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**Bruna Guidetti**
*University of Michigan*

**Edson Severnini**
*Carnegie Mellon University and IZA*

**Paula Pereda**
*University of Sao Paulo*

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ISSN: 2365-9793

**IZA – Institute of Labor Economics**

Schaumburg-Lippe-Straße 5–9
53113 Bonn, Germany

Phone: +49-228-3894-0
Email: publications@iza.org

www.iza.org

# ABSTRACT

# Health Shocks under Hospital Capacity Constraint: Evidence from Air Pollution in Sao Paulo, Brazil*

When a health shock hits a location, the healthcare infrastructure needs to be adjusted to meet the increased demand. This may be a challenge in developing countries because of limited hospital capacity. In this study, we examine the consequences of health shocks induced by air pollution in a megacity in the developing world: Sao Paulo, Brazil. Using daily data from 2015-2017, and an instrumental variable approach based on wind speed, we provide evidence that exposure to particulate matter (PM10) causes an increase in pediatric hospitalizations for respiratory diseases, which in turn leads to a decrease in hospital admissions for elective care – phimosis surgery and epilepsy-related procedures such as video-EEG (electroencephalograph) monitoring. Importantly, emergency procedures such as appendectomy and bone fracture repair are not affected. While strained Sao Paulo hospitals seem to absorb the increased demand induced by poor air quality, our results imply that the common practice of using health outcomes unrelated to pollution as "placebo tests" in studies on the effects of air pollution might be inadequate in settings with limited healthcare infrastructure. This is often the case in developing countries, where severe pollution is also ubiquitous, but also happens in deprived areas in the developed world.

**JEL Classification:** I15, Q53, Q56, O13

**Keywords:** air pollution, health outcomes, hospitalization for respiratory diseases and other causes, healthcare infrastructure, hospital capacity constraint

**Corresponding author:**
Edson Severnini
Heinz College
Carnegie Mellon University
4800 Forbes Avenue
Pittsburgh, PA, 15213
USA

E-mail: edsons@andrew.cmu.edu

* We are grateful for the invaluable comments by Ariaster Chimeli, Tatyana Deryugina, Josh Lewis, Nick Kuminoff, Matt Kotchen, Rudi Rocha, seminar participants at the University of Sao Paulo, and Yale University, and conference participants at the Annual Meetings of the Brazilian Econometric Society, and the Midwest Energy Fest, an annual workshop organized by the University of Chicago, Northwestern University, and University of Illinois Urbana-Champaign. The authors gratefully acknowledge financial support from the Heinz College at Carnegie Mellon University.

# 1 Introduction

The coronavirus pandemic has made it clear that when a health shock hits a location, all available healthcare infrastructure needs to be adjusted to meet the increased demand. Many hospitals have canceled or rescheduled elective and non-urgent procedures, for instance, to prepare for the influx of patients with coronavirus. While pandemics are rare, health shocks driven by air pollution are ubiquitous around the world, particularly in developing countries. There is a preponderance of evidence that air pollution is harmful to health (e.g., Chay and Greenstone, 2003; Currie and Neidell, 2005; Currie and Walker, 2011; Schlenker and Walker, 2016; Deschenes et al., 2017; Severnini, 2017; Deryugina et al., 2019). Short-term exposure has been associated with increased respiratory illness and duration of symptoms, exacerbation of asthma, and decline in lung function, among other outcomes (e.g., Pope et al., 1995). Developed countries seem to manage increased demand for health care relatively well in normal times (e.g., Giuntella et al., 2018). However, because of limited healthcare infrastructure, it is an open question whether developing countries such as Brazil can absorb increased demand during health shocks, such as those caused by poor air quality.

Figure 1 shows hospital beds and physicians per 1,000 population for the main developed and developing nations. These are two key measures of healthcare infrastructure. The number of physicians in Brazil seems to be adequate, since it is above the World Health Organization (WHO) recommendation of one physician per 1,000 inhabitants, but is lower than in France and Germany. Nevertheless, the number of hospital beds per 1,000 population is only larger than in India. Thus, response to pollution-driven health shocks may be challenging in developing countries, because it may overwhelm the healthcare system, preventing hospitalizations for other serious causes, and leading to the rescheduling of elective and non-urgent procedures. In this study, we examine how a large metropolitan area in Brazil copes with increased healthcare demand due to high levels of air pollution, under hospital capacity constraint.

In order to estimate the health effects of air pollution, we investigate how daily pediatric hospitalizations for respiratory diseases respond to short-term exposure to PM10 – particulate matter 10 micrometers or less in diameter – in the Sao Paulo Metropolitan Area (SPMA) over the period 2015-2017. The SPMA is among the ten largest metropolitan areas in the world, and the main urban agglomeration in South America. Among large cities, the level of PM10 in Sao Paulo is comparable to the levels in Mexico City and Istanbul, slightly less polluted than Johannesburg, and much less polluted than Delhi and Beijing.[1] However, the SPMA is more polluted than the metropolitan areas of London, Los Angeles, and New York City, as Figure 2 reveals. Although we look at recent data, our setting is comparable to developed countries decades ago, as well as China and India years ahead (Hanlon and Tian, 2015; Clay et al., 2016). The main sources of pollution in Sao Paulo are vehicle emissions, similar to urban areas in the United States (Currie and Walker, 2011; Marcus, 2017; Anderson, 2019; He et al., 2019).

Because pollutants are obviously not randomly assigned to individuals, we need to address the endogenous exposure to air pollution when identifying its effects on health outcomes (Currie et al., 2014; Dominici et al., 2014). To overcome such an endogeneity problem, we exploit an instrument capable of dealing with non-stationary sources: wind speed. This instrumental variable approach is similar to Deryugina et al. (2019).[2] The idea is that wind speed dissi-

[1] To be precise, we are comparing levels for the whole metropolitan region of Sao Paulo with levels for Mexico City, Istanbul, Johannesburg, Delhi and Beijing without taking into account their metropolitan area.

[2] Their innovative approach exploiting changes in wind direction does not require understanding the detailed layout of an area, or identifying the sources of air pollution. We show that our results are similar if we use both wind speed and direction as instruments. Other studies leveraging wind-related variables to identify effects of air pollution are Herrnstadt and Muehlegger (2015), Schlenker and Walker (2016), Barwick et al. (2018), Anderson (2019), and Rangel and Vogl (2019).

pates particle pollution, reducing PM10 concentration, but does not affect health directly.[3] We classify respiratory diseases into asthma, pneumonia, and influenza, in order to check whether the effects vary according to chronic versus infectious diseases. Usually, researchers would look at hospitalization for conditions unrelated to pollution as placebo outcomes. In our setting, those are phimosis surgery,[4] epilepsy-related procedures such as video-EEG (electroencephalograph) monitoring, appendectomy, and bone fracture repair; the first two are considered elective care procedures, while the last two are urgent procedures. We argue that in a context with hospital capacity constraint, hospitalizations for causes usually considered in "placebo tests" should be seen as outcome variables when evaluating the health impacts of air pollution. Because of strained hospitals, those procedures might be canceled or rescheduled, therefore indirectly affected by the health shocks caused by air pollution.

When it comes to data, the Environmental Protection Agency of the State of Sao Paulo (CETESB) tracks pollutants and meteorological variables hourly, using several monitors across the SPMA.[5] This is the same data used by Salvo and Geiger (2014), Salvo and Wang (2017), and He et al. (2019). The health data are collected and organized by the the Brazilian Hospital Data System (SIHSUS), which allow us to observe daily hospitalizations for respiratory diseases and other causes by zip code of residence. We perform our estimation in two stages using SPMA districts no further than 5 kilometers from any monitor as the unit of observation.[6] We link these districts to the nearest CETESB monitor, and calculate hospitalization rates per one million children for each day due to the diseases mentioned before as dependent variables. We focus our analysis on children up to five years old because they are more sensitive to air pollution, and usually more exposed due to more outdoor activities than other age groups (Currie et al., 2014). As it is well-known, air pollution exposure history for adults and the elderly is difficult to determine.[7]

Regarding our findings on the health shocks of air pollution, we first report that our first stage results indicate that wind speed is strongly negatively correlated to PM10, suggesting that the local air quality improves when heavy winds blow. Results for the second stage confirm that PM10 is harmful for health, consistent with previous studies, and does create a health shock in the SPMA. In fact, hospitalization rates for respiratory diseases rise when exposure to air pollution increases for children aged one to five years. We also find suggestive evidence that the length of hospital stay might be shorter, but the estimates are not statistically significant. Hospital admissions for asthma and pneumonia for that age group follow a similar pattern as for the overall hospitalization rate for respiratory diseases. On the other hand, admissions for influenza may be driven by infants. We caution, though, that our instrument may not be ideal for this last group because young children under one year of age may get less exposed given that they stay indoors more often. OLS estimation disregarding the endogeneity of exposure to air pollution seem to underestimate the parameters of interest in our context, and robustness checks support our main findings on the health impacts of air pollution.[8]

[3] Although there is weak evidence that wind speed is positively associated with chronic obstructive pulmonary disease (Ferrari et al., 2012), no biological mechanism has been proposed to explain such a correlation. To the best of our knowledge, we are the first to provide a credible mechanism behind wind-speed effects on health.

[4] Unlike in the United States, phimosis surgery (circumcision) is not a common practice in Brazil.

[5] CETESB has been monitoring some air pollutants since 1972, but only for a few monitors. Consistent measurement of pollutants started in 1981, but only after 2008 the monitoring network became denser.

[6] Results are qualitatively similar when we use the alternative radii of 3, 7, and 10 kilometers.

[7] Observe, however, that the percentage of the population ages 65 and above is usually much smaller in developing than in developed countries. For the countries mentioned in Figure 1, they are: Brazil 9%, China 11%, France 20%, Germany 21%, India 6%, South Africa 5%, United Kingdom 18%, and United States 16%.

[8] Among our robustness checks, we find that the contemporaneous model of pollution exposure may hide interesting patterns in parental behavior. Parents seem to delay visits to a doctor/hospital for children aged one to five years suffering the consequences of pollution exposure. In addition, because high ambient ozone concentration is also a problem in the SPMA (Salvo and Geiger, 2014; CETESB, 2016; Salvo and Wang, 2017; Salvo

Although strained, the SPMA healthcare system seems to absorb the increased demand caused by air pollution relatively well, but generates indirect effects on hospitalization for diseases unrelated to pollution. In Brazil, there is an universal, publicly-funded healthcare system (*Sistema Único de Saúde* – SUS), but there is also a market for private health insurance, clinics, and hospitals. Nevertheless, if a patient needs to be treated in a private establishment and cannot pay for it, the federal government reimburses those providers at the SUS rate. When air pollution increases in the SPMA, the public healthcare system appears to take in most of the additional hospitalizations for respiratory diseases. Because of increased demand due to pollution, the number of planned procedures such as phimosis surgery and video-EEG monitoring decreases in public hospitals. A proportion of those cases appear to be taken in by private hospitals, especially those with higher capacity, as measured by physicians and hospital beds per 1,000 population. On average, for every four additional pollution-related admissions in public hospitals, one elective care procedure was displaced. Since appendicitis and bone fracture usually need urgent treatment, those cases do not seem to be sent away by public hospitals, but some cases are treated by private hospitals. It is worth noting that we are not evaluating the quality of care due to data limitations, and we are only examining spillover effects on a limited number of diseases to make the analysis tractable, and still provide evidence supporting the general concept.[9]

This study makes two main contributions to the literature. First, it provides a unique look at how developing countries cope with limited healthcare infrastructure, especially in times of health shocks. Previous work examined the impacts of reduced supply of healthcare facilities in the United States due to hospital and clinic closures, or violence against abortion clinics, and found that constrained healthcare capacity led to treatment delays and worsened health outcomes, particularly for time-sensitive conditions (Buchmueller et al., 2006; Jacobson and Royer, 2011; Lu and Slusky, 2016; Carroll, 2019; Gujral and Basu, 2019). Healthcare demand shocks such as the ones driven by immigration were also found to increase waiting times for outpatient referrals in more deprived areas in the United Kingdom (Giuntella et al., 2018). In the SPMA, public hospitals seem to have absorbed the increased demand in days of relatively poor air quality by delaying elective and non-urgent care, or transferring patients to (publicly-reimbursed) private hospitals. Given the ubiquity of high levels of air pollution and limited healthcare infrastructure in the developing world, our findings are reassuring, but call for more research in other contexts.

The second contribution regards the implications of the indirect effects of air pollution on the treatment of health conditions unrelated to pollution. We provide evidence that air pollution caused not only hospitalization for respiratory diseases, but also led to the reallocation of elective and non-urgent care to other times, and from public to (publicly-reimbursed) private hospitals. Therefore, the common practice of using health outcomes unrelated to pollution as "placebo tests" in studies on the effects of air pollution might be inadequate in settings where there is limited healthcare infrastructure.[10] This is often the case in developing countries, where severe pollution is also ubiquitous, but also happens in deprived areas in the developed world.

et al., 2017), we check the robustness of our results using a multi-pollutant model including PM10 and ambient ozone, exploiting solar radiation as an additional instrumental variable. The PM effects on hospitalization rates in the multi-pollutant model are qualitatively similar to the ones in the single pollutant model, and we do not find a significant impact of exposure to ambient ozone. This is an interesting result because previous work did find an effect of exposure to ozone in a single-pollutant model (e.g., Neidell, 2009; Moretti and Neidell, 2011).

[9]This is related to the situation in the United States during this coronavirus pandemic, as discussed anecdotally by the New York Times: https://www.nytimes.com/2020/04/20/health/treatment-delays-coronavirus.html.

[10]Because the bias in placebo hospitalization regressions is negative in the presence of crowd-out, the results of those tests would be skewed in favor of finding no positive effects on outcomes unrelated to pollution. Therefore, in that context, even a zero effect could actually be evidence of a faulty research design.

This paper also adds to the literature comparing the impacts of air pollution in developed and developing countries. Air pollution effects may differ in those two different contexts because of a potential nonlinear dose-response function, or because of higher costs of avoidance behavior and lower willingness to pay in poorer countries due to low income levels (Greenstone and Jack, 2015; Arceo et al., 2016). Our estimates are on the same order of magnitude as those found by He et al. (2019) regarding the effects of NOx from diesel exhaust on hospitalizations for respiratory diseases in Sao Paulo, but not directly comparable to the other health effects of air pollution estimated for developing countries (e.g., Jayachandran, 2009; Cesur et al., 2017; Barwick et al., 2018; Rangel and Vogl, 2019). In addition, the paper contributes by providing estimates from a multi-pollutant model including particulate matter and ambient ozone, which has been considered challenging in the literature because of the usual high correlation among pollutants (e.g., Bell et al., 2007; Dominici et al., 2010). Our results for PM10 are robust to this co-variation, and we find no statistically significant effect of ambient ozone on hospitalization rates. From a policy point of view, the effects of these two pollutants are important to be investigated together because they are the main focus of environmental protection agencies around the world, among them CETESB and the U.S. EPA.

This paper is organized as follows: Section 2 sets up the background, discussing the problem of air pollution in the SPMA, the operation of public healthcare system in Brazil, and medical and epidemiological findings on the effects of pollutants on the human health. Section 3 presents the data sources, and some descriptive statistics. Section 4 explains the empirical strategy. Section 5 reports the main results, and robustness checks. Lastly, Section 6 provides some concluding remarks.

# 2 Background

## 2.1 Air pollution in Sao Paulo

The Sao Paulo Metropolitan Area (SPMA) is the largest metropolitan area in Brazil, with over 21 million people, representing approximately 10% of the Brazilian population.[11] It is among the 10 largest metropolitan areas in the world, and consists of 39 municipalities, where the country's largest industrial zone is located. In Brazil, Sao Paulo is popularly known for its traffic congestion and gray skies. Similar to most large urban areas, the region is also characterized by high building density, lack of green spaces, and poor air quality.

The two main sources of air emissions in the SPMA are automobiles and manufacturing (Braga et al., 2001).[12] While vehicle emissions are predominant in Sao Paulo city, industrial emissions dominate in the other municipalities of the metropolitan area. According to CETESB (2016), more than 7 million vehicles – automobiles, trucks, buses, and motorcycles – circulated in the region in 2015. Although the Sao Paulo vehicle fleet is not old (8.9 years, on average), CETESB (2016) draws attention to their contribution to local air pollution, especially particulate matter and ambient ozone, which usually reach maximum values in the SPMA.

Given the sources of emissions, the main air pollutants in the SPMA are particulate matter (PM), nitrogen oxides (NOx), carbon dioxide ($CO_2$), and volatile organic compounds (VOCs). Most of them are included in a group of pollutants whose "safe" thresholds for exposure are frequently updated in the WHO air quality guidelines, because of new evidence of effects on the human body even at low levels (WHO, 2016). The high emissions of NOx and VOCs favor

[11] Sao Paulo is the largest city in Brazil, with a population of over 12 million. Despite the distinction between the SPMA and Sao Paulo city, we will use the terms interchangeably.

[12] It is important to note that Brazil obtains 70% of its electricity from hydroelectric dams. That is the reason why electricity generation is not an important source of air pollution, despite other environmental problems hydroelectric development generates (Miranda et al., 2012).

ozone formation, which is an ambient pollutant not directly emitted, but rather formed by Leontief-like chemical reactions involving those two precursors in the presence of sunlight and warm temperatures (Orlando et al., 2010).

In the second half of the last century, the industrial activity in Sao Paulo was quite strong. While the city was growing, many manufacturing establishments migrated to the metropolitan area (Braga et al., 2001). Due to intense industrial activity and lack of environmental regulation, air quality deteriorated. To put in perspective, a series of newspaper articles pointed Cubatao – city in the state of Sao Paulo close to the SPMA – as the most polluted city in the world in the 1970s and 1980s.[13] Recently, industrial emissions have been under control as the main economic activity shifted from manufacturing to the service sector. On the other hand, the SPMA expanded with poor mass transit planning. Together with government incentives for car purchases, this resulted in a large fleet of private automobiles (Braga et al., 2001; Jacobi et al., 1997). According to CETESB (2016), almost all hydrocarbon and carbon monoxide emissions come from vehicles, and about a half of particulate matter comes from mobile sources, mainly associated with black carbon.[14]

Approximately 85% of the fleet in the SPMA consists of light-duty vehicles (LVDs) – cars, light trucks, and sport-utility vehicles (SUVs) (CETESB, 2016). About 60% of LDVs are flex-fuel vehicles, that is, capable of running on either gasohol or ethanol.[15] Although the latter is a biofuel, there is no consensus that flex-fuel vehicles running on ethanol generate less pollution (Niven, 2005; Coelho et al., 2006).[16] It does seem to reduce particulate matter emissions, but it may lead to an increase in ambient ozone concentrations (Salvo and Wang, 2017). On the other hand, ethanol consumption is strongly associated with the ethanol/gasoline relative price, which varies with business cycles and government interventions.

In an effort to control vehicle emissions, the National Environmental Council (CONAMA) established emission standards in 1986.[17] Partly because of these programs, carbon monoxide is no longer considered a serious concern in the SPMA. Another reason may be the agreement reached by the government and automakers in 1992 to produce only vehicles with emissions-reducing devices, such as electronic fuel injection and catalytic converters (Jacobi et al., 1997). Today, the state of Sao Paulo has the Plan for Reduction of Emissions from Stationary Sources (PREFE), created in 2014 to map emissions by subregions of the state. The city of Sao Paulo has also implemented the Vehicle Pollution Control Plan (PCPV) since 2011. Both programs aim at maintaining emission levels within the CONAMA requirements. Meanwhile, the En-

[13] Since the 1990s, the air pollution in Cubatao has been under control, as relatively weak regulations were imposed. Notwithstanding, particulate matter levels are usually close to the maximum established by the WHO, which still considers the annual exposure to PM2.5 well above the "safe" level.

[14] According to U.S. Environmental Protection Agency (EPA), black carbon is the sooty black material emitted from gas and diesel engines, coal-fired power plants, and other sources. It comprises a significant portion of particulate matter. Heavy-duty vehicles are the main sources of black carbon emissions in the SPMA.

[15] This may be a result of the Brazilian Alcohol Program (Proalcool), a government program created in 1975 in response to the 1970s oil shocks to stimulate the production of ethanol (Coelho et al., 2006).

[16] Note that we are not considering emissions from fuel production. For completeness, there are a few oil refineries in the SPMA, another source of air pollution. The state of Sao Paulo is also the largest producer of sugarcane in Brazil, the main input in ethanol production. The state has suffered from sugarcane burning during the harvest season, but the fires are not close to the SPMA, and the harvesting technology has become cleaner in the past decades.

[17] Two emissions control programs were introduced in May 1986 – PROCONVE for cars, trucks, buses and agricultural machinery, and PROMOT for motorcycles. They imposed emission limits and technological requirements for motor vehicles. In 1989, CONAMA created the National Program for Control of Air Pollution (PRONAR) in order to implement limits for the emission of air pollutants more broadly. PROCONVE was considered a successful proof of concept for the establishment of the National Program for the Control of Industrial Pollution (PRONACOP), National Air Quality Assessment Program, National Program for Inventory of Air Pollutants, and State Air Pollution Control Programs. Since then, CONAMA has been updating the emission limits for stationary sources of pollution, tightening the control by discriminating fuel sector limits, and requiring cleaner technologies.

vironmental Company of the State of Sao Paulo (CETESB) has been in charge of measuring the levels of pollutants in the air, leading an expansion in measurements throughout the state. Most of the monitors, however, are located in the SPMA.

## 2.2 The Brazilian healthcare system

In Brazil, public and private healthcare systems coexist. The Unified Healthcare System (*Sistema Único de Saúde* – SUS) was established by the 1988 federal constitution to provide universal preventive and curative care to the overall population (Paim et al., 2011). Unlike the American constitution, the Brazilian constitution expressly states in article 196 that "health is a right of all and a duty of the State and shall be guaranteed by means of social and economic policies." Yet, private care is available for those who can pay for faster access and usually better quality. The private healthcare system includes for-profit and non-profit hospitals and clinics, and a health insurance sector.

The private system can be complementary or supplementary to the public system. As complementary, purely private establishments provide services contracted by SUS, such as hospital beds and specific procedures. Also, although purely private, philanthropic hospitals allocate at least 60% of their hospitals beds to SUS and, in return, obtain exemption from federal taxes. SUS reimburses private and philanthropic hospitals by procedure, usually at standard fees below market prices, but they may agree to a higher negotiated price.[18] As supplementary, some private hospitals and clinics only provide services covered by health insurance plans or out-of-pocket expenses.

Figure 3 displays hospital beds per 1,000 population by bed type for the four largest metropolitan areas in Brazil. The smaller the share of beds in public hospitals, the larger the share of beds in philanthropic hospitals, suggesting that their relationship is more complementary than supplementary. In all four locations, the share of beds in private hospitals is in the range of a quarter to over a third. Zooming in the SPMA in Figure 4, we observe a substantial heterogeneity of hospital beds across municipalities. Only three municipalities have more than three hospital beds per 1,000 population, which is comparable to the average in the United States.

Facing the challenge of providing good-quality health care to the entire population, SUS has performed well in vaccination, and high-cost services and complex procedures such as hemodialysis and transplants, which are also used by private insured people according to Paim et al. (2011). SUS also manages the national HIV/AIDS prevention and control program, and the Popular Pharmacy Program (*Programa Farmácia Popular*), which provides subsidized medicines to treat the most common health conditions, such as diabetes, hypertension and asthma. The establishment of the Family Health Program (*Programa Saúde da Família* – PSF), with the purpose of providing primary health care through regular visits to households in order to reduce hospital demand, is also a SUS effort, which has resulted in reduction of child mortality, complications from chronic diseases, and hospitalizations that could be treated with ambulatory care (Macinko and Harris, 2015).[19]

Usually, the SUS system consists not only of hospitals, but also community health clinics called Basic Health Units (*Unidade Básica de Saúde* – UBS), and Emergency Care Units (*Unidade de Pronto Atendimento* – UPA), where regular appointments and emergencies of medium-high complexity are handled, respectively. In the city of Sao Paulo, the system also includes Ambulatory Medical Assistance (*Assistência Médica Ambulatorial* – AMA), and Specialty Medical

[18]The SUS reimbursement is similar to Medicare in the United States in that it is prospective, paying a preset amount for given procedures or diagnoses. Reimbursement is based on average hospital costs, thus not designed to cover the medical expenses of any given patient.

[19]SUS also manages other programs such as the More Physicians Program (*Programa Mais Médicos*), the Psychosocial Care Network (*Rede de Atenção Psicossocial*), and the Emergency Care Service (*Serviço de Atendimento Móvel de Urgência – SAMU*).

Ambulatory (*Ambulatório Médico de Especialidades* – AME) to meet the demand for outpatient procedures of medium complexity, and avoid overwhelming hospitals with cases in which the patient's life is not at risk. There are no AMA and AME out of the city of Sao Paulo. The Prefecture of Sao Paulo claims that public facilities, especially the Basic Health Units, are better distributed throughout the city than the private ones, although hospitals are concentrated in the center of the city (IVP, 2011).

Back to Figure 3, the SPMA has slightly fewer hospital beds per 1,000 population than the other large metropolitan areas. Thus, the available healthcare infrastructure may not be sufficient to meet Sao Paulo demands for healthcare assistance. It is possible, however, that the reduced number of beds may reflect improvements in the provision of primary care services, which may either reduce the demand for healthcare assistance in general or the demand for high complex services.

## 2.3 Air pollution and human health

In 2015, WHO organized a global consultation seeking expert opinion on the latest available evidence on the health effects of several ambient air pollutants. The goal was to contribute to the rationale behind a future update of the WHO Air Quality Guidelines (AQGs) (WHO, 2016). There was a general agreement among experts that short-term effects have proved quite significant, but sometimes with mixed evidence, while the long-term effects seem to be more robust. In addition, air pollution has been shown to affect health even at low levels. For classical pollutants – $NO_2$, $O_3$, PM, and $SO_2$ – the WHO argues that evidence of their effects on health has become stronger after 2006. Most studies find impacts on the respiratory and cardiovascular systems in the short and long term, and on lung function, lung carcinogenicity, and mortality in the long term.

The medical and economic literatures have reported more intensively on the impacts of particulate matter, especially the finer ones (e.g., Pope, 1989, 2000; Brauer, 2000; Braga et al., 2001; Chay and Greenstone, 2003; Currie and Neidell, 2005; Currie and Walker, 2011; Dominici et al., 2014; Schlenker and Walker, 2016; Deschenes et al., 2017; Deryugina et al., 2019). These particles arise from combustion from mobile or stationary sources, very common in urban centers. They can easily penetrate the tissues of the body, and increase blood coagulation, which can cause heart attack and lung problems (Brauer, 2000; Braga et al., 2001). Furthermore, constant exposure to high amounts of particulate matter in the air, more common in heavily polluted cities, increases the chance of developing chronic obstruction of airways (Churg et al., 2003). Braga et al. (2001) argue that many studies have found harmful health effects even when pollution concentrations were bellow the national air quality guidelines in Brazil, reason why the upper limit needs continuous update.

Besides affecting chronic obstructive pulmonary disease and allergic rhinitis hospitalizations, epidemiological and economic studies have linked air pollution to respiratory infectious diseases, such as influenza and pneumonia, once pollutants reach natural defenses of the lung (e.g., Kelly and Fussell, 2011; Clay et al., 2018, 2019). Moreover, Zelikoff et al. (2002) highlight a worsening of pneumonia in individuals exposed to particulate matter, beyond undermining pulmonary immune response, suggesting that pollutants can both facilitate and worsen infectious diseases. The risk and severity of respiratory tract viral infection have also been pointed out by the toxicology literature (Saravia et al., 2013).

Air pollution does not affect all individuals equally. Children and the elderly are more likely to suffer from poor air quality, because they have a more fragile immunological system. Other vulnerable groups include people with chronic diseases, such as asthma, who may have more acute episodes (Gouveia and Fletcher, 2000; Braga et al., 2001). In the long term, life expectancy in more polluted locations is significantly decreased (Pope, 2000). For children, we

should also take into consideration that both short- and long-term outcomes can be affected by pollution exposure (Currie et al., 2014; Isen et al., 2017).

Lastly, Kampa and Castanas (2008) survey studies that associate different pollutants to diseases, and conclude that all pollutants may impact the airways. Despite all efforts to determine the separate effects of each pollutant on health, WHO (2016) discuss the challenges of isolating these effects, because in most cases they are emitted simultaneously from the same source, and/or different sources emit similar pollutants. The existence of confounding effects due to high correlation between pollutants is also discussed by Bell et al. (2007) and Dominici et al. (2010).

# 3 Data

We use administrative data on hospital admissions from the Brazilian Hospital Data System (SIHSUS).[20] We observe all publicly funded or reimbursed hospital admissions by individuals' zip code of residence from January 2015 to December 2017. SIHSUS collects data on the date of admission, duration of hospitalization, total expenditure, and cause as diagnosed by a physician.[21] We consider admissions for the following respiratory diseases: pneumonia;[22] bronchitis; allergic rhinitis; asthma; pneumoconiosis due to inorganic dust; respiratory disease due to inhalation of chemical gases, fumes and vapors; respiratory failure; among others. In 2016, 87% of total hospital admissions in Brazil were funded by the public system. This percentage might be lower in Southern states where private markets are more prevalent (Paim et al., 2011).

We restrict our sample to hospitalizations of children aged one to five years. Adults and the elderly were not considered in the main analysis because we do not observe their pollution exposure history.[23] Adults might spend many hours at work and we do not observe their work location, while the elderly usually spend many hours indoors. On the other hand, children might spend more time engaged in outdoor activities and, therefore, might experience more intense air pollution exposure. Furthermore, health problems in children may have long term effects (Fletcher et al., 2010; Currie et al., 2014; Isen et al., 2017). We also exclude children under one as they might be less exposed to external agents, such as pollution, virus, and bacteria, because they spend more time indoors at home. In that sense, unexpected PM variation due to wind speed may be less relevant for infants.

We focus the analysis on individuals living in the Sao Paulo Metropolitan Area (SPMA) due to the availability of environmental data. The Environmental Company of the State of Sao Paulo (CETESB) collects hourly air pollution and weather variables using 30 monitors throughout the SPMA. Not every pollutant is measured by every monitor. Our pollution variable is particulate matter with less than 10 micrometers of diameter (PM10, in $\mu g/m^3$), because it is regularly collected by most of the monitors (24 monitors).[24] PM10 levels are high in the SPMA and highly correlated with PM2.5, as it contains PM2.5. PM2.5 data have too many missings in our data. The weather variables we use are relative humidity (in percentage), temperature (in degrees Celsius), wind speed (in m/s), and global solar radiation (in $W/m^2$).[25]

[20] As mentioned earlier, SUS is the Portuguese acronym for "Health Unified System," the Brazilian public healthcare system. SIHSUS is one of the several data systems of the Ministry of Health – named DATASUS – that provide data on health outcomes in Brazil.

[21] Causes are classified according to the International Statistical Classification of Disease and Related Health Problems – Tenth Revision (ICD-10).

[22] The ICD code for pneumonia is "pneumonia by unspecified microorganism," which is the most recurrent code for pneumonia.

[23] Nevertheless, we present estimates for all other age groups in the appendix.

[24] CETESB also collects data on $SO_2$, NO, $NO_2$, $NO_X$, CO, $O_3$, and a few volatile organic compounds (VOCs).

[25] Other meteorological variables collected are wind direction and air pressure. Later on, we perform robustness

The daily pollution and weather variables are the average of the day.[26] We spatially interpolate meteorological variables to get complete weather information for all monitors that measure PM10, by using an inverse distance criterion.[27] Due to evidence on the non-randomness of monitor siting, we do not spatially interpolate pollution data (Muller and Ruud, 2018). Because our data provider – CETESB – is an environmental inspection agency, and there are thresholds established for pollution monitoring, our panel is unbalanced due to missing pollution data.

Lastly, we match individuals' zip code of residence from the health data with the corresponding district in the SPMA. Environmental data are assigned to a district from the nearest monitor, limiting to a 5-kilometer radius. Figure 5 displays the location of the monitors, and the districts within the 5km radius.[28] Daily hospital admissions are expressed as hospitalization rates per one million children, and the length of stay is measured in days.[29] Our final dataset includes data from 85 SPMA districts.

Figure 6 displays the daily mean and maximum level of PM10 by monitor. The small numbers above the red line represent the number of days with average concentration above the World Health Organization (WHO) guidelines between 2015 and 2017 (WHO, 2016). PM levels draw attention because they are above the recommendation for almost all monitors.

Monitor location and district hospitalization rates for respiratory disease by quartile can be seen in Appendix Figure A1.[30] The darker the district, the larger the hospitalization rate. The relationship between hospitalization rate and air pollution is not directly seen in the map, although we can see that almost all western districts belong to higher quartiles. One of the two biggest airports in the SPMA is located near monitor 5, and the other is between monitors 6 and 7. Note that the main sources of pollutants are vehicles. One can infer that by examining Appendix Figure A2, which shows expressways and highways in the SPMA.

In general, the length of stay for pediatric hospitalizations due to respiratory diseases is relatively short. Appendix Figure A3 presents the frequency of length of stay, measured in days. For completeness, admissions for pneumonia represent 51.5% of all respiratory admissions. Asthma, a respiratory chronic disease, represents 10.25% of that total. Influenza hospitalizations are less common, 0.35% of all respiratory admissions, even considering the epidemic of 2016. The Brazilian government promotes influenza vaccination every year before winter (June in the southern hemisphere), and children are among the priority group.[31]

# 4 Empirical Strategy

To credibly identify the causal effects of exposure to air pollution on pediatric hospitalization, we must address several potential endogeneity issues. First, exposure to pollution is obviously not randomly assigned across locations. Although there is some evidence that individuals might not observe and/or react to some air pollutants (e.g., Currie et al., 2015), there

checks using wind direction variable as well.

[26] We exclude all days with less than twelve hours of data. We fill the missing hourly data by weighting the first hour before and after by 1-$\frac{|h_i - h_m|}{h_a - h_b}$, $i = a, b$, where $h_a$ is the hour after, $h_b$ is the hour before, and $h_m$ is the hour missing data. For example, if we have no information for 3PM and 4PM, but we have for 2PM and 5PM, we impute the value for 3PM by weighting the data for 2PM by $\frac{2}{3}$ and 5PM by $\frac{1}{3}$. On the same way, we weight 2PM by $\frac{1}{3}$ and 5PM by $\frac{2}{3}$ to fill the values of 4PM. For the missing days, we follow the same weighted scheme, but replacing hours with days.

[27] Previous studies have used inverse distance for similar purposes (Neidell, 2004; Schlenker and Walker, 2016).

[28] We perform robustness checks for other radii in the results section.

[29] Population data by age group are taken from the 2010 Population Census, conducted by the Brazilian Institute of Geography and Statistics (IBGE).

[30] The charts only have monitors that measure the respective pollutant, while the map has all monitors.

[31] Influenza vaccines are free of charge, and the entire population can be vaccinated after the priority group. Notwithstanding, the government does not always reach the vaccination targets for the priority groups.

is compelling evidence that individuals avoid outdoor activities during polluted days (Neidell, 2004, 2009; Moretti and Neidell, 2011), and remediate the consequences of exposure (Deschenes et al., 2017). Thus, disregarding defensive investments would induce *downward bias* in the estimated impacts of air pollution on hospital admissions. Second, the level of economic activity across districts is not easily observed, but it is likely positively associated with air pollution (Chay and Greenstone, 2003). Because there is strong evidence that health improves during recessions (Ruhm, 2000), failing to control for the state of the economy in the district would generate a *upward bias* on the air pollution effects on hospitalization. Third, because exposure to air pollution is not measured exactly where individuals live, but rather approximated by measurements in the closest available monitors, we introduce an unavoidable measurement error. To the extent that this is classical measurement error, there would be *attenuation bias* in the estimates of interest.[32]

To deal with these endogeneity issues, we propose an instrumental variable approach. Because vehicles are the main sources of air emissions in the SPMA,[33] we need an instrument capable of dealing with non-stationary sources of pollution. In that sense, we exploit wind speed as an instrument for air pollution à la Deryugina et al. (2019). Wind variables have been used as an important source of plausibly exogenous variation in air pollution in other recent studies (Allen et al., 2013; Herrnstadt and Muehlegger, 2015; Schlenker and Walker, 2016; Anderson, 2019). A key innovation of Deryugina et al. (2019) relative to previous quasi-experimental designs exploiting wind variation is that their approach does not require understanding the detailed layout of an area (e.g., locations of roads, rivers, and population centers) or identifying the sources of air pollution. All we need to know in our context is that wind speed directly influences the distribution and horizontal transport of air pollutants, and vertical dispersion in a region (Seaman, 2000). Regarding the exclusion restriction, wind speed does not seem to affect human health directly. As mentioned in the introduction, although there is weak evidence that wind speed is positively associated with chronic obstructive pulmonary disease (Ferrari et al., 2012), no biological mechanism has been proposed to explain such a correlation. To the best of our knowledge, we are the first to provide a credible mechanism behind wind-speed effects on human health.

To illustrate the impact of wind speed on particle pollution, Figure 7 displays daily wind speed and PM10 measurements from August 26-31, 2016. Red areas correspond to highly polluted districts on that day, while blue areas denote cleaner air. When wind speed increases (represented by the "+" sign), as for August 27, air pollution is dissipated on that day and the day after. Because pollution is fast-moving and also generated on a daily basis, mainly in the downtown areas located at the center of the figures, more wind is needed to clean the air when it accumulates. That is what happened on August 30-31.

We perform the estimation in two stages. First, we regress particulate matter on wind speed and controls. Second, we regress hospitalization rates on the fitted particular matter from the first stage, correcting the second stage standard errors. To improve the empirical model, taking advantage of pollutant persistence on time, as illustrated in Figure 7, we run an over-identified model with the wind speed on the day of hospital admission and its first lag as instruments. The regression models also include weather variables and fixed effects, according to the following equations:

$$PM_{it} = \alpha + \beta_1 WS_{it} + \beta_2 WS_{it-1} + X_{it}\pi + \mu_i + \theta_t + \nu_{it}, \quad \text{(1st stage)}$$

$$Hosp_{it} = \gamma + \delta \widehat{PM}_{it} + X_{it}\phi + \eta_i + \lambda_t + \varepsilon_{it}, \quad \text{(2nd stage)}$$

[32] This may be the case here because it is likely that the true exposure to air pollution faced by an individual is not correlated to the measurement error derived from the inverse distance approach used in our analysis. In fact, the pollution monitors seem to be distributed evenly across the SPMA, as shown in Figure 5.

[33] There are many roads in the region that can be heavily congested in peak times (see Appendix Figure A2).

where $i$ denotes districts, and $t$ calendar dates from 2015 to 2017. $PM$ is the level of PM10, and $WS$ is wind speed. $Hosp$ mainly represents the hospital admissions rate for children aged one to five years. $X$ represents time-varying controls, including a quadratic function of temperature, humidity, and their interaction. $\mu_i$ and $\eta_i$ represent district fixed effects, while $\theta_t$ and $\lambda_t$ represent time fixed effects to correct for potential seasonality and aggregate shocks (day-of-week, month-of-year, and year fixed effects). $\nu$ and $\varepsilon$ are idiosyncratic terms.

District fixed effects control for time-invariant characteristics of districts, such as socioeconomic status, topography, and other geographical features of the area. Furthermore, any urban spatial structure that may affect wind speed and pollution concentration, such as skyscrapers, is controlled by district fixed effects. In this sense, we explore variation within districts to estimate air pollution effects on pediatric hospitalizations.

As health outcomes, we use primarily pediatric hospitalization rates for all respiratory diseases per million children aged one to five years, but also consider length of stay. We also estimate the effects on specific respiratory diseases such as asthma, pneumonia, and influenza to investigate heterogeneous effects on chronic versus infectious diseases (bacterial, as pneumonia; and viral, as most influenza cases). To assess the potential for hospital admissions driven by air pollution to crowd out hospitalizations for causes unrelated to pollution, we also consider phimosis surgery, epilepsy-related procedures such as video-EEG (electroencephalograph) monitoring, appendectomy, and bone fracture repair; the first two are considered elective care procedures, while the last two are urgent procedures.

Since we include month-of-year and year fixed effects, the influenza epidemic of 2016 may also be controlled if we consider a homogeneous incidence across the SPMA.[34] In regards to vaccination campaigns, a possible occurrence of herd immunity is a limitation for estimating impacts of air pollution on influenza-related hospital admissions, but probably not on hospitalization for all respiratory diseases due to the low influenza incidence. There is also free-of-charge vaccine for bacterial pneumonia in Brazil, but it is not recommended for children under 2 years old. As this vaccine does not protect for all types of pneumonia, and it is not completely effective, we believe it is not a major concern for our identification strategy.

We now describe an augmented model used to assess the effects of the baseline hospital capacity on the PM-hospitalization relationship for non-respiratory causes. The idea is to evaluate the potential for the availability of healthcare infrastructure to reduce the crowd-out effect of air pollution on hospitalizations for causes unrelated to pollution. The second stage equation is augmented by adding an interaction of the PM variable with a baseline measure of hospital capacity. There are now two (unreported) first stage equations: one for PM, and another for the interaction term. The additional instruments are the original instruments – contemporaneous wind speed and its first lag – interacted with the baseline measure of hospital capacity. The second stage becomes

$$Hosp_{it} = \gamma + \delta_1 \widehat{PM}_{it} + \delta_2 \widehat{(PM_{it} \times 1[HCap]_i)} + X_{it}\phi + \eta_i + \lambda_t + \varepsilon_{it}. \qquad \text{(HCap 2nd stage)}$$

This second stage equation is identical to main one, except for the addition of the interaction variable between PM and the hospital capacity modifier $1[HCap]_i$. We explore the heterogeneity in the PM effects by three district-level hospital capacity indicators – pediatric hospital beds, general hospital beds, and family doctors – all in 2014, the year before our period of analysis. Each capacity is calculated as a rate per total district population. $1[HCap]_i$ is a dummy variable indicating that the capacity rate is above the median across the districts in the Sao Paulo Metropolitan Area. We understand the cross-section variation in the modifiers is not experimental, so it is natural to question whether the estimated interaction coefficients

[34] The influenza season in southern states in Brazil also happens in the winter months, which are June and July in the southern hemisphere (Alonso et al., 2007).

are likely to be unbiased. Because the modifiers are baseline infrastructure measures, they are captured by the district fixed effects. Thus, a number of unobserved time-constant determinants of hospitalization and hospital capacity are controlled for, as discussed above.

The coefficient associated with PM should now be interpreted as the impact of PM on admissions in hospitals with the measure of capacity below the median, and the coefficient of the interaction as the differential effect on admissions in hospitals with that measure above the median. If pollution-related hospitalizations crowd out hospital admissions for elective care, for instance, then the PM coefficient $\delta_1$ will be negative. If this PM impact is partially offset by the availability of hospital beds and doctors, the coefficient on the interaction term $\delta_2$ will be positive.

# 5 Results

In this section, we present the main results followed by a heterogeneity analysis of the air pollution effects and robustness checks. All regressions are weighted by district population, and all estimated coefficients are reported along with standard errors two-way clustered at the district and calendar date levels.

## 5.1 PM effects on pediatric hospitalizations for respiratory diseases

Table 1 reports first stage results. PM10 is regressed on instrumental variables – contemporaneous and lagged wind speed – district and time fixed effects, and the other covariates described in the previous section. First stage regressions examine whether wind speed is correlated with PM10, holding the other variables constant. A valid first stage is necessary for identification. As expected, the estimated coefficients of wind speed on the same day and one day before hospital admission are both negative and statistically significant. Because wind carries pollution, the stronger the wind blows, the more particulate matter is taken away, the cleaner the air. Not surprisingly, however, wind speed on the day before admission has a lower impact on the contemporaneous levels of air pollution when compared to its impact on the same day, as shown in the first column. The Kleibergen-Paap rk Wald F-statistics of the joint significance of instruments is over 84, indicating strong instruments.[35] With respect to the results for the linear probability model in the second column, the coefficients are on the same order of magnitude. Wind speed both on the day of admission and the day before seem to contribute similarly to the accumulation of local air pollutants, and consequently to the probability of crossing the PM10 threshold recommended by the WHO guidelines. The Kleibergen-Paap rk Wald F-statistics of the joint significance of instruments still indicates relatively strong instruments, but it is less than a third of the corresponding statistic in the regression in the first column using PM10 levels. We focus our analysis on the specification in levels.

Table 2 presents the second stage estimates of the PM10 effects on hospitalization rates and length of stay. Because each unit of PM10 in the regressions represents $10\mu g/m^3$ in this specification, all marginal effects should be interpreted as arising from a variation of $10\mu g/m^3$ in PM10 levels. The comparison between OLS and 2SLS estimates indicates the direction of the bias. Despite a few positive and significant OLS results, OLS estimates are much lower than 2SLS estimates, revealing a negative bias likely due to attenuation bias (e.g., pollution assigned to districts based on measurements in nearby monitors) and/or omitted variable bias (e.g, avoidance behavior, as children might have their outdoor activities interrupted in the most polluted days).

[35] In the robustness checks, we also include indicator variables for quadrants of wind direction. Results are discussed later on, but wind speed estimates are robust to the inclusion of wind direction.

We find significant impact of exposure to particulate matter on hospital admissions for all respiratory diseases, and asthma and pneumonia, in particular. As explained in the background section, exposure to a more polluted environment leads to a decline in children's immunity, favoring and/or worsening bacterial infection. Similarly, for chronic diseases such as asthma, exposure to poor air quality may increase the probability of children experiencing asthma attacks.

We do not find any significant effects of PM on the duration of hospitalization. This null effect on length of stay might indicate that respiratory diseases are not as severe as other diseases that cause pediatric hospitalizations. It is worth noting that hospital admissions for milder conditions may be underestimated, because we cannot control for the fact that individuals may not visit hospitals when the disease symptoms are not so strong, or if they believe it will take too long to see a doctor.[36]

Our results indicate that a $10\mu g/m^3$ increase in PM10 causes an increase in pediatric hospitalization rates for all respiratory diseases by 4.83 per million children aged one to five years. Given that the average hospitalization rate in that category is 67.63 per million children, that effect represents an increase of 7.1 percent. If we consider the number of children aged one to five years in the whole SPMA, the number of hospital admissions rises by approximately 6.4 children a day. If we do the same exercise for asthma and pneumonia, we find about 1.5 additional pediatric admissions for asthma a day, and 3.12 for pneumonia.

Back to the number of 6.4 additional children hospitalized for respiratory diseases per day, if we consider the average public cost of one day of hospitalization of \$61.58 (2016 USD),[37] additional expenditure would be \$394.11 per day. Considering that the median hospitalization in our data lasts 4 days, for each 6.4 children, the additional hospitalization costs are \$1,576.45 per day. In a year, the government expenditure would increase by \$575,403.52. This value would still be a lower bound estimate of the additional government spending in the SPMA, since we do not consider other health costs related to air pollution, and the average public expenditure does not reflect market prices in Brazil.[38] Nevertheless, this value represents 4.5% of all 2016 public expenditures with hospitalizations of children aged one to five years in the city of Sao Paulo, and approximately 12.5% of expenditures with hospital admissions due to respiratory diseases for this age group.

There is a preponderance of evidence in the economic literature that air pollution is harmful to health, but only a limited number of studies have examined the impacts of exposure to particulate matter on pediatric hospitalization rates. Most studies have looked at effects on infant mortality (e.g., Chay and Greenstone, 2003; Currie and Neidell, 2005; Jayachandran, 2009; Clay et al., 2016; Arceo et al., 2016). Neidell (2004) does look at asthma admissions for children, but finds no effect of PM. Although we investigate a different pollutant, we find a close proportion for asthma admissions as in Schlenker and Walker (2016). While those authors find effects representing 21% of average daily rates for all ages, we find 23.4% for children, considering an increase of one standard deviation in pollution. Also, Neidell (2004) and Jayachandran (2009) highlight stronger air pollution effects on infants from families of low socioeconomic status. We cannot examine the heterogeneity of effects according to different income levels due to data limitations,[39] but our data refer to admissions in public hospitals or in publicly-reimbursed philanthropic and private hospitals, therefore the focus is indirectly on hospital admissions of the lower-income population.

Finally, we investigate a potential nonlinearity of the impacts of air pollution by estimat-

[36]Second stage results are also robust to the inclusion of wind direction variables as additional instruments.

[37]We convert the R\$214.88 – the average in January 2016 (DATASUS) – in U.S. dollars using the average exchange rate of US\$1 = R\$3.4895.

[38]The costs used in our calculation are based on SUS reimbursements, which are far bellow market prices.

[39]We have no socioeconomic data at the level of the patients, only at the neighborhood level.

ing the PM effects on respiratory diseases in highly polluted days – pollution above the WHO recommended level of $50\mu g/m^3$ for a 24-hour mean. Table 3 shows that when a day is above the WHO guidelines, there are about 44 additional pediatric hospitalizations per million children aged 1-5 per day for all respiratory diseases, 11 for asthma, and 22 for pneumonia. These effects are much stronger than the ones reported in Table 2, given that the average daily PM10 is about $30\mu g/m^3$. Although only suggestive, this is a surprising result because Pope et al. (2015) find that most recent evidence indicates that the concentration-response function between PM2.5 air pollution and mortality risk may be supralinear (concave) across wide ranges of exposure.

## 5.2 PM (indirect) effects on the healthcare system

In Brazil, it is widely known that the public healthcare system has excess demand. One relevant question we ask is: what are the effects of a pollution-driven health shock on hospital demand? To answer this question, we propose two exercises. First, we run our main analysis separately for public hospitals and (publicly-reimbursed) private hospitals.[40] Results are reported in Table 4. Second, and most importantly, we examine potential impacts on hospital admissions for causes unrelated to air pollution. We keep the focus on children, because hospital beds for children are separated from all other age groups. If the number of hospital beds is not high enough to meet demand, what is supposed to be a placebo test becomes an outcome variable of interest. Results are reported in Table 5.

Table 4 suggests that our main estimates are driven by the response of the public healthcare system, since public hospitals seem to be absorbing admissions for all the respiratory diseases examined in this study. Therefore, public hospitals may be dealing with all the additional demand due to air pollution. It is important to acknowledge that although increasingly strained, public health services in Sao Paulo are still a reference for the whole country. For the record, almost 35% of hospital beds in the SPMA are in public hospitals, and more than 55% of total beds are publicly funded.[41] When it comes to pediatric beds, 61% of them are public.

Another feature that may be underlying our results is that some hospitals are specialized in specific procedures, which would explain the fact that we do not have a sample of pediatric hospital admissions in private hospitals large enough to carry out the analysis. Moreover, we are only able to estimate the effects for a few respiratory diseases. Investigation of the effects on other diseases would be required to rule out any other negative externality.

That said, we now examine the effects on pediatric hospitalization for the following other causes: phimosis surgery,[42] epilepsy-related procedures such as video-EEG (electroencephalograph) monitoring, appendectomy, and bone fracture repair. The results reported in Table 5 show negative and statistically significant impacts of PM exposure on hospital admissions for elective care procedures, such as phimosis and epilepsy, but only in public hospitals. On average, for every four additional admissions for respiratory diseases in public hospitals, one elective care procedure was displaced. Admissions for appendectomy and bone fracture repair, however, do not appear to be affected by higher levels of PM, probably due to their urgent nature. Nonetheless, we also find a positive effect of PM on appendectomy in private hospitals of about the same magnitude (but opposite signs) as the corresponding effects in public hospitals. This suggests that the public system needs to utilize beds out of its system to meet

[40] The public healthcare system owns hospitals, but also uses hospital beds in philanthropic health centers. Government only pays for those beds if they are used. As mentioned earlier, a private hospital must allocate at least 60% of its beds for public use to be exempt from federal taxes. For completeness, public and private hospitals/health centers were classified according to National Classification Commission – CONCLA.

[41] Around 35% of hospital beds in private health centers are publicly funded, and around 60% in philanthropic hospitals (DATASUS in 2016).

[42] Again, unlike in the United States, phimosis surgery (circumcision) is not a common practice in Brazil.

the increased demand caused by poor air quality. As explained in the background section, the public healthcare system prioritizes beds in public hospitals, but does use beds in the private system when necessary.

Regarding the heterogeneity of the PM impacts by hospital capacity, Tables 6 and 7 report larger negative effects in capacity constrained hospitals, i.e., those whose infrastructure indicators are below the median. In those tables, the coefficients associated with PM should be interpreted as the impact of PM on admissions in hospitals with the measure of infrastructure below the median, and the coefficients of the interactions as the differential effects on admissions in hospitals with those measures above the median. For example, the negative impact of PM on epilepsy-related procedures in Panel A of Table 6 is partially offset when the number of pediatric beds is above the median in the SPMA. Notice that the reduction in phimosis surgeries in public hospitals is partially compensated by an increase in publicly-reimbursed private hospitals with excess capacity, as shown in Table 7.

We also analyze the effects of pollution on childbirth deliveries, as they may also compete with pediatric beds. Appendix Table A1, in line with the results we find for other non-respiratory diseases, shows that planned Cesarean surgeries (C-sections) are negatively impacted by poor air quality, while natural birth deliveries are not, as they may be considered urgent. In unreported regressions, available upon request, we also find that duration of hospitalization is not impacted in public hospitals, as it seems that the adjustment is made in admissions for elective care procedures. However, we observe a small negative effect on duration of hospitalizations caused by natural births, as these procedures are normally less risky, and delivers more mature babies than C-sections.

To sum up, the health shocks triggered by highly-polluted days in the SPMA do seem to affect the operations of both the public and the private healthcare systems. Public hospitals appear to absorb the excess demand induced by air pollution, but elective care procedures may have to be rescheduled or transferred to private and philanthropic hospitals. In any case, hospitalization for causes unrelated to air pollution are also indirectly affected by it. These findings suggest that "placebo tests" usually reported in studies examining the impacts of air pollution should be interpreted with caution. Because the bias in placebo hospitalization regressions is negative in the presence of crowd-out, the results of those tests would be skewed in favor of finding no positive effects on outcomes unrelated to pollution. Therefore, in that context, even a zero effect could actually be evidence of a faulty research design. However, if the location under investigation does suffer from hospital capacity constraint, it is likely that researchers will find impacts on medical procedures unrelated to air pollution, especially those considered elective care.[43]

## 5.3 Robustness Checks

### Air pollution dissipation and cumulative exposure

Our first robustness check examines the lagged effect of wind speed on air pollution, and the cumulative effect of pollution on pediatric hospital admissions. In practice, we add three other lags of wind speed in our first stage, and three lags of PM10 in the second stage. The first stage now includes contemporaneous wind speed ($WS_t$), as well as four lags: $WS_{t-1}$, $WS_{t-2}$, $WS_{t-3}$, and $WS_{t-4}$. Results are reported in Table 8, and reveal that contemporaneous wind speed and its immediate lag are the ones that matter for PM10 concentration at any point in time. In fact, the cumulative effects reported at the bottom of the table – the sum of all wind speed coefficients – are almost identical to the sum of the coefficients of the contemporaneous

[43] Again, this is related to the situation in the United States during this coronavirus pandemic, as discussed anecdotally by the New York Times: www.nytimes.com/2020/04/20/health/treatment-delays-coronavirus.html.

variable and its first lag. In other words, particulate matter seems to dissipate in just a few days. Notice that we also end up checking whether wind speed days ahead correlate to previous PM10 levels. Once we control for contemporaneous wind, there does not appear to be any meaningful correlation between future wind and contemporaneous pollution.

The second stage results are reported in Table 9. They reveal an interesting pattern of behavioral responses: parents seem to delay a hospital visit when their children seem to be affected by exposure to air pollution. One may assume that the exact day of admission may vary with parental concerns about a child's health. Parents may take a child to the hospital as soon as the first symptoms appear, or may delay it for a few days if they believe it would not be a serious problem. Delays would underestimate the results of the contemporaneous model, since pollution seems to affect future hospitalization. On the other hand, exposure to consecutive days of poor air quality may have a larger effect on hospitalization in the first few days of a given period, because children with a more fragile health must be taken to the hospital sooner or later. In this case, parents would be anticipating a hospital visit that would occur anyway; hence, the contemporaneous model would overestimate the true impact of air pollution. In other words, if parents delay a hospital visit, we should expect positive coefficients for lagged pollution, but if they anticipate it, then those coefficients should be negative. This temporal displacement is also discussed by Schlenker and Walker (2016). Looking at the second stage results, the sum of all PM10 coefficients is double or triple the magnitude of the contemporaneous coefficient for all the respiratory diseases considered in our analysis. The positive and significant coefficients of lagged pollution suggest that parents do delay hospital visits, so contemporaneous model estimates may be underestimated. No statistically significant effect is observed for the length of hospital stay.

**A multi-pollutant model: particulate matter and ambient ozone**

We also run a multi-pollutant model considering the impacts of exposure to both PM10 and ambient ozone on pediatric hospitalizations. Solar radiation is used as the additional instrument to obtain *ceteris paribus* plausibly exogenous variation for each pollutant. Multi-pollutant models are still a challenge in the literature due to the difficulty in isolating the effects of each pollutant (Bell et al., 2007; Dominici et al., 2010). The correlation among air pollutants is usually high. Nevertheless, PM and $O_3$ are frequently pointed as the main problem in terms of air pollution in the SPMA, and the correlation between PM and ambient ozone in our sample is only approximately 0.17. Because $O_3$ is a secondary pollutant (not directly emitted), and its formation requires a combination of NOx and VOCs in the presence of sunlight, we leverage solar radiation as an additional instrument to separately identify the impact of each pollutant.

When including an additional endogenous variable in an econometric model, we need not only as many instruments as endogenous regressors, but also one instrument particularly strong for one and not so strong for other to isolate causal effects for both endogenous variables. This instrument would be responsible for capturing plausibly exogenous variation of an endogenous variable without varying the others in any substantial way. In that sense, we keep wind speed, contemporaneous and lagged, and add solar radiation in the instruments list. The first and second stage equations become:

$$\text{Poll}_{it} = \alpha + \beta_1 WS_{it} + \beta_2 WS_{it-1} + \beta_3 SR_{it} + X_{it}\pi + \mu_i + \theta_t + \nu_{it}, \quad \text{(1st stages)}$$
$$\text{Poll}_{it} \equiv PM_{it}, O3_{it},$$
$$\text{Hosp}_{it} = \gamma + \delta_1 \widehat{PM}_{it} + \delta_2 \widehat{O3}_{it} + X_{it}\phi + \eta_i + \lambda_t + \varepsilon_{it}. \quad \text{(2nd stage)}$$

Using solar radiation as instrument for ambient requires a few considerations. First, because ozone is destroyed during the night, only contemporaneous solar radiation should matter for ambient ozone concentrations that children are exposed to when engaging in outdoor

activities. Second, part of the solar radiation is reflected by clouds, so controls related to cloud coverage are crucial. We do not observe cloud coverage per se, but our main specification already includes relative humidity, which may be a good proxy for it. Third, some geoengineering studies show that solar radiation might be affected by air pollutants, either through absorption or reflection (e.g., Proctor et al., 2018). Considering that technically the purpose of first stage is to capture a correlation rather than a causal relationship, we recognize the limitation, but still proceed with the analysis. As a matter of fact, the first stage estimates that we discuss below show no correlation between PM10 and solar radiation. Lastly, solar radiation should affect pediatric hospitalizations for respiratory diseases only via exposure to ambient ozone. Although some studies have found a correlation between solar radiation and chronic pulmonary diseases and infectious diseases (e.g., Moan et al., 2009; Ferrari et al., 2012), we believe those associations are not causal due to the usual endogeneity issues. In this study, we are actually providing a mechanism behind those correlations.[44]

Table 10 reports the first stage results. It turns out that solar radiation is poorly correlated with PM10, but strongly associated with ambient ozone concentration. The wind speed association with PM remains the same as in previous regressions, but the wind speed correlation with ambient ozone is curious. Ozone formation depends on Leontief-like chemical reactions between NOx and VOCs (see Appendix Figure A4). Some studies indicate that ozone formation in the SPMA is primarily VOC-limited, with only a few NOx-limited places in the peripheral area (e.g., Martins and Andrade, 2008; Salvo and Wang, 2017). Since almost all monitors in our sample are located in the central area of the city, it is likely that the $O_3$ formation is primarily VOC-limited in our setting. When this is the case, it is possible that levels of ozone temporarily increase when NOx concentration decreases, as discussed by Martins and Andrade (2008) and Silva Junior et al. (2009). It is the so-called "weekend effect." In the end, contemporaneous wind speed might spread air pollution across the metropolitan area, and make the levels of NOx and VOCs more homogeneous. This might induce a reduction in NOx and an increase in VOCs around the monitors, resulting in enhanced $O_3$ formation. Because ozone is destroyed during the night, wind speed one day before has no effect on contemporaneous ozone formation, but might still reduce the baseline concentrations of ozone precursors in the next day.

Results for the second stage can be seen in Table 11. Notice that the numbers of districts and observations change because the analysis is restricted to monitors that measure both pollutants. Notwithstanding, comparing the OLS and 2SLS estimates for PM10, we observe the same pattern as in the single-pollutant model previously discussed. For $O_3$, 2SLS coefficients are larger for all diseases, except for influenza, but none of them are statistically or economically significant. In addition, there does not seem to be any considerable changes in the magnitude of the 2SLS PM10 estimates relative to the single-pollutant specification, which can be interpreted as a robustness of our main results. This is a surprising finding. Previous studies using a single-pollutant model for ambient ozone have provided evidence of substantial costs due to ozone-induced hospital admissions for asthma and other respiratory diseases (e.g., Neidell, 2009; Moretti and Neidell, 2011). Furthermore, recent studies using a multi-pollutant model with PM2.5 and O3 have found an increase in mortality among the Medicare population in the United States (Di et al., 2017). Our study highlights the need to use a multi-pollutant approach, and cautions that extrapolations of the estimated impacts of air pollution to different countries and age groups may be unwise.

### Wind direction, distance cutoffs, and age groups

As mentioned earlier, we carry out a robustness check leveraging plausibly exogenous variation arising from wind direction. Following Deryugina et al. (2019), we add three quadrants

[44] If anything, exposure to sunlight has been shown to protect against influenza (Slusky and Zeckhauser, 2018).

of wind direction as instruments in the first stage. Appendix Table A2 reports first stage results, and reveals that we do have variation in PM10 coming from changes in wind direction, consistent with and Deryugina et al. (2019). Nevertheless, the coefficients on those three quadrants are only marginally significant, leading to a reduction in the Kleibergen-Paap rk Wald F-statistic of the joint significance of instruments from about 84 to 40. That is the reason why we only use wind speed in the primary specification. Regarding second stage results, Appendix Table A3 shows that they are somewhat similar to our main findings.

We also check the sensitivity of our results with respect to choosing other radii when assigning air pollution data to SPMA districts. In Appendix Table A4, we report the second stage results considering the following cutoffs for the distance between district centroids and monitors: 3, 7, and 10km (5km is what we use in our preferred specification). The significance of 2SLS estimates is only slightly different, and not surprisingly PM10 impacts on hospital admissions reduces as the distance cutoff increases. It is likely that the pollution and weather variables assigned to the furthest districts do not represent the actual measures in those locations. This evidence corroborates the pattern reported in Figure 6, and suggests that PM10 levels do vary considerably across districts in the metropolitan area.

Finally, we investigate the effects of PM on hospitalization for respiratory diseases for other age groups (Appendix Table A5). For babies younger than one year old, hospital admissions by influenza are the only significantly affected by poor air quality. Pollution exposure for infants is difficult to determine, since they spend most of the time indoors. No significant effects are found for other age groups, probably because of the difficulty to assess the history of pollution exposure for older children and adults. For these groups, exposure at school/work represents an important unobserved confounder of the pollution exposure measure used in the analysis (Currie et al., 2014).

# 6 Concluding Remarks

Because air pollution affects the health of vulnerable population groups, such as children, they may create nontrivial health shocks. These shocks may in turn generate excess demand for hospitals, potentially affecting the treatment of other conditions not related to pollution. In this study, we examined the impact of PM10 on pediatric hospitalizations in the Sao Paulo Metropolitan Area (SPMA) from 2015-2017. We dealt with the endogeneity of air pollution exposure arising from avoidance behavior and attenuation bias, for instance, using wind speed as an instrumental variable for particle pollution. Our results showed that exposure to PM10 caused a large increase in pediatric hospitalization for respiratory diseases in the short term. In particular, we found an increase in hospital admissions for acute episodes of asthma and pneumonia.

An important follow-up result was related to the capacity constraints of the healthcare system in developing countries, such as Brazil. We found that in order to absorb the excess demand for hospital beds due to pollution-related hospitalization, the Brazilian public healthcare system may have had to postpone elective care procedures during highly-polluted days, or transfer patients to the publicly-reimbursed private system. In fact, we observed a reduction in admissions in public hospitals due to phimosis surgery and epilepsy-related procedures such as video-EEG (electroencephalograph) monitoring. At the same time, there was a slight increase in the number of those procedures in private and philanthropic hospitals. Therefore, it appears that the healthcare system absorbed the additional demand without imposing large costs to the SPMA society. Because hospitals were strained, but healthcare infrastructure in Brazil is still much better than other developing countries such as India, research in other contexts is warranted. In any case, these results highlight the shortcomings of using health outcomes unrelated to air pollution as "placebo tests" in studies examining the consequences

of exposure to pollution.

In light of our findings, policymakers in developing countries should weigh the costs of maintaining a healthcare system ready to meet potentially avoidable hospital demands, or imposing stricter air quality standards. It is important to mention, however, that our results consider only hospitalization costs. In a full cost-benefit analysis, additional costs may have to be considered, such as those related to ambulatory care visits, and prescription drugs, which were not part of our analysis due to data limitation. Recall that medications for chronic diseases such as asthma can be acquired either free-of-charge or at a subsidized price through the *Popular Pharmacy Program*. In addition, analysis on patient satisfaction regarding the quality of the public health care service should be taken into account.

Another limitation of our analysis is that we do not have information for out-of-pocket and insured hospitalizations. As a consequence, the wealthier portion of the population may not be represented in our findings. Despite this limitation, we believe that the air pollution impacts on the low income population are the key ones for policymaking. Lastly, because we only had georeferenced data for hospitalization, not mortality, we were not able to examine the air pollution effects on infant deaths within the SPMA. Therefore, our findings should be interpreted as a lower bound of the effects of particulate matter in Sao Paulo.

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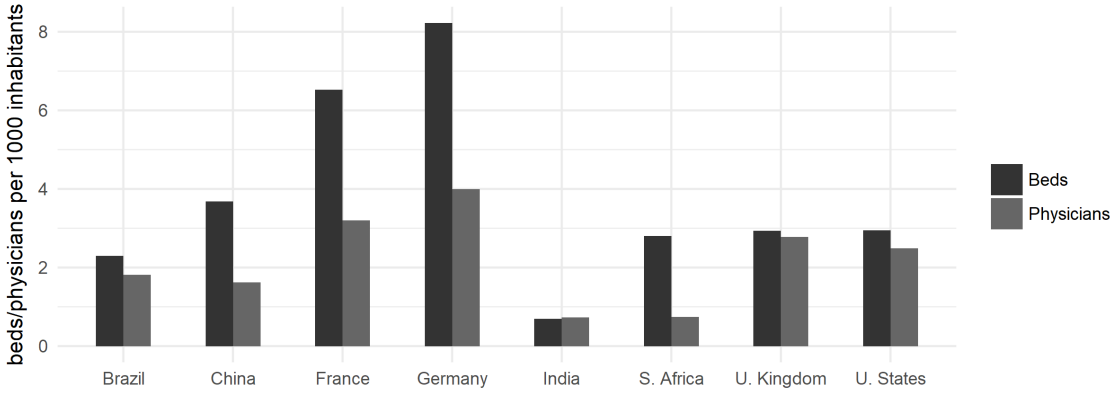
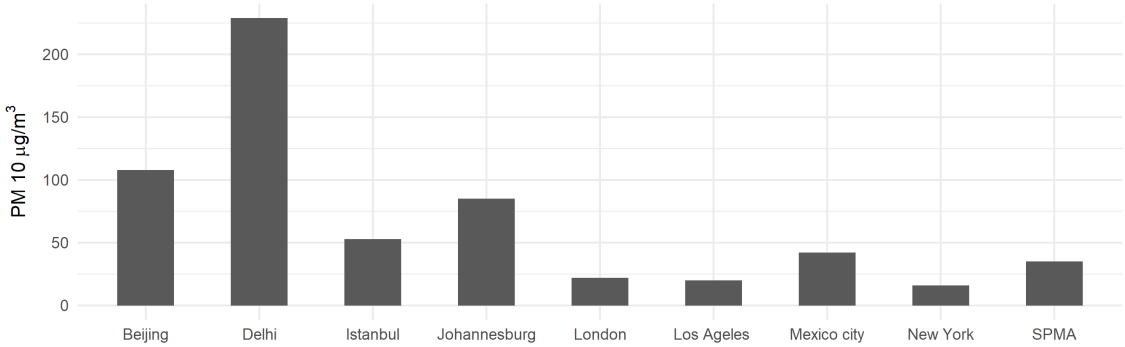
# Figures

Figure 1: Hospital beds and physicians per 1,000 population



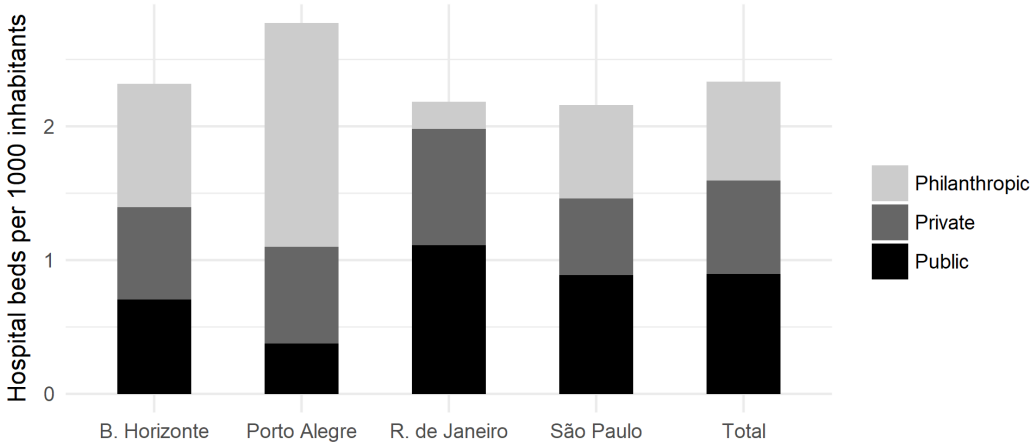

*Notes*: This figure displays the number of hospital beds and physicians per 1,000 population for a group of developing and developed countries. Reported values are averages for 2010-2012 (or available data between these years). For South Africa, the value is for 2005 (the last available). *Source*: World Health Organization.

Figure 2: Average PM10 concentration in large cities around the world



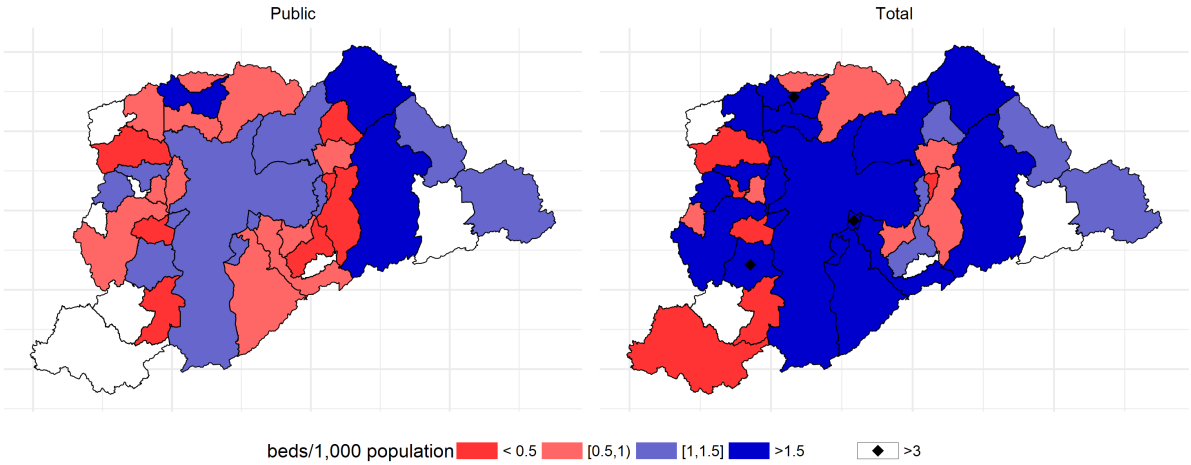

*Notes*: This figure displays the annual average of PM10 (in $\mu g/m^3$) for a group of large cities around the world. New York and Los Angeles refer to the metropolitan area. Reported values are for 2014 (Sao Paulo Metropolitan Area – SPMA, Los Angeles, New York, Mexico City), 2013 (Beijing and London), 2012 (Istanbul), and 2011 (Johannesburg). *Source*: World Health Organization.

Figure 3: Hospital beds per 1,000 population by bed type and MSA in Brazil



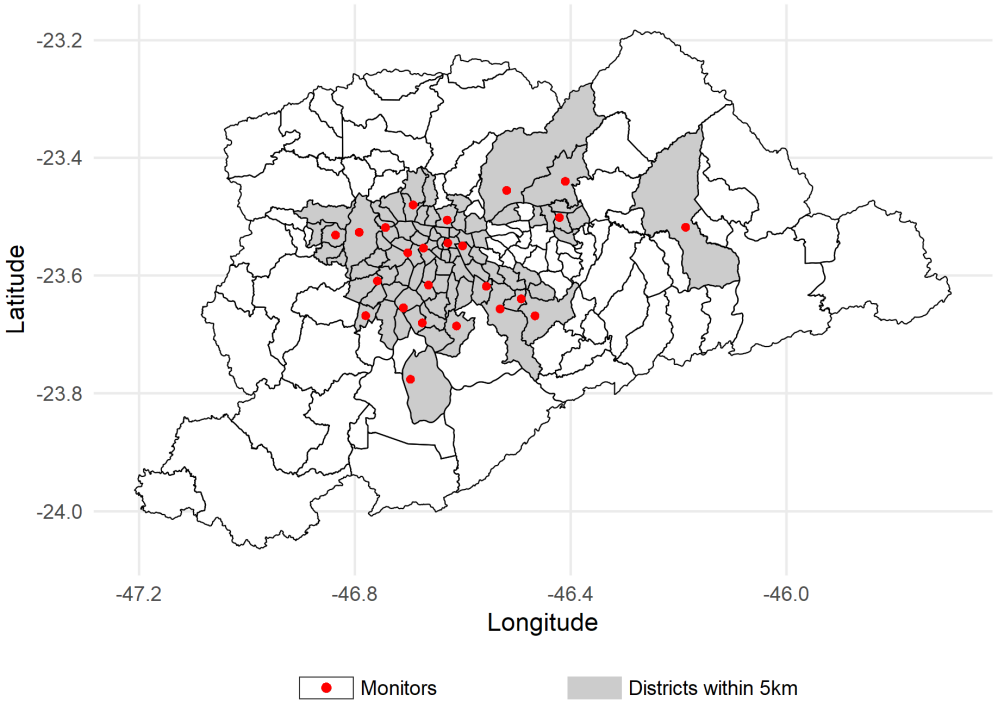

*Notes*: This figure displays the number of hospital beds per 1,000 population for the four largest metropolitan areas in Brazil. The hospital beds are classified into public, purely private, and philanthropic. *Source*: DATASUS.

Figure 4: Hospital beds per 1,000 population in Sao Paulo



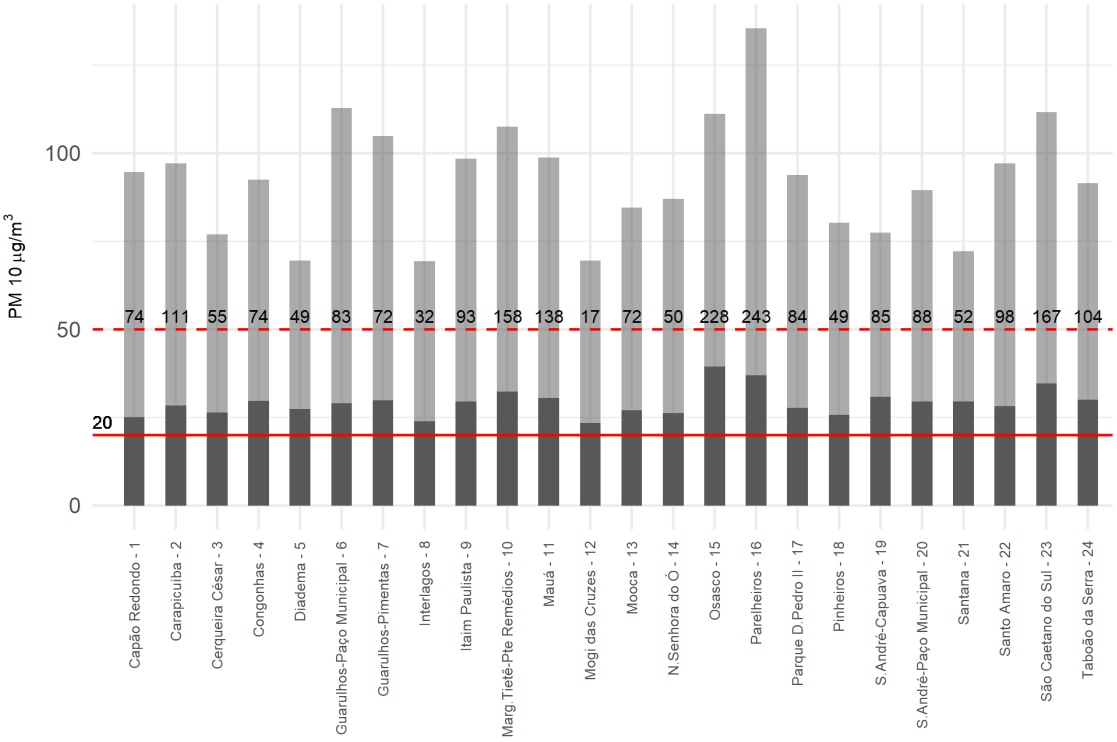

*Notes*: This figure displays the number of hospital beds per 1,000 population across municipalities in the Sao Paulo metropolitan area (SPMA). The left map shows hospital beds in public hospitals, and the right map show the total number of hospital beds, including purely private, and philanthropic hospitals.

Figure 5: Pollution monitors and districts within 5km from a monitor in Sao Paulo



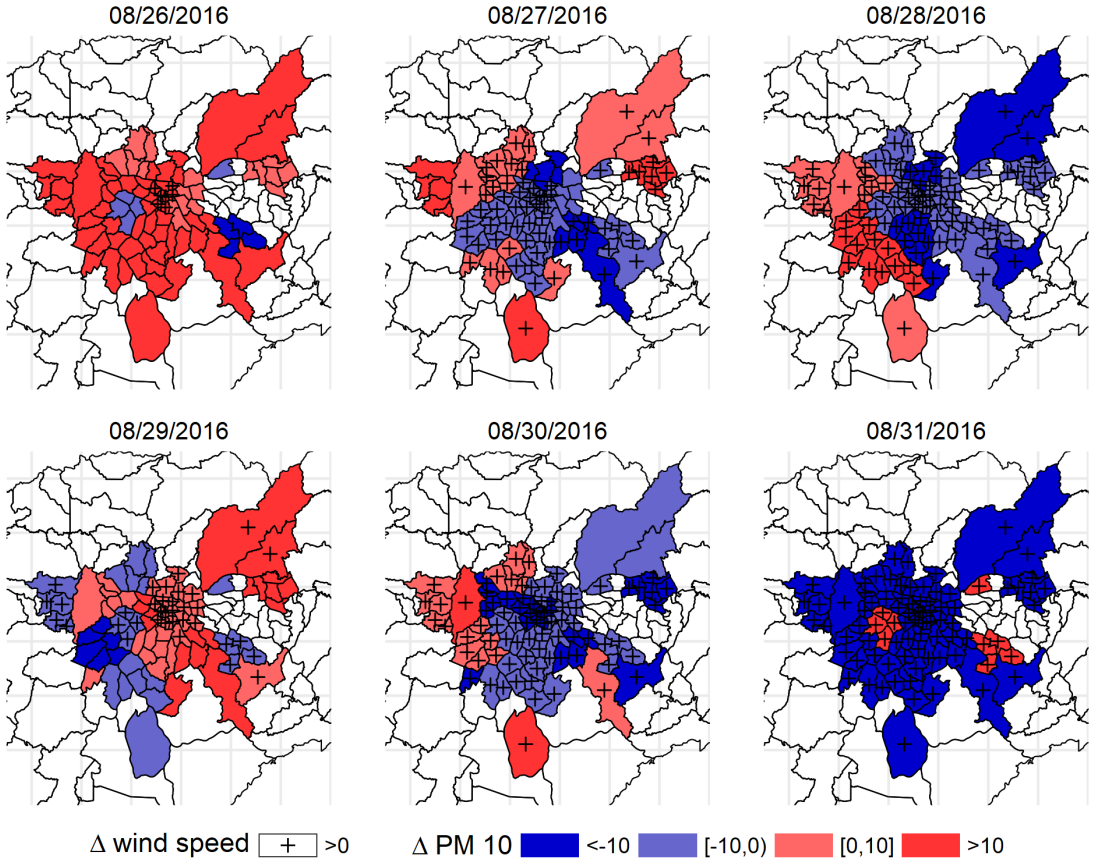

*Notes*: This figure displays the location of pollution monitors in the Sao Paulo Metropolitan Area (SPMA) (red dots), and the districts within 5km from any monitor (gray polygons).

Figure 6: Average and maximum PM10 concentration, and number of days above WHO guidelines in the SPMA




*Notes*: This figure displays the annual average of PM10 concentration (dark gray) and maximum daily averages (light gray) for each pollution monitor in the Sao Paulo Metropolitan Area (SPMA). The red horizontal lines indicate the values recommended by the WHO's guidelines: the dashed line represents the daily maximum for PM10 (maximum for short-term exposure), and the solid line the maximum annual average (maximum for long-term exposure). The numbers above the dashed line indicate the total number of days above the maximum recommended by the WHO by monitor, from 2015-2017.

Figure 7: Impact of wind speed on PM10 in Sao Paulo – First stage illustration




*Notes*: This figure illustrates the mechanism behind the first stage estimation. It represents daily PM10 and wind speed measurements from August 26-31, 2016, in the Sao Paulo Metropolitan Area (SPMA). Red areas correspond to highly polluted districts on a particular day, while blue areas denote cleaner air. The "+" sign represents increases in wind speed. As the winds blow stronger, as for example on August 27, air pollution is dissipated on that day and the day after.

# Tables

Table 1: Impacts of wind speed on PM10 and PM above WHO's guidelines – First stage

| | $PM_t$ | $1[PM_t > 50\mu g/m^3]$ |
|---|---|---|
| $WS_t$ | -0.68*** | -0.06*** |
| | (0.056) | (0.012) |
| $WS_{t-1}$ | -0.21*** | -0.05*** |
| | (0.050) | (0.011) |
| Dep. var. mean | 2.99 | 0.10 |
| Kleibergen-Paap rk Wald F-statistic | 84.39 | 24.83 |
| Number of districts | 85 | 85 |
| Number of days | 1.095 | 1.095 |
| Observations | 89.492 | 89.492 |

*Notes:* This table reports the first stage results for $PM_t$ (in $10\mu g/m^3$) and $1[PM_t > 50\mu g/m^3]$, a dummy variable indicating whether the PM10 level is above the WHO guidelines for short-term exposure. We use districts whose centroid is within 5km from a pollution monitor. Each column reports coefficients from a different regression. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** p<0.01, ** p<0.05, * p<0.1.

Table 2: PM impacts on hospitalization – Children 1-5 years old

| | Hospitalization rate – Respiratory | | | | Days |
|---|---|---|---|---|---|
| | All | Asthma | Pneumonia | Influenza | Respiratory |
| Panel A: OLS estimates | | | | | |
| $PM_t$ | 0.82* | 0.10 | 0.22 | 0.04** | 0.03 |
| | (0.480) | (0.117) | (0.271) | (0.019) | (0.033) |
| Panel B: IV estimates | | | | | |
| $PM_t$ | 4.83*** | 1.14*** | 2.36** | 0.08 | -0.03 |
| | (1.420) | (0.425) | (1.036) | (0.052) | (0.053) |
| Dep. var. mean | 67.63 | 7.30 | 35.54 | 0.22 | 2.96 |
| Number of districts | 85 | 85 | 85 | 85 | 85 |
| Number of days | 1.095 | 1.095 | 1.095 | 1.095 | 1.095 |
| Observations | 89.492 | 89.492 | 89.492 | 89.492 | 89.492 |

*Notes:* This table reports the PM10 impacts on hospitalization rates and length of stay for children between 1 and 5 years old. Hospitalization rate is measured as the number of hospital admissions per one million children, and length of stay is measured in days. Each column in each panel reports coefficients from a different regression. We use districts whose centroid is within 5km from a pollution monitor. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table 3: PM impacts on hospitalization – Children 1-5 years old, PM above WHO's guidelines

| | Hospitalization rate – Respiratory | | | | Days |
|---|---|---|---|---|---|
| | All | Asthma | Pneumonia | Influenza | Respiratory |
| Panel A: OLS estimates | | | | | |
| $1[PM_t > 50\mu g/m^3]$ | 3.81* | 0.62 | 1.38 | 0.07 | 0.11 |
| | (2.005) | (0.430) | (1.351) | (0.065) | (0.089) |
| Panel B: IV estimates | | | | | |
| $1[PM_t > 50\mu g/m^3]$ | 43.92*** | 11.06*** | 21.65** | 0.67 | -0.18 |
| | (12.442) | (3.852) | (8.819) | (0.416) | (0.492) |
| Dep. var. mean | 67.63 | 7.30 | 35.54 | 0.22 | 2.96 |
| Number of districts | 85 | 85 | 85 | 85 | 85 |
| Number of days | 1.095 | 1.095 | 1.095 | 1.095 | 1.095 |
| Observations | 89.492 | 89.492 | 89.492 | 89.492 | 89.492 |

*Notes:* This table reports the PM10 impacts on hospitalization rates and length of stay for children between 1 and 5 years old. Hospitalization rate is measured as the number of hospital admissions per one million children, and length of stay is measured in days. $1[(PM_t > 50\mu g/m^3)]$ is a dummy variable that indicates whether the PM10 level is above the WHO's guidelines for short-term exposure. $1[(PM_t > 50\mu g/m^3)]$ sample mean is 0.096. Each column in each panel reports coefficients from a different regression. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** p<0.01, ** p<0.05, * p<0.1.

Table 4: PM impacts on hospitalization – Children 1-5 years old, public vs. private system

| | Hospitalization rate – Respiratory | | | | Days |
|---|---|---|---|---|---|
| | All | Asthma | Pneumonia | Influenza | Respiratory |
| Panel A: Public Health System (SUS) | | | | | |
| $PM_t$ | 4.55*** | 0.99** | 2.35** | 0.08* | 0.02 |
| | (1.346) | (0.408) | (0.998) | (0.044) | (0.067) |
| Panel B: Private Health System (publicly reimbursed) | | | | | |
| $PM_t$ | 0.29 | 0.15 | 0.01 | 0.00 | -0.14 |
| | (0.263) | (0.089) | (0.152) | (0.023) | (0.102) |
| Dep. var. mean (public) | 62.57 | 6.63 | 34.21 | 0.18 | 2.83 |
| Dep. var. mean (private) | 5.06 | 0.67 | 1.32 | 0.04 | 0.34 |
| Number of districts | 85 | 85 | 85 | 85 | 85 |
| Number of days | 1.095 | 1.095 | 1.095 | 1.095 | 1.095 |
| Observations | 89.492 | 89.492 | 89.492 | 89.492 | 89.492 |

*Notes:* This table reports the second stage results for children between 1 and 5 years old, considering the hospitalization rate and length of stay in public and private healthcare systems. Hospitalization rate is measured as the number of hospital admissions per one million children, and length of stay is measured in days. Each column in each panel reports coefficients from a different regression. We use districts whose centroid is within 5km from a pollution monitor. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table 5: PM impacts on hospitalization – Children 1-5 years old, other diseases, public vs. private

| | Hospitalization rate | | | |
|---|---|---|---|---|
| | Elective care | | Urgent care | |
| | Phimosis | Epilepsy | Appendicitis | Bone Fracture |
| Panel A: Public Health System | | | | |
| $PM_t$ | -0.84* | -0.28** | -0.06 | 0.00 |
| | (0.490) | (0.127) | (0.088) | (0.097) |
| Panel B: Private Health System (publicly reimbursed) | | | | |
| $PM_t$ | 0.06 | 0.06 | 0.05** | -0.02 |
| | (0.049) | (0.055) | (0.023) | (0.033) |
| Dep. var. mean (public) | 7.60 | 2.32 | 0.82 | 0.94 |
| Dep. var. mean (private) | 0.22 | 0.28 | 0.07 | 0.11 |
| Number of districts | 85 | 85 | 85 | 85 |
| Number of days | 1.095 | 1.095 | 1.095 | 1.095 |
| Observations | 89.492 | 89.492 | 89.492 | 89.492 |

*Notes:* This table reports the PM10 impacts on the hospitalization rate for other diseases. Hospitalization rate is measured as the number of hospital admissions per one million children. The results are split into hospital admissions that occurred in public and private hospitals (but reimbursed by the government). The non-respiratory causes of hospitalization considered in this table are phimosis surgery, epilepsy-related procedures such as video-EEG (electroencephalograph) monitoring, appendectomy, and bone fracture repair; the first two are considered elective care procedures, while the last two are urgent procedures. Each column in each panel reports coefficients from a different regression. We use districts whose centroid is within 5km from a pollution monitor. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table 6: PM impacts on hospitalization – Children 1-5 years, public beds

| | Hospitalization rate | | | |
|---|---|---|---|---|
| | Elective care | | Urgent care | |
| | Phimosis | Epilepsy | Appendicitis | Bone Fracture |
| Panel A: Pediatric Beds | | | | |
| $PM_t$ | -1.56* | -0.87*** | 0.07 | -0.02 |
| | (0.913) | (0.198) | (0.186) | (0.179) |
| $PM_t * 1$[high pediatric beds] | 0.47 | 0.39*** | -0.09 | 0.02 |
| | (0.449) | (0.139) | (0.101) | (0.115) |
| Panel B: All Beds | | | | |
| $PM_t$ | -1.56 | -0.53** | 0.08 | 0.12 |
| | (0.958) | (0.233) | (0.189) | (0.176) |
| $PM_t * 1$[high general beds] | 0.47 | 0.17 | -0.09 | -0.08 |
| | (0.443) | (0.156) | (0.105) | (0.116) |
| Panel C: Family Doctors | | | | |
| $PM_t$ | -1.33 | -0.59** | 0.13 | 0.05 |
| | (1.028) | (0.224) | (0.205) | (0.191) |
| $PM_t * 1$[high family doctors] | 0.31 | 0.20 | -0.12 | -0.03 |
| | (0.478) | (0.148) | (0.109) | (0.116) |
| Dep. var. mean | 7.60 | 2.32 | 0.82 | 0.94 |
| Number of districts | 85 | 85 | 85 | 85 |
| Number of days | 1.095 | 1.095 | 1.095 | 1.095 |
| Observations | 89.492 | 89.492 | 89.492 | 89.492 |

*Notes:* This table reports the PM10 impacts on hospitalization rate for other diseases in public hospitals. Hospitalization rate is measured as the number of hospital admissions per one million children. The non-respiratory causes of hospitalization considered in this table are phimosis surgery, epilepsy-related procedures such as video-EEG (electroencephalograph) monitoring, appendectomy, and bone fracture repair; the first two are considered elective care procedures, while the last two are urgent procedures. We explore the heterogeneity of the results by hospital capacity indicators for beds and doctors by district in 2014, the year before our period of analysis. Each capacity rate is calculated per total district population. 1[high] is a dummy variable indicating that the capacity measure is above the median. Each column in each panel reports coefficients from a different regression. We use districts whose centroid is within 5km from a pollution monitor. The Kleibergen-Paap rk Wald F-statistics of the first stages are 88.27 (Panels A, B, and C for the first-stage results for $PM_t$), 83.36 (Panel A, first stage results for $PM_t * 1$[high pediatric beds]), 84.75 (Panel B, first stage results for $PM_t * 1$[high general beds]), and 91.45 (Panel C, first stage results for $PM_t * 1$[high family doctors]). We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** p<0.01, ** p<0.05, * p<0.1.

Table 7: PM impacts on hospitalization – Children 1-5 years old, publicly-funded beds in private hospitals

| | Hospitalization rate | | | |
|---|---|---|---|---|
| | Elective care | | Urgent care | |
| | Phimosis | Epilepsy | Appendicitis | Bone Fracture |
| Panel A: Pediatric Beds | | | | |
| $PM_t$ | 0.02 | 0.17 | 0.06 | -0.00 |
| | (0.076) | (0.118) | (0.036) | (0.064) |
| $PM_t * 1$[high pediatric beds] | 0.02 | -0.07 | -0.00 | -0.01 |
| | (0.058) | (0.066) | (0.026) | (0.044) |
| Panel B: All Beds | | | | |
| $PM_t$ | -0.11 | 0.22* | 0.03 | -0.06 |
| | (0.082) | (0.123) | (0.043) | (0.070) |
| $PM_t * 1$[high general beds] | 0.11** | -0.10 | 0.01 | 0.03 |
| | (0.052) | (0.070) | (0.027) | (0.045) |
| Panel C: Family Doctors | | | | |
| $PM_t$ | -0.13 | 0.19 | 0.04 | -0.03 |
| | (0.079) | (0.125) | (0.045) | (0.069) |
| $PM_t * 1$[high family doctors] | 0.12** | -0.08 | 0.01 | 0.01 |
| | (0.055) | (0.069) | (0.027) | (0.043) |
| Dep. var. mean | 0.22 | 0.28 | 0.07 | 0.11 |
| Number of districts | 85 | 85 | 85 | 85 |
| Number of days | 1.095 | 1.095 | 1.095 | 1.095 |
| Observations | 89.492 | 89.492 | 89.492 | 89.492 |

*Notes:* This table reports the PM10 impacts on hospitalization rate for other diseases in private hospitals, but publicly funded by the Brazilian Healthcare System. Hospitalization rate is measured as the number of hospital admissions per one million children. The non-respiratory causes of hospitalization considered in this table are phimosis surgery, epilepsy-related procedures such as video-EEG (electroencephalograph) monitoring, appendectomy, and bone fracture repair; the first two are considered elective care procedures, while the last two are urgent procedures. We explore the heterogeneity of the results by hospital capacity indicators for beds and doctors by district in 2014, the year before our period of analysis. Each capacity rate is calculated per total district population. 1[high] is a dummy variable indicating that the capacity measure is above the median. Each column in each panel reports coefficients from a different regression. We use districts whose centroid is within 5km from a pollution monitor. The Kleibergen-Paap rk Wald F-statistics of the first stages are 88.27 (Panels A, B, and C for the first-stage results for $PM_t$), 83.36 (Panel A, first stage results for $PM_t * 1$[high pediatric beds]), 84.75 (Panel B, first stage results for $PM_t * 1$[high general beds]), and 91.45 (Panel C, first stage results for $PM_t * 1$[high family doctors]). We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** p<0.01, ** p<0.05, * p<0.1.

Table 8: Impacts of wind speed on PM10 – First stage with 4 lags of speed

| | $PM_t$ | $PM_{t-1}$ | $PM_{t-2}$ | $PM_{t-3}$ |
|---|---|---|---|---|
| $WS_t$ | -0.68*** | -0.02 | 0.00 | 0.08* |
| | (0.056) | (0.044) | (0.044) | (0.043) |
| $WS_{t-1}$ | -0.22*** | -0.67*** | -0.01 | -0.03 |
| | (0.053) | (0.064) | (0.048) | (0.044) |
| $WS_{t-2}$ | 0.01 | -0.24*** | -0.68*** | -0.00 |
| | (0.049) | (0.052) | (0.065) | (0.048) |
| $WS_{t-3}$ | 0.04 | 0.01 | -0.24*** | -0.68*** |
| | (0.050) | (0.048) | (0.052) | (0.065) |
| $WS_{t-4}$ | 0.04 | 0.06 | 0.03 | -0.24*** |
| | (0.046) | (0.045) | (0.046) | (0.051) |
| Cumulative effects | -0.811*** | -0.853*** | -0.887*** | -0.874*** |
| | (0.090) | (0.085) | (0.083) | (0.079) |
| Dep. var. mean (in $10\mu g/m^3$) | 2.99 | 2.99 | 2.99 | 2.99 |
| Kleibergen-Paap rk Wald F-statistic | 35.34 | 35.52 | 35.26 | 35.51 |
| Number of districts | 85 | 85 | 85 | 85 |
| Number of days | 1.092 | 1.092 | 1.092 | 1.092 |
| Observations | 89.245 | 89.245 | 89.245 | 89.237 |

*Notes:* This table reports alternative first stage results. We include 4 lags of wind speed, and vary the time dimension of the dependent variable. Each column reports coefficients from a different regression. Cumulative effects are the sum of all wind speed coefficients. We use districts whose centroid is within 5km from a pollution monitor. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** p<0.01, ** p<0.05, * p<0.1.

Table 9: PM impacts on hospitalization – Children 1-5 years, with 3 lags of PM

| | Hospitalization rate – Respiratory | | | | Days |
|---|---|---|---|---|---|
| | All | Asthma | Pneumonia | Influenza | Respiratory |
| $PM_t$ | 4.63*** | 0.79* | 2.41** | 0.12* | -0.01 |
| | (1.716) | (0.450) | (1.182) | (0.068) | (0.072) |
| $PM_{t-1}$ | 2.88 | 1.22*** | 1.67 | -0.01 | 0.00 |
| | (1.991) | (0.446) | (1.333) | (0.072) | (0.124) |
| $PM_{t-2}$ | -1.22 | -0.54 | -1.17 | 0.02 | 0.01 |
| | (1.968) | (0.512) | (1.214) | (0.069) | (0.085) |
| $PM_{t-3}$ | 5.06*** | 1.07*** | 3.65*** | 0.14** | 0.12 |
| | (1.578) | (0.374) | (0.982) | (0.055) | (0.075) |
| Cumulative effects | 11.35*** | 2.54*** | 6.56*** | 0.26*** | 0.13 |
| | (2.397) | (0.673) | (1.598) | (0.084) | (0.098) |
| Dep. var. mean | 67.72 | 7.30 | 35.58 | 0.23 | 2.97 |
| Joint F-statistic (1st stage) | 20.24 | 20.24 | 20.24 | 20.24 | 20.24 |
| Number of districts | 85 | 85 | 85 | 85 | 85 |
| Number of days | 1.092 | 1.092 | 1.092 | 1.092 | 1.092 |
| Observations | 89.237 | 89.237 | 89.237 | 89.237 | 89.237 |

*Notes:* This table reports alternative second stage results for children between 1 and 5 years old, considering the hospitalization rate and length of stay. The new feature is the inclusion of 3 lags of exposure to PM10. Hospitalization rate is measured as the number of hospital admissions per one million children, and length of stay is measured in days. Each column reports coefficients from a different regression. Cumulative effects are the sum of all PM coefficients. We use districts whose centroid is within 5km from a pollution monitor. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table 10: Impacts of wind speed and solar radiation on PM10 and ozone – First stage, multi-pollutant model

| | $PM_t$ | $O3_t$ |
|---|---|---|
| $WS_t$ | -0.66*** | 2.25*** |
| | (0.062) | (0.583) |
| $WS_{t-1}$ | -0.21*** | -1.20** |
| | (0.048) | (0.494) |
| $SR_t$ | -0.00 | 0.06*** |
| | (0.001) | (0.007) |
| Dep. var. mean | 2.91 | 37.08 |
| Kleibergen-Paap rk Wald F-statistic | 52.50 | 32.05 |
| Number of districts | 61 | 61 |
| Number of days | 1.095 | 1.095 |
| Observations | 63.160 | 63.160 |

*Notes:* This table reports the first stage results for the multi-pollutant model. We include contemporaneous solar radiation together with wind speed as instruments for PM10 and ambient ozone. PM10 average (in $10\mu g/m^3$): 2.91. O3 average (in $\mu g/m^3$): 37.08. Each column reports coefficients from a different regression. We use districts whose centroid is within 5km from a pollution monitor. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

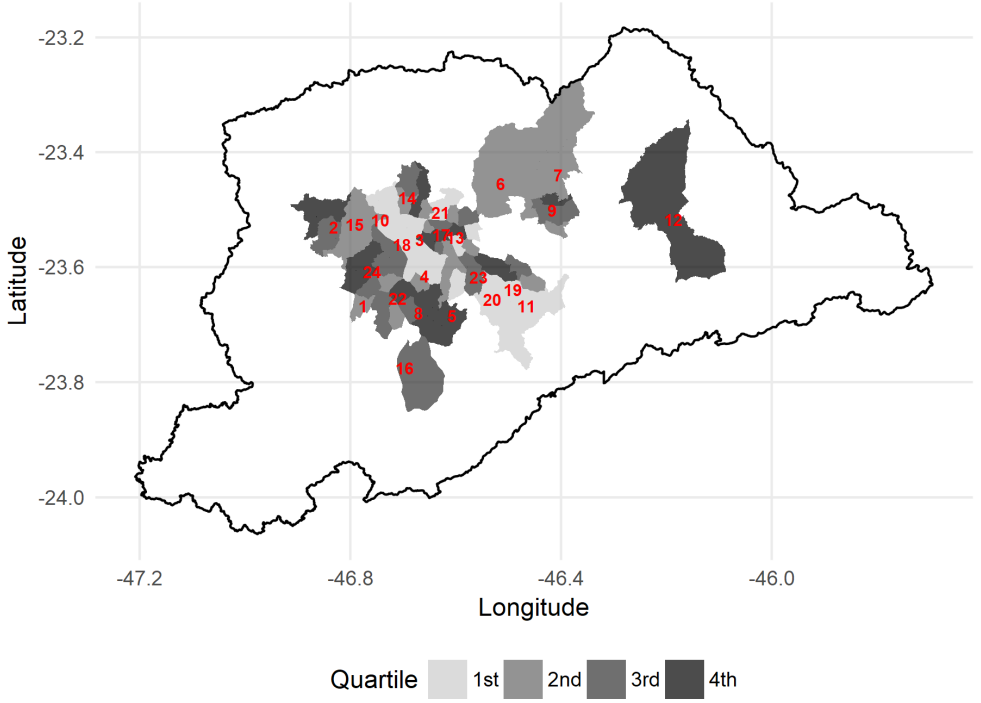
Table 11: PM and ozone impacts on hospitalization – Children 1-5 years old

| | Hospitalization rate – Respiratory | | | | Days |
|---|---|---|---|---|---|
| | All | Asthma | Pneumonia | Influenza | Respiratory |
| Panel A: OLS estimates | | | | | |
| $PM_t$ | 0.82 | 0.07 | 0.31 | 0.06** | 0.03 |
| | (0.585) | (0.136) | (0.321) | (0.022) | (0.027) |
| $O3_t$ | -0.07 | -0.01 | -0.02 | 0.00 | -0.00 |
| | (0.047) | (0.012) | (0.037) | (0.003) | (0.003) |
| Panel B: IV estimates | | | | | |
| $PM_t$ | 4.38*** | 1.10** | 2.28* | 0.06 | -0.05 |
| | (1.582) | (0.508) | (1.201) | (0.056) | (0.060) |
| $O3_t$ | 0.10 | 0.02 | 0.01 | -0.01 | -0.00 |
| | (0.236) | (0.063) | (0.160) | (0.009) | (0.009) |
| Dep. var. mean | 70.95 | 7.35 | 38.20 | 0.22 | 2.98 |
| Number of districts | 61 | 61 | 61 | 61 | 61 |
| Number of days | 1.095 | 1.095 | 1.095 | 1.095 | 1.095 |
| Observations | 63.160 | 63.160 | 63.160 | 63.160 | 63.160 |

*Notes:* This table reports the second stage results of the multi-pollutant model. We consider the impacts of both PM10 and ambient ozone on hospitalization rates and length of stay for children between 1 and 5 years old. Hospitalization rate is measured as the number of hospital admissions per one million children, and length of stay is measured in days. Each column in each panel reports coefficients from a different regression. We use districts whose centroid is within 5km from a pollution monitor. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

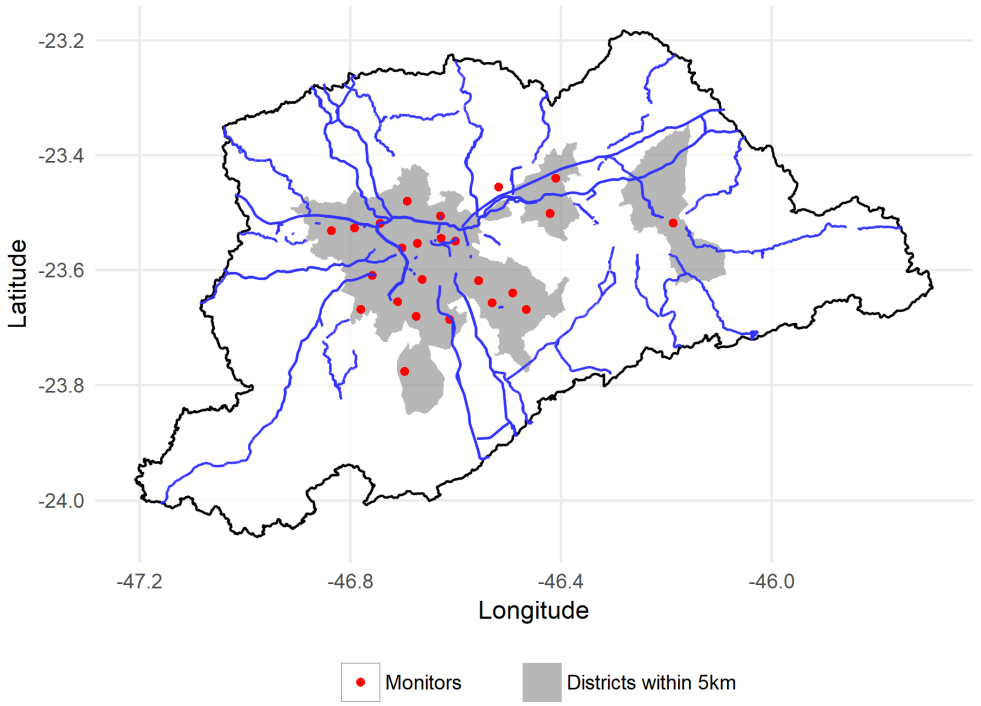
# Appendix Figures

Figure A1: Pediatric hospitalization rate and pollution monitors in Sao Paulo



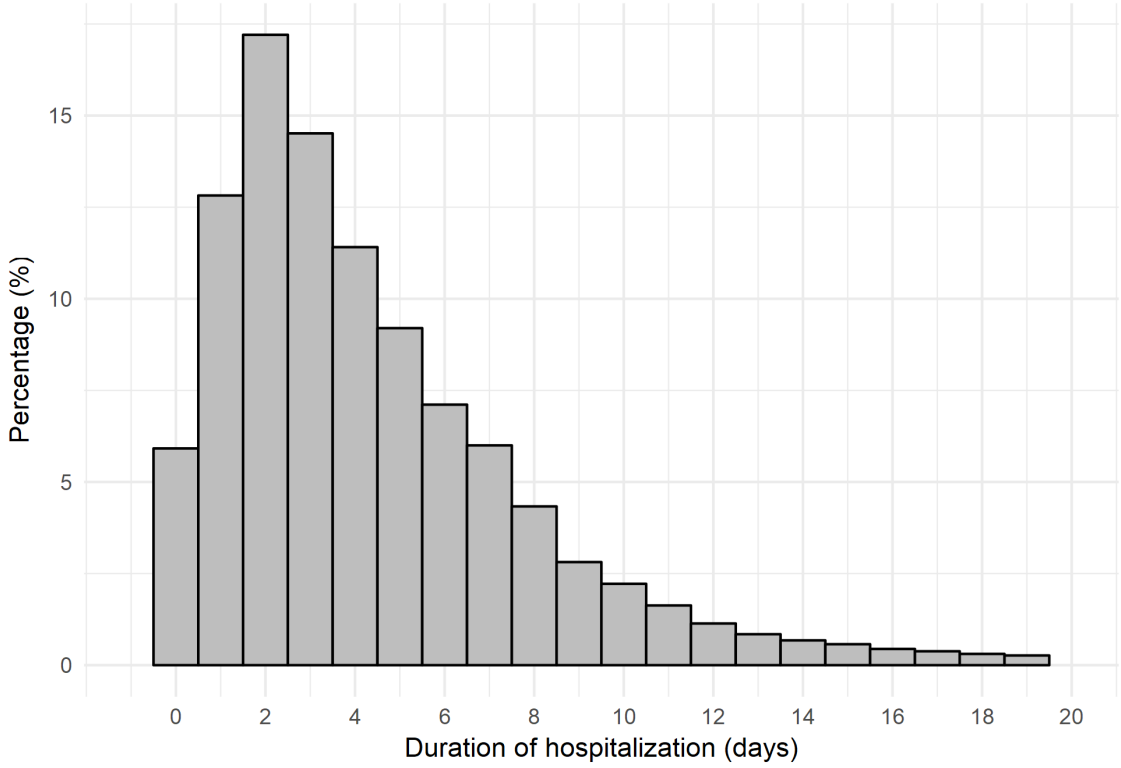

*Notes*: This map displays a representation of average pediatric hospitalization rates from 2015-2017 by quartile in the Sao Paulo Metropolitan Area (SPMA). Hospitalization rate is measured as the number of hospital admissions per one million children 1-5 years old. Darker gray shades indicate higher rates. The red numbers correspond to the pollution monitors enumerated in Figures 6 and A5.

Figure A2: Expressways and highways in Sao Paulo



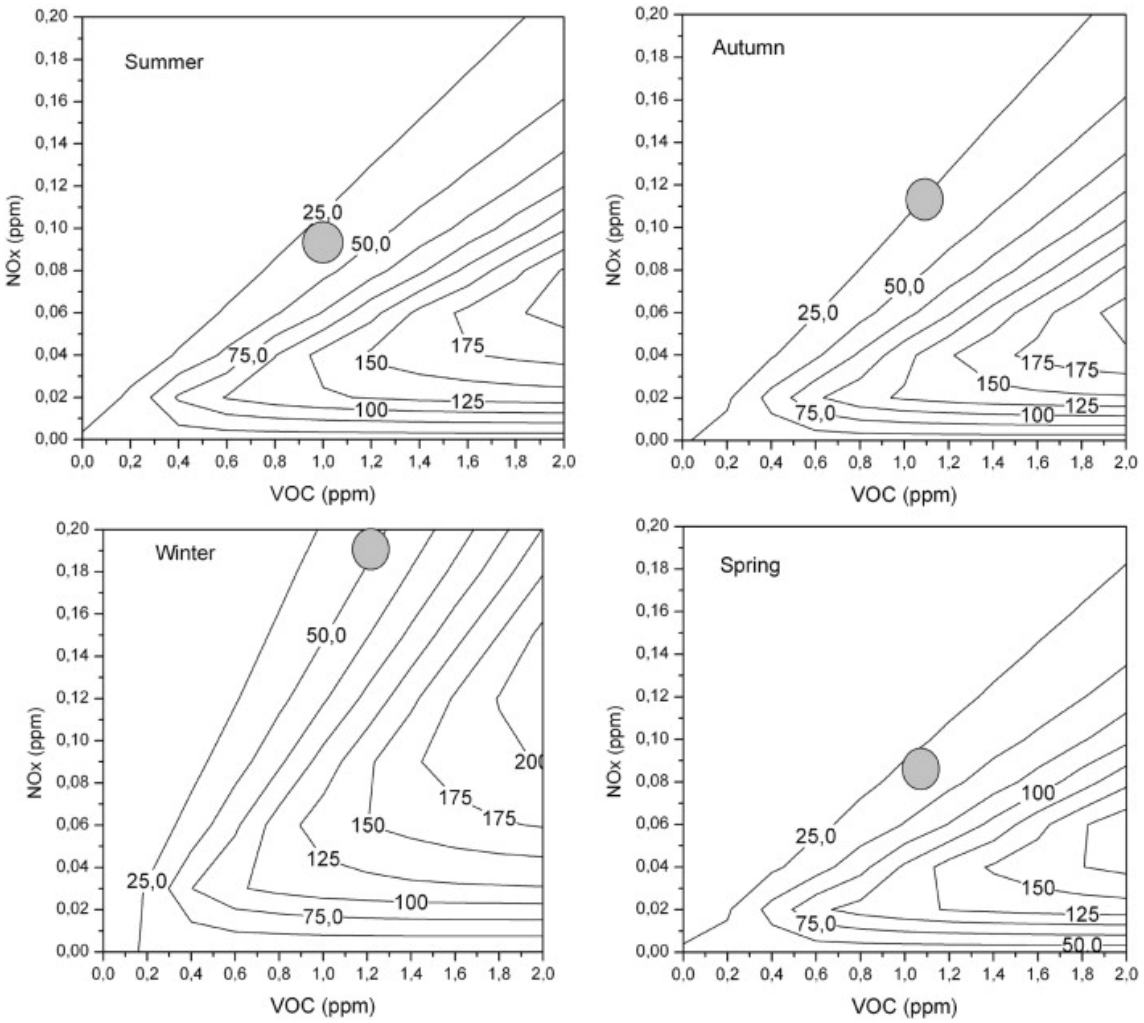

*Notes*: This map displays expressways and highways (blue lines) together with pollution monitors (red dots) and the districts within 5km from any monitor (gray area) in the Sao Paulo Metropolitan Area (SPMA).

Figure A3: Duration of pediatric hospitalizations in Sao Paulo



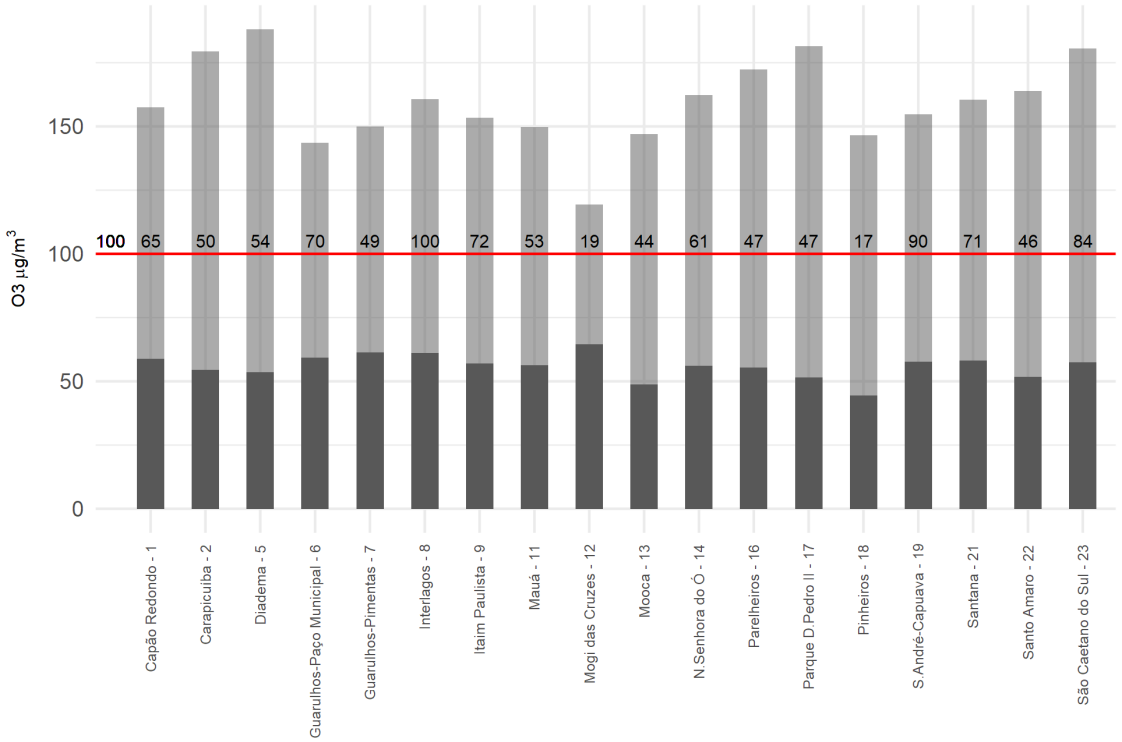

*Notes*: This figure displays the histogram for duration of pediatric hospitalizations, measured in days, in the Sao Paulo Metropolitan Area (SPMA). Hospitalization rate is measured as the number of hospital admissions per one million children 1-5 years old.

Figure A4: Ozone formation – Illustration




*Notes*: This figure illustrates the Leontief-like production function of ozone. Each quadrant displays the concentration of NOx (y-axis) and VOCs (x-axis) – measured in parts per million (ppm) – for a season of the year. The gray lines represent the $O_3$ isopleths for several VOCs and NOx concentrations. The gray circles represent the average $O_3$ concentration for each season of the SPMA. *Source*: Orlando et al. (2010).

Figure A5: Average and maximum ambient ozone concentration, and number of days above WHO guidelines in the SPMA




*Notes*: This figure displays the annual 8-hour average of ambient ozone concentration (dark gray), as well as the annual maximum 8-hour average (light gray), and the number of days with ozone concentration above the daily maximum recommended by the WHO (numbers above red line), by monitor in the Sao Paulo Metropolitan Area (SPMA), from 2015-2017. The red line represents the maximum daily 8-hour average of ambient ozone concentration that the WHO's guidelines recommend for relatively "safe" exposure. We use ozone measurements from 9AM to 5PM to construct the daily 8-hour average for this figure.

# Appendix Tables

Table A1: PM impacts on childbirth hospitalization – Public vs. private health system

| | Public System | | Private System | |
|---|---|---|---|---|
| | Natural birth | C-section | Natural birth | C-section |
| Panel A: Average | | | | |
| $PM_t$ | -0.00 | -0.13** | 0.01 | 0.02 |
| | (0.154) | (0.065) | (0.079) | (0.037) |
| Panel B: Pediatric Beds | | | | |
| $PM_t$ | -0.01 | -0.17 | 0.08 | 0.13 |
| | (0.309) | (0.109) | (0.155) | (0.094) |
| $PM_t * 1$[high pediatric beds] | 0.00 | 0.03 | -0.05 | -0.08 |
| | (0.189) | (0.067) | (0.078) | (0.047) |
| Panel C: All Beds | | | | |
| $PM_t$ | -0.18 | -0.25** | 0.11 | 0.15 |
| | (0.333) | (0.113) | (0.162) | (0.112) |
| $PM_t * 1$[high general beds] | 0.11 | 0.07 | -0.07 | -0.08 |
| | (0.194) | (0.062) | (0.086) | (0.055) |
| Panel D: Family Doctors | | | | |
| $PM_t$ | -0.07 | -0.22* | 0.17 | 0.16 |
| | (0.341) | (0.117) | (0.161) | (0.118) |
| $PM_t * 1$[high family doctors] | 0.05 | 0.06 | -0.10 | -0.09 |
| | (0.195) | (0.063) | (0.085) | (0.057) |
| Dep. var. mean | 16.01 | 1.98 | 3.09 | 0.58 |
| Number of districts | 85 | 85 | 85 | 85 |
| Number of days | 1.095 | 1.095 | 1.095 | 1.095 |
| Observations | 89.492 | 89.492 | 89.492 | 89.492 |

*Notes:* This table reports the PM10 impacts on C-section and natural birth hospitalization rate, by public hospitals and publicly-funded beds in private hospitals. We consider the pregnancy of women aged 18 to 65 years to calculate the hospitalization rate related to childbirth. We explore the heterogeneity of the results by hospital capacity indicators for beds and doctors by district in 2014, the year before our period of analysis. Each capacity rate is calculated per total district population. 1[high] is a dummy variable indicating that the capacity measure is above the median. Each column in each panel reports coefficients from a different regression. We use districts whose centroid is within 5km from a pollution monitor. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. The Kleibergen-Paap rk Wald F-statistics of the first stages are 88.27 (Panels A, B, C and D for the first-stage results for $PM_t$), 83.36 (Panel B, first stage results for $PM_t * 1$[high pediatric beds]), 84.75 (Panel C, first stage results for $PM_t * 1$[high general beds]), and 91.45 (Panel D, first stage results for $PM_t * 1$[high family doctors]). Standard errors in parentheses are two-way clustered by district and calendar date. *** p<0.01, ** p<0.05, * p<0.1.

Table A2: Impacts of wind speed and wind direction on PM10 – First stage

| | Main – $PM_t$ on WS | $PM_t$ on WS & WD |
|---|---|---|
| $WS_t$ | - 0.681*** | - 0.680*** |
| | (0.056) | (0.057) |
| $WS_{t-1}$ | - 0.211*** | - 0.212*** |
| | (0.050) | (0.049) |
| 1[Wind Direction$_t$ – 1st quadrant] | | 0.012 |
| | | (0.100) |
| 1[Wind Direction$_t$ – 2nd quadrant] | | 0.148** |
| | | (0.072) |
| 1[Wind Direction$_t$ – 3rd quadrant] | | 0.109* |
| | | (0.056) |
| Dep. var. mean (in $10\mu g/m^3$) | 2.99 | 2.99 |
| Kleibergen-Paap rk Wald F-statistic | 84.39 | 39.84 |
| Number of districts | 85 | 85 |
| Number of days | 1.095 | 1.095 |
| Observations | 89.492 | 89.492 |

*Notes:* This table reports the main first stage estimates, and results from an alternative specification using wind speed and wind direction as instruments. Following Deryugina et al. (2019), wind direction is represented by three dummy variables informing the quadrant of the direction. Each column reports coefficients from a different regression. We use districts whose centroid is within 5km from a pollution monitor. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** p<0.01, ** p<0.05, * p<0.1.

Table A3: PM impacts on hospitalization – Children 1-5 years old, IV estimates

| | Hospitalization rate - Respiratory | | | | Days |
|---|---|---|---|---|---|
| | All | Asthma | Pneumonia | Influenza | Respiratory |
| Panel A: Wind Speed | | | | | |
| $PM_t$ | 4.83*** | 1.14*** | 2.36** | 0.08 | -0.03 |
| | (1.420) | (0.425) | (1.036) | (0.052) | (0.053) |
| Panel B: Wind Speed & Direction | | | | | |
| $PM_t$ | 5.22*** | 1.16*** | 2.60** | 0.10* | -0.01 |
| | (1.435) | (0.419) | (1.062) | (0.054) | (0.058) |
| Dep. var. mean | 67.63 | 7.30 | 35.54 | 0.22 | 2.96 |
| Number of districts | 85 | 85 | 85 | 85 | 85 |
| Number of days | 1.095 | 1.095 | 1.095 | 1.095 | 1.095 |
| Observations | 89.492 | 89.492 | 89.492 | 89.492 | 89.492 |

*Notes:* This table reproduces the IV estimates of Table 2, and compares them with IV estimates after including wind direction (dummy variables for quadrants) as additional instruments. Hospitalization rate is measured as the number of hospital admissions per one million children 1-5 years old, and length of stay is measured in days. Each column in each panel reports coefficients from a different regression. We use districts whose centroid is within 5km from a pollution monitor. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table A4: PM impacts on hospitalization – Children 1-5 years old, alternative samples varying the distance of districts to pollution monitors

| | Hospitalization rate – Respiratory | | | | Days |
|---|---|---|---|---|---|
| | All | Asthma | Pneumonia | Influenza | Respiratory |
| Panel A: 3 km | | | | | |
| $PM_t$ | 6.24*** | 1.13** | 3.23*** | 0.09 | - 0.06 |
| | (1.368) | (0.487) | (1.051) | (0.081) | (0.071) |
| Dep. var. mean | 64.93 | 6.91 | 33.96 | 0.23 | 2.74 |
| Number of districts | 48 | 48 | 48 | 48 | 48 |
| Panel B: 5 km | | | | | |
| $PM_t$ | 4.83*** | 1.14*** | 2.36** | 0.08 | - 0.03 |
| | (1.420) | (0.425) | (1.036) | (0.052) | (0.053) |
| Dep. var. mean | 67.63 | 7.30 | 35.54 | 0.22 | 2.96 |
| Number of districts | 85 | 85 | 85 | 85 | 85 |
| Panel C: 7 km | | | | | |
| $PM_t$ | 3.62** | 0.98*** | 2.13** | 0.08* | - 0.05 |
| | (1.541) | (0.368) | (0.994) | (0.043) | (0.046) |
| Dep. var. mean | 67.04 | 7.14 | 35.19 | 0.22 | 2.97 |
| Number of districts | 106 | 106 | 106 | 106 | 106 |
| Panel D: 10 km | | | | | |
| $PM_t$ | 2.55 | 0.83** | 1.69* | 0.10** | - 0.06 |
| | (1.552) | (0.331) | (0.930) | (0.044) | (0.043) |
| Dep. var. mean | 66.32 | 6.85 | 35.03 | 0.24 | 2.90 |
| Number of districts | 126 | 126 | 126 | 126 | 126 |
| Number of days | 1.095 | 1.095 | 1.095 | 1.095 | 1.095 |

*Notes:* This table reproduces the IV estimates of Table 2, whose sample uses only districts within 5km from a pollution monitor (Panel B), and compares them with IV estimates arising from alternative samples varying the distance from the district centroid to the nearest pollution monitor – 3, 7, and 10km. Hospitalization rate is measured as the number of hospital admissions per one million children 1-5 years old, and length of stay is measured in days. Each column in each panel reports coefficients from a different regression. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table A5: PM impacts on hospitalization – All age groups

| | Hospitalization rate – Respiratory | | | | Days |
|---|---|---|---|---|---|
| | All | Asthma | Pneumonia | Influenza | Days |
| Panel A: Ages 0-1 | | | | | |
| $PM_t$ | 1.70 | 0.06 | 0.48 | 0.15* | 0.07 |
| | (1.120) | (0.147) | (0.568) | (0.079) | (0.057) |
| Dep. var mean | 13.21 | 0.29 | 5.30 | 0.12 | 0.31 |
| Panel B: Ages 1-5 | | | | | |
| $PM_t$ | 4.83*** | 1.14*** | 2.36** | 0.08 | -0.03 |
| | (1.420) | (0.425) | (1.036) | (0.052) | (0.053) |
| Dep. var. mean | 67.63 | 7.30 | 35.54 | 0.22 | 2.96 |
| Panel C: Ages 5-17 | | | | | |
| $PM_t$ | 0.35 | 0.10 | 0.31 | 0.01 | 0.04 |
| | (0.332) | (0.089) | (0.191) | (0.014) | (0.164) |
| Dep. var. mean | 13.24 | 1.85 | 6.21 | 0.05 | 2.16 |
| Panel D: Ages 18-65 | | | | | |
| $PM_t$ | -0.05 | -0.02* | -0.02 | 0.00 | 0.04 |
| | (0.077) | (0.014) | (0.053) | (0.006) | (0.104) |
| Dep. var. mean | 4.90 | 0.13 | 2.40 | 0.03 | 3.72 |
| Panel E: Ages 66 + | | | | | |
| $PM_t$ | 0.38 | -0.07 | 0.18 | -0.00 | -0.05 |
| | (0.680) | (0.058) | (0.485) | (0.019) | (0.081) |
| Dep. var. mean | 29.64 | 0.32 | 19.26 | 0.07 | 3.61 |
| Number of districts | 85 | 85 | 85 | 85 | 85 |
| Number of days | 1.095 | 1.095 | 1.095 | 1.095 | 1.095 |
| Observations | 89.492 | 89.492 | 89.492 | 89.492 | 89.492 |

*Notes:* This table reports the second stage results for all age groups. Hospitalization rate is measured as the number of hospital admissions per one million individuals in each age group, and length of stay is measured in days. Each column in each panel reports coefficients from a different regression. We use districts whose centroid is within 5km from a pollution monitor. We include district, day-of-week, month-of-year, and year fixed effects. We also add temperature and humidity in quadratic form as controls. Standard errors in parentheses are two-way clustered by district and calendar date. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.