

The health data classifies urgent care visits by different age brackets, allowing us to analyze urgent care visits for the following age groups: infants (individuals less than

1 year old), toddlers (individuals 1 to 4 years old), children (individuals 5 to 14 years old), adults (individuals 15 to 64 years old), and elderly (individuals 65 and older).

In addition, the data on health outcomes allows urgent care visits to be characterized as respiratory visits and cardiovascular and circulatory visits according to the leading physician's diagnostic. Respiratory visits can be further classified into different types: upper respiratory, lower respiratory, pneumonia, influenza and other causes.⁴⁷ More specifically, I classify urgent care visits according to 10th version of the International Classification of Diseases (ICD-10). Upper respiratory visits refer to classifications J00-J06 for acute upper respiratory infections⁴⁸. Lower respiratory visits refer to classifications J40-J47 for chronic lower respiratory diseases⁴⁹ and classifications J20-J21 for other acute lower respiratory infections⁵⁰. Respiratory visits associated to pneumonia and influenza refer to classifications J12-J18⁵¹ and J09-J11⁵², respectively. Other respiratory visits refer to classifications J22, J30-J39, J47 and J60-

⁴⁷ Ibabaca et al. (1999) audited the quality of the record summary of emergency visits in Santiago with data from original medical records and found classification errors in less than 2 percent of the total records.

⁴⁸ These include: acute nasopharyngitis (common cold); acute sinusitis; acute pharyngitis; acute tonsillitis; Acute laryngitis and tracheitis; acute obstructive laryngitis [croup] and epiglottitis; and, Acute upper respiratory infections of multiple and unspecified sites.

⁴⁹ These include: bronchitis, not specified as acute or chronic; simple and mucopurulent chronic bronchitis; unspecified chronic bronchitis; emphysema; other chronic obstructive pulmonary disease; and, asthma.

⁵⁰ These include acute bronchitis and acute bronchiolitis

⁵¹ These include: viral pneumonia, not elsewhere classified; pneumonia due to streptococcus pneumoniae; pneumonia due to hemophilus influenza; bacterial pneumonia, not elsewhere classified; pneumonia due to other infectious organisms, not elsewhere classified; pneumonia in diseases classified elsewhere; and, pneumonia, unspecified organism.

⁵² These include: influenza due to certain identified influenza viruses; influenza due to other identified influenza virus; and, influenza due to unidentified influenza virus,

J98⁵³. On the other hand, cardiovascular and circulatory visits refer to classifications I11, I21, I47, I49, and I64⁵⁴.

4. Descriptive Statistics

a. Health Data

Table 7 below presents descriptive statistics (means and standard deviations) of daily respiratory and cardiovascular and circulatory visits by age group and type of respiratory illness, for the period May 1st through August 15th, 2014.

Whereas most of the urgent care visits are by adult individuals, respiratory visits are more frequent among the very young and the very old. Table 7 shows that, out of the 5,010 total average daily respiratory visits, 2,211 visits (representing 44.1 percent of the total) are by adult individuals. However, as a proportion of the number individuals in each age group, most respiratory visits are by infants, toddlers and the elderly. The breakdown by respiratory type shows that the most frequent respiratory visits are those due to upper respiratory diseases, especially among children and adults. Table 7 show that daily average upper respiratory visits represent 57.8 percent of the total

⁵³ These include: unspecified acute lower respiratory infection; other diseases of upper respiratory tract; bronchiectasis; lung diseases due to external agents; other respiratory diseases principally affecting the interstitium; suppurative and necrotic conditions of the lower respiratory tract; other diseases of the pleura; intraoperative and postprocedural complications and disorders of respiratory system, not elsewhere classified; and other diseases of the respiratory system.

⁵⁴ These include hypertensive heart disease; myocardial infarction; paroxysmal tachycardia; other cardiac arrhythmias; and cerebral infarction.

(2,895 out of 5,010 visits). This proportion of upper respiratory visits is even larger for children, 68.0 percent (615 out of 904), and for adults, 62.5 percent (1,381 out of 2,211). The second most frequent type of respiratory visits is that due to lower respiratory diseases, representing 21.4 percent of the total daily average respiratory visits (1,074 out of 5,010). Lower respiratory visits are more frequent among infants (43.8 percent of the total respiratory visits within this age group), toddlers (28.7 percent of the total respiratory visits within this age group), and the elderly (32.8 percent of the total respiratory visits within this age group). Although respiratory visits due to pneumonia represent only 3.9 percent of the total respiratory visits, most of these visits are by the elderly, with 37.8 percent of total respiratory visits due to pneumonia (73 out of 193). Finally, respiratory visits due to Influenza represent only 1.3 percent of the total respiratory visit, most of them (60.6 percent, or 40 out of 66) by adults.

On the other hand, almost all of the 268 total average daily visits for cardiovascular and circulatory diseases are by adults and the elderly, representing 59.3 percent and 39.6 percent of the total, respectively.

Table 7: Descriptive Statistics of Respiratory Urgent Care Visits for Santiago by Type and Age Group. Mean and Standard Deviation (in parenthesis).

	Respiratory Urgent Care Visits					Total	Cardio. & Circ. Visits	Obs.
	Upper	Lower	Pneumonia	Influenza	Other			
Less than 1 year old	169 (51)	183 (79)	21 (13)	2 (2)	44 (16)	418 (141)	0.3 (0.8)	106
1 to 4 years old	624 (225)	326 (148)	39 (22)	9 (9)	136 (56)	1,134 (436)	0.7 (1.3)	106
5 to 14 years old	615 (306)	120 (75)	10 (6)	14 (14)	145 (83)	904 (475)	2.3 (3.3)	106
15 to 64 years old	1,381 (397)	332 (132)	50 (19)	40 (35)	408 (136)	2,211 (696)	159 (23)	106
65 and older	106 (42)	113 (50)	73 (30)	2 (3)	49 (19)	344 (135)	106 (15)	106
All Age Groups	2,895 (934)	1,074 (449)	193 (80)	66 (58)	782 (284)	5,010 (1,740)	268 (36)	106

b. Pollution Data and Other Covariates

As mentioned earlier, PM_{2.5} pollution as well as other variables may affect respiratory and cardiovascular and circulatory visits. Table 8 below presents descriptive statistics for PM_{2.5} pollution, seasonal viral indexes and weather variables.⁵⁵ Table 8 shows that the average concentration of PM_{2.5} pollution for Santiago, over the period May 1st to August 31st 2014, largely exceeds the World Health Organizations guidelines as well as Chile's own standards.⁵⁶

Table 8: Descriptive Statistics of PM_{2.5} Pollution, Seasonal Viral Diseases and Weather Variables for Santiago (period May 1st through August 30th, 2014).

Variable	Mean	Std. Dev.	Obs.
PM _{2.5} (µg/m ³)	46.09	17.93	106
Syncytial Virus (%)	26.88	13.23	106
Type A Flu (%)	2.62	3.00	106
Type B Flu (%)	0.10	0.20	106
Para Flu (%)	2.87	1.83	106
Adenovirus (%)	1.10	0.67	106
Metavirus (%)	0.40	0.52	106
Temperature (Cº)	9.98	2.77	106
Precipitations (mm)	0.05	0.17	106
Relative Humidity (%)	76.67	9.23	106

⁵⁵ PM_{2.5} pollution is measured as micrograms per cubic meter (µg/m³) of airborne particles with a diameter of 2.5 micrometers or less. Seasonal virus indexes are measured as percentage of positive tests per calendar week for each virus (Syncytial, Type A Flu, Type B Flu, Para Flu, Adenovirus and Metavirus).

⁵⁶ World Health Organization's guidelines for PM_{2.5} pollution are 10 µg/m³ as annual mean, and 25 µg/m³ as a 24-hour mean. Chile's standard for PM_{2.5} pollution are 20 µg/m³ as annual mean and 50 µg/m³ as 24-hour mean.

To gain an idea of the serial correlations Figure 5 below presents a time plot of $PM_{2.5}$ pollution and total respiratory visits. Figure 5 presents LOWESS-smoothed time series plots of total respiratory visits and $PM_{2.5}$ pollution for the period May 1st to August 15th, 2014. I also add in Figure 5 a plot of weekly index for the prevalence of Type A Flu during the same period of time. Figure 5 shows a similar time pattern for all these three variables, showing a high correlation, particularly, regarding the peak that all three variables reach between late-June and early-July. This suggests that, when examining the effects of $PM_{2.5}$ pollution concentrations on respiratory visits, it is important to account for the prevalence of seasonal respiratory viruses (such as Type A Flu), since much of the variation in respiratory visits may be accounted for variation of these seasonal viruses, rather than by variation in concentrations of $PM_{2.5}$ pollution alone.

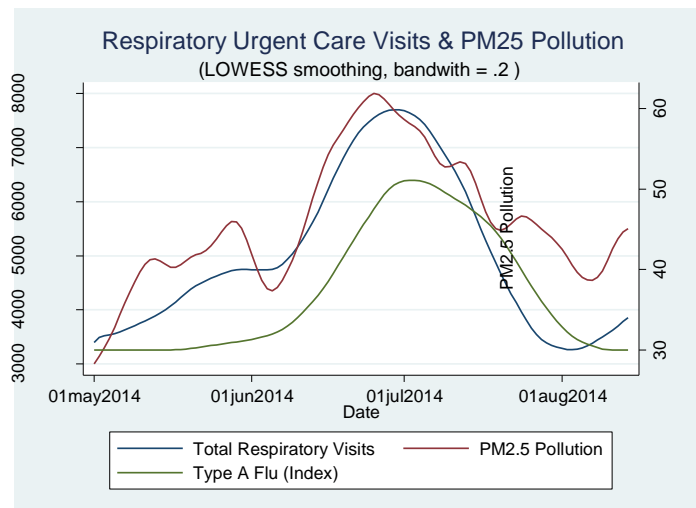


Figure 9 : Time Series of Total Respiratory Urgent Care Visits and Average $PM_{2.5}$ Pollution for Santiago. Period: May 1st through August 15th, 2014.

In addition, Figure 6 below shows LOWESS-smoothed time series respiratory visits by age group (left) and type of respiratory visit (right). In order to make the series comparable and show them in the same axis, I plot the standardized series for each category. The plot in the left shows that respiratory visits vary by age group, particularly between the very young, adults and the elderly. For infants, toddlers and children respiratory visits increase and reach a peak earlier than it does for adults and the elderly. Similarly, the plot in the right shows that respiratory urgent care visits vary by type, particularly between those upper, lower or other respiratory visits and those respiratory visits associated to influenza or pneumonia.

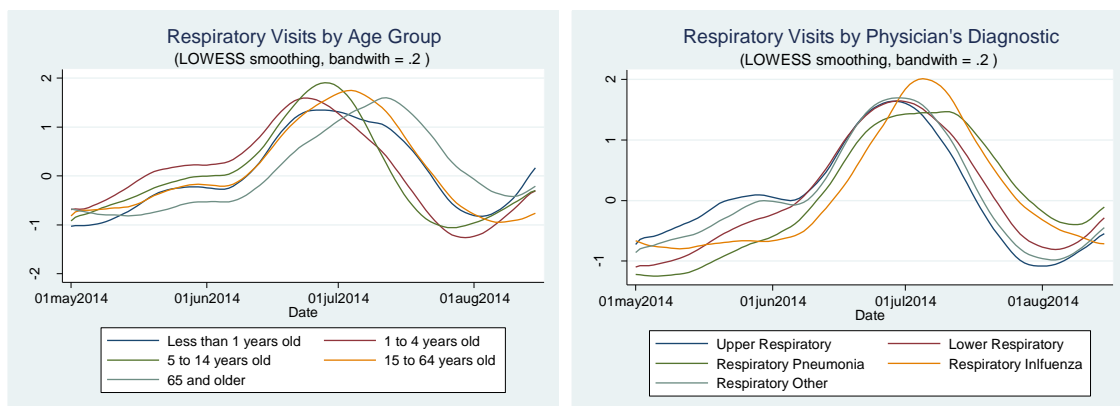


Figure 10: Time Series of Standardized Total Respiratory Visits by Age Group (left,) and Standardized Respiratory Visits by Care Professional’s Leading Diagnosis (right). Period: May 1st through August 30th, 2014.

i. FIFA World Cup Games and $PM_{2.5}$ Pollution

In Figure 11 below I use hourly average $PM_{2.5}$ pollution data to construct 24-hour average $PM_{2.5}$ pollution (red line) and plot it alongside with 24-hour windows around

WC weekend games (gray bars). The figure shows that $PM_{2.5}$ pollution peaks when there is a WC weekend game, and that the only times that 24-hour average $PM_{2.5}$ pollution reaches levels above $80 \mu g/m^3$ is around a WC weekend game. I believe that massive grill-outs on occasion of these WC weekend games create sharp increases in particulate pollution emissions. Hence, I observe these spikes in $PM_{2.5}$ concentrations around these WC games.

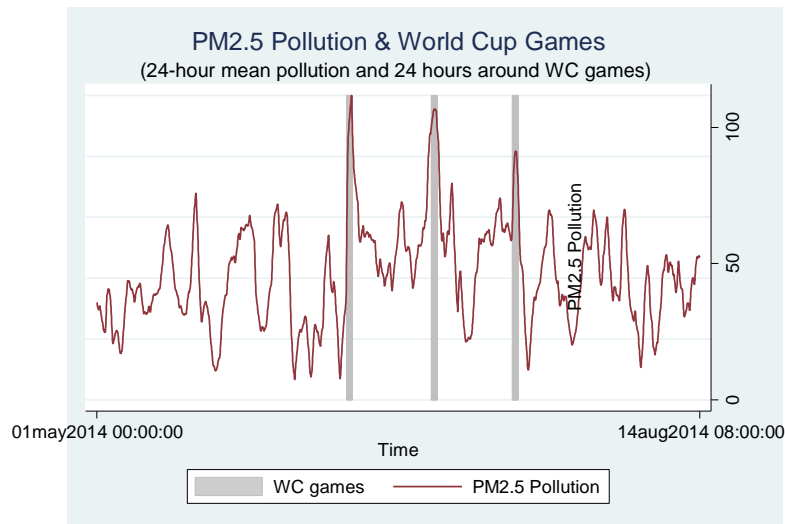


Figure 11: Time Series of $PM_{2.5}$ Pollution and Weekend Games. May 1st through August 30th 2014.

ii. Thermal Inversions, Precipitations and $PM_{2.5}$ Pollution

World Cup games are not the only reason to observe sudden changes in $PM_{2.5}$ pollution in Santiago. In addition to pollution emissions, ventilation of Santiago's air basin is one of the key determinants of $PM_{2.5}$ pollution concentrations. Both, thermal inversions as well as precipitations are major drivers of ventilation of Santiago's air

basin. The left graph in Figure 12 below presents the 24-hour average $PM_{2.5}$ pollution only for the month of the FIFA 2014 World Cup, June 10th to July 13th, 2014 (left). Furthermore, the right graph in Figure 12 shows 24-hour average temperature difference between the city elevation and the meteorological station located 500 meter above the city (blue line), precipitations (red bars), and 24-hour average temperature at the city elevation (green line). Figure 12 shows that 24-hour average $PM_{2.5}$ pollution increases at the time of thermal inversions (high 24-hour average temperature difference) and drops dramatically with precipitations, even if not strong ones.

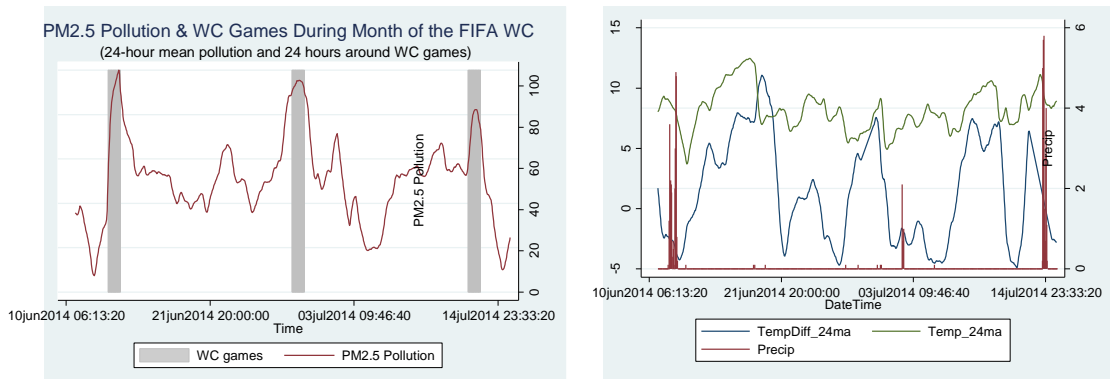


Figure 12: Time Series of $PM_{2.5}$ Pollution and Weekend Games (left), and Meteorological Variables (right) during the time of the FIFA World Cup 2014 (Jun 10th through July 15th, 2014).

5. *Empirical Strategy*

To estimate the effect of $PM_{2.5}$ pollution on urgent care visits—both respiratory and cardiovascular and circulatory visits—I ran two-stage least squares (2SLS) regressions using as instruments for $PM_{2.5}$ pollution concentrations both thermal

inversions as well as WC games. Furthermore, I contrast the 2SLS estimates with those from estimating by ordinary least squares (OLS). Equation (2) below sets the framework for analysis. I want to estimate the effects of the concentrations of $PM_{2.5}$ on day t (P_t) on urgent care visits on day t (Y_t), where Y_t can be either respiratory visits or cardiovascular and circulatory visits. This effect is captured by the parameter β . I control for a Seasonal Viral Index for week w (SVI_w), Precipitations on day t ($Precip_t$), and a vector of temperature, precipitation and relative humidity on the previous day (summarized by the vector W_{t-1}). Furthermore, I add a vector of dummies for each day of the week (D_t), and a vector of dummies for each month (M_t). Any unobservable effect, not accounted for in the variables listed above, is captured by the error term (ε_t).

$$Y_t = \alpha + \beta P_t + \gamma_1 SVI_w + \gamma_2 Precip_t + \gamma_3 W_{t-1} + \gamma_4 D_t + \gamma_5 M_t + \varepsilon_t \quad (2)$$

If there is any correlation between one of the variables in the right hand side and the error term then the entire vector of parameter estimates ($\alpha, \beta, \gamma_1, \gamma_3, \gamma_4$, and γ_5) turns not only biased but also inconsistent. This problem is known as omitted variable bias and it is likely to arise in the presence of confounders that are not accounted for in the model.⁵⁷ A well know technique to address this problem is by means of using an *instrumental variable*, say a variable z_t , that is not correlated with the error term, $Cov(z_t, \varepsilon_t) = 0$, but that is correlated with the endogenous variable x_t , $Cov(z_t, x_t) \neq$

⁵⁷ Furthermore, it is usually the case that if say, an endogenous variable x_t correlates with the error term, say $Cov(x_t, \varepsilon_t) \neq 0$, then the parameter estimate associated to such variable, is “greatly” biased and inconsistent.

0. Thus, by regressing by OLS x_t on z_t , conditional on the other variables in the model, I can predict values for x_t , and obtain \hat{x}_t . This is known as the first stage. Then, in the second stage regress Y_t on \hat{x}_t and the other variables in the model. This technique is commonly known as two-stage least squares (2SLS).

I use G_t and TI_t as instruments for P_t , where G_t is a vector of daily dummies for WC weekend games; and TI_t is a vector with variables *temperature difference* and *temperature difference squared* that capture the presence and intensity of thermal inversions of Santiago's air shed. Thereby, by estimating the equation below by OLS I obtain \hat{P}_t .⁵⁸

$$P_t = \lambda + \phi G_t + \phi TI_t + \psi_1 SVI_w + \psi_2 Precip_t + \psi_3 W_{t-1} + \psi_4 D_t + \psi_5 M_t + v_t$$

Finally, in the second stage I substitute \hat{P}_t for P_t in equation (2) above, and regress Y_t on \hat{P}_t and the other variables in the model (SVI_w , W_{t-1} , D_t , and M_t).

Therefore, even in the presence of possible confounding effects that may bias the estimates of β , if the underlying assumptions for the instruments G_t and TI_t hold true (that is, that they are correlated with $PM_{2.5}$ pollution but uncorrelated with the error term ε_t), then 2SLS yields unbiased and consistent estimates for β .⁵⁹

⁵⁸ $\hat{P}_t = \hat{\lambda} + \hat{\phi} G_t + \hat{\phi} TI_t + \hat{\psi}_1 SVI_w + \hat{\psi}_2 Precip_t + \hat{\psi}_3 W_{t-1} + \hat{\psi}_4 D_t + \hat{\psi}_5 M_t$

⁵⁹ For example, as explained earlier, an example of a confounder effect might be the presence of avoidance behavior. On the other hand, even though we control for seasonal viral indexes, the viral indexes that we use in this paper are only a proxy for the widespread prevalence of seasonal viruses

In addition to presenting parameter estimates for $\hat{\beta}$, in the next section I also present results in terms of *relative risk* estimates and its 95% confidence interval. The relative risk is the estimated change in the dependent variable—with respect to its mean value—for a change in PM_{2.5} equal to one standard deviation.⁶⁰ That is,

$$Relative\ Risk = \frac{1 + \hat{\beta}\sigma_P}{\bar{y}}$$

Furthermore, I examine the effects of cumulative exposure to PM_{2.5} pollution over different periods of exposure, from 1-day exposure to up to 14-day exposure. To do so, I modify the econometric framework laid out in equation (2) to better capture the effect of exposure over consecutive days, as in equation (3) below.

$$Y_t = \alpha + \beta AvgP_{(t-j ; t)} + \gamma_1 SVI_w + \gamma_2 Precip_t + \gamma_3 W_{t-1} + \gamma_4 D_t + \gamma_5 M_t + \xi_i \quad (3)$$

Where $AvgP_{(t-j ; t)}$, ($j = 0, \dots, 13$), denotes $(j+1)$ -day average PM_{2.5} pollution, over days $t - j$ to t . To obtain predicted values for $AvgP_{(t-j ; t)}$ in the 2SLS estimation, I use the lag of instruments, both FIFA 2014 World Cup games as well as thermal inversions, over days $t - j$ to t . This means that, for $j = 0$ (that is, 1-day exposure) I use basically the same estimating equation for the 2SLS as in equation (2) above; for $j = 1$ (that is, 2-day exposure) I use both contemporaneous as well as 1-day

that may affect the outcome variable Y_t . These variables are built upon the proportion of positive tests, for specific types of seasonal viruses, from a relatively small random sample of young children (mostly less than 5 years old). Therefore, they may imperfectly control for the presence of viruses among different cohorts of Santiago's population, particularly among older individuals. Furthermore, there might be non-viral effects (such as bacteriological diseases), not accounted for the viral indexes above, that while correlating with PM_{2.5} pollution, might also determine the outcome variable Y_t

⁶⁰ Recall from Table 7 that the standard deviation of PM_{2.5} in our sample is 17.93.

lagged instruments; for $j = 2$ (that is, 3-day exposure) I add 2-day lagged instruments, and so on. Furthermore, when computing relative risks, I calculate the standard deviation for the corresponding period of exposure to $PM_{2.5}$ pollution, that is I calculate $\sigma_{P(t-j ; t)}$.

6. Results

a. Effects of Contemporaneous Exposure to $PM_{2.5}$ Pollution

i. Contemporaneous Effect on Total Respiratory Visits, All Age Groups

Table 9 below shows parameter estimates for the effect of $PM_{2.5}$ on total respiratory visits for different specifications of the regression equation (2) above. Whereas columns 1 through 4 present results without day-of-week and month dummies, columns 5 through 8 present results when further adding time controls. Furthermore, results from OLS regressions are presented in columns 1 and 5, whereas results from 2SLS regressions are presented in columns 2, 3, 4, 6, 7 and 8. For the 2SLS results, columns 2 and 6 present results using only WC games as instruments; similarly, columns 3 and 7 present results using only thermal inversions as instruments; and, columns 4 and 8 present results using both WC games and thermal inversions as instruments. In all eight columns of Table 9 below I control for seasonal viral indexes (Syncytial virus, Type A Flu, Type B Flu, Para Flu, Adenovirus and Metavirus) and

weather variables (temperature, precipitation and relative humidity). Throughout, I present standard errors that are robust to heterogeneity and autocorrelation (HAC).⁶¹

Results from Table 9 show that, overall, PM_{2.5} pollution has a statistically significant effect on total respiratory visits (although results presented in column 6 turn non-significant). The OLS estimate ($\hat{\beta}_{OLS}$) from column 1 (without day-of-week and month dummies) suggests that a one-standard-deviation increase in PM_{2.5} pollution is associated with a 6.2 percent increase in respiratory visits (relative risk estimate 1.062). However when estimating by 2SLS, this effect turns much larger, suggesting the presence of important confounding effects. Columns 2 through 4 show that a one-standard-deviation increase in PM_{2.5} pollution increases respiratory visits by slightly more than 15 percent. Notice that this result holds regardless of the set of instruments I use. That is, whether I use only WC games as instruments (column 2), only thermal inversions as instruments (column 3), or both (column 4). The main difference of using different sets of instruments comes from the gains in precision of the $\hat{\beta}_{2SLS}$ estimate when using thermal inversions as compared to WC games, or better yet, using both. The standard error of the corresponding $\hat{\beta}_{2SLS}$, shrinks from 11.974 (column 2) to 11.152 (column 3) to 9.080 (column 4). The 95% confidence interval for the corresponding relative risk estimate shrinks accordingly.

⁶¹ We use Newey-West (1994)'s optimal lag method to correct the variance-covariance matrix.

Table 9: Parameter and Relative Risk Estimates of the Effect of PM_{2.5} Pollution on Total Respiratory Urgent Care Visits, All Age Groups.

	Total Respiratory Visits, All Age Groups							
	OLS (1)	2SLS (2)	2SLS (3)	2SLS (4)	OLS (5)	2SLS (6)	2SLS (7)	2SLS (8)
PM _{2.5}	17.264*** (3.605)	42.437*** (11.974)	43.826*** (11.152)	42.740*** (9.080)	10.008** (4.366)	18.078 (12.088)	25.925*** (5.330)	24.024*** (6.376)
Relative Risk	1.062 [1.04, 1.09]	1.152 [1.07, 1.24]	1.157 [1.08, 1.24]	1.153 [1.09, 1.22]	1.036 [1.01, 1.07]	1.065 [0.98, 1.15]	1.093 [1.06, 1.13]	1.086 [1.04, 1.13]
Day and Month Dummies	No	No	No	No	Yes	Yes	Yes	Yes
Instruments								
World Cup Games		Yes	No	Yes		Yes	No	Yes
Thermal Inversions		No	Yes	Yes		No	Yes	Yes
R-squared	0.678	0.639	0.635	0.638	0.870	0.867	0.856	0.859
Observations	106	106	106	106	106	106	106	106

HAC robust standard errors in parentheses (Bartlett kernel with 20 lags chosen by Newey-West's (1994) method). Relative risks's 95% confidence Intervals in brackets. *** p<0.01, ** p<0.05, * p<0.1.

On the other hand, as shown by the R-squared statistics, notice that the model without day-of-week and month dummies accounts for more than 60 percent of the variation of the dependent variable (columns 1 through 4). Adding time controls (columns 5 through 8), the model accounts for more than 85 percent of the variation of the dependent variable. Although adding day-of-week and month dummies allows for capturing unobserved time-specific effects, adding time controls yields smaller $\hat{\beta}$ estimates and smaller relative risks estimates, both when estimating by OLS as well as when estimating by 2SLS. The results of the OLS estimation accounting for time controls presented in column 5 suggest that a one-standard-deviation increase in PM_{2.5} pollution is associated with a 3.6 percent increase in respiratory visits. Similarly, the results for the 2SLS estimations accounting for time controls (columns 6 through 8) suggest that a one-standard-deviation increase in PM_{2.5} pollution increases respiratory visits by 6.5 percent to 9.3 percent, depending on the sets of instruments used in the 2SLS estimation. Since the model that uses all available instruments (column 8) yields a higher R-squared statistics, I choose the estimate from this model as the preferred estimate for the effect of PM_{2.5} pollution on total respiratory visits. That is, the results suggest that a one-standard-deviation increase in PM_{2.5} pollution increases respiratory visits by 8.6 percent.

In what follows, I use the 2SLS specification that uses all available instruments (both WC games as well as thermal inversions) to examine the effect of PM_{2.5} pollution on respiratory visits across different age groups (subsection 6.a.ii.), across different types

of respiratory diseases (subsection 6.a.iii.), and across both different age groups and different types of respiratory diseases (subsection 6.a.iv.). Furthermore, I present results for the effect of $PM_{2.5}$ pollution on cardiovascular and circulatory visits (subsection 6.a.v.). However, before moving on to those sections, in the next section I examine the first stage estimation and discuss the validity of the instruments.

1. First Stage Regressions

Table 10 below presents the first stage regressions of the 2SLS estimation of Table 9 discussed in the previous section. Whereas columns 1 through 3 of Table 10 present the first stage regressions without day-of-week and month dummies (corresponding to columns 2 through 4 of Table 9 above), columns 4 through 6 present the first stage regressions when adding time controls (corresponding to columns 6 through 8 of Table 9 above). Likewise, columns 1 and 4 of Table 9 present the first stage results when using only WC games as instruments, and columns 2 and 5 present results when using only thermal inversions as instruments. Columns 3 and 6 present results using both WC games and thermal inversions as instruments.

Table 10 shows that the instruments, both WC games and thermal inversions, have a strong and statistically significant effect in explaining the variation of $PM_{2.5}$ pollution. The parameter estimates associated to these instruments turn highly significant across all six columns. Furthermore, although same-day precipitations have a strong effect on explaining the variation of $PM_{2.5}$, I believe that precipitations also affect

respiratory and cardiovascular and circulatory visits directly⁶², so that I do not use same-day precipitations as instrument for PM_{2.5} pollution.

The Wu-Hausman test statistic in columns 2, 3, 5 and 6 of Table 10 below show that the 2SLS estimation yields a parameter estimate $\hat{\beta}_{2SLS}$ that is statistically different from that of OLS, $\hat{\beta}_{OLS}$. The Wu-Hausman p-value is less than 0.05, the conventional statistical significance. However, this test statistic is only weakly significant in the specification presented in column 1, when using only WC games and not adding time controls. Adding day-of-week and month dummies in column 4, the Wu-Hausman test statistic turns non-significant. Controlling for these time dummies may cancel out most of the effect of the WC games as instruments because all WC weekend games took place almost on the same day of the week (either Friday night or Saturday). Overall, however, the results from the Wu-Hausman test statistics suggests that, when estimating the effect of PM_{2.5} pollution on respiratory visits (equation 1 above), there may be important confounding effects that are left out in the initial model and may generate a downward bias in the OLS estimates.

⁶² I believe that precipitations affect respiratory visits via two channels. The most direct one is due to individuals choosing not to go to a respiratory urgent care visits on days of rain. I believe that those individuals that may feel already sick may prefer not to go to an urgent care facility in days of rain, because of concerns that exposure to outside rain may make them sicker. Moreover, mobility and transportation is relatively limited in those rainy days. The second reason is that precipitations in the days before attending an urgent care facility may correlate with overall air humidity that makes it easy for spreading and aggravation of bacterial diseases.

Table 10: First Stage Regressions for Estimating the Effect of PM_{2.5} Pollution on Total Respiratory Urgent Care Visits.

VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
FIFA 2014 World Cup Games						
Game #1 (Chile weekend game), lagged	44.466***		37.100***	45.673***		37.179***
Game #2 (Chile weekend game)	40.660***		29.856***	39.540***		26.742***
Game #2 (Chile weekend game), lagged	37.836***		25.803***	38.860***		32.523***
Game #3 (Finals weekend)	23.982***		24.997***	22.872***		20.895***
Thermal Inversions						
Temperature Difference (1084m vs 560m)		3.154***	2.797***		3.296***	2.966***
Temperature Difference , squared		-0.266***	-0.253***		-0.255***	-0.239***
Seasonal Viral Index (weekly)						
Syncytial virus	0.162	-0.241	-0.209	0.241	-0.264	-0.197
Type A Flu	0.553	1.413	1.368*	0.223	0.580	0.256
Type B Flu	17.170**	15.490*	16.249**	21.544**	23.970**	26.986***
Para Flu	-0.385	-1.425	-0.550	-0.105	-1.176	-0.367
Adenovirus	7.190***	8.081***	7.154***	6.496**	7.311***	6.928***
Metavirus	1.578	-0.322	2.437	3.179	2.182	4.751
Weather Controls						
Temperature, lagged	-1.217	-2.284***	-1.363**	-1.081	-2.307***	-1.530**
Precipitations	-34.381***	-20.703***	-19.968***	-30.069***	-16.979**	-15.571**
Precipitations, lagged	-0.618	-7.284	-2.693	2.968	-3.516	0.014
Relative Humidity, lagged	-0.323**	0.078	0.051	-0.352*	0.117	0.122
Day and Month Dummies						
	No	No	No	Yes	Yes	Yes
Additional Statistics						
Wu-Hausman p-value	0.0571	<0.01	<0.01	0.469	<0.01	<0.01
F test on instruments	44.30	28.43	34.45	19.42	25.11	26.44
OverID p-value	0.481	0.433	0.718	0.315	0.459	0.475
Partial R-squared	0.238	0.357	0.502	0.222	0.379	0.516
R-squared	0.563	0.632	0.715	0.593	0.675	0.747
Observations	106	106	106	106	106	106

*** p<0.01, ** p<0.05, * p<0.1

In order to further test the validity of the instruments I ran a generalized methods of moments (GMM) instrumental variables regression and tested the null hypothesis that all instruments are valid, against the alternative hypothesis that at least one of the instruments is not valid. In all six specifications, (columns 1 through 6 of Table 10) the p-values for the test of over identified restrictions leads us not to reject the null hypothesis. Therefore, I conclude that all instruments are valid. Furthermore, the F-test statistic for the null that all instruments are equal to zero is larger than 20 across all six specifications of the 2SLS estimation (with the exception of the specification in column 5, where the F-test statistic turns slightly less than 20). This means that the finite sample bias of the 2SLS estimate ($\hat{\beta}_{2SLS}$) is less than 5 percent of the finite sample bias of the OLS estimate ($\hat{\beta}_{OLS}$). In other words, this suggests that 2SLS estimation yields an estimate that is considerable closer than the OLS estimation to the true value of β .

On the other hand, the partial R-squared statistic, \bar{R}^2 , shows the proportion of the total variance of PM_{2.5} pollution that is accounted for by the instruments in the model (WC games and thermal inversions). The results from Table 10 above show that, whereas WC games account for about 22 percent of the daily variation of PM_{2.5} pollution during the period May 1st through August 15th (columns 1 and 4), thermal inversions account for about 36 percent of the variation in PM_{2.5} pollution during the same period of time (columns 2 and 5). Using all instruments together, they account for more than 50 percent of the daily variation in PM_{2.5} pollution (columns 3 and 6).

Additionally, the partial R-squared statistic has a direct relationship with the precision of the 2SLS estimate. Specifically, the standard error of the $\hat{\beta}_{2SLS}$ is inversely proportional to the squared root of the partial R-squared statistic. More precisely $S.E.(\hat{\beta}_{2SLS}) \cong \sqrt{1/\bar{R}^2} S.E.(\hat{\beta}_{OLS})$. However, this relationship does not fully hold in Table 9 above since I correct the variance-covariance matrix according to Newey-West(1994)'s method in order to obtain heterogeneity and autocorrelation consistent standard errors.

On the other hand, Table 9 provides point estimates of the direct effect of WC games on concentrations of PM_{2.5} pollution. Estimates presented in column 6 of Table 9 suggest that WC games increase PM_{2.5} pollution by 20 $\mu g/m^3$ to 37 $\mu g/m^3$, and by 29 $\mu g/m^3$ in average. This corresponds to a 63 percent increase with respect to average PM_{2.5} pollution for this period (see Table 8).

ii. Contemporaneous Effect on Total Respiratory Visits by Age Group

In this section I analyze how the effect of PM_{2.5} on total respiratory visits varies for different subgroups of the population according to the age groups presented in Table 7. In Table 11 below I present results in a different panel for each age group. Panel A presents estimates for infants (those less than 1 year old); Panel B presents estimates for toddlers (those 1 to 4 years old); Panel C presents estimates for children (those 5 to 14 years old); Panel D presents estimates for adults (those 15 to 64 years old); and,

Panel E presents estimates for the elderly (those 65 and older). Whereas column 1 presents OLS estimates, column 2 presents 2SLS estimates. Furthermore, the regressions presented in both columns 1 and 2 control for day-of-week and month dummies (which account for those time-specific unobserved effects). All regressions in Table 11 control for seasonal viral indexes (Syncytial virus, Type A Flu, Type B Flu, Para Flu, Adenovirus and Metavirus) and weather variables (temperature, precipitation and relative humidity).

The R-squared statistics presented in Table 11 below show that the model accounts for 83 to 88 percent of the variation of the total respiratory visits by age group when controlling for day-of-week and month dummies.⁶³ Also, and similar to the results presented in Table 9 above, adding time controls yields more conservative estimates for β .

The 2SLS estimates presented in Table 11 (column 2) are not just consistently larger than their OLS counterparts (columns 1), but as shown by their corresponding Hausman tests statistic, they are also statistically different from their OLS counterparts across all age groups.⁶⁴ In fact, the 2SLS estimates are about two to three times larger than that of OLS, suggesting that there may be important confounding effects that bias down the OLS estimates.

⁶³ Before introducing time controls (not shown in Table 11), the R-squared statistic falls within .43 and .77.

⁶⁴ Except from the results presented in Panel C for children (age group 5 to 14 years old), where according to the Hausman tests statistic the 2SLS is different from OLS only at 80% confidence. However, 2SLS and OLS estimates are statistically different when not introducing time controls.

Table 11: Parameter and Relative Risk Estimates of the Effect of PM_{2.5} on Total Respiratory Urgent Care Visits, by Age Group.

Total Respiratory Visits, by Age Group		
	OLS	2SLS
	(1)	(2)
Panel A: Less than 1 year old		
PM _{2.5}	0.520 (0.390)	1.866*** (0.486)
Relative Risk	1.022 [0.99, 1.06]	1.080 [1.04, 1.12]
R-squared	0.844	0.829
Hausman test on PM _{2.5}		21.65
Panel B: 1 to 4 years old		
PM _{2.5}	2.176 (1.400)	5.734*** (1.964)
Relative Risk	1.034 [0.99, 1.08]	1.091 [1.03, 1.15]
R-squared	0.844	0.832
Hausman test on PM _{2.5}		6.673
Panel C: 5 to 14 years old		
PM _{2.5}	2.541 (1.573)	4.596** (2.099)
Relative Risk	1.050 [0.99, 1.11]	1.091 [1.01, 1.17]
R-squared	0.836	0.833
Hausman test on PM _{2.5}		2.187
Panel D: 15 to 64 years old		
PM _{2.5}	3.960*** (1.304)	9.262*** (2.398)
Relative Risk	1.032 [1.01, 1.05]	1.075 [1.04, 1.11]
R-squared	0.885	0.875
Hausman test on PM _{2.5}		6.944
Panel E: 65 and older		
PM _{2.5}	0.810*** (0.279)	2.566*** (0.599)
Relative Risk	1.042 [1.01, 1.07]	1.134 [1.07, 1.2]
R-squared	0.831	0.802
Hausman test on PM _{2.5}		10.95
Day and Month Dummies	Yes	Yes
Observations	106	106

HAC robust standard errors in parentheses. Relative risks's 95% confidence intervals in brackets. *** p<0.01, ** p<0.05, * p<0.1.

The results presented in Table 11 show that a one-standard-deviation increase in $PM_{2.5}$ pollution increases total respiratory visits from 7.5 percent for adults (Panel D), to 13.4 percent for the elderly (Panel F). Furthermore, notice that the relative risk estimates for infants (Panel A) is smaller than the relative risk estimates of those slightly older, both toddlers (Panel B) as well as children (Panel C).⁶⁵ Yet, although the effect of $PM_{2.5}$ pollution on total respiratory visits varies across different age groups, these effects are not statistically different from each other.⁶⁶

iii. Contemporaneous Effect on Total Respiratory Visits by Type

In this section I analyze the effect of $PM_{2.5}$ pollution on the different type of respiratory visits according to the leading care professional's diagnosis, as shown in Table 7 above. Table 12 below presents both OLS and 2SLS estimates for: upper respiratory (columns 1 and 2), lower respiratory (columns 3 and 4), pneumonia (columns 5 and 6), respiratory related to influenza (columns 7 and 8), and other respiratory problems (columns 9 and 10). All while controlling for seasonal viral indexes, weather variables and time dummies.

⁶⁵ One reason for this result may be that parents of these very young and vulnerable individuals tend to keep them in very safe and protected environments, particularly during the winter time in Santiago, when it is cold and humid outside, thus avoiding exposing them to high levels of air pollution.

⁶⁶ Hausmann-type tests on relative risk estimates (using the Delta method) show that relative risks estimates are not statistically different across groups.

Table 12: Parameter and Relative Risk Estimates of the Effect of PM_{2.5} on Respiratory Urgent Care Visits, by Care Professional's Leading Diagnosis.

	Respiratory Visits by Care Professional's Leading Diagnosis									
	Upper Respiratory		Lower Respiratory		Pneumonia		Resp. Influenza		Resp. Other	
	OLS (1)	2SLS (2)	OLS (3)	2SLS (4)	OLS (5)	2SLS (6)	OLS (7)	2SLS (8)	OLS (9)	2SLS (10)
PM _{2.5}	5.609** (2.836)	12.405*** (4.627)	1.959* (1.076)	6.092*** (1.484)	0.250 (0.166)	1.207*** (0.335)	0.070 (0.095)	0.215** (0.104)	2.119*** (0.615)	4.105*** (0.708)
Relative Risk	1.035 [1, 1.07]	1.077 [1.02, 1.13]	1.033 [1, 1.07]	1.102 [1.05, 1.15]	1.023 [0.99, 1.05]	1.112 [1.05, 1.17]	1.019 [0.97, 1.07]	1.058 [1, 1.11]	1.049 [1.02, 1.08]	1.094 [1.06, 1.13]
R-squared	0.846	0.837	0.885	0.870	0.891	0.867	0.850	0.849	0.851	0.842
Hausman test on PM _{2.5}		3.454		16.34		10.76		12.13		31.79
Day and Month Dummies	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	106	106	106	106	106	106	106	106	106	106

HAC robust standard errors in parentheses. Relative risks's 95% confidence Intervals in brackets. *** p<0.01, ** p<0.05, * p<0.1.

Overall, the 2SLS parameter estimates turns statistically different from their OLS counterparts, and about two to four times larger than that of OLS.⁶⁷ The 2SLS estimates show that the effect of PM_{2.5} pollution varies across different types of respiratory visits. In particular, the relative risk estimates show that the effect of PM_{2.5} on lower, pneumonia and other respiratory visits (columns 4, 6 and 10 of Table 12, respectively) are larger than those results for total respiratory visits discussed earlier (presented in column 8 of Table 9 above). The estimates suggest that a one-standard-deviation increase in PM_{2.5} pollution increases lower, pneumonia and other respiratory visits by about 10 percent (relative risk estimates are 1.101, 1.112 and 1.094, respectively). This contrasts with the effect of the same increase in PM_{2.5} pollution on overall respiratory problems (8.4 percent, according to column 8 of Table 9 above)

On the other hand, although most respiratory visits, are due to upper respiratory diseases (57 percent of total, according to Table 7), the effect of PM_{2.5} pollution on respiratory visits due to upper respiratory problems is smaller than the overall effect presented in Table 9 above. Table 12 shows that a one-standard-deviation increase in PM_{2.5} pollution increases the relative risk of upper respiratory visits by 7.7 percent (relative risk estimate is 1.077).

iv. Contemporaneous Effect on Total Respiratory Visits by Age Group and Type

⁶⁷ The 2SLS estimate for type upper respiratory is statistically different from that of OLS at 90%.

Next, I look at the estimates across different age groups and types of respiratory visits. Table 13 below presents 2SLS parameter and relative risk estimates for the effect of $PM_{2.5}$ pollution on respiratory visits across different age groups (horizontal panels A through F) and across different types of respiratory diseases (columns 1 through 5). All regressions control for seasonal viral indexes, weather and time dummies.

Similar to what we saw in section 6.a.iii. and Table 12 above, the effect of $PM_{2.5}$ pollution on respiratory visits is largest among the elderly (Panel F). Particularly, for lower respiratory, respiratory due to pneumonia and upper respiratory visits (relative risks estimates of 1.15, 1.142 and 1.133, respectively). Recall from Table 7 above that most of those individuals that experience respiratory diseases associated with pneumonia are in the age group 65 and older (37.8 percent of those with respiratory problems due to pneumonia). Thereby, these results suggest that many individuals in this age group suffer from respiratory diseases due to pneumonia because of exposure to $PM_{2.5}$ pollution.

The effect of $PM_{2.5}$ pollution on lower respiratory diseases (columns 3 and 4) among children (Panel C) is also considerable large (relative risk estimate of 1.133). However, as can be seen from Table 7 above, very few individuals in this age group suffer from lower respiratory diseases (only 13.3 percent of total across all types of respiratory diseases for children). Therefore, although the effect of $PM_{2.5}$ pollution on

lower respiratory visits for children is relatively large, relatively few individuals in this age group suffer that type of respiratory disease.

On the other hand, the effect $PM_{2.5}$ pollution on upper respiratory visits (Columns 1 and 2) is relatively small among adults (Panel E), where the results report relative risk estimates of 1.066. As shown in Table 7 above most individuals fall into this age group (44.1 percent of all individuals) and that upper respiratory is the most prevalent respiratory disease (57.8 percent of total). Similarly, the effect $PM_{2.5}$ pollution is also relatively small among infants (Panel A), particularly for lower respiratory (relative risks estimate 1.071). Table 7 above also shows that, for infants, most urgent care respiratory visits are associated with lower respiratory diseases (43.8 percent of total across all types for this age group). Thereby, these results suggest that, for the most prevalent type of respiratory diseases among infants, the effect of $PM_{2.5}$ pollution is relatively small.

Table 13 : Parameter and Relative Risk Estimates of the Effect of PM_{2.5} on Respiratory Urgent Care Visits, by Age Group and Care Professional's Leading Diagnosis.

	Respiratory Visits by Age Group and Care Professional's Leading Diagnosis				
	Upper Respiratory	Lower Respiratory	Pneumonia	Resp. Influenza	Resp. Other
	2SLS (1)	2SLS (2)	2SLS (3)	2SLS (4)	2SLS (5)
Panel A: Less than 1 year old					
PM ₂₅	0.769*** (0.235)	0.723*** (0.267)	0.102 (0.078)	0.026*** (0.010)	0.246*** (0.054)
Relative Risk	1.082 [1.03, 1.13]	1.071 [1.02, 1.12]	1.087 [0.96, 1.22]	1.251 [1.07, 1.43]	1.101 [1.06, 1.14]
R-squared	0.683	0.869	0.691	0.316	0.720
Panel B: 1 to 4 years old					
PM ₂₅	2.942** (1.147)	1.765** (0.698)	0.263*** (0.066)	-0.023 (0.048)	0.786*** (0.202)
Relative Risk	1.084 [1.02, 1.15]	1.097 [1.02, 1.17]	1.122 [1.06, 1.18]	0.953 [0.76, 1.15]	1.103 [1.05, 1.16]
R-squared	0.805	0.840	0.856	0.623	0.805
Panel C: 5 to 14 years old					
PM ₂₅	2.861* (1.626)	0.891*** (0.245)	0.046 (0.037)	0.077 (0.054)	0.721** (0.316)
Relative Risk	1.083 [0.99, 1.18]	1.133 [1.06, 1.2]	1.083 [0.95, 1.22]	1.102 [0.96, 1.24]	1.089 [1.01, 1.17]
R-squared	0.823	0.835	0.688	0.714	0.815
Panel E: 15 to 64 years old					
PM ₂₅	5.050*** (1.893)	1.768*** (0.377)	0.218** (0.089)	0.138 (0.086)	2.089*** (0.355)
Relative Risk	1.066 [1.02, 1.11]	1.096 [1.06, 1.14]	1.078 [1.02, 1.14]	1.062 [0.99, 1.14]	1.092 [1.06, 1.12]
R-squared	0.857	0.870	0.808	0.869	0.832
Panel F: 65 and older					
PM ₂₅	0.783*** (0.177)	0.945*** (0.213)	0.579*** (0.190)	-0.005 (0.015)	0.263*** (0.086)
Relative Risk	1.133 [1.07, 1.19]	1.150 [1.08, 1.22]	1.142 [1.05, 1.23]	0.966 [0.76, 1.18]	1.097 [1.04, 1.16]
R-squared	0.733	0.795	0.774	0.619	0.698
Day and Month Dummies	Yes	Yes	Yes	Yes	Yes
Observations	106	106	106	106	106

HAC robust standard errors in parentheses. Relative risks's 95% confidence Intervals in brackets. *** p<0.01, ** p<0.05, * p<0.1.

v. Contemporaneous Effect on Cardiovascular and Circulatory
Visits

The data allows us to also examine effects on urgent care visits due to cardiovascular and circulatory disease during the period May 1st to August 15th, 2014 for Santiago. Table 1 above shows the mean number of visits due to cardiovascular and circulatory problems by age group. Almost all urgent care visits due to this cause are by adults (59.3 percent) and the elderly (39.6 percent). I use the same analytical framework of equation (1) above to obtain parameter estimates $\hat{\beta}$ and relative risk estimates of the effect of PM_{2.5} pollution on cardiovascular and circulatory visits. However, on days of WC games individuals may experience cardiovascular and/or circulatory problems due to causes other than air pollution, such as problems due to fatigue because of excess eating at barbecues and/or stress associated with watching the games. This would invalidate the use of WC games as instruments since they will fail to drive cardiovascular and circulatory visits *only* via the effect of WC games on PM_{2.5} pollution. Therefore, for examining the effect of PM_{2.5} pollution on cardiovascular and respiratory visits I only use thermal inversions as instruments in the 2SLS estimations. Table 14 below presents results for the aggregate across all age groups (Panel A) as well as for adults (Panel B), and for the elderly (Panel C).⁶⁸ Panel A of Table 14 shows that, for all age groups, a one-standard-deviation increase in PM_{2.5} pollution increases cardiovascular and circulatory visits by 4.4 percent (relative risk estimate in column 4 is 1.044). Panel B shows that, although weakly significant, such

⁶⁸ For brevity, I omit results for the other age groups. Estimates for those age groups turn statistically non-significant.

an increase in $PM_{2.5}$ pollution increases cardiovascular and circulatory visits for adults by 3.4 percent (relative risk estimate in column 4 is 1.032). I find the largest effect on cardiovascular and circulatory visits among the elderly. Panel C shows that a one-standard-deviation increase in $PM_{2.5}$ increases cardiovascular and circulatory visits by 6.3 percent for this age group (relative risk estimate in column 4 is 1.063). Given that those individuals in older cohorts are more susceptible of suffering from cardiovascular and circulatory problems, we may expect that the deterioration of air quality may trigger larger adverse effects on their health condition. In fact, using the Delta method I find that the relative risk estimate for the elderly (1.063 in column 2 of Panel C) is statistically larger than the relative risk estimate for adults (1.032 in column 2 of panel B).

Table 14: Parameter and Relative Risk Estimates of the Effect of PM_{2.5} Pollution on Cardiovascular and Circulatory Urgent Care Visits, by Age Groups.

	Cardiovascular & Circulatory Visits, by Age Group	
	OLS (1)	2SLS (2)
Panel A: All age groups		
PM _{2.5}	0.280** (0.126)	0.657*** (0.220)
Relative Risk	1.019 [1, 1.04]	1.044 [1.02, 1.07]
R-squared	0.439	0.420
Hausman test on PM _{2.5}		4.355
Panel B: 15 to 64 years old		
PM _{2.5}	0.076 (0.078)	0.300* (0.165)
Relative Risk	1.009 [0.99, 1.03]	1.034 [1, 1.07]
R-squared	0.386	0.371
Hausman test on PM _{2.5}		2.362
Panel C: 65 and older		
PM _{2.5}	0.230*** (0.077)	0.372*** (0.104)
Relative Risk	1.039 [1.01, 1.06]	1.063 [1.03, 1.1]
R-squared	0.485	0.470
Hausman test on PM _{2.5}		4.176
Day and Month Dummies	Yes	Yes
Observations	106	106

HAC robust standard errors in parentheses. Relative risks's 95% confidence Intervals in brackets. *** p<0.01, ** p<0.05, *

vi. Contemporaneous Effect on Urgent Care Visits Due to
External Causes

The data also allows us to look at the effect of PM_{2.5} pollution on urgent care visits due to external causes (other than respiratory or cardiovascular and circulatory). These refer to urgent care visits due to traffic accidents and minor accidents such as cuts and contusions. The results presented in Table 15 below show that exposure to PM_{2.5} pollution has no significant effect on urgent care visits due to external causes. This ‘falsification test’ suggests that I am correctly identifying the pathway in which PM_{2.5} pollution affects urgent care visits, which is, either via respiratory visits or via cardiovascular and circulatory visits.

Furthermore, these results provide indirect evidence of the absence of avoidance behavior of the individuals in the sample from Santiago. If individuals were to adjust their behavior to reduce exposure on days of high concentrations of PM_{2.5} pollution, then they would restrict their outdoor activities and will stay indoors, which would be reflected on a negative relationship between PM_{2.5} pollution and urgent care visits due to external causes. The results suggest that there is no evidence of avoidance behavior.

Table 15: Parameter and Relative Risk Estimates of the Effect of PM_{2.5} Pollution on Urgent Care Visits Due to External Causes, All Age Groups.

Ugent Care Visits Due to External Causes, by Age Group				
	OLS	2SLS	OLS	2SLS
	(1)	(2)	(3)	(4)
Panel A: All age groups				
PM _{2.5}	0.212 (1.209)	3.560 (2.365)	1.047 (1.092)	1.786 (1.458)
Relative Risk	1.003 [0.97, 1.04]	1.050 [0.99, 1.12]	1.015 [0.99, 1.05]	1.025 [0.99, 1.07]
R-squared	0.487	0.450	0.744	0.742
Hausman test on PM _{2.5}		2.711		0.585
Day and Month Dummies	No	No	Yes	Yes
Observations	106	106	106	106

HAC robust standard errors in parentheses. Relative risks's 95% confidence Intervals in brackets. *** p<0.01, ** p<0.05, * p<0.1.

b. Effect of Cumulative Exposure to PM_{2.5} Pollution

i. Cumulative Effect on Total Respiratory Visits

So far, I have focused only on the acute effects of contemporaneous exposure to PM_{2.5} pollution. However, it is likely that most respiratory and cardiovascular diseases that result in these urgent care visits are not a consequence of such an immediate response to same-day pollution exposure. Exposure to high levels of PM_{2.5} pollution for an extended period of time (longer than one day) may result in a steady worsening of an individual's health, to a point at which this individual (or his/her guardian) eventually decides to go to an urgent care facility. Most of the existing literature has not looked into the varying effects of different periods of exposure to air

pollution. In this section I examine the effects over different periods of exposure to shed some light on what should be the most relevant period of exposure when estimating the effects PM_{2.5} pollution on respiratory and cardiovascular diseases. That is, in this section I examine the effects of exposure to PM_{2.5} pollution over different periods of time, from a 1-day (contemporaneous) exposure to 14-day (cumulative) exposure.

In figures 13 through 17 below I present results in terms of relative risk estimates and 95% confidence intervals only from 2SLS estimations with day-of-week and month controls. In Figure 13 I plot the relative risks estimates (vertical axes) and periods of exposure to PM_{2.5} pollution (horizontal axis). I also present detailed results in terms of parameter estimates, standard errors and R-squared statistics in a accompanying table under Figure 13. The relative risk estimate is the change in the dependent variable, with respect to its mean value, for a change in PM_{2.5} equal to one standard deviation. Thereby, for purposes of computing the relative risk estimate of the cumulative effect of PM_{2.5} pollution, I calculate the standard deviation for each period of cumulative exposure to PM_{2.5} pollution. The row “SD of Cumm. PM_{2.5}”, in the accompanying table (right below Figure 13), presents this standard deviation over $j + 1$ days ($j = 0, \dots, 13$) of cumulative exposure to PM_{2.5} pollution.

Whereas Figure 13 presents relative risk estimates for overall respiratory visits, Figure 14 presents results by age group and Figure 15 presents results for type of respiratory visit. Figure 16 presents results by both age group and type of respiratory

visit. Furthermore, Figure 17 presents relative risk estimates for cardiovascular and circulatory visits. Throughout, to calculate these relative risk estimates I use the standard deviation of cumulative exposure to PM_{2.5} pollution presented in row “SD of Cumm. PM_{2.5}” of the accompanying table of Figure 13.

Figure 13 below shows that the effect of PM_{2.5} pollution on overall respiratory visits varies depending on period of exposure. The effect of same-day as well as 2-day average exposure turns statistically significant (weakly significant for 2-day exposure). Whereas the effect of exposure over 3 to 9 days turns non-significant, the effect of exposure over 10 to 13 days turns statistically significant (although not statistically different from that of 1-day exposure). These results may reflect both an acute effect on respiratory visits due to exposure to short lasting peaks in PM_{2.5} pollution concentrations as well as an effect on health of exposure to PM_{2.5} pollution over a longer period time. Short-lasting changes to PM_{2.5} pollution concentrations, such as those sharp peaks during WC games, may have a strong adverse acute effect on respiratory visits (same-day as well as 2-day average exposure). However, once those short-lasting peaks in PM_{2.5} pollution concentrations die out, the effect of exposure to PM_{2.5} pollution vanishes out. However, the cumulative effect of exposure to PM_{2.5} pollution over a longer period of time (over 10 to 13 days) remains significant.

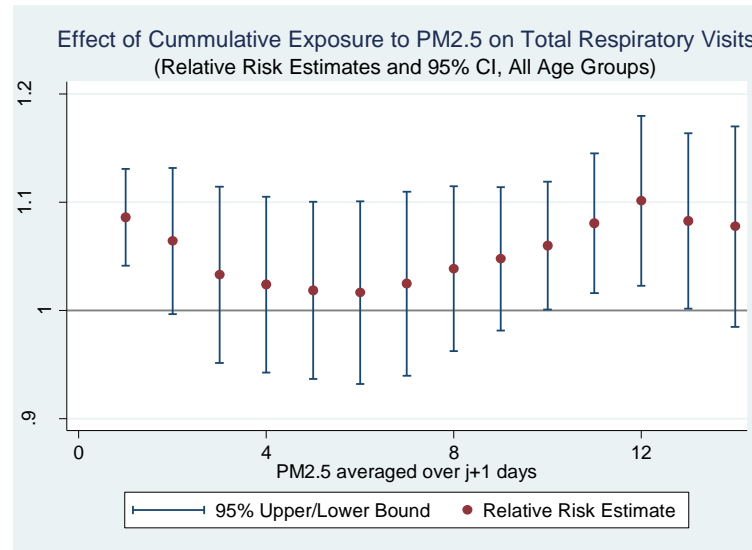


Figure 13: 2SLS Relative Risk Estimates (and 95% confidence intervals) of the effect of cumulative exposure to PM_{2.5} on Total Respiratory Urgent Care Visits, All Age Groups.

Effect of Cumulative Exposure to PM _{2.5} on Total Respiratory Visits, All Age Groups														
	1 day (j = 0)	2 days (j = 1)	3 days (j = 2)	4 days (j = 3)	5 days (j = 4)	6 days (j = 5)	7 days (j = 6)	8 days (j = 7)	9 days (j = 8)	10 days (j = 9)	11 days (j = 10)	12 days (j = 11)	13 days (j = 12)	14 days (j = 13)
Cumulative PM _{2.5}	24.02*** (6.38)	19.87* (10.68)	11.25 (14.32)	9.01 (15.72)	7.66 (17.21)	7.19 (19.04)	11.65 (20.48)	19.15 (19.39)	24.86 (17.66)	32.64** (16.41)	45.71** (18.7)	59.5** (23.54)	50.02** (25)	47.84 (29.18)
Relative Risk	1.086 [1.04, 1.13]	1.064 [1, 1.13]	1.033 [0.95, 1.11]	1.024 [0.94, 1.11]	1.019 [0.94, 1.1]	1.016 [0.93, 1.1]	1.025 [0.94, 1.11]	1.038 [0.96, 1.11]	1.048 [0.98, 1.11]	1.06 [1, 1.12]	1.081 [1.02, 1.15]	1.101 [1.02, 1.18]	1.083 [1, 1.16]	1.077 [0.98, 1.17]
SD of Cumm. PM _{2.5}	17.93	16.17	14.56	13.22	12.15	11.32	10.62	10.06	9.6	9.19	8.83	8.52	8.28	8.11
R-squared	0.859	0.861	0.864	0.865	0.866	0.866	0.866	0.867	0.869	0.871	0.872	0.874	0.871	0.869
Observations	106	106	106	106	106	106	106	106	106	106	106	106	106	106

HAC robust standard errors in parentheses. Relative risks's 95% confidence Intervals in brackets. *** p<0.01, ** p<0.05, * p<0.1.

ii. Cumulative Effect on Total Respiratory Visits by Age Group

Similar to section 6.a.ii. above, I also examine the cumulative effect of PM_{2.5} pollution exposure on respiratory visits across different age groups. Figure 14 below shows results, relative risks estimates and 95% confidence intervals across different periods of exposure in one quadrant for each age group. As I showed in Table 5 above, Figure 14 shows that the effect on respiratory visits of acute same-day exposure to PM_{2.5} pollution turns statistically significant across all age groups. However, for those very young and very old individuals, I do not find statistically significant effect of PM_{2.5} pollution over more than 1 or 2-day exposure. These findings suggest that these vulnerable individuals are negatively affected mostly by acute episodes of PM_{2.5} pollution (such as sharp increases in PM_{2.5} pollution on days of WC games), but they seem less affected to exposure over a relatively longer period of time, over more than two consecutive days.

The results presented in Figure 14 below, however, show a different pattern for children. For these individuals, these results suggest that there is a significant effect of PM_{2.5} pollution over consecutive days of exposure. Furthermore, the adverse effect on respiratory visits seems to increase over longer periods of exposure. For example, for these individuals, the relative risk estimate for the effect exposure to 12-day average PM_{2.5} pollution on respiratory visits is 1.244. This means that a one-standard-deviation in 12-day average PM_{2.5} pollution increases respiratory visits by 24.4 percent. This figure is more than twice as large as the effect of acute same-day

exposure to $PM_{2.5}$ pollution for this same group of individuals (8 percent, as reported in Table 11 above). Since these individuals are of an age that they should be attending school (Pre-K to middle school), it might be that they spend relatively more time outdoors (probably playing and doing sports) than other individuals of different age. These outdoor activities may make them more susceptible to suffering from exposure to air pollutants over a longer period of time.

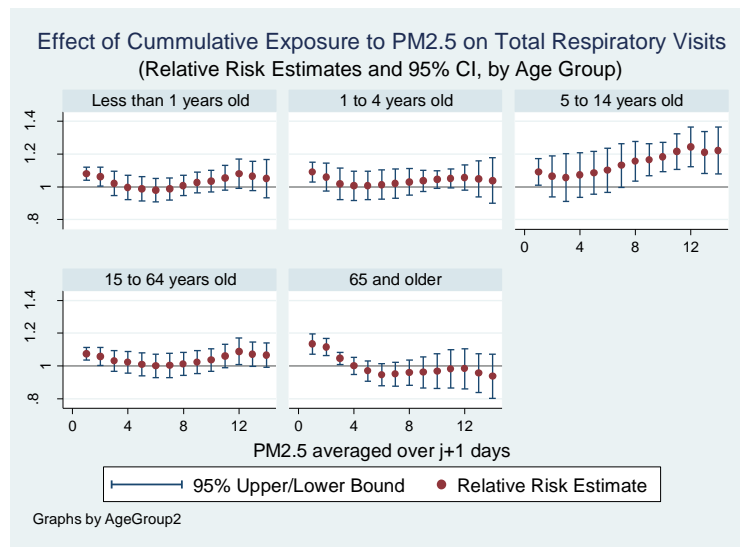


Figure 14: 2SLS Relative Risk Estimates (and 95% confidence intervals) of the effect of cumulative exposure to $PM_{2.5}$ on Total Respiratory Urgent Care Visits, by Age Group.

iii. Cumulative Effect on Total Respiratory Visits by Type

In this section I examine the effect of cumulative exposure to $PM_{2.5}$ pollution on respiratory urgent care visits by type of respiratory disease. Figure 15 below shows that whereas same-day exposure to $PM_{2.5}$ pollution has statistically significant effect across all types of respiratory visits, 2 and 3-day exposure have significant effects

only on lower, pneumonia and other respiratory visits. On the other hand, exposure to 12-day average $PM_{2.5}$ pollution has a significant effect on all respiratory visits except from those associated with pneumonia. For most types of respiratory visits the effect of exposure to $PM_{2.5}$ pollution over 2 to 14 days seems not to be different to the effect of same-day exposure (with the exception of respiratory visits due to influenza).

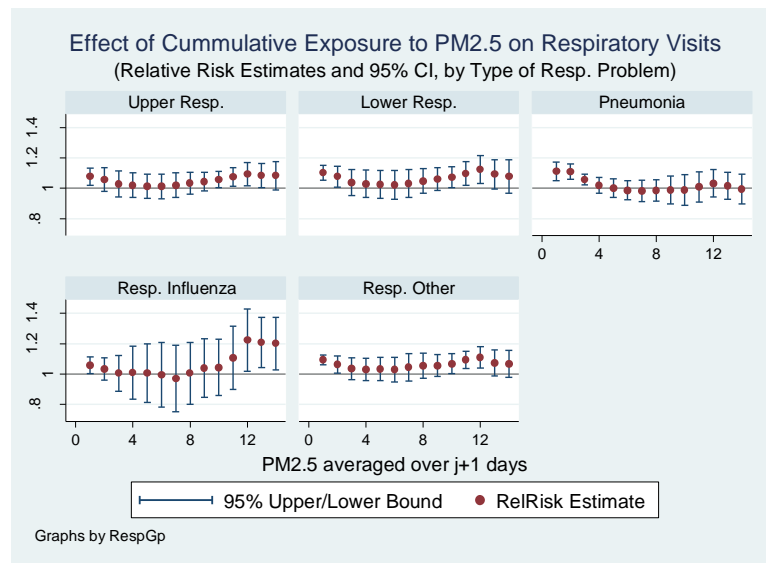


Figure 15: 2SLS Relative Risk Estimates (and 95% confidence intervals) of the effect of cumulative exposure to $PM_{2.5}$ on Total Respiratory Urgent Care Visits, by Type of Respiratory Problem.

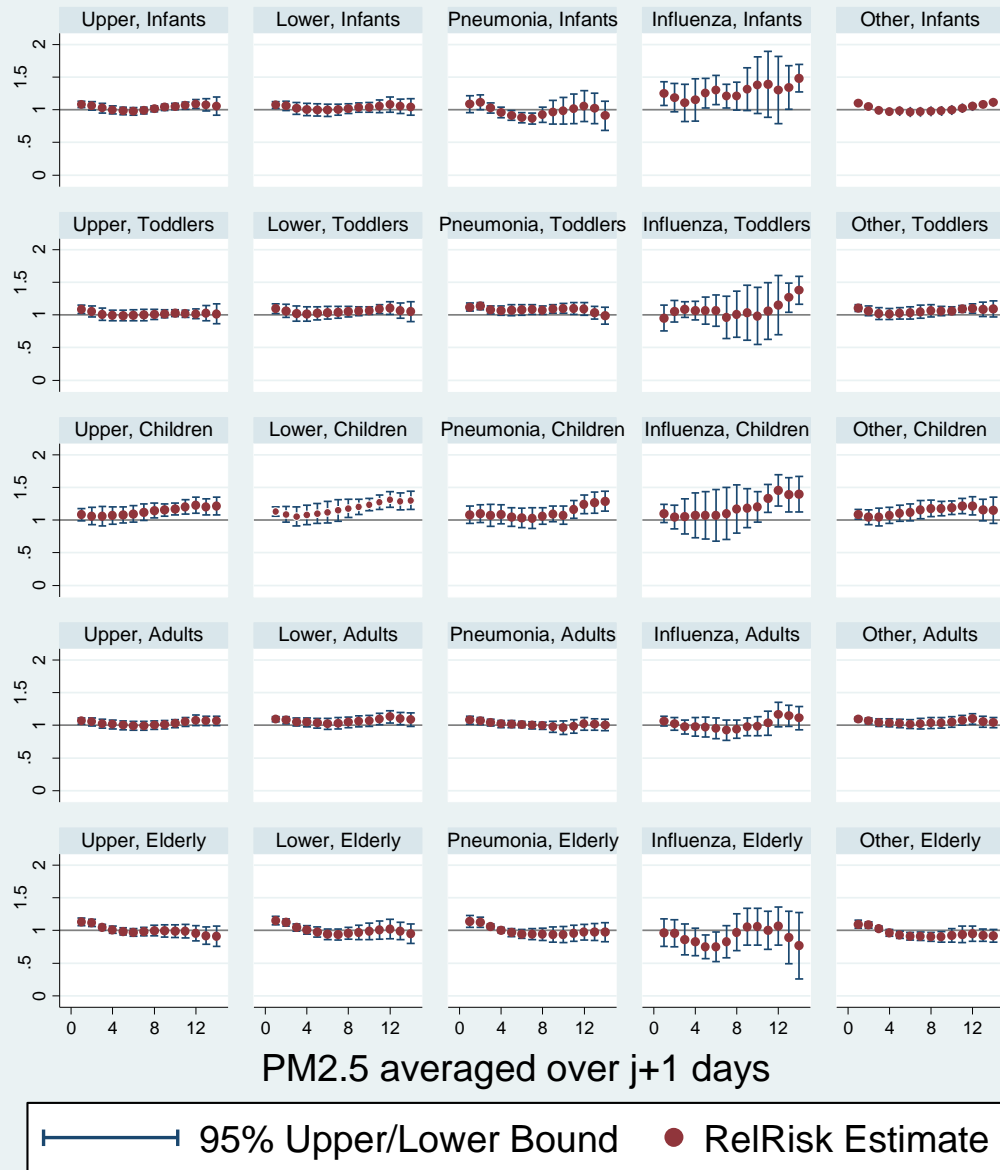
iv. Cumulative Effect on Total Respiratory Visits by Age Group and Type

In this section I examine the effect of cumulative exposure to $PM_{2.5}$ pollution on respiratory visit by age group and type of respiratory visit. Figure 16 below presents results for the effect of cumulative exposure to $PM_{2.5}$ pollution across both age groups

(row plots) and type of respiratory visits (column plots). The results basically reflect what was shown in figures 6 and 7 above. That is, the effect of cumulative exposure to $PM_{2.5}$ pollution is largest for children, particularly for lower respiratory visits. Figure 12 shows that the relative risk estimate of 12-day average exposure on lower respiratory visits for this age group is 1.317, more than twice as large as the relative risk estimate of same-day exposure, 1.133, reported in Table 13 above. Furthermore, the large effect of 12-day average exposure to $PM_{2.5}$ pollution for influenza visits for children seems to drive the large effect of respiratory visits due to influenza across all age groups shown in Figure 15 above. In fact, the effect of cumulative exposure to $PM_{2.5}$ pollution on respiratory visits due to influenza is only significant for children, and it turns non-significant for all other age groups.

Effect of Cumulative Exposure to PM_{2.5} on Respiratory Visits

(Relative Risk Estimates and 95% CI, by Type of Resp. Visit and Age Group)



Graphs by RespGp and AgeGroup2

Figure 16: 2SLS Relative Risk Estimates (and 95% confidence intervals) of the effect of cumulative exposure to PM_{2.5} on Respiratory Urgent Care Visits, by Type of Respiratory Visit and Age Group.

v. Cumulative Effect on Cardiovascular and Circulatory Visits

Similar to section 6.a.v., in this section I look at the effects of cumulative exposure to $PM_{2.5}$ on cardiovascular and circulatory visits. Figure 17 below presents relative risk estimates and 95% confidence intervals for the effect of cumulative exposure to $PM_{2.5}$ on cardiovascular and circulatory visits for all age groups. Whereas the relative risk of one-day exposure is 1.047, the relative risk for 2 and 3-day exposure turns larger, at 1.064 and also significant. This means that a one-standard-deviation increase in 2 or 3-day average $PM_{2.5}$ pollution increases cardiovascular and circulatory urgent care visits by 6.4 percent. On the other hand, the relative risk estimate for the elderly turns statistically significant for 1 to 14-day exposure to $PM_{2.5}$ pollution (not shown in Figure 16), and the relative risk estimate for a 2 to 3-day exposure for the elderly turns 1.08 and significant (not shown in Figure 16).

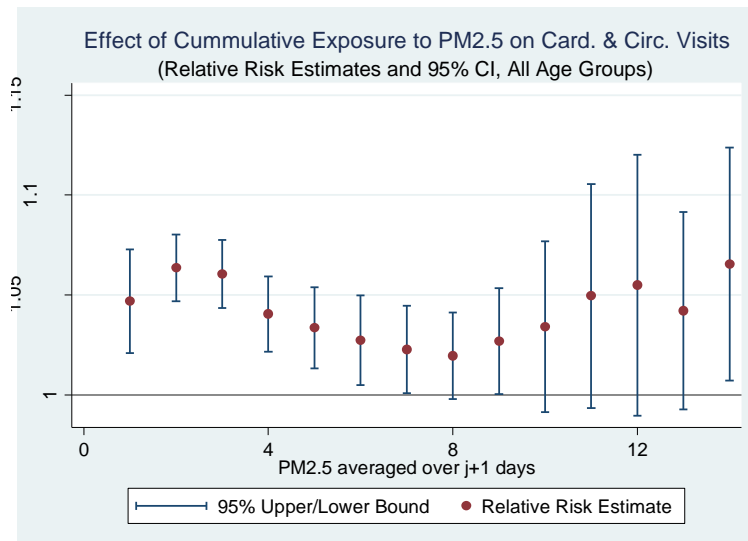


Figure 17: 2SLS Relative Risk Estimates (and 95% confidence intervals) of the effect of cumulative exposure to $PM_{2.5}$ on Cardiovascular and Circulatory Urgent Care Visits, All Age Groups.

7. *Robustness Check: Adding Respiratory Visits in Valparaiso-Viña Metropolitan Area as Control Variable*

To check the robustness of the 2SLS estimates I introduce as an explanatory variable the correspondent outcome variable for Chile's second largest metropolitan area: the metropolitan area that includes the cities of Valparaiso, Viña del Mar, Quilpué, y Villa Alemana (I refer to it as Valparaiso-Viña, for short). The Valparaiso-Viña metropolitan area has a very similar climate, and is about 120 kilometers (75 miles) west of Santiago. Furthermore, there is high connectivity between these two metropolitan areas to the extent that many people even commute every day to work in Santiago while living in Valparaiso-Viña (or vice versa). The main difference between these two large metropolitan areas is that, unlike Santiago, Valparaiso-Viña is not enclosed by mountains, and as a consequence, air pollutants do not accumulate as dramatically as they do in Santiago.⁶⁹ Instead, Valparaiso-Viña is located off the coast of Chile and has geographical conditions that facilitate the ventilation of air pollutants. Chile's Coastal Mountain Range (Cordillera de la Costa) separates Santiago from Valparaiso-Viña metropolitan area.

Thereby, the variable for respiratory visits in Valparaiso-Viña may capture unobserved confounding effects that are not accounted for in the regressions and results presented earlier (such as Table 9 above). Table 16 below presents results from OLS (columns 1 and 3) and 2SLS (columns 2 and 4) for respiratory visits across

⁶⁹ Similarly, the concentrations of air pollutants in Valpo-Viña are not affected by thermal inversions.

all age groups, adding the outcome variable in Valparaiso-Viña as a control. Table 16 presents both estimates for β , R-squared statistics and Hausman test statistics comparing to the corresponding β estimates in Table 9 above. Furthermore, I present results with and without day-of week and month dummies (columns 1 and 2 and columns 3 and 4, respectively). The results in Table 16 show that, when adding day-of-week and month dummies (column 4), the 2SLS estimate is not statistically different from that in Table 9 above (Hausman test statistic on $PM_{2.5}$ smaller than 3.86). I conclude that, when accounting for time-specific dummies, further controlling for respiratory visits in Valparaiso-Viña does not introduce new information that significantly changes the results of the model in Table 9. This suggests that I am correctly identifying the effect of $PM_{2.5}$ pollution on respiratory visits and that the β estimates are robust to possible effects captured by respiratory visits in Valparaiso-Viña metropolitan area.

Table 16: Parameter Estimates of the Effect of $PM_{2.5}$ on Total Respiratory Urgent Care Visits, for All Age Groups. Controlling for Outcome Variable in Valparaiso-Viña Metropolitan Area.

	Total Respiratory Visits (with Region V), All Age Groups			
	OLS (1)	2SLS (2)	OLS (3)	2SLS (4)
$PM_{2.5}$	10.778* (5.520)	26.802*** (8.430)	4.186 (2.768)	17.999*** (3.642)
R-squared	0.837	0.821	0.925	0.915
Hausman on $PM_{2.5}$ (w.r.t. Table 3)	1.98	4.44	2.76	1.09
Day and Month Dummies	No	No	Yes	Yes
Observations	106	106	106	106

HAC robust standard errors in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

8. Discussion

On October 2016 Chile's Congress passed new legislation to regulate air pollution emissions in Santiago's Metropolitan Area. The legislation is part of the Air Pollution Control and Prevention Plans ("*Plan De Prevención y Decontaminación Ambiental, PPDA*", in Spanish) that have been articulated by Chile's Ministry of Environment to protect the environment under Chile's current legislation. This latest PPDA, aims to reduce annual PM_{2.5} pollution concentrations by 12 $\mu\text{g}/\text{m}^3$, and thus bring Santiago's PM_{2.5} concentration below Chile's standard for annual PM_{2.5} pollution concentrations.⁷⁰

The Ministry of Environment's report for Santiago's PPDA ("*General Analysis of Economic and Social Impact of Santiago's PPDA*", henceforth AGIES, according to the acronym in Spanish) conducts a cost-benefit analysis in which it estimates that the new PPDA for Santiago will have a net social benefit of USD 6,965 million over the course of the next 10 years, with a cost-to-benefit ratio of 1:6. Key to calculate these benefits are the expected reductions in PM_{2.5} concentrations and its corresponding health impacts on Santiago's population. The AGIES estimates a constant reduction of PM_{2.5} concentrations over the next ten years that is expected to translate into 241,404 fewer respiratory urgent care visits over the next 10 years. To arrive to this figure, the AGIES uses a concentration-response estimate from Norris et al. (1999) of

⁷⁰ Chile's standards for PM_{2.5} concentration are 50 $\mu\text{g}/\text{m}^3$ for the daily average and 20 $\mu\text{g}/\text{m}^3$ for the annual average.

16.5 percent fewer respiratory visits, for children less than 18 years old, to a 10- $\mu\text{g}/\text{m}^3$ change in $\text{PM}_{2.5}$ pollution concentrations (Norris et al. 1999).

The estimate presented in Table 3 above suggests that a 10- $\mu\text{g}/\text{m}^3$ change in $\text{PM}_{2.5}$ pollution concentrations would yield a 4.8 percent reduction in respiratory visits for the total population. Furthermore, the estimates presented in Table 5 above suggest that a 10- $\mu\text{g}/\text{m}^3$ change in $\text{PM}_{2.5}$ pollution concentrations would yield 4.5 percent reduction in respiratory visits for infants and a 5.1 percent reduction for toddlers and children. The concentration-response estimates in this chapter are less than a third as large as those used to calculate the cost-benefit analysis in the AGIES report for Santiago's PPDA (estimates from Norris et al., 1999, for children only). However, Norris et al. (1999)'s estimates used in Santiago's AGIES report are closer to the estimates of the cumulative exposure to $\text{PM}_{2.5}$ pollution for children (the largest estimates). For 12-day exposure to $\text{PM}_{2.5}$ pollution I estimate that a 10- $\mu\text{g}/\text{m}^3$ reduction in $\text{PM}_{2.5}$ pollution will reduce respiratory visits by 13.6 percent for children.

On the other hand, for a 10- $\mu\text{g}/\text{m}^3$ change in $\text{PM}_{2.5}$ pollution concentrations Ilabaca et al. (1999) estimates a concentration-response of 0.6 percent for respiratory-related emergency visits and 1.48 percent for visits for pneumonia for children under 15 years old in Santiago.⁷¹ The concentration-response estimates for those individuals

⁷¹ Similarly, for a 10 $\mu\text{g}/\text{m}^3$ change in PM_{10} pollution concentrations, Ostro et al. (1999) estimates for lower respiratory visits a 0.5 percent change for children under 2 years old and a 0.74 percent change for children 3 to 15 years old. Jans, Johansson and Nilsson (2014) estimate that respiratory hospital admissions in Sweden (both in-patients as well out-patients) increase by 7.9 percent by each 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} pollution. This effect is larger for admissions due to asthma (10.7 percent),

under 15 years old presented in this chapter are about eight times larger than Ilabaca et al. (1999)'s estimates for respiratory-related emergency visits and about four times larger for visits due to pneumonia. Moreover, in a meta-analysis of 26 previous studies for children, Lim et al. (2016) estimate a concentration-response of 2.7 percent fewer respiratory visits for children, for a $10\text{-}\mu\text{g}/\text{m}^3$ change in $\text{PM}_{2.5}$ pollution concentrations. Similarly, in a meta-analysis of 87 previous studies for asthma-related respiratory visits among the total population, Zheng et al. (2015) estimate a concentration-response of 1.7 percent fewer asthma-related respiratory visits for total population, for a $10\text{-}\mu\text{g}/\text{m}^3$ change in $\text{PM}_{2.5}$ pollution concentrations. As compared to these estimates, the concentration-response estimates reported in this chapter are more than twice as large as those by Zheng et al. (2015) and those reported by Lim et al. (2016).

9. Concluding Remarks

In this chapter I look at the effect of exposure to fine particulate matter ($\text{PM}_{2.5}$) on respiratory and cardiovascular and circulatory visits in Santiago, Chile. I look at effects for all visits as well as by age group and by type of respiratory visits, and estimate effects for both same-day exposure to $\text{PM}_{2.5}$ pollution as well as exposure over consecutive days.

bronchitis (10.1 percent) and pneumonia (9.4 percent) and for individuals 6 to 18 years old (10.4 percent).

To identify a causal relationship and account for possible confounding effects I use exogenous variation on concentrations of PM_{2.5} pollution from thermal inversions and sharp increases in pollution emissions due to massive grill outs on occasion of FIFA 2014 World Cup games. I find no evidence of avoidance behavior that may act as a confounding factor and, furthermore, I check that the estimates are robust to the presence of other possible confounders that should also be present in a nearby large metropolitan area (Valparaiso-Viña del Mar Metropolitan Area). Therefore, I believe that I have identified causal estimates of the effect of PM_{2.5} pollution on respiratory and cardiovascular and circulatory visits.

I find that a one-standard-deviation increase in PM_{2.5} pollution increases daily respiratory visits by 8.6 percent, and daily cardiovascular and circulatory visits by 4.7 percent. I estimate a larger effect for lower respiratory visits (10.2 percent), visits for pneumonia (11.2 percent) and for the elderly (13.4 percent for respiratory and 6.3 percent for cardiovascular and circulatory visits). Furthermore, I find larger effects for exposure over consecutive days. I estimate that a one-standard-deviation increase in 12-day exposure to PM_{2.5} pollution increases respiratory visits by 10.1 percent (by 24.4 percent for children). Similarly, I estimate that a one-standard-deviation increase in 2-day exposure to PM_{2.5} pollution increases cardiovascular and circulatory visits by 4.4 percent (by 6.3 percent for the elderly).

Chapter 3: Adverse Effects of Air Pollution on the Probability of Stillbirth Delivery: Evidence from Central Chile

1. Introduction

There is a large literature documenting the effects of exposure to airborne air pollutants on human health across several cities and regions in both developing as well as the industrialized world (Nadadur and Hollingsworth 2015; Šrám et al. 2005). Much of the literature focuses on the adverse effects on particularly vulnerable subpopulations, namely infants and the elderly. In this chapter, we examine the adverse effects of airborne air pollution on a fundamental pregnancy outcome; that is, whether a pregnancy results in a livebirth delivery, or whether a fetal death occurs so that the pregnancy ends in a stillbirth delivery. Although there is a growing literature documenting the adverse effects of air pollution on several pregnancy outcomes — such as low birth weight and preterm birth—there is little evidence on the adverse effects of air pollution on the probability of pregnancy ending in a stillbirth delivery (Nadadur and Hollingsworth 2015). In fact, the empirical literature is, so far, inconclusive on whether air pollution has any significant effect on fetal deaths and stillbirth deliveries (Veras et al. 2015).

In this chapter, we look at the effects of two airborne pollutants, particulate matter of diameter less than 10 microns (PM_{10}) and Carbon Monoxide (CO) on the probability of pregnancy resulting in a stillbirth delivery. We examine the effects of these two pollutants throughout two distinctive periods of exposure: *chronic* exposure, that is, exposure throughout the entire length of pregnancy, and *acute* exposure, exposure on the week of delivery (either a livebirth or stillbirth).

On the other hand, as air pollution diminishes the capacity of the pregnant woman to transmit nutrients and oxygen to the fetus, a severe inhibition of this capacity may result in a fetal death due to the lack of oxygen in the fetus. Therefore, we also examine the effects of these two pollutants and these two periods of exposure on the most likely consequences of air pollution, a stillbirth due to hypoxia.

We find evidence of adverse effects of *acute* exposure to both PM_{10} and CO pollution on the probability of stillbirth. Furthermore, we find larger effects for the probability of stillbirth due to hypoxia. Conversely, we find only weak evidence of *chronic* exposure to these air pollutants.

The next section briefly reviews the existing literature. We present the data we employed in section 3 and in section 4 we explain the statistical methods we use for econometric analysis. Section 5 presents econometric results and in section 6 we contrast these results with those of the existing related literature. We discuss on the

policy implications of our results in section 7 and present concluding remarks in section 8.

2. Literature Review

There is a growing body of literature that looks at the effects of air pollution on births' health outcomes. For instance, Edwards et al. (2015) reviews 139 articles and extracts extensive information about the effects of exposure to criteria pollutants (carbon monoxide, particulate matter, nitrogen dioxide, sulfur dioxide, ozone) polycyclic aromatic hydrocarbons and traffic-related air pollutants. Edwards et al. (2015)'s review examines six pregnancy outcomes: gestational-age, preterm birth, small for gestational-age, full term low birth-weight and both continuous and threshold low birth-weight. However, few studies look at the association between maternal exposure to air pollutants and fetal death or stillbirth delivery. Moreover, most of these studies examine the effects of exposure to air pollution during specific periods of the pregnancy (the days immediately before delivery, specific months and trimesters of pregnancy) as well as exposure during the entire length of pregnancy. The table in Appendix II summarizes the existing related literature and its main findings.

The existing literature that looks at the effects of exposure to air pollution on the probability of stillbirth finds (i) significant effects of *acute* exposures (just a few days immediately before delivery) to *peak* levels of carbon monoxide pollution (Faiz et al.

2013)⁷²; or, (ii) only weak evidence of exposure to air pollution when measured in levels, significant only at 90% confidence (Pereira et al. 1998)⁷³. Regarding exposure to particular matter, both PM₁₀ and PM_{2.5} (also known as fine particulate matter), the literature finds significant effects of different windows of exposure (third trimester, first two months, and entire length of pregnancy) and at different intensities. Whereas Kim et al. (2007) find statistical evidence on the probability of stillbirth from exposure to PM₁₀ during the third trimester of pregnancy in Seoul, Korea; Hwang et al. (2011) find evidence of exposure during the first two months of pregnancy in Taiwan. Furthermore, De Franco et al. (2015) find evidence of exposure to *peak* levels of PM_{2.5} during the third trimester of pregnancy in the State of Ohio; and Green et al. (2015) find only weak evidence of exposure to levels of PM_{2.5} for the entire pregnancy in the State of California.

However, because of to the focus on trimester-specific exposure, the existing literature misses those stillbirth deliveries that could have been due to exposure to air pollution but did not lasted through the third trimester of pregnancy. This misses those pregnancies that may have already resulted in a stillbirth but that did not last passed the second trimester of pregnancy.⁷⁴ If those early stillbirth deliveries are due to exposure to air pollution, then this selection of observations introduces a (selection) bias. This bias would lead us to accept the null hypothesis of no

⁷² *Peak* levels of pollution refers to pollution events in which pollution is higher than the sum of the mean pollution and inter-quantile range ($> \text{mean} + \text{IQR}$). If pollution has a normal distribution, this would be equivalent to episodes of pollution in the top 2.5% percent of its distribution.

⁷³ Notice that Pereira et al. (1998) is an *ecological study*. Most ecological studies have only one city-wide measure of pollution exposure and do not include mother-specific controls, such as mother's medical history, demographics and socioeconomic characteristics.

⁷⁴ In fact, as I will show below, about half of the stillbirth deliveries in our sample to not last passed the second trimester of pregnancy (passed the twenty sixth week of pregnancy).

statistically significant effect of air pollution on stillbirth deliveries. Same is true for those studies that focus on month-specific exposure through the eighth and ninth month of pregnancy.

Moreover, the existing literature does not find conclusive evidence of an effect of exposure to CO pollution on stillbirth deliveries (other than at *peak* levels), nor does it find evidence of acute exposure to PM₁₀ pollution on stillbirth deliveries. In this paper we examine the effects on the probability of stillbirth of both *acute* and *chronic* exposure to PM₁₀ and CO pollution. Moreover, the existing literature fails to control for potential confounders such as those given by seasonal patterns of conception and delivery, and, most importantly, for possible location-specific unobservable effects (for example, municipality-specific confounders that may introduce bias to the estimation of the effect of exposure to air pollution). In this paper we address all these issues and, in doing so, we do not introduce a selection bias due to missing observations on pregnancies that resulted in stillbirth before reaching the third trimester of pregnancy.⁷⁵ Furthermore, we control for potential seasonal and location-specific confounders.

Moreover, in this paper we also look at cause-specific stillbirths by focusing on those stillbirths that are most likely to be driven by exposure to air pollutants; stillbirths due to hypoxia.⁷⁶ Thus, in this paper we also we provide supporting evidence of the more

⁷⁵ We refer to acute exposure as exposure to air pollution on the week of delivery, and to chronic exposure as to average exposure throughout the entire duration of the pregnancy.

⁷⁶ Hwang et al. (2011) and Green et al. (2015) suggest that one possible pathway linking air pollution exposure and stillbirth would be via the oxygen uptake by the fetus. They suggest that pollutants may

likely pathway in which air pollution may affect the fetus, via severely inhibiting the capacity of the pregnant woman to provide the necessary nutrients and oxygen to the fetus. Therefore, we also examine whether exposure to air pollution is more likely trigger stillbirths due to hypoxia.

3. Data

We obtained individual-level data on stillbirth deliveries from official records of the Department of Health Statistics of Chile's Ministry of Public Health. This data contains nationwide hospitals' records on those women who have delivered livebirths and stillbirths. The data records the municipality where the mother lived at the time of delivery as well as the date of delivery. This data also provides information on mother's pregnancy histories (number of previous livebirths deliveries, whether she had delivered a stillbirth in the past, etc), and mother's personal characteristics (mother's age, years of schooling, etc). The data also reports the number of weeks of pregnancy at the time of delivery. We restrict our data to pregnancies lasting more than thirteen weeks as miscarriages of pregnancies lasting less than that often go

increase maternal methemoglobin levels, which can oxidize fetal hemoglobin levels and inhibit the oxygen transport needed by the fetus (Green et al., 2015). Thereby, a severe lack of oxygen by the fetus could potentially result in fetal death due to hypoxia. Furthermore, Veras et al. (2015) suggest that air pollution can affect the utero-placental and umbilical cord flow and consequently the transport of glucose and oxygen through the placenta. Airborne particulate matter can affect pregnancy outcomes due to inflammation of the placenta, which could impair transplacental nutrient exchange thus affecting nutrition of the fetus and reducing oxygenation of maternal blood Kannan et al. (2007). Similarly, Maisonet et al. (2004) suggest that alveolar inflammation can lead to increased difficulties with blood flow, impacting placental functions.

unreported.⁷⁷ In this way, we have data on 4,915 stillbirth deliveries and 857,820 livebirth deliveries over the period 2008-2015, yielding a rate of 5.7 stillbirths for every thousand pregnancies lasting more than thirteen weeks.

Additionally, the health data allows us to categorize those stillbirths according to the leading cause of death as diagnosed by the physician at the time of delivery. When a pregnancy ends in a delivery of a stillbirth, the physician must write down the leading diagnostic according to the tenth version of the international classification of disease (ICD-10). In our dataset, of those 4,915 stillbirth deliveries in our data, 1,365 correspond to stillbirth due to hypoxia whereas 3,550 correspond to stillbirths due to other causes (different from hypoxia). This yields a rate of 1.6 stillbirths due to hypoxia out of every thousand pregnancies (lasting more than thirteen weeks).

Air pollution data comes from Chile's Air Quality National Information System; a network of air quality monitoring stations of Chile's Environmental Ministry. The monitoring stations provide daily records of both particulate matter with diameter less than 10 microns per cubic meter (PM₁₀) and carbon monoxide concentrations (CO) in parts per million. We build a municipality-week level dataset by assigning pollution exposure to each municipality for each week as follows. First, we take the week-average pollution from each monitoring station. Second, we construct a spatial mapping of pollutants by using the geographical coordinates of the monitoring stations to impute pollution levels to spatial reference points. As we assume that most

⁷⁷ We also conducted analysis restricting the sample to those pregnancies that lasted more than twenty weeks. The results remain largely unchanged.

people live within certain vicinity of schools we use the geographical location of schools within a given municipality as spatial reference points to spatially impute air pollution concentrations.⁷⁸ That is, we impute air pollution concentrations to the geographical location of each school within a municipality and then we average the imputed air pollution data across all (selected) schools for each municipality.⁷⁹ In this way we generate a municipality-week dataset for air pollution data that we then merge with the individual-level pregnancy data outlined above.

a. Correlation between Air Pollution and Pregnancy Outcomes

Our data allows us to assign municipality-level PM_{10} and CO pollution on the week of delivery of each pregnancy (either a livebirth or stillbirth). We refer to this as *acute* exposure. Furthermore, since we also have data on the number of weeks of pregnancy, we also assign average PM_{10} and CO pollution exposure throughout the entire length of pregnancy, to which we refer to as *chronic* exposure. Figure 18 below plots average chronic and acute exposure to PM_{10} pollution (left plot) and average chronic and acute exposure to CO pollution (right plot) for each pregnancy outcome

⁷⁸ Furthermore, we restrict our sample of schools to those that are located (i) at a distance of no more than 5 kilometers from the nearest air quality monitoring station, and, (ii) between 5 to 10 kilometers to the nearest monitoring station, but no more than 20 kilometers to the second-nearest monitoring station. If there is more than one monitoring station within a 20 kilometer radius from the school, we take the average by weighting the pollution data of each station by the inverse of the distance to the school. This methodology for spatial imputation of pollution data is known as the Inverse of the distance as weights (IDW) and has been previously been employed in (Currie and Neidell 2005) and (Arceo, Hanna, and Oliva 2016), to name a few.

⁷⁹ We only consider those municipalities for which we have pollution data for at least 70% of the weeks of any given year.

(either a livebirth or a stillbirth delivery). The figure also adds its corresponding 95% confidence intervals.

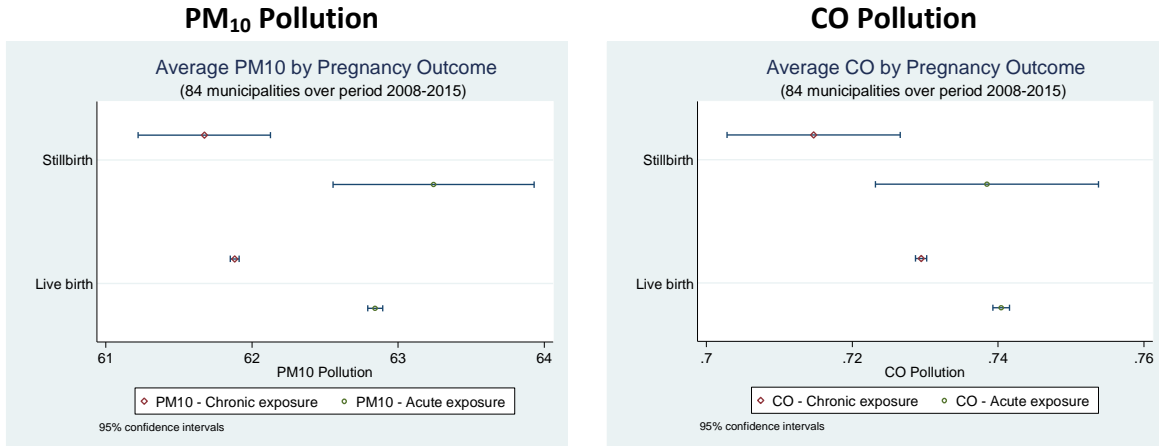


Figure 18: Mean PM₁₀ and CO Pollution, by Pregnancy Outcomes

The left plot of Figure 18 shows that the average *chronic* exposure to PM₁₀ pollution of those livebirths is not statistically different from the average *chronic* exposure to PM₁₀ pollution of those pregnancies that ended in a stillbirth. The same is true for the average *acute* exposure to PM₁₀ pollution. However, this figure is somehow different for CO pollution. The right plot of Figure 18 shows that the average chronic exposure to CO pollution of those livebirths is higher than that of stillbirths, whereas this difference becomes statistically insignificant for *acute* exposure to CO pollution.

We also look at cause-specific stillbirth, by distinguishing between those stillbirths diagnosed as due to hypoxia from those diagnosed as due to causes other than hypoxia ('SB Other' in Figure 19 below). The left plot of Figure 19 below shows that the average chronic exposure to PM₁₀ pollution for those stillbirths due to hypoxia is

statistically higher than that of both stillbirth due to causes other than hypoxia (SB Other) and for livebirths. This difference is even larger for acute exposure to PM₁₀. On the other hand, although the right plot of Figure 19 shows that the average chronic exposure to CO pollution for those stillbirths due to hypoxia is not statistically different from that of livebirths. However, the average acute exposure to CO pollution is statistically higher than both that of stillbirths due to other causes and that of livebirths.

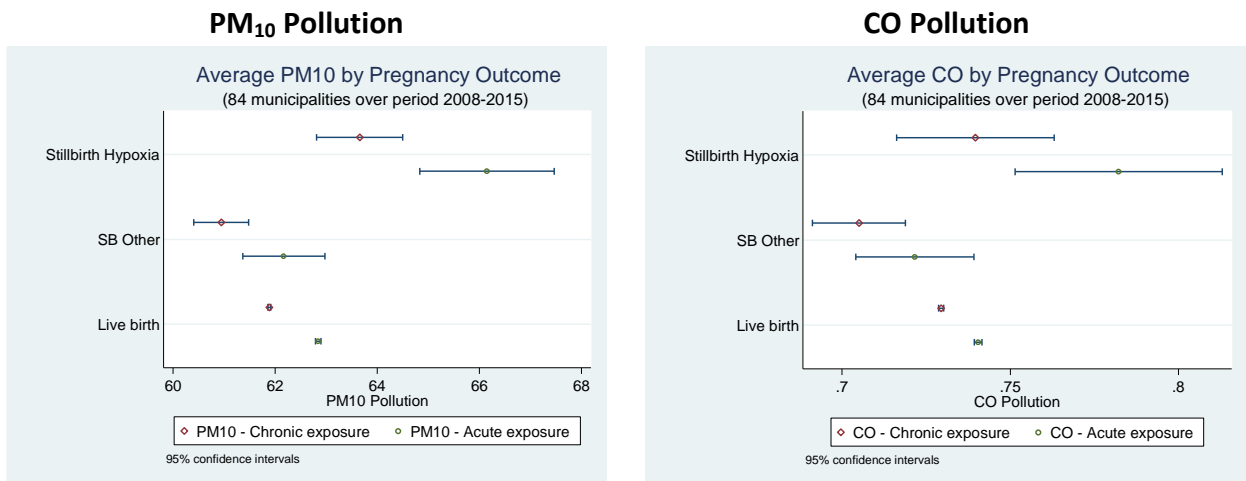


Figure 19: Mean PM₁₀ and CO Pollution, by Causes of Stillbirth Delivery

In addition, Table 17 below shows both mean and standard deviation of PM₁₀ and CO pollution (both for chronic and acute exposure), for pregnancies that resulted in a livebirth or a stillbirth, and within those stillbirths, whether the stillbirth was due to hypoxia or due to other causes. These are the same numbers that we use to generate

both Figure 18 and Figure 19. Also, Table 17 presents the number of observations for each category.⁸⁰

Table 17: PM₁₀, CO Pollution and Covariates, by Pregnancy Outcome.

	Stillbirths						Live Births	
	All Stillbirths (N = 4,915)		Stillbirths Hypoxia (N = 1,365)		Stillbirths Other (N = 3,550)		(N = 857,820)	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
PM10 - Chronic exposure	62.51	16.03	64.17	15.52	61.87	16.18	62.79	0.36
PM10 - Acute exposure	64.41	24.32	67.13	24.09	63.35	24.33	63.90	23.29
CO - Chronic exposure	0.718	0.425	0.740	0.441	0.709	0.418	0.730	0.363
CO - Acute exposure	0.741	0.548	0.781	0.577	0.726	0.535	0.741	0.541
Mother's age	28.7	7.5	27.8	7.5	29.0	7.4	27.7	6.7
Mother's past stillbirths	0.041	0.199	0.044	0.205	0.040	0.196	0.019	0.136
Marital status (0=married, 1=single)	0.39	0.49	0.36	0.48	0.40	0.49	0.33	0.47
Mother's years of education	12.1	3.0	11.6	2.9	12.3	3.1	12.6	2.9
Season of Conception								
Summer	0.22	0.42	0.23	0.42	0.22	0.42	0.23	0.42
Autumn	0.23	0.42	0.21	0.41	0.24	0.43	0.26	0.44
Winter	0.29	0.45	0.31	0.46	0.28	0.45	0.27	0.45
Spring	0.25	0.43	0.25	0.43	0.25	0.43	0.24	0.42
Season of Delivery								
Summer	0.27	0.44	0.27	0.45	0.27	0.44	0.26	0.44
Autumn	0.28	0.45	0.30	0.46	0.28	0.45	0.28	0.45
Winter	0.23	0.42	0.23	0.42	0.23	0.42	0.24	0.43
Spring	0.22	0.41	0.20	0.40	0.22	0.42	0.23	0.42

In addition to presenting statistics for PM₁₀ and CO pollutants by birth outcome, Table 17 above also presents descriptive statistics (means and standard deviations) for mother's personal characteristics (mother's age, history of past stillbirths, marital status and years of education) and the season of the year at both the beginning and at the end of the pregnancy (season of conception and season of delivery). Table 17 above shows that the average age at time of delivery is statistically higher for those

⁸⁰ So that 95% confidence intervals shown in figures 18 and 19 can easily be calculated by using the simple formula $Mean \pm SD/\sqrt{N}$.

pregnant women that delivered a stillbirth than for those that delivered a livebirth. However, when looking at the average age of those that delivered a stillbirth due to hypoxia, the average age of those women is not statistically different from the average age of those women who delivered a livebirth. Those who delivered a stillbirth due to causes other than hypoxia (SB Other) are statistically older than those that delivered livebirths. Table 17 also shows that those women that delivered a stillbirth (either due to hypoxia or due to other causes) present a higher incidence of history of delivering stillbirths in the past (41 per every thousand pregnant women) than those that delivered a livebirth (19 per every thousand pregnant women). Those women that delivered a stillbirth are more likely to be single (39 percent) and have fewer years of education (12.9) than those that delivered a livebirth (of which 33 percent are single and have an average of 12.6 years of education). Finally, the bottom panel of Table 17 shows descriptive statistics for season of conception and season of delivery. Among those that delivered a stillbirth, 29 percent of them became pregnant during the winter, which is statistically higher than that of those women that delivered a stillbirth and became pregnant during any other season of the year. This difference is further accentuated when focusing on those stillbirths due to hypoxia (31 percent). In sum, Table 17 shows that there are important differences in the personal characteristics and the patterns of pregnancy between those women that delivered a stillbirth and those that delivered a livebirth. In assessing the effect of air pollution exposure on pregnancy outcomes we explicitly control for the personal characteristics of the woman as well as for the seasonal pattern of the pregnancy.

b. Stillbirth Rates and Air Pollution across Municipalities and Time

To further explore the relationship between air pollution and pregnancy outcomes we look at the average stillbirths rates and pollution exposure both across different municipalities and across time, for the period 2008-2015. Figure 20 below presents average acute exposure to PM_{10} and CO pollution and stillbirth rate (upper plots) and rate of stillbirth due to hypoxia (lower plots) for the 84 municipalities in our dataset, where each blue dot represents average for each municipality. We have added a trend (red line) to highlight the possible correlations between these variables. The figure suggests that, when aggregated at the municipality level, there is little correlation between average acute exposure to PM_{10} pollution and average stillbirth rate across municipalities (upper left plot). However, the figure suggests a weak positive correlation between average acute exposure to CO pollution and stillbirth rate across municipalities (upper right plot). The lower plots of Figure 20, however, show a relatively stronger positive correlation between average acute exposure to PM_{10} and CO pollution and stillbirth rate due to hypoxia across municipalities (lower plots). On the other hand, the plots for chronic exposure to both PM_{10} and CO pollution (omitted here) present very similar patterns as those of acute exposure, in Figure 20.

Acute exposure to PM_{10}

Acute exposure to CO

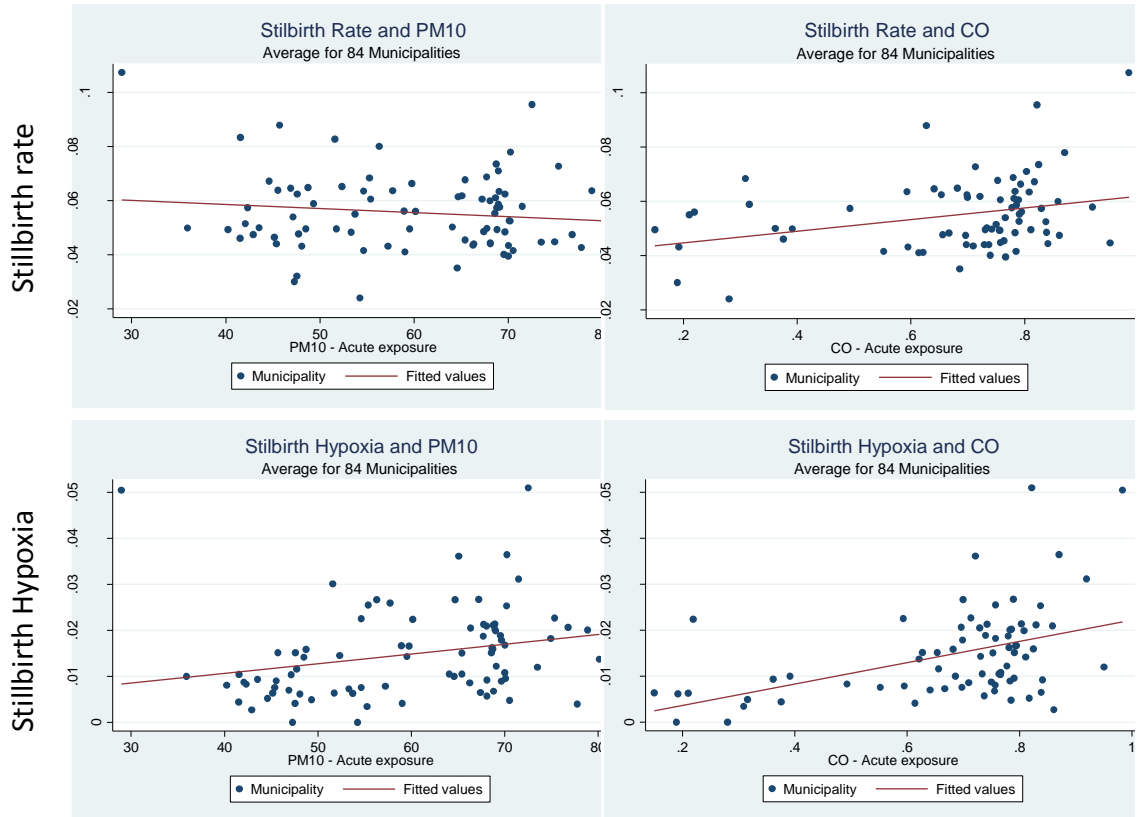


Figure 20: Municipality-level Stillbirth Rate, Stillbirth Due to Hypoxia and Acute Exposure to PM₁₀ and CO Pollution.

On the other hand, Figure 20 presents month-average acute exposure to PM₁₀ and CO pollution and stillbirth rates and stillbirth due to hypoxia, for the period 2008-2015. The figure suggests little serial correlation between acute exposure to PM₁₀ and CO pollution and stillbirth (upper plots), as well as little serial correlation between acute exposure air pollution and stillbirth due to hypoxia (lower plots).

Acute exposure to PM₁₀

Acute exposure to CO

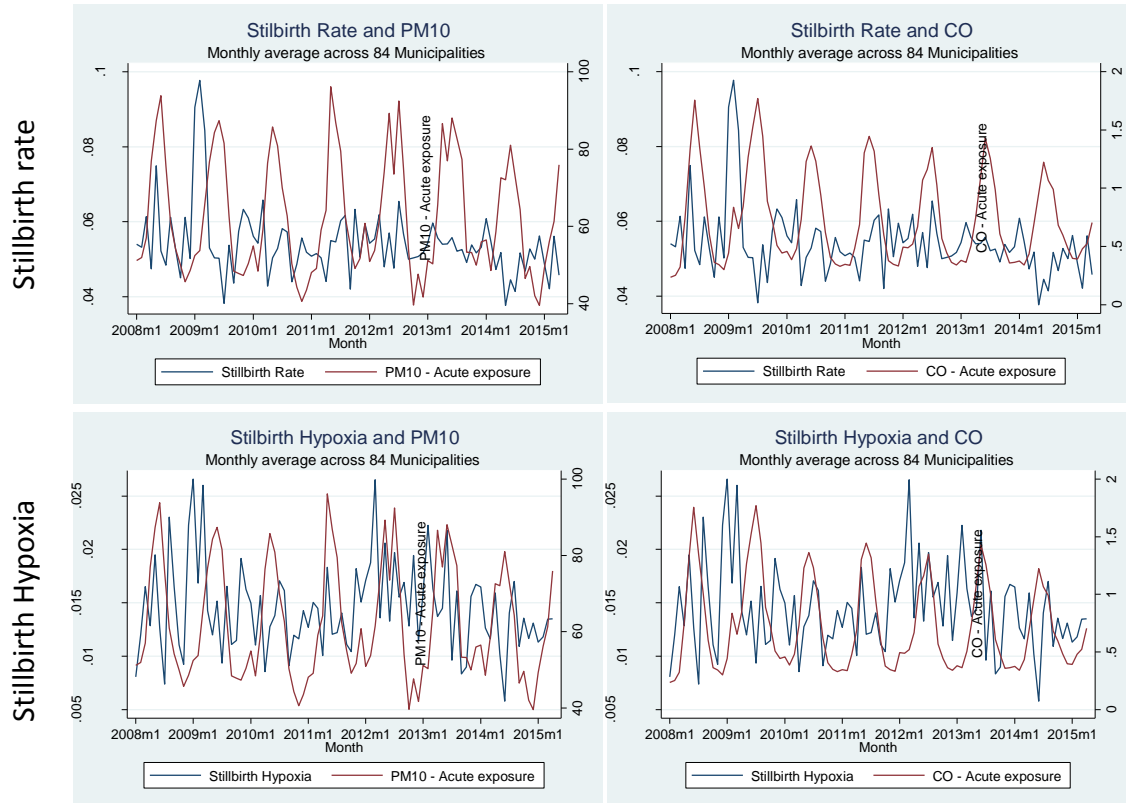


Figure 21: Monthly average Stillbirth Rate, Stillbirth Due to Hypoxia and Acute Exposure to PM₁₀ and CO Pollution.

c. Variation of Air Pollutants and Stillbirth

Our analysis thus far suggests that there is a positive correlation between acute exposure to air pollution and stillbirth delivery, and a stronger positive correlation between acute exposure to air pollution and stillbirth due to hypoxia. In this section we further examine how the *variation* in exposure to air pollution relates to the variation in stillbirth rate and stillbirth due to hypoxia.⁸¹ Table 18 below presents means and standard deviations of stillbirth rate for all causes, stillbirth rate due to

⁸¹ The regression analysis discussed in detail in the subsequent section exploits this variation to examine how exposure to air pollutants affects stillbirth rates.

hypoxia and both chronic as well as acute exposure to PM₁₀ and CO pollution for the sample of 84 municipalities over the period 2008-2015. The table shows that the stillbirth rate (all causes) in our sample is 0.057, and the stillbirth rate due to hypoxia is 0.016.

Also, as it can be expected, Table 18 shows that both chronic and acute exposure yield pretty similar means but they differ in their variation in terms of their respective standard deviations (where the more disaggregated week-level acute pollution exposure presents much larger standard deviations).

Table 18 also presents a decomposition of the standard deviation across municipalities and weeks. The table shows that the overall variation of stillbirth rate and stillbirth due to hypoxia is evenly distributed across the space and time dimension of our data (that is, across both municipalities and weeks), ranging from 9 percent of the overall variation (for municipality-level variation of stillbirth rate) to 14 percent (for both the municipality-level variation of stillbirth due to hypoxia as well as for the week-level variation of stillbirth). For chronic exposure to PM₁₀ pollution, the variation is also evenly distributed across municipalities and time (weeks) at 67 percent and 65 percent of the total variation, respectively. However, the more disaggregated measure for PM₁₀ pollution, acute exposure, varies largely across time (weeks), representing 79 percent of the overall variation of acute exposure to PM₁₀ pollution. On the other hand, most of the variation of exposure to CO pollution occurs

across weeks for both chronic exposure to CO (68 percent) as well as for acute exposure to CO (77 percent).

Table 18: Descriptive Statistics for Stillbirth, Stillbirth due to Hypoxia, PM₁₀ & CO Pollution Over Length of Pregnancy and on Week of Birth or Fetal Death

Variable	Mean	Standard Deviation			Obs.
		Overall	Municipality	Week	
Stillbirth rate (all causes)	0.0057	0.106	0.009	0.015	90,697
Stillbirth Hypoxia	0.0016	0.058	0.008	0.008	90,697
PM10 - Chronic exposure	62.77	13.87	9.26	9.05	90,697
PM10 - Acute exposure	63.93	23.34	9.22	18.44	90,697
CO - Chronic exposure	0.73	0.37	0.11	0.25	90,697
CO - Acute exposure	0.74	0.54	0.11	0.42	90,697

4. *Duration of Pregnancies and Hazard Function Approach*

a. Duration of Pregnancies by Pregnancy Outcome

One of the fundamental differences between stillbirths and livebirths is that the pattern of duration of pregnancy of those stillbirths is remarkable different to that of livebirths. Most fetal deaths and stillbirths deliveries occur before full-term gestation.⁸² Figure 22 below presents histograms for both livebirths and stillbirths according to the number of weeks of pregnancy at the time of delivery. The figure

⁸² Full term pregnancy is defined in the medical literature as those pregnancies that last at least 36 weeks since conception.

below show that whereas most livebirth deliveries occur between weeks 38 and 40 (indeed, more than 75 percent of total livebirth deliveries), very few stillbirths occur during that period of gestation (less than 10 percent of total stillbirth deliveries). Stillbirth deliveries seem to distribute relatively evenly over the entire pregnancy, with a relatively larger amount of stillbirth deliveries concentrated between the 16th and 24th week of pregnancy and another (although smaller) concentration of stillbirth deliveries in the weeks before the completion of a full term of pregnancy.⁸³ On the other hand, the distribution of pregnancies varies little between those stillbirths due to hypoxia and stillbirths due causes other than hypoxia (not shown here).

⁸³ The remarkable differences in the distribution of weeks of pregnancies for livebirths and stillbirths seem to have been overlooked by the existing literature that examines the relationship between air pollution and pregnancy outcomes. As explained earlier, this omission is likely to have introduced selection bias in previous studies. We should also note that the duration of the pregnancy is intrinsically connected with the pregnancy outcome (either a livebirth or a stillbirth) and those observed or unobserved factors that determine the duration of pregnancies are very likely to also determine the pregnancy outcome. Thus, if not properly controlling for these factors, this would introduce further biases in the estimation of the effects of exposure to air pollution on pregnancy outcomes.

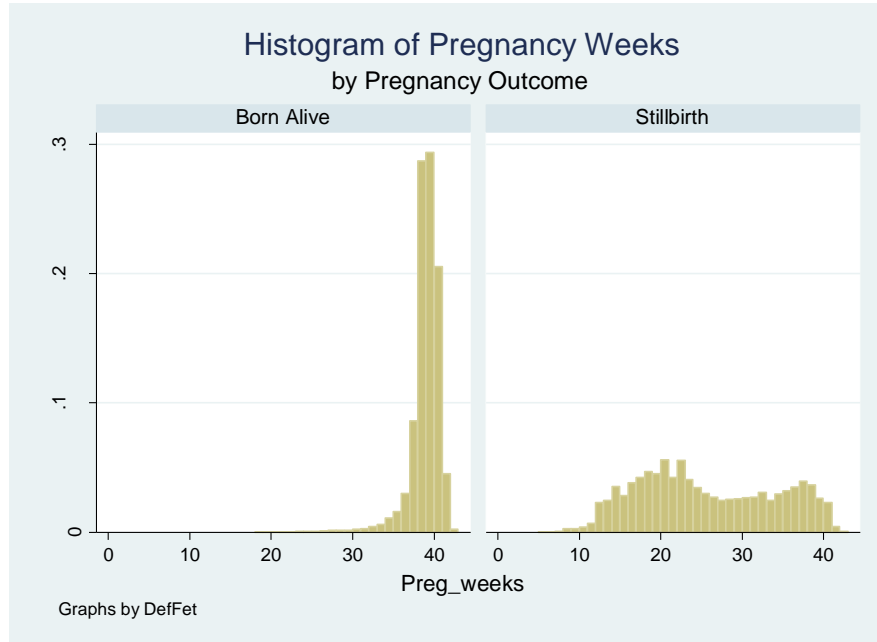


Figure 22: Histogram of Livebirths and Stillbirths.

In addition, for each week of actual delivery, Figure 23 below shows the ratio of those pregnancies ending in a stillbirth in week t over all pregnancies ending in week t , and contrasts the time pattern of those pregnancies that end in a Stillbirth delivery to those that end in a livebirth delivery. The figure shows that whereas most pregnancies that end early (say, before the 26th week of pregnancy) present a very large probability of ending on a stillbirth (indeed, the probability is always greater than .8), those pregnancies that end close to full term (after the 36th week of pregnancy) have a very low probability of ending in a stillbirth (probabilities lower than .05).

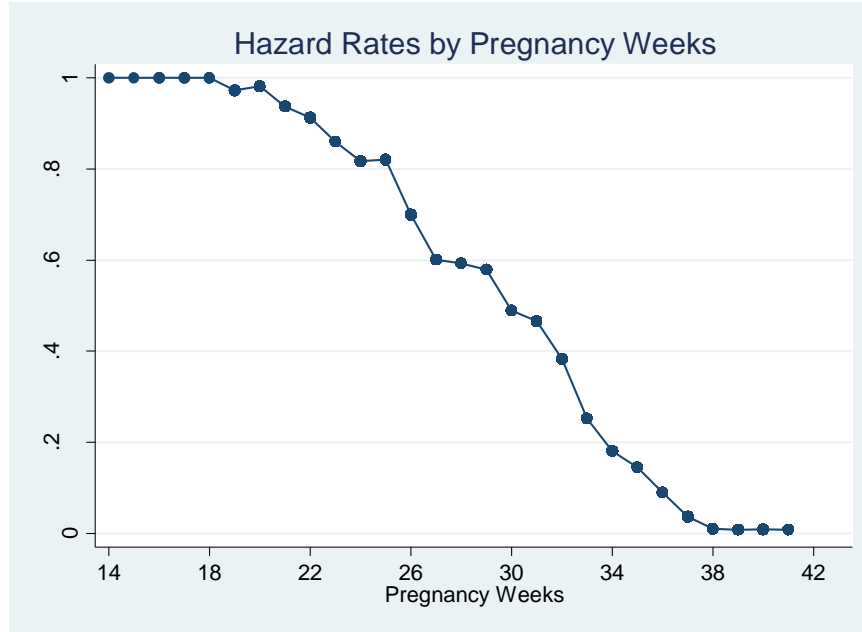


Figure 23: Hazard of Stillbirth Delivery for Pregnancies Ending at a Given Week t .

b. Hazard Function Approach

To estimate the effect of air pollution exposure on the probability of stillbirth in this paper we employ the hazard function approach. The hazard function approach allows to explicitly account for the *time at risk* throughout the duration of the pregnancy, and to gauge how exposure to air pollution during the pregnancy may affect whether a pregnancy results in a stillbirth or livebirth delivery.⁸⁴

In terms of the biostatistics terminology, a stillbirth or livebirth delivery can be thought of as a transition out of a current state of pregnancy, resulting in an

⁸⁴Currie and Neidell (2005) employ a linear hazard functions approach to estimate the effect of PM₁₀, O₃ and CO on infant mortality in California. While controlling for location specific unobserved factors at the zip-code level, the authors find significant effects of acute (week-level) exposure to CO.

interruption of the pregnancy. In this paper, we focus on how exposure to air pollution affects these transitions out of pregnancy and how such exposure to air pollution may make it more likely for any specific pregnancy outcome to occur (either a livebirth or a stillbirth delivery).

The hazard function approach consists on estimating the probability that the status of a pregnant woman changes, at a given week (say week T), from pregnant to no longer pregnant, where this no-longer-pregnant status can be either a livebirth or a stillbirth delivery. More formally, the hazard function approach consists on estimating the probability that a given pregnancy that has lasted through period $T \geq t - 1$, transitions out of its current state at period $T = t$. In other words, this approach consists of estimating the (discrete-time) hazard function $h(X, t)$ defined by

$$h(X, t) = \frac{f(X, t)}{\text{Prob}(X, T > t - 1)} = \frac{f(X, t)}{1 - F(X, t - 1)} \quad (4)$$

where $f(\cdot)$ denotes the (discrete-time) probability distribution function and $F(\cdot)$ denotes the (discrete-time) cumulative distribution function. Both need to be specified.

Whereas the numerator in equation (4) denotes the unconditional probability of a transition out of a state of pregnancy, the denominator in equation (4) denotes the *survival probability*, that is, the probability that the pregnancy lasts at least up to period $T \geq t - 1$. Hence, the hazard $h(t)$ denotes the probability of a transition out of

a state of pregnancy at a given period $T = t$, occurs, given that such a transition has not occurred yet.

Under this framework, exposure to air pollution may have two effects that are captured simultaneously by the hazard of stillbirth in equation (4). First, exposure to air pollution may shorten the elapsed time in the current state (shorten the duration of pregnancies), which is captured by a decrease in the survival probability (the denominator in equation (4)). Second, for those pregnancies that end at a given period $T \geq t - 1$, exposure to air pollution may increase the probability that those transitions out of pregnancies result in stillbirth deliveries (as opposed to resulting in livebirth deliveries), yielding an increase in the numerator of equation (4) above.

In order to assess the effects of covariates on the hazard probability, it is common practice in the empirical literature to assume that the underlying continuous-time model follows a proportional hazard rate $\theta(X, t) = \lambda(X)\theta_0(t)$, so that the hazard rate of transitioning out of the current state depends on both the elapsed time in that state (pregnancy time, $\theta_0(t)$) as well as on covariates X (a function $\lambda(X)$). Where $\theta_0(t)$ is known as the baseline hazard, and $\lambda(X) = e^{\beta'X}$ denotes a vector of covariates where each covariate X_i has a proportional effect on the hazard probability via β_i . Assuming a logit specification for $f(\cdot)$ in (1) above, the proportional hazard rate assumption in a leads to the *complementary log-log* regression model discrete-time setting (Jenkins 2005):

$$h(X, t) = 1 - \exp[-\exp(\beta'X + \gamma_i(t))]$$

or

(5)

$$\text{Log}[-\text{Log}[1 - h(t, X)]] = \beta'X + \gamma_i(t)$$

where $\gamma(t)$ is the cumulative hazard ($\gamma(t) = \int_{t-1}^t \theta_0(u)du$) that depends on elapsed time only (not on covariates X s).

Using this hazard function approach, in the next section we estimate both the probability of a pregnancy transitioning out to a stillbirth (that is, the probability of Stillbirth) and the probability of a pregnancy transitioning to a stillbirth due to hypoxia (Stillbirth Hypoxia). When estimating such probabilities we focus in particular on how both chronic and acute exposure to PM₁₀ and CO pollution shapes this probability. That is, for any given week of pregnancy in which the pregnant mother is *at risk* of suffering from the effects of exposure to air pollution, we focus on how such exposure may affect the probability of her pregnancy ending in a stillbirth delivery.

From equation (5) above, the regression framework for assessing the effect of air pollution on probability of stillbirth can be expressed as follows.

$$\begin{aligned} \text{Log}[-\text{Log}[1 - h_i(t, X)]] &= \beta'X_i + \gamma(t)_i \\ &= \beta_1 P_{it} + \beta_2 z_i + \beta_3 w_t \\ &\quad + \gamma_i(t) \end{aligned} \tag{5'}$$

where P_{it} denotes exposure to air pollution over period t (chronic or acute exposure to either PM_{10} or CO pollution); z_i denotes mother's characteristics (such as age, history of past stillbirth deliveries, marital status and years of education) as well as controls for seasonality of the pregnancy (season of conception and season of delivery); and w denotes a set of month and year dummies. Furthermore, $\gamma_i(t)$ is a function that captures the effect of the duration of the pregnancy (in weeks) on the hazard of stillbirth delivery. In this case, we use a semi-parametric piece-wise constant hazard with breaks at pregnancy weeks 21, 28, and 36.

To implement this discrete-time estimation strategy, we treat a pregnancy that lasted for n weeks as if it contributes n observations to the sample.⁸⁵ Thus, this model can be implemented by estimating equation (5') using a *complementary log-log* regression model where the dependent variable takes on value 1 when a pregnancy ends in a stillbirth and on value 0 in all other cases (both when a pregnancy continues from one week to the next one, and when a pregnancy ends in a livebirth delivery). Alternatively, in the presence of competing end states (say, a stillbirth delivery or a livebirth delivery) the *complementary log-log* regression model reduces to a multinomial logit (Jenkins 2005). In our case, the multinomial logit has three potential outcomes. Whereas the base outcome is that of remaining pregnant (from one week to the following one), the two other outcomes are a stillbirth delivery and a livebirth delivery. Therefore, to estimate the effect of exposure to pollution on the probability of stillbirth as a transition out of a state of pregnancy we could use either

⁸⁵ In this framework, we calculated chronic exposure to air pollution as the moving average of the week-level air pollution variable, averaging out up to the n -th week of pregnancy.

the complementary log-log model or the multinomial model (they indeed yield both the same results).⁸⁶

In the next section, using this hazard function approach, we estimate the effect of air pollution on the probability of a pregnancy transitioning out to a stillbirth by estimating a multinomial logit. Furthermore, we also estimate the effect on the probability of a pregnancy transitioning to a stillbirth due to hypoxia by estimating a multinomial logit with three end states (namely, stillbirth delivery due to hypoxia, stillbirth delivery due to other than hypoxia and livebirth delivery). When estimating these multinomial logits we focus in particular on how both chronic and acute exposure to PM₁₀ and CO pollution shapes these probabilities for any given week of pregnancy.

5. *Econometric Analysis*

In this section we present the parameter estimates of the model outlined above. We first examine the effect of air pollution on the probability of a pregnancy ending in a stillbirth delivery and in the next sub-section we look at the effect of air pollution on the probability of a pregnancy ending in stillbirth due to hypoxia.

⁸⁶ By changing the base outcome to that of a livebirth, the multinomial logit also allows to estimate the effect of exposure to air pollution on—once a delivery occurs— whether that delivery is a stillbirth or a livebirth. If both stillbirth and livebirth deliveries were to occur around the same week of pregnancy, then contrasting stillbirth deliveries directly with livebirth deliveries would be the appropriate model. However, as Figure 5 shows, most of stillbirth deliveries do not occur around the same week of pregnancy as livebirths deliveries do (livebirths occur around the 38th week of pregnancy). Therefore, in this paper we focus on estimating the effect of exposure to air pollution on a transition out of pregnancy towards a stillbirth delivery, and contrast that with the probability of remaining pregnant on that particular week.

a. Effect of Exposure to Air Pollution on Probability of Stillbirth

Table 19 below presents hazard function parameter estimates for the effect of exposure to air pollution on the probability of stillbirth (the β_1 s). Each cell presents parameter estimates associated to exposure to PM₁₀ and CO pollution (and its corresponding standard error) from an independent regression. Each regression introduces only one pollutant (first two rows for PM₁₀ and third and fourth rows for CO), and only one specification for each period of exposure to each pollutant (either chronic exposure or acute exposure). That is, two pollutants and two periods of exposure to each pollutant. For the estimates presented in column 1 we do not introduce any additional controls. The estimates presented in column 2 introduce controls for pregnancy weeks (piecewise hazard with breaks at 21, 28, and 36 pregnancy weeks), mother's age, whether the mother has experienced stillbirth deliveries in the past, marital status and years of education.⁸⁷ In column 3 we introduce dummies for months, year and seasonality of pregnancy (both for the season of conception as well as for the season of delivery). Column 4 introduces all the controls from columns 2 and 3. Finally, Column 5 adds dummies by municipality to control for unobserved municipality-specific effects. Due to computer processing capacity the regressions used only a 10 percent random sample of those pregnancies that resulted in a livebirth delivery. Accordingly, regressions coefficients and

⁸⁷ Although we do not directly observe the smoking habits of the pregnant woman, mother's years of education, marital status and age, together, can act as a good proxy for smoking habits of the pregnant woman.

standard errors were adjusted by frequency weights in order to account for this sampling of livebirth deliveries.

The results presented in Table 19 show that the parameter estimates associated to acute exposure to PM₁₀ pollution (second row of Table 19) are statistically significant across all specifications, as shown in columns 1 through 5. Furthermore, the parameter estimates associated to acute exposure to CO pollution (fourth row of Table 19) are statistically significant only across those specifications that introduce time and municipality-specific controls. On the other hand, the results shown in Table 19 show that both chronic exposure to PM₁₀ and CO pollution have mostly a non-significant effect on the probability of stillbirth. Most parameter estimates on chronic exposure to both PM₁₀ and CO pollution are statistically non-significant at conventional levels.

Table 19: Effect of PM₁₀ and CO pollution on the probability of stillbirth. Hazard estimates.

	(1)	(2)	(3)	(4)	(5)
PM10 - Chronic exposure	-0.00212** (0.00100)	-0.00170 (0.00116)	0.000116 (0.00149)	0.000589 (0.00176)	0.00196 (0.00270)
PM10 - Acute exposure	0.00112* (0.000581)	0.00118** (0.000600)	0.00343*** (0.000759)	0.00354*** (0.000758)	0.00449*** (0.000893)
CO - Chronic exposure	-0.0882* (0.0455)	-0.0937* (0.0488)	-0.0617 (0.0676)	-0.0641 (0.0688)	-0.0908 (0.0736)
CO - Acute exposure	-0.000205 (0.0311)	-0.00334 (0.0302)	0.0859*** (0.0302)	0.0999*** (0.0310)	0.0976*** (0.0321)
• Controls: Pregnancy weeks, Mother's age, Past stillbirth delivery, Marital Status and Years of education.	N	Y	N	Y	Y
• Dummies for Month, Year and Seasonality of pregnancy (season of the year for both conception & delivery)	N	N	Y	Y	Y
• Municipality Dummies	N	N	N	N	Y
Obs. expanded by pregnancy weeks	2,261,543	2,261,543	2,261,543	2,261,543	2,261,543
Observations	90,697	90,697	90,697	90,697	90,697

Standard errors clustered by municipality. *** p<0.01, ** p<0.05, * p<0.1.

The hazard estimates of the parameter associated to acute exposure to PM₁₀ vary from .00112 (column 1) to 0.00449 (column 5), depending on the set of controls we introduce. When introducing only acute exposure to PM₁₀ pollution (column 1), we obtain that the parameter estimate is small and only weakly significant. Adding further controls for mother's characteristics (column 2), dummies for month, year, season of conception and delivery (columns 3 and 4), and dummies for municipality (column 5) yields larger and more significant estimates. Moreover, once we introduce time and municipality controls, the parameter estimates turn relatively stable across all these different specifications (columns 3, 4 and 5).

According to the parameter estimate in column 5 (which introduces all available controls), our estimate suggests that the probability that a pregnancy ends in a stillbirth (as opposed to remaining pregnant) increases by .449 percent due to a 1-unit change in acute exposure to PM₁₀. Table 18 shows that the standard deviation of acute exposure to PM₁₀ is 23.34 and that the stillbirth rate (baseline probability of stillbirth) in our sample is 5.69 per every thousand pregnancies. Thereby, our estimate suggests that a one-standard-deviation increase in acute exposure to PM₁₀ pollution would increase the stillbirth rate by 10.5 percent, bringing it up to 6.29 per every thousand pregnancies [$6.29 = 5.69 * (1 + 23.34 * .00449)$]. Thereby, for our sample (spanning 84 municipalities over the years 2008-20015), a one-standard-deviation increase in acute exposure to PM₁₀ pollution would result in 508.4 additional stillbirth deliveries over the period [$508.4 = 0.00629 * (4,915 + 857,820) - 4,915$], an average of 63.6 additional stillbirths a year.

On the other hand, the hazard estimates associated to the effect of acute exposure to CO pollution on probability of stillbirth varies from -.000205 (column 1) to .0999 (column 4), depending on the set of controls we introduce. Once we introduce controls for month, year, season of conception and delivery (columns 3 and 4), and dummies for municipality (column 5) the parameter estimates turn statistically significant and relatively stable across all these specifications.

The parameter estimate that introduces all available controls (column 5) yields an estimate for the effect of acute exposure on the probability of stillbirth of .0976.

Thereby, this estimate suggests that a one-standard-deviation increase in acute exposure to CO pollution (see Table 18) would increase the stillbirth rate by 5.3 percent, bringing it up to 5.99 per every thousand pregnancies $[=0.00529*(1 + .54*.0976)]$.⁸⁸ Therefore, such an increase in acute exposure to CO pollution would result in 252.7 additional stillbirth deliveries over the period considered in this paper $[=0.00599*(4,915+857,820) - 4,915]$, an average of 31.6 additional stillbirths a year.

b. Effect of Exposure to Air Pollution on Probability of Stillbirth Due to Hypoxia

One of the channels that air pollution is thought to adversely affect the fetus during pregnancy is via the nutrients and oxygen that the pregnant woman transmits to the fetus. Thereby, exposure to air pollution is likely to restrict her capacity to properly transmit nutrients and oxygen to the fetus. In situations where this inhibition is severe enough to end the pregnancy in a stillbirth, it is likely that the cause of fetal death is diagnosed by the physician as hypoxia. Therefore, the adverse effects of exposure to air pollution on pregnancies are likely to be more directly reflected in those fetuses that die due to hypoxia.

In Table 20 below we present estimates of the hazard function regression analysis for stillbirths due to hypoxia. Similar to Table 19, the first column presents estimates without introducing any controls, and then columns 2 to 5 introduce further controls.

⁸⁸ Table 18 shows that the standard deviation of acute exposure to CO pollution is .54.

Each cell in Table 20 refers to an independent regression with only one pollutant (and only type exposure) where the first statistic refers to the parameter estimate associated to that pollutant and the second statistic refers its corresponding standard error.

Table 20: Effect of PM₁₀ and CO pollution on the probability of stillbirth due to hypoxia. Hazard estimates.

	(1)	(2)	(3)	(4)	(5)
PM10 - Chronic exposure	0.00326 (0.00205)	0.00436* (0.00227)	0.00976*** (0.00305)	0.0119*** (0.00361)	0.00181 (0.00530)
PM10 - Acute exposure	0.00565*** (0.00147)	0.00560*** (0.00144)	0.00874*** (0.00181)	0.00878*** (0.00179)	0.00633*** (0.00198)
CO - Chronic exposure	0.0344 (0.0806)	0.0220 (0.0840)	0.194* (0.102)	0.198* (0.111)	0.0808 (0.109)
CO - Acute exposure	0.131** (0.0591)	0.111** (0.0552)	0.252*** (0.0651)	0.255*** (0.0659)	0.212*** (0.0660)
• Controls: Pregnancy weeks, Mother's age, Past stillbirth delivery, Marital Status and Years of education.	N	Y	N	Y	Y
• Dummies for Year and Seasonality of pregnancy (season of the year for both conception & delivery)	N	N	Y	Y	Y
• Municipality Dummies	N	N	N	N	N
Obs. expanded by pregnancy weeks	2,261,543	2,261,543	2,261,543	2,261,543	2,261,543
Observations	90,697	90,697	90,697	90,697	90,697

Standard errors clustered by municipality. *** p<0.01, ** p<0.05, * p<0.1.

The estimates presented in Table 20 show that acute exposure to PM₁₀ and CO pollution has a statistically significant effect on the probability of stillbirth due to hypoxia. Furthermore, these parameter estimates turn relatively stable throughout all different specifications of Table 20 (columns 1 through 5). On the other hand, the estimates for the effect of chronic exposure to PM₁₀ and CO pollution are not consistently significant throughout different model specifications in Table 20.

The estimates presented in column 5 of Table 20 suggest that the probability of a pregnancy ending in a stillbirth due to hypoxia increases by .633 percent due to a 1-unit increase in acute exposure to PM₁₀. Given that the stillbirth rate due to hypoxia is 1.58, per every thousand (see Table 18), a one-standard-deviation increase in acute PM₁₀ pollution would bring this rate up to 1.82 per every thousand $[=.00158*(1+23.34*.00633)]$, which corresponds to a 14.8 percent increase. This means that a one-standard-deviation increase in acute exposure to PM₁₀ pollution would result in 201.7 additional stillbirth deliveries due to hypoxia, $[=0.00182*(4,915+857,820)-1,365]$, an average of 25 additional stillbirths due to hypoxia a year.

Similarly, the estimates for acute exposure to CO pollution in column 5 show that a one-standard-deviation increase in acute CO pollution would bring the probability of stillbirth due to hypoxia up to 1.76 per every thousand $[=.00158*(1+.54*.212)]$, which corresponds to a 11.4 percent increase. That is, such an increase in acute exposure to CO pollution would result in additional 154.2 stillbirths due to hypoxia $[=0.00182*(4,915+857,820) - 1,365]$, an average of 19.3 additional stillbirth due to hypoxia a year.

Our findings support the notion that the effect of exposure to air pollution is stronger among those stillbirths due to hypoxia. Table 18 shows that stillbirths due to hypoxia amount to only 27.8 percent of total stillbirths (1,365 out of 4,915). However, the additional stillbirths due to hypoxia that would result from an increase in pollution

exposure amounts to a much larger proportion. Whereas the additional stillbirths due to hypoxia that would result from a one-standard-deviation increase PM_{10} pollution would represent 37.9 percent of the total additional stillbirths, the figure from an increase in one-standard-deviation in CO pollution would represent 61 percent of the total additional stillbirths. This finding support our prior that the pathway in which air pollution adversely affects pregnancies is primary manifested through those fetal deaths that are caused by hypoxia.

An additional finding that is worth noticing is that the hazard estimates for the probability of stillbirth due to hypoxia are quite similar for both chronic as well as acute exposures to both PM_{10} as well as CO pollution. This is particularly true for columns 3 and 4 of Table 4 (first and second row for PM_{10} pollution, and third and fourth row for CO pollution). This suggests a consistent estimate for the effects of both PM_{10} and CO pollution regardless of whether it is chronic exposure or acute exposure. This may be because the aggregation of the pollution exposure variable throughout many weeks of pregnancy (in the case of chronic exposure) reduces the variability of the pollution variable, which, in turn, generates larger standard errors in the parameter estimate. This suggests that our statistical methods may be limited when it comes to reliable reporting significant effects of chronic exposures to air pollutants. Although chronic exposure may actually have significant adverse effects on pregnancies, our statistical methods cannot actually capture that effect.

6. Discussion

In this chapter we estimate that acute exposure to a 10-unit increase in acute exposure to PM₁₀ pollution increases the probability of stillbirth by 4.49 percent. On the other hand, about half of the stillbirth deliveries in our sample occur during the third trimester of pregnancy—after the 26th week of pregnancy (see Figure 22). Thereby, our measure of acute exposure to PM₁₀ can be somewhat compared to measures of exposure to PM₁₀ pollution in the last trimester of pregnancy of those stillbirth deliveries (either second or third trimester of pregnancy). For instance, Kim et al. (2007) find that a 10-unit increase exposure to PM₁₀ pollution in the third trimester of pregnancy increases the probability of stillbirth by 8 percent. However, whereas the stillbirth rate that reported in Kim et al. (2007) for the city of Seoul is 4.45 percent, the stillbirth rate in our data is only .57 percent. For stillbirth rates similar to the one reported in our paper, Hwang et al. (2011) find that only exposure to *peak* levels of PM₁₀, during the first and second month of pregnancy, increases the probability of stillbirth by about 2 percent.⁸⁹ Moreover, in a study that looks at the effects of exposure to fine particulate matter (PM_{2.5}), De Franco et al. (2015) find that exposure to *peak* levels of PM_{2.5} during the third trimester of pregnancy increase the probability of stillbirth by 42 percent. Similarly, Green et al. (2015) find some evidence that exposures to *peak* levels of PM_{2.5} during the entire pregnancy increase the probability of stillbirth by 6 percent.

⁸⁹ *Peak* levels of pollution refers to pollution events in which pollution is higher than the sum of the mean pollution and inter-quantile range ($> \text{mean} + \text{IQR}$). If pollution has a normal distribution, this would be equivalent to episodes of pollution in the top 2.5% percent of its distribution.

7. Policy Implications

Air pollution is a growing global concern, particularly in middle income and emerging economies. Consequently, estimating the effects of air pollution on human health can provide policy makers with the necessary tools to back efforts to reduce concentrations of air pollutants.

For example, Chile's environmental authority has developed Air Pollution Control and Prevention Plans (PPDAs) for Santiago since the late 1990s, and more recently also for other smaller cities throughout the rest of the country. PPDAs consist of a battery of provisions for reducing air pollution. As of today, Chile's environmental authority has developed PPDAs for 18 cities, covering largely the same geographical area as the set of municipalities considered in this paper.

The goal of these PPDAs is to bring air pollution concentrations below Chile's national standard, which is 50 micrograms per cubic meter for PM₁₀. These PPDAs are evaluated ex-ante estimating social costs and benefits of expected improvements in air quality using estimates from different international studies that look at the effect of changes in pollution concentrations on specific health outcomes (these estimates are known in the literature as *dose-response functions*). However, the effect of air pollutants on stillbirth deliveries has not yet been included in the evaluation of Chile's PPDAs. As a consequence, it is likely that the benefits from the efforts to

reduce air pollution concentration have been largely underestimated in the cost-benefit analysis for Chile's PPDA's.

Indeed, the average PM₁₀ concentration in our sample is 63.93, exceeding Chile's standard for this pollutant (see Table 18). To bring PM₁₀ concentrations to meet Chile's standard for PM₁₀ would require an average reduction in concentration of 13.93 units. According to our estimates from Table 3, such a reduction would translate into 300 fewer stillbirth deliveries over the period 2008-2015, and an average of 37.6 fewer stillbirth deliveries a year. Chile's Minister of Social Development has recently set the official value of statistical life at US\$ 0.431 million (MINDES 2017). Therefore, the value of those fewer stillbirth deliveries resulting from reducing concentrations of PM₁₀ pollution to meet Chile's standard would amount to US\$16.2 million a year. These benefits have not been valued in the cost-benefit evaluations of the efforts to reduce air pollution concentrations under Chile's PPDA's.

8. Concluding Remarks

In this chapter we examined the relation between exposure to airborne air pollution and the effect that this may have on the probability of a pregnant woman ending their

pregnancy in a stillbirth delivery. The existing literature shows only weak evidence of the relationship between exposure to air pollution and stillbirth delivery.⁹⁰

We looked at the effect of both chronic as well as acute exposure to PM₁₀ and CO pollutants on the probability of stillbirth by estimating hazard functions that explicitly account for the *time at risk* throughout the duration of the pregnancy. We controlled for number of weeks of pregnancy, mother's characteristics, seasonal pregnancy characteristics, month and year-specific dummies, and municipality-specific dummies. We find that acute exposure to PM₁₀ has an adverse effect on the probability of stillbirth. We estimate that a one-unit change in acute exposure to PM₁₀ pollution increases the probability of stillbirth by .449 percent. This means that, for our sample of 84 municipalities over the period 2008-2015 in Chile, a one-standard-deviation change (increase/decrease) in PM₁₀ concentrations would translate into 63.6 (additional/fewer) stillbirths a year.

Furthermore, to the best of our knowledge, the existing literature does not examine the effect of exposure to air pollution on the probability of cause-specific stillbirths, and the likely pathway of the effects of pollution exposure on fetal deaths that result in a stillbirth delivery. In this paper we looked at the effects of PM₁₀ and CO on the probability of stillbirth due to hypoxia (those fetuses that die due to the lack of oxygen). We find that acute exposure to both PM₁₀ and CO increase the probability of

⁹⁰ As noted earlier, only exposure to very high levels of air pollutants is associated to a statistically significant higher probability of stillbirth (De Franco et al., 2015).

stillbirth due to hypoxia and has a larger effect than that of the one on stillbirth due to all causes.

On the other hand, it could be argued that the lack of controls for smoking habits of the pregnant woman may introduce bias in our parameter estimates. However, as long as smoking habits does not systematically correlate with exposure to air pollution after controlling for mother's years of education, age, marital status and location of residence (municipality of the mother), the lack of control for smoking habits should not introduce bias in our estimates.

Moreover, as with most of epidemiological studies in the literature, this study assigns exposure to air pollutants only imperfectly. In our case, we assign exposure based on a sound average of the pollution concentration at the municipality of residence. A more accurate measure of pollution exposure would be such that it would allow to record air quality at an exact location for each pregnant woman in each period of time during her pregnancy. Unfortunately, our data does not allow for such accurate recordings. Furthermore, it is likely that pregnant women are more aware of the health risks of the surrounding environment. Pregnant women attend regular checkups with their gynecologist and health practitioners who may alert them of the health hazards of exposure to high levels of air pollution. They may also be more alert at the news and warnings of the local environmental and health authorities. As a consequence, pregnant women may change their behavior to limit their exposure to air pollutants. For example, they may choose not to exercise and to reduce time spent

outside in days of high pollution concentrations. They may even be inclined to wear a breathing or dust mask during days of high pollution concentrations, although this would be rare in a country like Chile. Nevertheless, both assigning only an imperfect measure of actual pollution exposure as well as a possible change in behavior to minimize pollution exposure would bias our results towards the null of no effect. The fact that we find a statistically significant effect under these limitations likely represents a lower bound of the true effect of air pollution on the probability of stillbirth.

Appendices

1. Appendix I – The Kriging Method

With the exact coordinate location of schools and monitoring stations, data were interpolated using the Kriging method.⁹¹ Kriging interpolates using the spatial variation of air pollution based on the empirical semi-variogram:

$$\gamma(h) = \frac{1}{2M(h)} \sum_{j=1}^{M(h)} \{z(x_i) - z(x_i + h)\}^2$$

where h is the distance, $z(x_i)$ the observed value at location x_i and $M(h)$ are the number of pairs. The Kriging method utilizes the semi-variogram to determine the nature of variance and localized variability of data to generate values on a surface taking into account localized spatial trends. Kriging involves estimating values for any location using weights, which are optimized according to the semi-variogram model, the location of the samples and all the relevant inter-relationship between known and unknown values. With ordinary Kriging, the variable Z at a given location x_0 is written as a weighted linear function of the N neighboring values:

$$Z(x_0) = \sum_{i=1}^N \omega_i z(x_i)$$

The interpolation was performed day-by-day but conditioning to those days with at least five reports from different monitoring stations. The parameters for the variogram model were estimated using a likelihood-based method with an exponential correlation function and then were interpolated with an ordinary Kriging.

⁹¹ We used geoR package in R software, which makes it possible to handle spatial data and undertake several geostatistical procedures.

2. Appendix II – Summary of Current Literature for Chapter 3

Authors	Location and Years	Pollutants & Imputation of Exposure	Length of Exposure in the Analysis	PM and CO Mean (SD)	Stillbirth rate (%)	Methodology and Econometrics	Controls	Effects on Stillbirth (Odd Ratios)
Kim et. al. (2007)	Seoul, Korea. May 1 st 2001 – May 31 st 2004.	PM ₁₀ Nearest monitoring station to mother's address using 27 monitoring stations in Seoul.	Trimesters, Last six weeks. Months,	89.3 (45.7)	4.4	Hospital-based cohort study. Logistic regression	Demographics, mother's SES, medical history, pregnancy complications, exposure to smoking, alcohol use	1.1 for Third trimester
Pereira et. al. (2008)	Sao Paulo, Brazil. Jan 1991 – Dec 1992.	PM ₁₀ , CO (also SO ₂ , NO ₂ & O ₃). City-wide daily average using 13 and 5 monitoring stations for PM ₁₀ and CO, respectively.	3 and 14 days before delivery for CO and PM ₁₀ , respectively.	CO: 5.73 (1.89) PM ₁₀ : 65.0 (27.3)	8.36	Citywide ecological study. Poisson regression.	Season (month and day dummies) and weather (temperature, relative humidity).	CO: Positive & significant at 90%
Hwang et. al. (2011)	Taiwan. Jan 1 st 2001- Dec 31 st 2007.	PM ₁₀ , CO (also SO ₂ , NO ₂ & O ₃) Interpolated using IDW to postal-code area (one block face) using 72 monitoring stations.	Months, Trimesters and entire pregnancy.	PM ₁₀ : 72.9 (23.3) CO: 0.66 (0.18)	0.62	Nationwide population-based. Logistic regression.	Sex, maternal age, gestational age, municipality-level SES, season of conception and year of birth.	PM ₁₀ : 1.02 for First and Second Month
Faiz et. al. (2012)	State of New Jersey, USA. Jan 1 st 1998 – Dec 31 st 2004.	PM _{2.5} , CO (also SO ₂ & NO ₂) Nearest monitoring station to mother's address (10Km and 5Km) using 25 and 16 monitoring stations for PM _{2.5} and CO, respectively	Trimesters and entire pregnancy	PM _{2.5} : 13.8 (1.6) CO: 0.92 (0.3)	0.42	Statewide population-based. Logistic regression	Maternal age, race and educational attainment, participation in prenatal care and self-reported smoking. Also, neighborhood SES, mean temperature, calendar year and month of conception.	CO: 1.14 of peaks (> mean+IQR) during Second and Third trimesters
Faiz et. al. (2013)	State of New Jersey, USA. Jan 1 st 1999 – Dec 31 st 2004	PM _{2.5} , CO (also SO ₂ & NO ₂) Nearest monitoring station to mother's address (10Km and 5Km) using 5 and 13 monitoring stations for PM _{2.5} and CO, respectively	2 to 6 days before delivery	PM _{2.5} : 14.7 (8.8) CO: 0.85 (0.4)	0.42	Statewide population-based. Logistic regression	Maternal age, race and educational attainment, participation in prenatal care and self-reported smoking. Also, mean apparent temperature.	CO: 1.20 and 1.17 of peaks (> mean+IQR) for lag-2 days and lag-3 days, respectively.
De Franco et. al. (2015)	State of Ohio, USA. 2006 - 2010	PM _{2.5} Nearest monitoring station to mother's address (10Km) using 57 monitoring stations.	Trimesters and entire pregnancy	PM _{2.5} : 13.3 (1.8)	0.53	Statewide population-based. Logistic regression.	Maternal age, race, educational attainment, prenatal care and season of conception.	PM _{2.5} : 1.42 of peaks (> mean+IQR) during Third trimester.
Green et. al. (2015)	State of California, USA. Jan 1 st 1999 – Dec 31 st 2009	PM _{2.5} , CO (also SO ₂ , NO ₂ & O ₃) Nearest monitoring station to the centroid of mother's postal-code. 20Km and 5Km for PM _{2.5} and CO, respectively.	Trimesters and entire pregnancy	PM _{2.5} : 15.2 (5.1) CO: 1.29 (0.67)	0.46	Statewide population-based. Logistic regression.	Maternal education, race/ethnicity and age. Season and year of conception, sex of the infant/fetus, relative humidity, and air basin.	PM _{2.5} : 1.06 of peaks (> mean+IQR) for entire pregnancy (significant at 90%)

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