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The Impact of Air Pollution on Hospital Admissions: Evidence from Italy

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Abstract

In this paper we examine the relationship between air pollution and hospital admissions for chronic obstructive pulmonary disease in Italy, at province level, over the period 2004-2009. To this end, we use information on annual mean concentrations of carbon monoxide, nitrogen dioxide, particulate matter, and ozone measured at monitoring station level to build province-level indicators. In our model for hospital admissions, we allow pollution measures to be subject to measurement error and possibly correlated with the error term. By adopting an instrumental variables approach, we find that higher levels of particulate matter and carbon monoxide are associated with higher hospitalisation for children, while ozone has an influence on hospital admissions of the elderly. Other factors that appear to have an important role are the rainfall and the level of education.

Keywords: airborne pollutants; hospital admission; instrumental variables.
JEL codes: I120, I180, Q530.

1 Introduction

Over the past decade, a substantial scientific literature has documented the size and seriousness of the impact of atmospheric pollution on the environment and the health of people. Air pollutions in Europe varies substantially over time and across territory. According to the European Environment Agency many air pollutants have decreased substantially over time, resulting in improved air quality across territory. However air quality problems still persist, as air pollutant concentrations have not sufficiently declined, and a large proportion of Europe's population lives in urban areas where emission limits set by the EU National Emission Ceilings Directive are regularly exceeded. A recent report on the quality of air in Europe (Istat, 2010) shows that Italy is ranked as the third most polluted country in Europe, after Bulgaria and Greece, with more than half of the 30 most polluted cities being Italian. In particular, in the year 2008, Turin, Brescia, and Milan have the highest levels of overall air pollution in Europe, after the Bulgarian city, Plovdiv. Turin is also the city with the highest concentration of tropospheric ozone, although this has been reducing over time, while Naples is leading for the highest annual concentration of nitrogen dioxide, responsible for acid rains.

Atmospheric pollution threats public health with both short- and long-term effects. The former may include irritation to the eyes, nose and throat, and upper respiratory infections such as bronchitis and pneumonia. Long-term health effects can include chronic respiratory disease, lung cancer, heart disease, and even damage to the brain, nerves, liver, or kidneys. Some groups of the population may be more sensitive to pollutants than are others, such as young children and the elderly, or people with pre-existing health problems. Medical conditions arising from atmospheric pollution can be very expensive. Health care costs, lost productivity in the workplace, and human welfare impacts cost billions of dollars each year.

This paper focuses on the impact of air pollutants on hospital admissions in Italy, at provincial level. Specifically, we study the effects of a range of different pollutants, namely particular matter of size smaller than about 10 micrometers (PM10), nitrogen dioxide (NO2), carbon monoxide (CO),and ozone (O3) on hospital admissions for chronic obstructive respiratory diseases (COPD), for young children and elderly people living in 99 Italian provinces in the period from 2004 to 2009. Respiratory illness are amongst the most common chronic diseases in the Italian population, including younger age, and cause of premature mortality, with high socio-economic costs. Despite advances in therapy, COPD still represents, by number, the third cause of death in Italy, after circulatory diseases and cancer. Chronic bronchitis and asthma affect more than 20 per cent of the population aged 65 years and represent the third leading cause of chronic disease after osteoarthritis/arthritis and hypertension (Istat, 2009). Although cigarette

smoking is considered the major cause of COPD, recent studies have shown that sustained exposure to exhaust fumes from both motor vehicles and industrial plants may cause development or exacerbation of chronic respiratory diseases (Gauderman, 2007; Kunzli et al. 2009; Ko and Hui, 2012).

One main feature of our work is that we explicitly control for possible measurement errors in our provincial measure of pollution. Indeed, pollution readings from monitoring stations may not reflect the exact amount of pollution to which people have been exposed, given that people live at different distances from stations, and they may move across territory. This issue has been pointed by a recent literature in economics (e.g. Graff Zivin and Neidell, 2009; Knittel et al., 2011; Moretti and Neidell, 2011; Schlenker and Walker, 2011). In order to alleviate this problem, we have decided to adopt a two stage approach. We first study the main drivers of pollution, for each pollutant. A large variety of factors are responsible for the releasing of pollutants into the atmosphere. These can be classified into two major categories: anthropogenic sources, i.e., generated by human activity, mostly related to burning different kinds of fuel, and natural sources such as dust, smoke and carbon monoxide from wildfires, volcanic activity. In our first step, we estimate a regression model for pollution, including province effects, to account for geographical time invariant characteristics, and a set of factors that are recognized to be linked to pollution, such as the number of cars. We also allow for spatio-temporal dynamics to control for persistence over time of pollution, as well as its concentration across space. Hence, in the second stage regression, we use the predicted values of the first stage regression to estimate the impact of pollution on hospital admission. To estimate these models, we adopt a set of econometric techniques recently advanced within the spatial econometrics literature.

The remainder of the paper is organized as follows. Section 2 provides a review of the literature on the effects of pollution on mortality rate and hospital admissions Section 3 introduces our econometric specification and outlines our estimation strategy. Section 4 describes the data, while Section 5 comments on the empirical findings. Section 6 concludes.

2 Background literature

Over the past decade, a wide scientific literature has been documenting the size and seriousness of the impact of atmospheric pollution on the health of people. Most of the studies have focused on the effect of air pollutants on health outcomes, using data at the city, county or region level to test for the effects of prolonged exposure to air pollution on mortality rates or hospital admissions. Early works on the link between urban air pollution and chronic respiratory illness have been carried by Portney and Mullhay (1986, 1990), for the US. The authors combine

information from the Health Interview Survey for a set of people with data on air pollution from the pollution monitors nearest their residences. Results show a positive relationship between ozone concentrations and sickness. Alberini and Krupnick (1998) analyze the health status of over 900 residents of three urban area in Taiwan, and show that the incidence of illness may be related to the ambient concentration levels of PM10. Pope et al. (2002) explore the effects of particulate on long cancer and cardiopulmonary mortality. Chay and Greenstone (2003a) use air quality changes across US counties attributable to the effects of the 1981-82 recession, to estimate the relationship between infant mortality and particulate air pollution. The authors find that a 1 $\mu g/m^3$ reduction in particulate results in about 4-8 fewer infant deaths per 100,000 live births. The estimated effects are driven almost entirely by fewer deaths occurring within one month and one day of birth, suggesting that fetal exposure to pollution has adverse health consequences (see also Chay et al., 2003).

Samakovlis et al. (2005) investigate the relationship between air pollution and respiratory diseases in Sweden. In particular they find that NO2 may increase risk for asthma, bronchitis and hay fever nasal problems. Jerrett et al. (2005) study the health effects of chronic air pollution exposure within industrial cities, using a geostatistical technique in combination with small-area data. Their results suggest that chronic air pollution exposure significantly increases the risk of premature cardiorespiratory and cancer mortalities. Subsequent studies have also found significant associations between ozone (Bell et al., 2005) and nitrogen dioxide (Nafstad et al., 2004) on higher mortality rates.

Currie et al. (2009) explore the impact of air pollutants on infant health, measured by birth weight, gestation and mortality, in New Jersey in the 1990s. The paper combines information about mother's residential location from birth certificates with information on air quality monitors. Results suggest negative effects of exposure to carbon monoxide on children heath, both during and after birth, even in areas at low levels of pollution. Few of the pollution measures are statistically significant, and when they are, are likely to suggest positive effects on birth weight and gestation. The authors also report some evidence of significant effects of particulate matter and ozone on health at birth (see also Currie and Naidell (2005)).

Janke et al. (2009) study the relationship between common sources of airborne pollution and population mortality for England. Using data at Local Authority level over the period 1998 to 2005, they examine whether current levels of airborne pollution are associated with excess deaths. After controlling for unobserved heterogeneity, the authors find that higher levels of particulate matter and ozone are associated with higher mortality rates, arguing that the effect sizes are considerably larger than previously estimated.

Agarwal et al. (2010) study the effect of exposure to a set of toxic pollutants from manu-

facturing facilities on county-level infant and fetal mortality rates in the United States between 1989 and 2002. Results show a significant adverse effects of toxic air pollution concentrations on infant mortality rates. Beatty and Shimshack (2011), examine the impact of school bus emissions reductions programs on health outcomes in the State of Washington. They use a data set on bus retrofits, and detailed information such as retrofit type, retrofit date, and retrofit cost, along with morbidity and demographic data at the school district level. The authors exploit a natural experiment and employ a differences-in-differences research design to help isolate causal impacts. Results suggest that school bus retrofits induced reductions in bronchitis, asthma, and pneumonia incidence for at-risk populations. Coneus and Spiess (2012) study the influence of outdoor pollution and parental smoking on children's health from birth until the age of three years, in Germany, using data from the German Socio-Economic Panel, on the cohort 2002-2007, combined with measures for five pollutants. Results suggest a significantly impact of some pollutants on infant health, with high exposure to carbon monoxide prior to birth causing loss of birth weight, and high ozone levels causing respiratory diseases in toddlers.

Few studies have focused on the effects of air pollution on hospital admissions. Delfino et al. (1994) carry out a study on the relationship of urgent hospital admissions for respiratory illnesses to air pollution in Montreal. Schwartz and Morris (1995) examine the association between air pollution and cardiovascular hospital admissions for people aged 65 years and older in the Detroit, Michigan, metropolitan area during the years 1986-1989. After controlling for seasonal and other long-term temporal trends, temperature, and dewpoint temperature, particulate matter was associated with daily admissions for ischemic heart disease, while sulphur dioxide, carbon monoxide, and ozone made no independent contribution to ischemic heart disease admissions. Linn et al. (2000) evaluate associations between ambient carbon monoxide, nitrogen dioxide, particulate matter and hospital admissions for cardiopulmonary illnesses in metropolitan Los Angeles during 1992-1995. They observe association of pulmonary diseases with PM10 and NO2, and of cardiovascular diseases with CO.

Neidell (2004) studies the influence of air pollution on child hospitalizations for asthma in California, at zip code level, in the period between 1992 to 1998. Results show that, among the pollutants considered in the analysis, only carbon monoxide has a significant effect on hospital admissions for children, with a greater effect for children of lower socio-economic status. Another important finding of this paper is that households seem to respond to information about pollution with avoidance behavior when high levels of pollutions are registered. It is interesting to observe a weak evidence of the impact of particulate matter, ozone and nitrogen dioxid on child hospitalisation.

Dominici et al. (2006) examine risks of cardiovascular and respiratory hospital admissions

associated with PM2.5 , at county level, for American Medicare enrollees, in the period between 1999 to 2002. Results show a short-term increase in hospital admission rates associated with PM2.5. Further, cardiovascular risks tend to be higher in counties located in the Eastern region of the United States. Jayaraman and Nidhi (2008) suggest that air pollution levels in Delhi, specifically of O3, NO2 and PM10 have a significant impact on human health in terms of an increase (24%, 13% and 3%, respectively) in respiratory diseases related hospital visits. Namdeo et al. (2011) demonstrate association of short-term variation in pollution and health outcomes in the northern part of the UK. Results show that PM10 and O3 are positively associated with respiratory hospital admissions in the elderly. Rava et al. (2011) show that proximity to wood industries is associated with a higher risk of hospitalization for respiratory diseases and respiratory symptoms in children.

A recent related literature has emphasized that the majority of the works we have reviewed may suffer for a problem of measurement errors, thus leading to bias estimates. It is likely that people have a different exposure to the amount of solution detected from the monitoring stations. Indeed, people live at different distances from these stations, with some residing close while others far apart. Further, some people may be more mobile than others, also because of avoidance behaviour. In other words, a mismatch is likely to exist between the amount of pollution detected and the exposure of the population to such pollution. Lleras-Muney (2010) finds that estimates are very sensitive to the technique used to impute pollution at aggregate level, and that the measurement error is not normally distributed, making the direction of the bias on estimates ambiguous. Chay and Greenstone (2003b) use a natural experiment to look at the relationship between pollution and infant mortality rate. In their framework, the infant mortality rate depends on level of pollution, but because unobserved factors are likely to be correlated with both the level of pollution and the infant mortality rate, any estimation would lead to a biased estimation of the health production function. Thus the authors use the Clean Air Act of 1970 as an instrument to estimate effect of pollution on the infant mortality rate. Moretti and Neidell (2011), using zip code for the months April-October for the years 1993-2000, study the relationship between ozone and infant mortality rate. To alleviate possible bias resulting from the measurement error, they adopt an instrumental variables (IV) approach, using timing of Port of Los Angeles traffic and distance to port as an instrument for ozone concentrations. The authors conclude that estimated effects of ozone on health are large, and that simple correlations are significantly biased by unobserved avoidance behavior and/or measurement error. Evidence of avoidance behavior can also be found in the paper by Graff Zivin and Neidell (2009). This study shows that people reallocate activities across time when faced with bad air quality air. Knittel et al. (2011) investigate the relationship between traffic,

weather, pollution, and infant outcomes in California, using zip code-week level data for the years 2002-2007. The authors perform an IV approach to deal with measurement error and find that ambient pollution levels have a large impact on weekly mortality rates.

3 Empirical model

The dependent variable of our model is hospital admissions caused by COPD. COPD is a disease state characterized by airflow limitation that is not fully reversible, and progressive lung function decline, and is known to be exacerbated by air pollution. Another reason for focusing on this variable is that, worldwide, COPD is ranked as the sixth leading cause of death in 1990, and it is projected to be the fourth leading cause of death worldwide by 2030 (Mathers and Loncar, 2006). As noted by Bellander et al. (1999), and Samakovlis et al. (2005), hospitalisation may capture only part of the total effect of moderate air pollution, since most effects are less severe. Indeed, it is possible that pollutants affect the respiratory system without resulting in hospitalisations. However, we believe that this is an important measure of public health, also reflecting the consumption of health care resources.

As described in Section 4, data at monitoring station level are used to build annual province-level indicator of pollution. Hence, our variable is likely to be subject to measurement errors. To deal with these issues, we estimate our model for COPD by adopting an Instrumental Variables (IV) approach in a two stage regression. In the first step, we assume that the kth pollutant, with k = PM10, NO2, CO, and O3, is driven by the following spatio-temporal process with unobserved heterogeneity:

$$p_{k,it} = \mu_i + \delta p_{k,it-1} + \eta \overline{p}_{k,it} + \boldsymbol{\gamma}' \mathbf{z}_{it} + \varepsilon_{it}, \tag{1}$$

where $\overline{p}_{k,it}$ is the spatial lag of $p_{k,it}$, given by $\overline{p}_{k,it} = \sum_{j=1}^{N} s_{it} p_{k,jt}$,and s_{ij} are elements of a spatial weights matrix, where $s_{ij} = 1/d_{ij}$ where d_{ij} is the distance in kilometers between centroids of provinces i and j. We have adopted a dynamic specification since the sources of pollution, such as an industry, generally continue over time, making pollution a phenomenon persistent over time. We also include $\overline{p}_{it}$ under the assumption that an air pollutant originating in a particular point in space, due for example to car emissions, may propagate across a wider geographical area, given the absence of physical boundaries. It is reasonable to think that such propagation will depend on the physical characteristics of the territory, for example, the altitude, the presence of mountains, or the proximity to the sea. The province-coefficients, μ_i, may capture such time-invariant, unobserved characteristics of provinces that explain permanent

differences in pollution across provinces. The vector $\mathbf{z}_{it}$ contains a set of variables that are likely to have an impact on the dependent variable. We expect climate conditions to have a composite impact on the concentration of pollutants. While on one hand side, pollution particles may be washed out by precipitation, they may also grow due to the absorption of water. For instance higher temperature may increase the oxidation rates of sulfur dioxide, nitrogen oxides and organic compounds, and promote particulate matter formation by boosting the concentration of oxidants in the air. However, higher temperature can shift the particle phase of more volatile compounds e.g., nitrate and volatile organic compound, to the gas phase, inhibiting for example PM formation and growth. Another factor that is likely to have an influence on the concentration of pollution is the amount of green present in the area. We also include the number of cars per inhabitant, and the number of people employed in the manufacturing sector. Cars contribute to particulate matter through fuel combustion or via the friction resulting from the contact of wheels to the road, which creates and spreads road dust. Fuel combustion also produces nitrogen oxides, volatile organic compounds, and CO. Various photochemical reactions between these three pollutants can result in the formation (or destruction) of O3. Larger manufacturing industries may lead to higher emissions of pollutants.

We note that the presence of the spatial lag of the dependent variable amongst the regressors in equation (1) generates an endogeneity problem in addition to the standard endogeneity issues arising in dynamic panels. Hence, we have decided to estimate this model by Generalised Method of Moments (GMM), following the approach proposed by a very recent literature (see, among others, Kukenova and Monteiro (2009)). In particular, we have adopted the GMM by Arellano and Bond (1991), where the standard set of instruments is augmented by the spatial lags of the regressors. We note that we have decided not to use the system GMM estimator in our study, in order to avoid a proliferation in the number of instruments.

In the second step, on the basis of the literature discussed in the preceding section, we consider the following model for hospital admission in province i at time t, adm_{it}:

$$adm_{it} = \alpha_i + \gamma t + \boldsymbol{\lambda}'\mathbf{p}_{it} + \boldsymbol{\beta}'\mathbf{x}_{it} + u_{it}, \tag{2}$$

where the province-specific coefficients, α_i, may capture time-invariant, unobserved characteristics of provinces, t is a time trend, $\mathbf{p}_{it}$ is a vector of pollutants, and $\mathbf{x}_{it}$ is a vector of control variables that may affect the dependent variable. Specifically, following previous literature, we have included the average temperature and precipitation as proxies of weather conditions, since low temperatures and high precipitations may contribute to deteriorate the health status of an individual thus increasing hospital admissions. We have also controlled for socio-economic characteristics of the area, by including unemployment rate, education, and population density

in our regression (see Janke et al., 2009). We have added the percentage of people regularly smoking, as this is known to be a major determinant of respiratory diseases. Finally, we have included a variable that measures the regional health deficits published annually by the Italian National Audit office (Corte dei Conti, 2010).

The dependent variable is hospital admissions due to COPD for children aged between 0 and 14 years old, and for people aged 65 and over, divided by total population. Since hospital admission is expressed as a rate, and therefore is bounded between 0 and 1, we take the logistic transformation and use as dependent variable $y_{it} = ln(adm_{it}/(1 - adm_{it}))$. As noted by Janke et al. (2009), the effects of pollution may be over-estimated if temporary elevated levels of pollution worsen the health of frail persons, for example the elderly, who would have been hospitalised anyway. While this problem may be severe when taking as dependent variable hospitalisation of the elderly, we believe that it is milder when focusing on hospital admissions for children. As for the selected pollutants, we check the effect of PM10, NO2, CO,and O3 included one by one in model (2), to isolate the impact of specific pollutants, and then simultaneously, to allow for correlation between them (Salam et al., 2005; Ritz et al., 2007; Bell et al., 2007; Coneus and Spiess, 2012).

We will estimate equation (2) by instrumental variables approach, where instruments are given by the vector $\mathbf{z}_{it}$ in (1), as well as the temporal lag, $p_{k,it-1}$, and the spatial lag, temporally lagged, $\overline{p}_{k,it-1}$. We also allow for spatial and serial correlation in the error term, using robust spatial correlation, heteroskedasticity-consistent (SHAC) standard errors for estimates, following the approach outlined in Moscone and Tosetti (2012). In the computation of SHAC standard errors we use the Parzen kernel function. Adopting SHAC standard errors is a very flexible approach that does not require specifying a spatio-temporal process for the error term (see also, Kelejian and Prucha (2007) on this).

4 Data

Data on air pollution are extracted from the AIRBASE database mantained by the European Environment Agency (EEA), while data on health outcomes and risk factors, as well as environmental data are gathered from the Italian Office of National Statistics, Istat. We refer to Table 1 for a formal definition of the variables involved in our study. Data provide information on pollution concentration from a total of 592 monitoring stations spread across Italy. From Figure 1, it is evident that stations distribute more densely in the North of Italy, while they are more sparsely spread in the Centre and South regions, and islands. Starting from the information at station level, we have computed a measure of pollution at provincial level adopting a procedure

similar to that advanced by Currie and Neidell (2005). In particular, for province i in year t we take the annual average daily concentration for each pollutant, registered by all monitoring stations whose distance to the centroid of the province is less than 30 kilometers (less than 15 kilometers for Milan and Rome where there are many monitoring stations within relatively small distances). By taking this approach, in certain years there are no stations around the centroid of some provinces, and therefore these provices will display missing values for our index. In line with previous studies (Neidell, 2004; Janke et al., 2009) we have considered several pollutants (PM10, NO2,CO, O3) that may cause problems of respiratory morbidity in the population.

Figure 1: Location of monitoring stations

Table 1: Definition of variables

Variables	Unit	Description
COPD		Chronic obstructive pulmonary disease
0-14	n./10,000 inhab.	n. cases in population aged 0-14
Over 65	n./10,000 inhab.	n. cases in population aged 65 and over
PM10	$\mu g/m^3$	Annual daily average of PM10
NO2	$\mu g/m^3$	Annual daily average of NO2
CO	$\mu g/m^3$	Annual daily average of CO
O3	$\mu g/m^3$	Annual daily average of O3
Precipitation	ml	Annual average precipitation
Temperature	$^\circ C$	Annual average maximum temperature
Smoking	n./100 inhab.	% of people smoking
Unemployment	n./100 inhab.	Unemployment rate
Green	m^2/inhab.	Green area per inhabitants
Cars	n./1000 inhab.	Cars per inhabintants
Manufact.	100 employ.	People employed in the industry sector
Education	n./100 inhab	% of people who completed high school
Pop. dens.	n./km^2	Population density
Deficit	1,000s Euro	Regional health deficits

Table 2 shows a set of descriptive statistics for the variables involved in our analysis. From this table, it emerges that the average daily concentration of PM10 within the year is 33 $\mu g/m^3$, with a maximum value of 61 $\mu g/m^3$ exceeding the limit of 50 $\mu g/m^3$ set by the European Community[1]. Nitrogen dioxide (NO2) has an average of 35.49 $\mu g/m^3$, with a peak of 68.14 $\mu g/m^3$, higher than the limit value of 40 $\mu g/m^3$, established by the European Community. The main artificial sources of NO2 are the central heating plants, some industrial processes and the exhaust gases of motor vehicles. Carbon monoxide (CO) can be generated by the incomplete combustion of materials containing carbon (e.g. fuels). It can also be emitted from combustion sources such as heating gas or motor vehicles. Its average concentration is 0.76 $\mu g/m^3$ with a maximum of 8.64 $\mu g/m^3$. The ozone (O3) in the atmosphere, is a important component of photochemical smog that even in low concentrations may cause respiratory irritation. The average daily concentration of O_3 within the year is 51.63 $\mu g/m^3$, with maximum points of 108.8 $\mu g/m^3$.

[1]The limits for the protection of health are set by Ministerial Decree 60/2002 for PM10 and NO2 and Legislative Decree 183/2004 for ozone.

Table 2: Descriptive statistics

Variables	Obs.	Mean	Std. Dev.	Min.	Max.
COPD 0-14	618	29.60	22.10	12.11	126.38
COPD 65 and over	618	76.15	37.10	11.30	265.91
PM10	491	33.26	8.19	5.48	61.51
NO2	409	35.89	11.15	4.14	68.14
CO	406	0.76	0.48	0.01	8.64
O3	398	51.63	11.44	16.03	108.8
Precipitation	618	780.89	173.68	406.00	1,378.70
Temperature	618	18.08	2.90	5.50	23.40
Smoking	618	7.24	1.64	3.90	11.12
Unemployment	618	7.33	4.28	1.85	21.60
Green	618	161.25	377.94	0.20	2,853.00
Cars	618	632.08	146.33	411.45	2,104.30
Manufact.	618	503.97	615.04	43.00	4,876.00
Education	618	27.43	3.90	20.31	37.96
Pop. dens.	618	1,213.19	1,374.12	78.50	8,508.70
Deficit	618	343.60	373.39	13.15	1,786.52

Figure 2 shows the quantile distribution of pollutants in 2009 (the last year of our analysis). The graphs show that largest concentrations of pollutants occur in areas around large cities and industrial districts, such as Turin and Naples.

In our study, hospital admissions due to COPD is used as an indicator of morbidity, differentiating between infant and elderly population.Admission rates for the elderly are considerably high, with an average of more than 76 individuals out of 10,000, reaching peaks of 266 admissions in Bari, a province in the South-East of Italy (see also Figure 3).

5 Results

Table 3 shows results from estimation of the first stage regression, model (1). The coefficient attached to $p_{i,t-1}$ is positive and significant for all pollutants, ranging between 0.254 for CO and 0.668 for PM10. This coefficient measures how persistent is the pollution over time, and is likely to reflect the enduring effects of the sources of pollution, both observable and unobservable. The coefficient attached to $\overline{p}_{it}$ is positive and significant for all pollutants except for O3. This

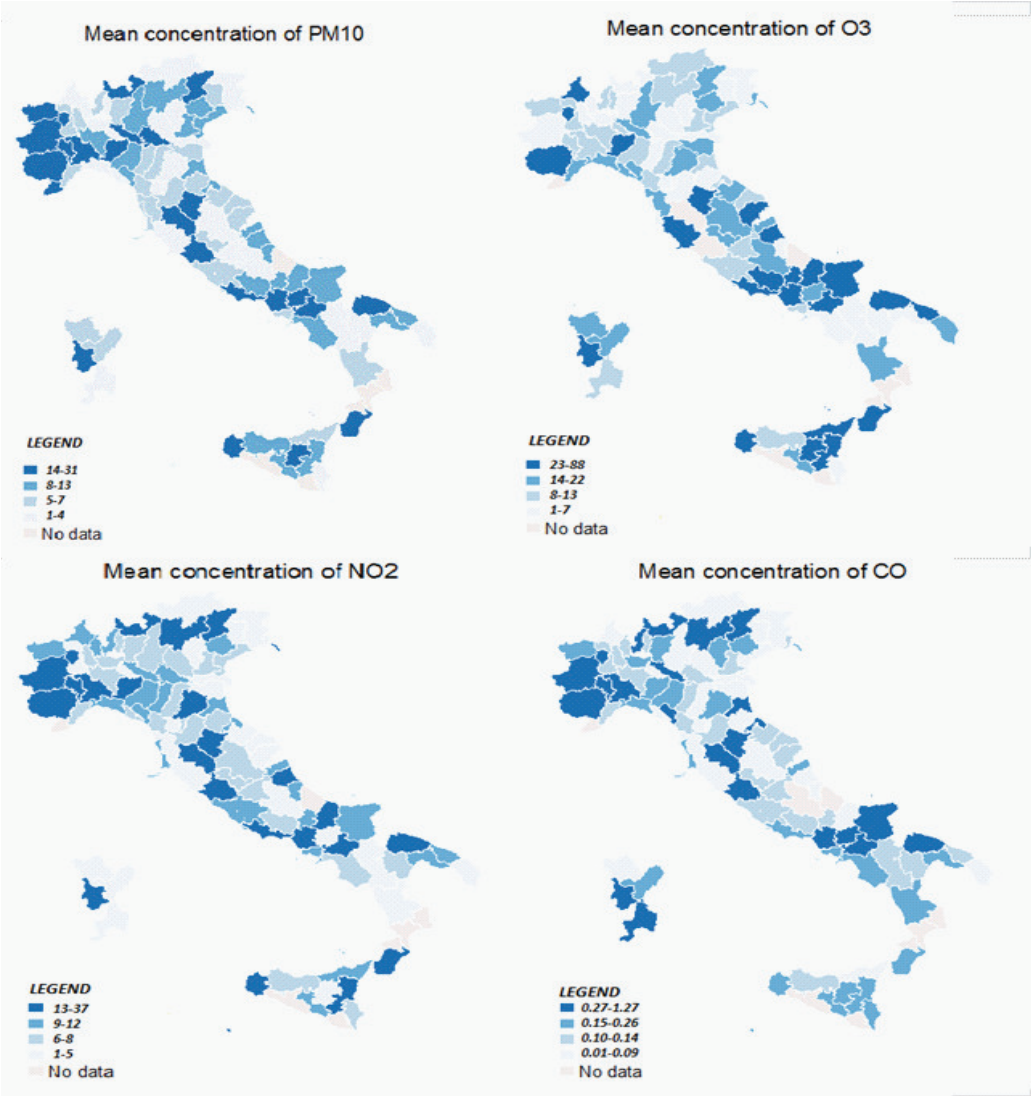

Figure 2: Quantile distribution of pollutants in 2009

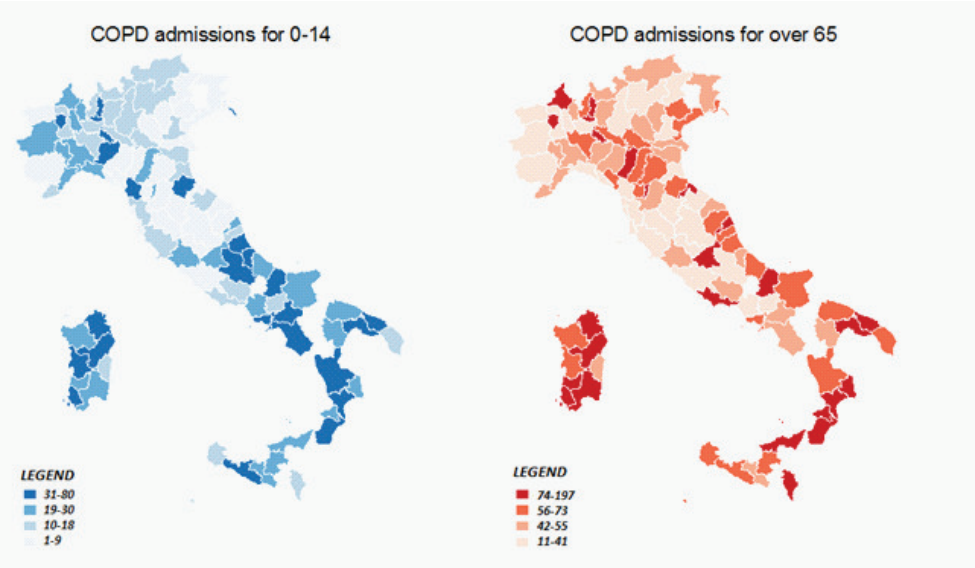

Figure 3: Quantile distribution of COPD in 2009 for people aged 0-14 and people aged 65 and over

result shows that pollution generated in one point in space is likely to diffuses across a wider area, which may include cities within the same region or from different regions. The spatial effect is particularly strong for NO2 and CO, with coefficients 0.729 and 0.603, repectively. As for the remaining determinants, wider green areas tend to reduce PM10 and CO, while the presence of manufacturing industry increases the concentration of these pollutants in the air; the number of cars boosts NO2. It is interesting to observe that O3 does not seem to be affected by any of these variables. One reason for this result is that this pollutant is not emitted directly by car engines or by industrial operations, but rather formed by the reaction of sunlight on air containing hydrocarbons and nitrogen oxides.

The reported Sargan tests does not reject the null hypothesis that the instruments adopted in GMM estimation are valid. Further, while, as expected, there is evidence of serial correlation of first order, we do not observe second-order serial correlation.

Table 3: Determinants of pollution

	PM10		**NO2**		**CO**		**O3**	
Variables	Coeff.	std.err.	Coeff.	std.err.	Coeff.	std.err.	Coeff.	std.err.
$p_{i,t-1}$	0.668*	0.109	0.419*	0.136	0.254*	0.122	0.564*	0.127
$\overline{p}_{it}$	0.331*	0.119	0.729*	0.249	0.603*	0.184	0.041	0.212
Green	-0.184*	0.053	-0.084	0.085	-0.004*	0.002	0.371	0.464
Temperature	-0.615	0.413	-1.042*	0.394	-0.004	0.008	-1.022	0.716
Precipitations	-0.001	0.003	1.543	2.232	-0.028	0.040	0.157	5.674
Cars	-0.010	0.032	0.049*	0.019	0.000	0.000	-0.034	0.071
Manufact.	0.015*	0.007	0.004	0.005	0.001*	0.000	-0.010	0.016
Sargan	34.29	[0.071]	23.452	[0.43]	32.50	[0.10]	27.57	[0.23]
Ser. corr								
AR(1)	-3.1882	[0.001]	-2.021	[0.043]	-2.033	[0.042]	-2.364	[0.018]
AR(2)	1.0473	[0.295]	1.149	[0.250]	-0.635	[0.524]	1.482	[0.138]

Notes: (*): significant at the 5 per cent significance level. p-values in square brackets.

Estimation of the second stage regression - defined in equation (2) - are presented as follow. Table 4 shows the output for COPD hospitalisation of people aged 0 to 14 years old, while Table 5 reports results for COPD hospitalisation of people aged 65 and over. Results show that PM10 and CO have an impact on hospital admissions for children while O3 has an influence on hospitalisation of the elderly. The results indicate that NO2 does not significantly affect hospital admissions by COPD. The variable precipitation is statistically significant in all regressions with a positive coefficient that ranges between 0.16 and 0.36. Density of population has a negative impact on admissions for young people in almost all models. This negative sign may be associate to a constrain capacity of the hospitals. However, we have failed to find a similar patterns for the elderly. Education seems to play a role in explaining variation in youth hospital admissions; a higher level of education in a province is associated with a higher probability of being admitted. More educated parents may have easier access on medical information, for example by consulting a General Practitioner (GP) or specialist, and therefore more able to identify and treat their children health conditions. The variable trend has a negative effect on all specifications. This may in part explain the the effect of medical technologies (e.g. the adoption of pharmaceuticals such as Bronchodilators, Steroids, etc) over time, that has reduced hospital admissions for both vulnerable categories of the population.

The Moran tests at the bottom part of the table, which have been performed on the residuals of the fixed effects, are positive and statistically significant. This confirms the appropriateness of the use of HAC standard errors.

Table 4: Determinants of COPD admission for people aged 0 to 14 years old

	p_{it} =**PM10**		p_{it} =**O3**		p_{it} =**CO**		p_{it} =**NO2**	
Variables	Coeff.	std.err.	Coeff.	std.err.	Coeff.	std.err.	Coeff.	std.err.
p_{it}	0.010*	0.002	0.0017	0.0022	0.0101*	0.0037	0.1903	0.1596
Temperature	-0.024	0.017	-0.0319	0.0203	-0.0191	0.0170	-0.0272	0.0189
Precipitation	0.342*	0.096	0.3420*	0.0947	0.3683*	0.0937	0.2941*	0.1010
Smoke	0.010	0.011	0.0137	0.0110	0.0116	0.0113	0.0148	0.0104
Unempl. rate	0.001	0.001	0.0001	0.0006	0.0003	0.0006	0.0000	0.0006
Pop. density	-0.025*	0.010	-0.0253*	0.0120	-0.0314*	0.0104	-0.0222	0.0114
Education	0.009*	0.003	0.0097*	0.0037	0.0083*	0.0037	0.0092*	0.0038
Deficit	0.001	0.000	0.0000	0.0002	0.0000	0.0002	0.0000	0.0002
Trend	-0.079*	0.010	-0.0878*	0.0110	-0.0789*	0.0095	-0.0844*	0.0095
Moran's I	5.07*	[0.00]	7.10*	[0.00]	12.11*	[0.00]	8.09*	[0.00]

Notes: (*): significant at the 5 per cent significance level. p-values in square brackets.

Table 5: Determinants of COPD admission for people aged 65 years and over

	p_{it} =**PM10**		p_{it} =**O3**		p_{it} =**CO**		p_{it} =**NO2**	
Variables	Coeff.	std.err.	Coeff.	std.err.	Coeff.	std.err.	Coeff.	std.err.
p_{it}	0.0047	0.0032	0.0059*	0.0026	0.0099	0.0057	0.1424	0.1050
Temperature	0.0363	0.0221	0.0395	0.0205	0.0489	0.0257	0.0382	0.0227
Precipitation	0.1619*	0.0635	0.2227*	0.0598	0.2788*	0.0520	0.2279*	0.0463
Smoke	0.0080	0.0068	0.0036	0.0087	0.0050	0.0092	0.0027	0.0092
Unempl. rate	-0.0008	0.0006	-0.0008	0.0006	-0.0007	0.0006	-0.0009	0.0006
Pop. density	0.0041	0.0077	0.0054	0.0083	0.0058	0.0075	0.0059	0.0083
Education	-0.0030	0.0024	-0.0029	0.0025	-0.0024	0.0025	-0.0024	0.0021
Deficit	-0.0002	0.0001	-0.0002	0.0001	-0.0003	0.0002	-0.0002	0.0002
Trend	-0.0946	0.0128	-0.1020	0.0112	-0.0981	0.0111	-0.1014	0.0132
Moran's I	6.90*	[0.00]	4.89*	[0.00]	7.87*	[0.00]	10.21*	[0.00]

Notes: (*): significant at the 5 per cent significance level. p-values in square brackets.

6 Concluding remarks

In this work, we have analysed the impact of pollutants on hospital admission for diseases related to chronic respiratory diseases in Italy. To control for possible measurement errors in our pollution variables, we have adopted a two stage estimation procedure. In the first stage, we have estimated a spatio-temporal model for our pollutants. Hence in the second stage regression, we have used as instruments the determinants of pollution to estimate its impact on hospital admissions. Our results show that PM10 and CO impact significantly on hospitalisation of young population, while O3 increases hospitalisation of the elederly. Other factors that appear to have an important role are rainfall and level of education. Our findings also show that air pollution seems to mostly determined by the presence of industrial plants, while the presence of green areas in cities lessens its concentration. For example, a recent study by Marinaccio et al. (2011) has indicated that Taranto, which is the province with the smallest green area per inhabitants while having a large manifacturing industry and a small residential vicinity to polluting facilities, shows high and increasing trends of pleural and lung cancers. Traffic does not seem to have a significant impact on pollution and therefore according to our results policies of alternating plates taken in many Italian cities may not be effective. The strong spatial effects

detected in pollution seem to suggest that any policy against pollution, to be effective, should be taken by a set of contiguous cities, rather than one single city. Also, our findings indicate that policies oriented to increase the presence of green, such as the creation of "green belts" around cities, like in Anglosaxon countries, may be effective in mitigating pollution generated within urban zone and therefore in the number of hospitalizations for respiratory deseases.

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