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Working Paper 17/279
November 2017

Economics Working Paper Series

Eidgenössische Technische Hochschule Zürich
Swiss Federal Institute of Technology Zurich

The Impact of Ambient Air Pollution on Hospital Admissions

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Abstract

Ambient air pollution is the environmental factor with the greatest impact on human health. Several epidemiological studies provide evidence for an association between ambient air pollution and human health. However, the recent economic literature has challenged the identification strategy used in these studies. This paper contributes to the ongoing discussion by investigating the association between ambient air pollution and morbidity using hospital admission data from Switzerland. Our identification strategy rests on the construction of geographically explicit pollution measures derived from a dispersion model that replicates atmospheric conditions and accounts for several emission sources. The reduced form estimates account for location and time fixed effects and show that ambient air pollution is strongly correlated with hospital admissions. In particular, we find that SO2 and NO2 are positively associated with admission rates for coronary artery and cerebrovascular diseases. As a robustness check, we adopt instrumental variable methods to account for the possible endogeneity of pollution measures. These results may contribute to a more accurate evaluation of future environmental policies aiming at a reduction of ambient air pollution exposure.

Keywords: Ambient air pollution, dispersion model, hospital admissions, count panel data

JEL Codes: I10, Q51, Q53

Acknowledgement: We thank the Swiss Federal Office for the Environment (FOEN) for providing the ambient air pollution data and the Swiss Federal Statistical Office (FSO) for making available the hospitalization data. The corresponding author of this paper is Sandro Steinbach. *Massimo Filippini*: Department of Management, Technology and Economics (D-MTEC), Swiss Federal Institute of Technology in Zurich (ETH Zurich), Zurich, Switzerland, and Institute of Economics (IdEP), Università della Svizzera Italiana (USI), Lugano, Switzerland *email*: mfilippini@ethz.ch. *Giuliano Masiero*: Institute of Economics (IdEP), Università della Svizzera Italiana (USI), Lugano, Switzerland, and Department of Management, Information and Production Engineering (DIGIP), University of Bergamo, Bergamo, Italy, *email*: giuliano.masiero@usi.ch. *Sandro Steinbach*: Department of Management, Technology and Economics (D-MTEC), Swiss Federal Institute of Technology in Zurich (ETH Zurich), Switzerland, *email*: ssteinbach@ethz.ch.

1. Introduction

Even though air quality has improved substantially in the last decades, ambient air pollution is still the environmental factor with the greatest health impact in developed countries. The World Health Organization (WHO) has estimated that exposure to ambient air pollution is responsible for health care expenses of more than US$ 1.27 trillion in Europe alone (WHO Regional Office for Europe and OECD, 2015). The significant decrease in pollution exposure in industrialized countries is largely due to stricter environmental regulations and technological progress. However, a substantial proportion of the European population is still exposed to levels of air pollution that are above national and international air quality standards (European Environmental Agency, 2015). Among those air pollutants, particulate matter (PM), nitrogen dioxide (NO2), sulfur dioxide (SO2), and ground-level ozone (O3) are considered to have the largest health impacts. These pollutants are associated with higher mortality and morbidity rates (WHO, 2005). Although the literature on the relationship between ambient air pollution and mortality is extensive, the empirical evidence on the association between ambient air pollution and morbidity is still far from being conclusive. The limited evidence is mainly due to restricted access to patient-level data with sufficient geographical resolution. Even when detailed data are available, the identification strategy is challenged by imprecise pollution measures and unobserved factors that are correlated with the treatment variable (Knittel et al., 2016; Schlenker and Walker, 2016).

This paper builds on recent advances in the economic literature and aims at identifying the relationship between air pollution exposure and morbidity in the general population. We exploit space and time variation in hospital admissions data for specific disease categories covering the entire Swiss population between 2001 and 2013. Moreover, we use a novel approach to measure pollution exposure which builds on a mathematical simulation model that replicates the atmospheric conditions and simultaneously accounts for various emission sources. The geographical resolution of our analysis is the MedStat region, a spatial concept used by the Swiss authorities to anonymize patient-level data. This resolution allows for a more accurate assignment of pollution measures and a more

precise identification of the treatment effect as compared to previous studies. The level of aggregation is prone to systematic measurement error, as a single monitoring site for pollution is assumed to be representative of a large and likely heterogeneous area. On average, the MedStat regions have a size of about 12,000 inhabitants, which is substantially more detailed than the usual level of aggregation which is at the zip code, county or even city level (e.g., Currie et al., 2009; Luechinger, 2014; He et al., 2016; Knittel et al., 2016; Schlenker and Walker, 2016).[1]

Our contribution to the growing economic literature on the relationship between air quality and human health is threefold. First, we explore the association between ambient air pollution and hospital admissions that gained little attention in the literature so far. Second, we address the measurement error in the treatment variable using geographically explicit air pollution measures derived from a dispersion model. Prior studies solely rely on the inverse distance interpolation approach to compute measures of local pollution exposure which can lead to systematic estimation bias if the monitoring network is coarse. Third, we investigate differences in the treatment effect for major air pollutants at the disease level. Although previous studies recognized this issue, they usually look at a single pollutant and do not account for the wide range of air pollutants.

The economic literature on the relationship between ambient air pollution and human health is extensive. A large body of this literature is concerned with the impact of ambient air pollution on infant health and general mortality (e.g., Chay and Greenstone, 2003; Currie et al., 2009; Luechinger, 2014; Sanders and Stoecker, 2015; He et al., 2016; Knittel et al., 2016). These studies use explicit location information to show that ambient air pollution has a negative and lasting impact on birth outcomes, fetal death rates, and general mortality. The recent interest on the impact of ambient air pollution on morbidity is mainly due to better access to patient-level data. For instance, Schlenker and Walker (2016) investigate the impact of air pollution on morbidity using individual-level data

[1] For instance, the study by He et al. (2016) relies on Chinese city-level mortality data. Because a city in China can be relatively large and heterogeneous, the estimated level of pollution exposure may be significantly different to the true level of pollution exposure. Schlenker and Walker (2016) conduct their analysis using zip code level data for California. The average size of a zip code in California is above 37,000 inhabitants, ranging between 11,000 and more than 100,000 inhabitants.

from California. They find that carbon monoxide exposure is positively associated with hospitalization rates. These findings support the results of Beatty and Shimshack (2014) who estimate the impact of carbon monoxide exposure on respiratory health outcomes among children based on cohort data from England.

A general concern of the literature is the potential measurement error of pollution exposure. Two approaches are commonly used to compute measures of pollution exposure at the location level. The prevalent approach builds on the assumption that the concentration of air pollutants is homogenous within a given region, implying that a monitoring site is representative of a wide geographical area. The homogeneity assumption is violated whenever the topography has a strong effect on the dispersion of air pollutants. Therefore, this approach can induce systematic measurement bias in the estimation of the treatment effect. As an alternative, spatial interpolation methods are used to address the homogeneity issue. Although various interpolation methods are applied in the literature, these methods differ only in the choice of sample weights. The most frequently used weighting method is the inverse distance approach (e.g., Currie et al., 2009; Lagravinese et al., 2014; Knittel et al., 2016; Schlenker and Walker, 2016). This method attributes higher weight to monitoring sites that are close to the site where the prediction is made. A downside of the inverse distance approach is that it does not account for emission sources and atmospheric conditions. Therefore, both approaches are prone to measurement error and have the potential to induce systematic bias in the estimation of the treatment effect.

The economic literature has resorted to instrumental variable (IV) estimation techniques to address the endogeneity issue arising from measurement error. For instance, Knittel et al. (2016) use variation in traffic shocks and local weather conditions, and Schlenker and Walker (2016) use airport congestion and weather conditions as instrumental variables. Lagravinese et al. (2014) choose a different route by instrumenting spatial and temporal lags of the interpolated pollution measures. Although the IV approach is a viable option to address the endogeneity issue, it is only applicable when appropriate instruments are available. In this paper, we propose to solve the measurement problem at the source instead of relying on statistical methods. We introduce a novel approach to compute geographically

explicit and reliable measures of pollution exposure derived from a dispersion model. This approach allows for a more accurate estimation of the treatment effect as compared to previous studies.

Our reduced form estimates account for location and time fixed effects and show that ambient air pollution is positively associated with hospital admissions for cardiovascular and respiratory diseases in the general population. Moreover, our results show that the inverse distance approach is prone to measurement bias, leading to negative and significant coefficient estimates for some pollutants. The results for the dispersion model approach are robust to different distributional assumptions and non-linearity in the treatment effect. As a further robustness check, we account for omitted variables with IV methods. These control function estimates convey a similar picture as our baseline results. In particular, we find that the impact of SO2 and NO2 on admissions for cardiovascular diseases is statistically significant and robust. Lastly, by distinguishing between elective and emergency admissions, we find that the positive treatment effect mainly operates through emergency admissions.

The remainder of the paper is organized as follows. Section 2 describes the data used in the empirical analysis. We first introduce the dispersion model approach and show how this approach addresses the endogeneity issue provoked by measurement error. We then discuss our choice of morbidity data, explain the selection of causes of hospital admissions, and introduce the covariates used in the empirical analysis. Section 3 explains the empirical model and our estimation strategy. We summarize the estimation results in Section 4 and also discuss a variety of robustness checks. Section 5 provides some conclusions.

2. Data

To assess the relationship between ambient air pollution and hospital admissions, it is necessary to carefully define the geographical level at which the analysis should be performed. Ideally, we would measure pollution exposure at the patient level. However, such detailed patient information is not available due to privacy concerns. The most detailed geographical resolution at which hospital admission data are available in Switzerland is the

MedStat region level. The MedStat regions are a geographical concept used by the FSO to anonymize individual-level hospital admission data. An advantage of these data is that the 604 MedStat regions are homogenous with respect to the population size, with each of them containing about 12,000 people. It is important to note that the administrative definition was updated in 2008 to account for population growth. Based on postal codes for 2007, the old MedStat regions were split up or combined to form new MedStat regions. Therefore, it is impossible to study hospital admissions over the structural break without reassigning the data from the new to the old definition of MedStat region. We accomplish this task by matching postal codes underlying the MedStat regions over the structural break. We obtained detailed information on the general population at the postal code level for 2010 from the FSO. We use this information to create weights and recode the location information in order to obtain a match between the new and the old definition. We then reassign the morbidity data over the structural break using population weights.[2]

2.1 Ambient air pollution data

To calculate the measures of pollution exposure for the dispersion model approach, we obtained geographically explicit data on ambient air pollution from the Swiss Federal Office for the Environment (FOEN, 2016). These data are prepared by a mathematical simulation model, which is described in Heldstab et al. (2013). The model simulates the dispersion of air pollutants in Switzerland using a two-part procedure. The first part of the procedure is concerned with the emission modeling. Emission inventories are prepared on rectangular grids with a geographical resolution of 200 meters, taking into account all major emission sources. These sources are road traffic, households, agriculture and forestry, railway and aviation, as well as industrial and commercial activities. The model considers both domestic and foreign emission sources. It is necessary to account for these sources because a considerable share of emissions in Switzerland has a foreign origin.

The second part of the procedure is concerned with the concentration modeling. The

[2] We perform a number of robustness checks to ensure that the reassignment method does not affect the identification. For instance, we use gridded housing data from the Swiss land register to accomplish the recoding of location information. The estimation results are available upon request from the authors. These results are similar to the estimates obtained with our baseline specification.

dispersion model uses pollutant-specific transfer functions to replicate the atmospheric dispersion of PM10, NO2, SO2, and O3, providing measures of annual concentration for each pollutant. A Gaussian plume dispersion is applied to generate these functions. Each transfer function measures the average impact of an emission source on the surrounding area. The model also accounts for topographical variability by constructing separate transfer functions for each of the four main topographical areas in Switzerland.[3] Moreover, the transfer functions consider atmospheric conditions, which include wind speed and direction, air temperature and mixing height. The correlation between observed and predicted pollution concentrations for PM10, NO2, SO2, and O3 is above 80 percent. Therefore, we believe that the dispersion model approach produces a more precise measure of local pollution exposure than the inverse distance approach, resolving the endogeneity issue that arises from measurement error at the source. Conversely, the inverse distance approach is less precise because the pollution concentration is solely determined by the inverse distance of a location centroid to a set of monitoring sites.

For the purpose of comparison, we also calculate pollution exposure for PM10, NO2, SO2, and O3 using the inverse distance approach:

$$\widehat{p}_{it} = \frac{\sum_{j=1}^{n} \frac{1}{d_{ij}} p_{jt}}{\sum_{j=1}^{n} \frac{1}{d_{ij}}}, \tag{1}$$

where $\widehat{p}_{it}$ is the interpolated pollution level for the centroid of each MedStat region. We denote the distance between a region centroid i and a pollution monitoring site j with d_{ij}. The monitoring-site pollution data are also obtained from the FOEN. We follow the literature and limit the interpolation to monitoring sites with a Euclidian distance less than 30 km to the location where the prediction is made (Currie et al., 2009; Knittel et al., 2016). The geographical extent of the pollution monitoring network in Switzerland is illustrated in the online supplementary material (Figure A1).

[3] The main topographical areas are the Swiss plateau (North of the Alps), the Basel region with specific climate conditions due to the Rhine valley, the Alpine region (valley floors of all alpine valleys), and the remaining parts. Additional information on these regions are provided in Heldstab et al. (2013).

Because pollution data from the dispersion model are available at a more detailed geographical resolution than the MedStat region level, we compute a representative measure of air pollution exposure for each MedStat region. To uniquely assign each grid cell to the corresponding MedStat region, we use a Geographic Information System (GIS). If a grid cell overlaps two or more regions, we assign the cell to the MedStat region that contains the larger part of the cell area. To obtain a measure of air pollution for each MedStat region, we calculate a population-weighted measure of pollution concentration. We exclude grid cells without population, using information from the Swiss land register (FSO, 2016*a*). It is necessary to exclude these cells because people spend most of their time in populated areas, implying that a pollution measure based on all grid cells would understate the actual pollution exposure, particularly in mountainous regions. Consequently, the estimates of the treatment effect would be systematically biased.

We use the annual arithmetic mean of the daily pollution exposure because this measure is a major legislative target in the Swiss federal law on air pollution (Federal Council, 2016).[4] The average concentration of ambient air pollution is calculated for each MedStat region and year. We illustrate the geographical variation in the pollution exposure for PM10, NO2, SO2, and O3 in Figure 1. The four maps show the average daily pollution exposure by MedStat region in the period 2001 to 2013. To enable the visual comparison between pollutants, we use a grouping algorithm to find natural breaks in the pollution data. For each pollutant, we identify ten classes, where the bright green color stands for the lowest class and the dark red color for the highest class. The data indicate distinct patterns of air pollution exposure in Switzerland. The exposure is clearly higher in urban areas, and the Southern cantons exhibit the highest pollution exposure. The descriptive statistics of the treatment measures, as well all the other variables considered in the econometric analysis, are presented in the online supplementary material (Table A1). With the exception of O3 exposure, all pollutants show a negative time trend, and the average pollution levels are below the threshold defined by the Swiss air pollution legislation.

[4] Other moments of the pollution distribution function (e.g., annual median, minimum and maximum) could be also relevant for hospital admissions. However, the use of other moments of pollution exposure is limited by a data protection agreement between the FOEN and external data providers.

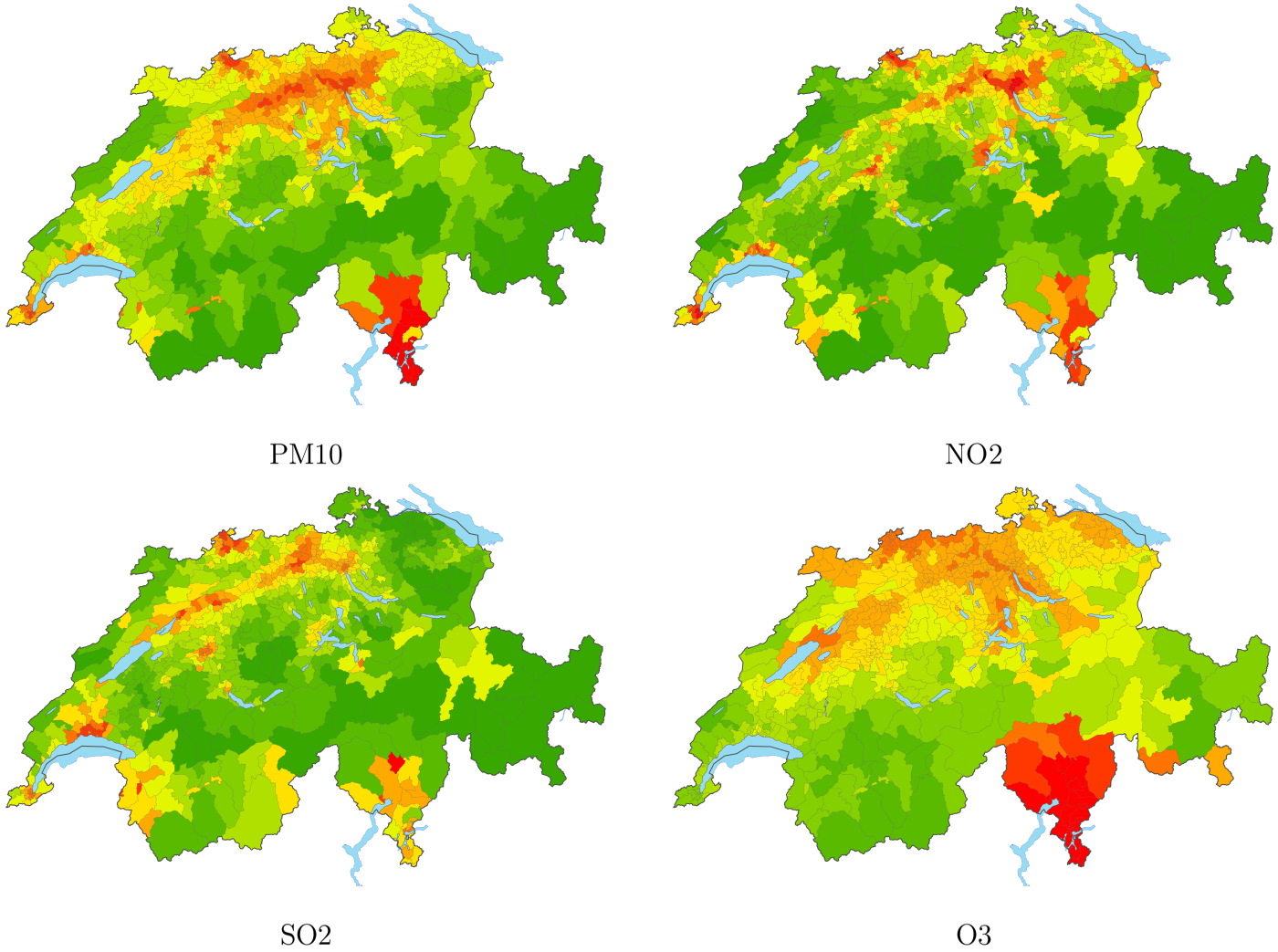

Figure 1: Geographical variation of pollution exposure (average for the period 2001 to 2013).

Note: The four maps show the average daily pollution exposure by MedStat region. The bright green color indicates the lowest level of pollution whereas the dark red color indicates the highest level of pollution.

2.2 Hospital admission data

We obtained hospital admission data from the Medical Statistics of the Hospitals maintained by the FSO (2016*b*). These data are collected by the Swiss cantons and include a wide array of information on people that are admitted to the hospital. Since 1998, Swiss hospitals are obliged by the federal law to submit patient-level data. According to the FSO, the dataset covers 99.9 percent of hospital admissions. Because the quality of data is restricted before 2001, we drop earlier years and focus on patients who were stationary treated in the period 2001 to 2013. Following Schlenker et al. (2015), we select patients based on their main and secondary diagnosis and include both emergency and elective admissions. The causes of hospital admissions considered in this analysis are listed in Table 1. The table provides information on the cause of hospital admissions, the relevant ICD-10 codes, and a brief description of each cause of hospital admission. We select these causes based on the extensive literature review in WHO (2005) and European Environmental Agency (2015). Therefore, we focus on hospital admissions for cardiovascular and respiratory diseases. We also look at more disaggregated causes of hospital admissions, allowing for a better understanding of the disease-specific treatment effects. To ensure the validity of our identification strategy, we include two negative control outcomes. These common health outcomes are diabetes and diseases of middle ear and mastoid.

Table 1: Investigated causes of hospital admissions

Cause of hospital admissions	ICD-10 code	Description
All cardiovascular diseases	I00-I99	All diseases that are related to the cardiovascular system.
Coronary artery disease	I20-I25	Stable angina, unstable angina, myocardial infarction, and sudden coronary death.
Cerebrovascular disease	I60-I69	Vascular disease of the cerebral circulation.
All respiratory diseases	J00-J99	All conditions of the upper respiratory tract, trachea, bronchi, bronchioles, alveoli, pleura and pleural cavity, and the nerves and muscles of breathing.
Pneumonia	J12-J18, P23	Inflammatory condition of the lung.
COPD	J40-J44	Obstructive lung disease characterized by chronically poor airflow.
Asthma	J45-J46	Chronic inflammatory disease characterized by variable and recurring symptoms, reversible airflow obstruction and bronchospasm.
Diabetes	E10-E14	Metabolic disease in which there are high blood sugar levels over a prolonged period.
Diseases of middle ear and mastoid	H65-H75	All diseases related to the middle ear and mastoid.

2.3 Control variables

The relationship between ambient air pollution and hospital admissions may be confounded by factors that vary across MedStat regions over time. Among others, such factors are population characteristics, economic conditions and access to outpatient and hospital care facilities.[5] To account for population characteristics, we use registry information from the FSO. We compute a measure of population size to capture changes in the demand for hospital care that are unrelated to changes in pollution exposure. We also consider the share of foreigners, the share of females, and the share of the working-age population in the total population. These variables are supposed to account for migration patterns and the effect of age and gender composition on hospital admissions. Moreover, we include a number of economic covariates: the average household income, an income equality measure, and the unemployment rate. Household income and inequality data are obtained

[5] As for possible border effects, note that the dispersion model already accounts for these effects by construction, since it considers emissions sources in adjacent regions.

from the Swiss Federal Tax Administration (FTA), and unemployment data from the Swiss State Secretariat for Economic Affairs (SECO). These variables are supposed to capture changes in the financial abilities of the population. Finally, we account for access to outpatient care with a measure of the number of ambulatory doctors, and for access to hospital care with a measure of the number of stationary doctors.[6]

Some additional factors may complicate the identification of the relationship between ambient air pollution and hospital admissions. On the one hand, people living in regions with poor air quality may have a worse health status for reasons that are unrelated to ambient air pollution. For instance, accessing preventive medical care services can be more difficult in certain regions. This could induce a systematic bias in the parameter estimates. On the other hand, people may live in regions with good air quality because they derive utility from unobserved location characteristics that are confounded with air quality. Among others, such characteristics are the availability of recreational infrastructure and a lower density of commercial and industrial infrastructure. When these factors are not accurately taken into account, we could obtain spuriously large estimates of the air pollution effect on hospital admissions. Given our limited knowledge of factors affecting the selection of people into certain geographical locations, standard ordinary least squares (OLS) estimates are likely biased. The potential for selection bias calls for an identification strategy that captures the influence of confounding factors.

[6] Ideally, we would account for access to hospital care with a measure of distance to the nearest hospital. However, such data are not available for the entire study period.

3. Empirical approach

To account for unobserved factors, we exploit the panel structure of our data and include both location and time fixed effects in the following count model:[7]

$$adm_{it} = \exp(\alpha_i + \beta p_{it} + \boldsymbol{X}_{it}\boldsymbol{\gamma}_x + \delta_t)\eta_{it}, \tag{2}$$

where i is the MedStat region and t is the year. We denote the location fixed effects by α_i, and time fixed effects by δ_t. These variables are supposed to account for the influence of unobserved confounding factors. The location fixed effects address unobserved heterogeneity between MedStat regions. $\boldsymbol{X}_{it}$ is the matrix of covariates that vary at the region level over time, and η_{it} is the multiplicative error term. The treatment variable is p_{it}, measuring the average pollution exposure for PM10, NO2, SO2, and O3, and the key parameter of interest is β. This parameter is supposed to measure the effect of ambient air pollution on hospital admissions.[8]

We consider two model specifications to address unobserved heterogeneity over time. Our baseline specification includes common time fixed effects, whereas our preferred specification includes canton-time fixed effects.[9] We prefer the specification with flexible time fixed effects because the Swiss cantons have some autonomy in designing health policy instruments. In this way, we can account for shocks generated by cantonal health policies.[10] Moreover, the canton-time fixed effects are supposed to control for other time-variant factors, such as the progression of diseases, that are predictive of the outcome

[7] Another approach would be to include spatial effects in the regression model. For this reason, we also estimate spatial lag panel models for count data (see e.g., Cameron and Trivedi, 2013). The spatial estimates are very similar to the results of our main model and indicate that spatial lags are of minor relevance for most causes of hospital admissions.

[8] A potential source of bias is the correlation of pollution measures. To address this issue, we estimate the relationship between pollution exposure and hospital admissions using single regressors of pollution. Therefore, Equation 2 estimates different models for each pollutant. This approach is required because the linear correlation coefficient for pollution measures varies between 0.3 and 0.7. As emission sources are correlated in space and over time, a positive correlation between PM10, NO2, SO2, and O3 is expected.

[9] Switzerland is a federal state of 26 cantons. Each canton has its own constitution, legislature, and government. Among others, the cantons are responsible for healthcare services, welfare, law enforcement and public education.

[10] For instance, Switzerland has recently introduced a new hospital financing system to promote cost-effective health care. Although the system was simultaneously introduced in all cantons, the reimbursement rates for medical treatment are different between cantons.

and correlated with the treatment. Among others, such unobserved variables are the relocation of sicker people into areas with better air quality and the access to transport facilities. Ideally, we would like to track people over time and space to explore the temporal component of pollution exposure and, therefore, account for the possible progression of diseases. However, such detailed information is not available for the Swiss population. In any case, the location and canton-time effects should allow us to circumvent the endogeneity problem. Furthermore, we can exclude that sick people are more likely to move because of easier hospital admissions since waiting times in Switzerland are generally absent.

Following Schlenker et al. (2015), the outcome variable in our regression model is denoted by adm_{it}, representing the non-negative integer count of hospital admissions.[11] One might transform the outcome variable and then estimate the relationship using a linear regression model. Although this approach is practicable for particular types of data, it is inappropriate when the outcome is a count. As discussed in Wooldridge (1999), the linear regression model does not ensure positivity for the predicted values of the count outcome. Moreover, the discrete nature of the count outcome makes it difficult to find a transformation with a conditional mean that is linear in parameters. Finding such a transformation is a particular problem in the presence of heteroskedasticity as the transformed errors would be correlated with the covariates, leading to inconsistent estimates of the treatment effect. Even when the transformation of the conditional mean is correctly specified, it would be impossible to measure the relationship of primary interest. Hence, we model the relationship between the health outcome and the covariates directly, using the exponential form to ensure positivity for the covariates as shown in Equation 2. An advantage of the exponential form is that the response variable can follow different distributional assumptions.

To explore the relationship between treatment and count outcome, we use the Poisson pseudo-maximum likelihood (PML) estimator (Gong and Samaniego, 1981; Gourieroux

[11] We are aware that several studies in the health economics literature use admissions per population as the outcome variable. However, the absolute number of admissions is more appropriate in this context since it allows to use a count-data model that reflects the data generating process of hospital admissions due to pollution exposure.

et al., 1984). The Poisson PML estimator is consistent in the presence of heteroskedasticity, and even if the conditional variance is not proportional to the conditional mean, the Poisson PML estimator is consistent (Wooldridge, 1999; Cameron and Trivedi, 2013). Because the Poisson PML estimator makes no assumption on the dispersion of the fitted values, we do not need to test for this aspect. An advantage of the Poisson PML estimator is that the scale of the dependent variable has no effect on the parameter estimates, which is a challenge for the Negative Binomial PML estimator. Moreover, as long as the conditional mean is correctly specified, the Poisson PML estimator yields estimates that are similar in size to the estimates of both the Gaussian and Negative Binomial PML estimators. To ensure that the distributional assumption has no impact on the parameter estimates, we also estimate Equation 2 using these alternative PML estimators (see the online supplementary material). Lastly, to address heteroskedasticity in the error term, we use a robust variance estimator and account for clustering at the MedStat region level (Cameron and Miller, 2015).

4. Results

We first explore the relationship between ambient air pollution and hospital admissions with the baseline specification and then extend this analysis by comparing the effect of different distributional assumptions and testing for non-linearity in the treatment effect. We also use the control function approach to account for omitted variables. These variables may induce an endogeneity issue that could affect the validity of our parameter estimates. Lastly, we compare the effect of ambient air pollution for elective and emergency hospital admissions.

4.1 The effect of ambient air pollution on hospital admissions

We commence our empirical analysis by exploring the relationship between ambient air pollution and hospital admissions in the general population. Table 2 summarizes the Poisson PML estimates for the investigated causes of hospital admission. All specifications include covariates and fixed effects for MedStat regions and time.[12] We report the estimates of the treatment effect measuring the air pollution using the inverse distance approach in columns 2-5, and the dispersion model approach in columns 6-9.[13] As suggested earlier, our regression results indicate an endogeneity problem for the inverse distance approach as most estimates have a negative sign or are not statistically significant at the 10 percent confidence level.[14] Moreover, the statistically significant and negative parameter estimates for diseases of the middle ear and mastoid provide further evidence for an endogeneity issue induced by the inverse distance approach. This inconsistency is the primary concern of our analysis and the reason why we advance the use of a dispersion model approach to solving the endogeneity issue. Therefore, the remaining discussion is solely concerned with the parameter estimates of the dispersion model approach as these estimates are not affected by measurement bias.

[12] We do not report the estimates of the control variables because of space limitations. The table shows the estimates of 72 (9x8) regressions. The estimates including all covariates are available upon request from the authors.

[13] Note that the effects of different pollutants are comparable since they are all measured in $\mu g/m^3$.

[14] The negative parameter estimates for some types of pollution are likely caused by insufficient variation in the measures of exposure. For instance, since there are only a few SO2 monitoring sites, the within variation in pollution exposure is limited, inducing collinearity between the fixed effects and the measures of pollution exposure.

Table 2: Baseline specification with MedStat region and time fixed effects (Poisson PML estimates)

Cause of hospital admissions	Inverse distance approach				Dispersion model approach			
	PM10	NO2	SO2	O3	PM10	NO2	SO2	O3
All cardiovascular diseases	0.214	−0.146	−0.216	−0.030	1.676***	1.848**	1.528*	0.217***
	(0.224)	(0.144)	(0.420)	(0.047)	(0.435)	(0.891)	(0.789)	(0.075)
Coronary artery disease	0.365	−0.104	−1.078*	−0.100	1.954***	2.974***	3.636***	0.241**
	(0.278)	(0.203)	(0.634)	(0.064)	(0.584)	(1.079)	(1.099)	(0.094)
Cerebrovascular disease	0.088	−0.270	0.041	0.001	1.510*	2.602**	1.950*	0.277**
	(0.385)	(0.221)	(0.562)	(0.074)	(0.777)	(1.251)	(1.029)	(0.122)
All respiratory diseases	0.078	0.191	−0.595	−0.108**	0.848	−0.737	−0.525	0.135*
	(0.317)	(0.169)	(0.408)	(0.052)	(0.595)	(0.871)	(0.842)	(0.079)
Pneumonia	0.168	0.175	−1.162**	−0.052	0.480	−0.508	−1.473	0.143
	(0.338)	(0.217)	(0.477)	(0.070)	(0.776)	(1.151)	(1.151)	(0.099)
COPD	−0.463	−0.394	−2.330**	−0.225**	−0.914	−1.324	−0.995	0.067
	(0.486)	(0.295)	(0.908)	(0.109)	(1.055)	(1.406)	(1.387)	(0.148)
Asthma	0.644	−0.805*	−1.309	−0.680***	3.681**	1.049	−1.569	−0.264
	(0.734)	(0.487)	(1.105)	(0.158)	(1.753)	(1.752)	(2.288)	(0.220)
Diabetes	−1.115**	−0.719**	−1.146	−0.159	0.616	−0.125	0.587	0.220
	(0.444)	(0.325)	(0.988)	(0.130)	(1.212)	(1.345)	(2.389)	(0.165)
Diseases of middle ear and mastoid	−2.919***	−0.232	−2.159**	−0.566***	−2.620	−2.708	−3.547	−0.379*
	(0.871)	(0.477)	(1.096)	(0.155)	(1.792)	(2.150)	(2.794)	(0.221)

Note: This table reports the estimates of the treatment effect obtained by estimating different models for each pollutant. All estimates and standard errors are rescaled (x100). The sample consists of 604 MedStat regions for the period 2001 to 2013. We assume that the data generating process follows a Poisson distribution. All regressions include control variables and both year and MedStat region fixed effects. The regression results are sorted by cause of hospital admissions. Columns 2-5 report the estimates of the treatment effect by pollutant for the inverse distance approach, and columns 6-9 the results for the dispersion model approach. The heteroscedasticity-robust standard errors are provided in parenthesis, and are adjusted for within cluster correlation at the MedStat region level. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent, respectively.

The Poisson PML estimates based on the dispersion model approach explain between 72 and 86 percent of the variation, and the pollution measures account for 0.4 to 7.2 percent of the overall variation in hospitalization data.[15] The estimates provide evidence for a significant association between ambient air pollution and hospital admissions for cardiovascular diseases, but only weak evidence for respiratory diseases. Except for PM10 exposure and hospital admissions for asthma, all estimates for respiratory diseases are not statistically significant. Conversely, we find evidence for a positive association between pollution exposure and hospital admissions for cardiovascular diseases. The strongest association is found for SO2, for which a 1 unit increase in pollution exposure is associated with a 3.7 percent increase in the incidence of hospital admissions for coronary artery diseases. Although the estimates for O3 exposure are all statistically significant, the treatment effect is in general much smaller than for PM10, NO2 and SO2.

Although our baseline specification with MedStat region and time fixed effects provides evidence for a positive association between ambient air pollution and hospital admissions for cardiovascular diseases, it is possible that unobserved location characteristics varying between cantons and over time are correlated with the measures of air pollution. To account for these factors, we allow the time fixed effects to be flexible and estimate Equation 2 with canton-time fixed effects. The regression results are provided in Table 3. Again, we compare the estimates using the inverse distance approach with the estimates of the dispersion model approach. The negative and non-significant estimates for the inverse distance approach provide further evidence of an endogeneity issue. Conversely, the estimates for the dispersion model approach show the expected sign and are statistically significant.

[15] As a measure of explanatory power, we use the Pseudo R-squared value, which is defined as the squared correlation between predicted and observed count outcome.

Table 3: Preferred specification with MedStat region and canton-time fixed effects (Poisson PML estimates)

Cause of hospital admissions	Inverse distance approach				Dispersion model approach			
	PM10	NO2	SO2	O3	PM10	NO2	SO2	O3
All cardiovascular diseases	0.153	−0.128	−0.151	−0.082	1.071	2.681***	3.369***	0.112
	(0.241)	(0.173)	(0.362)	(0.076)	(0.808)	(1.013)	(1.151)	(0.134)
Coronary artery disease	−0.091	−0.139	−1.266**	−0.147	1.200	4.845***	3.502***	0.216
	(0.370)	(0.257)	(0.540)	(0.108)	(1.156)	(1.345)	(1.343)	(0.198)
Cerebrovascular disease	0.533	−0.503**	−0.708	−0.197**	0.047	3.433**	4.456***	−0.080
	(0.430)	(0.250)	(0.591)	(0.097)	(1.409)	(1.569)	(1.686)	(0.268)
All respiratory diseases	−0.243	0.176	−0.517	−0.170**	−0.792	0.343	2.058*	−0.095
	(0.294)	(0.177)	(0.471)	(0.084)	(0.896)	(1.077)	(1.181)	(0.154)
Pneumonia	0.435	0.019	−1.590***	−0.118	−1.250	−0.180	0.461	−0.053
	(0.436)	(0.268)	(0.592)	(0.113)	(1.445)	(1.572)	(1.418)	(0.238)
COPD	−0.520	−0.689	−1.840*	−0.169	−2.250	−0.610	1.543	−0.087
	(0.620)	(0.452)	(1.109)	(0.176)	(2.032)	(2.054)	(2.100)	(0.365)
Asthma	−0.168	−1.246*	−1.324	−0.405	2.720	−2.962	1.059	−0.044
	(1.007)	(0.737)	(1.582)	(0.248)	(3.926)	(3.244)	(3.336)	(0.550)
Diabetes	−1.206**	−0.373	0.096	0.043	−2.571	−0.182	0.047	−0.368
	(0.567)	(0.349)	(1.071)	(0.163)	(2.122)	(1.973)	(2.274)	(0.419)
Diseases of middle ear and mastoid	−3.319***	0.261	−2.302	0.224	−3.178	−1.705	0.554	0.023
	(0.871)	(0.646)	(1.406)	(0.209)	(3.391)	(3.460)	(3.050)	(0.583)

Note: This table reports the estimates of the treatment effect obtained by estimating different models for each pollutant. All estimates and standard errors are rescaled (x100). The sample consists of 604 MedStat regions for the period 2001 to 2013. We assume that the data generating process follows a Poisson distribution. All regressions include control variables and both canton-year and MedStat region fixed effects. The regression results are sorted by cause of hospital admissions. Columns 2-5 report the estimates of the treatment effect by pollutant for the inverse distance approach, and columns 6-9 the results for the dispersion model approach. The heteroscedasticity-robust standard errors are provided in parenthesis, and are adjusted for within cluster correlation at the MedStat region level. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent, respectively.

The results of the dispersion model approach indicate that PM10 exposure has no statistically significant effect on hospital admissions for cardiovascular and respiratory diseases in Switzerland. However, we cannot exclude that these results are influenced by the introduction of canton-time fixed effects. Therefore, we cannot conclude that current PM10 exposure does not affect hospital admissions. On the contrary, the estimates of the treatment effect for NO2 and SO2 indicate a positive association between pollution exposure and hospital admissions. Both pollutants have statistically significant and economically meaningful effects on hospital admissions for cardiovascular diseases. The treatment effect is larger for SO2 than for NO2. We find that the incidence of hospital admissions for cardiovascular diseases is 2.7 percent higher for NO2, and 3.4 percent higher for SO2, when the pollutant exposure increases by 1 unit. The SO2 effect on hospital admissions for respiratory diseases is smaller with the incidence rate increasing most for chronic obstructive pulmonary diseases (COPD). Moreover, we find no evidence for a statistically significant association between pollution exposure and hospital admissions for asthma. The last column of Table 3 reports the parameter estimates for O3. We find no evidence for a significant effect of O3 exposure on hospital admissions, which is likely because summer spikes are captured insufficiently by our annual pollution measure. Lastly, the negative control outcomes (diabetes and diseases of the middle ear and mastoid) provide no evidence for a significant effect of ambient air pollution on hospital admissions, which further ensures the validity of our identification strategy (see the bottom lines of Table 3).

To ensure the validity of our estimation results, we conducted two additional robustness checks. First, we extended the analysis by comparing the effect of different distributional assumptions. The estimates of the treatment effect for the Gaussian and the Negative Binomial PML estimators are provided in the online supplementary material (Tables A2 and A3). We find that all estimates are similar to the estimates for the Poisson distribution regarding the significance level, but are larger regarding the size of the treatment effect. Second, we account for the potential effect of non-linearity in the treatment effect. We classify each treatment variable for the dispersion model approach into quartiles and interact the quartile dummies with the treatment measure. We find no

compelling evidence for non-linearity in the PM10 treatment effect. The Poisson PML estimates confirm that PM10 exposure has no statistically significant effect on hospital admissions in Switzerland. Additionally, the quartile regression results indicate that both NO2 and SO2 have statistically significant effects on hospital admissions. We find no evidence for non-linearity in the treatment effect for NO2, and only limited evidence for SO2. Overall, the largest estimates are observed for the last quartile. The estimation results for O3 are similar to those presented in our main regression table, providing no evidence for a statistically significant treatment effect.[16]

4.2 Accounting for endogeneity of the air pollution measure

We now adopt IV methods to account for potential correlation between the error term and the measures of pollution exposure. Although we believe that the dispersion model approach resolves the endogeneity issue provoked by the measurement error, there is still potential for estimation bias due to omitted variables. To account for this source of endogeneity, we adopt the control function method by generalizing the conditional Poisson model to an IV setting (Mullahy, 1997; Terza et al., 2008; Wooldridge, 2015). We apply the control function method to account for the endogeneity of pollution measures in the inverse distance approach and the dispersion model approach. We follow the argumentation of Lagravinese et al. (2014) and include the spatial and temporal lags of the endogenous variable, in addition to control variables ($\boldsymbol{Z}_{it}$) and fixed effects (α_i and δ_{ct}), in the first stage regression:

$$p_{it} = \alpha_i + \delta_{ct} + \beta_1 \bar{p}_{it} + \beta_2 p_{i,t-1} + \boldsymbol{Z}_{it}\boldsymbol{\gamma}_z + u_{it}. \tag{3}$$

The spatial lag ($\bar{p}_{it}$) is defined as the inverse distance weighted pollution exposure in adjacent MedStat regions. As adjacency criterion, we consider MedStat regions that share a common border with the region of interest.[17] We believe that the spatial lag will help us to solve the endogeneity issue because unobserved emission sources are likely to spread pollutants across administrative borders. Moreover, we believe that the temporal lag is a

[16] The results are available upon request from the authors.
[17] We follow Waller and Gotway (2004) assuming that all touching polygons are neighbors.

reliable instrument because air pollution is a time persistent problem.[18] Following **?**, we include the first stage residuals in the baseline specification (Equation 2). Although the control function approach allows us to obtain consistent estimates of β, we need to adjust the standard errors for the estimation error in $\widehat{u}_{it}$. To account for this error, we apply a block-bootstrap procedure with replacement, randomly drawing 200 samples from the entire history of each MedStat region (Cameron and Trivedi, 2013).

Table 4 summarizes the second stage parameter estimates for the investigated causes of hospital admissions for both measures of air pollution exposure. Our instruments are highly relevant, which is indicated by the significant parameter estimates of the first stage regression (provided in Table A4 in the online supplementary material). Most treatment estimates for the inverse distance approach are not statistically significant. Moreover, the SO2 treatment effect for cardiovascular and respiratory diseases is negative and statistically significant, which indicates that the IV approach is incapable of accounting for the systematic measurement bias provoked by the inverse distance approach. Then again, the parameter estimates for the dispersion model approach convey a similar picture as for the baseline specification. The treatment effects are in general larger and exhibit smaller standard errors. We find that both NO2 and SO2 exposure have a significant impact on hospital admission for cardiovascular and respiratory diseases. The incidence rate for coronary artery diseases is 6.9 percent higher for NO2 and 3.1 higher for SO2 when the pollution exposure increases by 1 unit. Again, the negative control outcomes provide no evidence for a significant association between ambient air pollution and hospital admissions, ensuring the reliability of our identification strategy.

[18] We test various combinations of spatial and temporal lags as instrumental variables but find no significant differences in the estimates of the treatment effect. Since the use of temporal lags may be questioned, we also estimate the model only with spatial lags, which do not affect directly the number of admissions. The results are confirmed and are available upon request from the authors.

Table 4: IV estimates of the effect of ambient air pollution on hospital admissions (spatial and temporal lag instruments)

Cause of hospital admissions	Inverse distance approach				Dispersion model approach			
	PM10	NO2	SO2	O3	PM10	NO2	SO2	O3
All cardiovascular diseases	0.288	−0.122	−0.531	−0.033	1.747	4.545***	4.028***	0.118
	(0.293)	(0.229)	(0.393)	(0.094)	(1.131)	(1.365)	(1.507)	(0.151)
Coronary artery disease	0.273	−0.226	−1.982***	−0.138	1.960	6.647***	3.082*	0.220
	(0.435)	(0.323)	(0.566)	(0.116)	(1.615)	(1.796)	(1.794)	(0.226)
Cerebrovascular disease	0.989*	−0.389	−1.549***	−0.194	0.487	5.318**	4.153**	−0.137
	(0.548)	(0.367)	(0.599)	(0.124)	(1.932)	(2.152)	(1.999)	(0.280)
All respiratory diseases	−0.239	0.571**	−0.554	−0.241**	−1.462	1.247	2.675*	−0.217
	(0.364)	(0.236)	(0.505)	(0.100)	(1.280)	(1.385)	(1.546)	(0.176)
Pneumonia	0.656	0.364	−1.816***	−0.179	−2.466	0.206	0.563	−0.252
	(0.541)	(0.349)	(0.670)	(0.140)	(1.983)	(2.030)	(1.901)	(0.252)
COPD	−0.190	−0.376	−2.165*	−0.319	−2.874	1.933	1.721	−0.058
	(0.766)	(0.514)	(1.123)	(0.199)	(2.719)	(2.593)	(2.718)	(0.420)
Asthma	0.652	−0.855	−2.128	−0.412	2.631	−7.092*	−1.448	0.115
	(1.151)	(1.061)	(1.823)	(0.321)	(4.702)	(4.239)	(4.047)	(0.598)
Diabetes	−1.502**	−0.031	0.292	0.115	−1.099	0.278	0.307	0.115
	(0.694)	(0.496)	(1.243)	(0.201)	(3.181)	(2.605)	(2.693)	(0.437)
Diseases of middle ear and mastoid	−4.765***	0.147	−3.280**	0.106	2.381	−0.268	−0.590	0.557
	(1.127)	(0.830)	(1.565)	(0.266)	(4.584)	(4.405)	(3.663)	(0.623)

Note: This table reports the second stage IV estimates of the treatment effect for the spatial and temporal lag instruments obtained by estimating different models for each pollutant. All estimates and standard errors are rescaled (x100). The sample consists of 604 MedStat regions for the period 2001 to 2013. We assume that the data generating process follows a Poisson distribution. All regressions include control variables and both year-by-canton and MedStat region fixed effects. The regression results are sorted by cause of hospital admissions. Columns 2-5 report the estimates of the treatment effect by pollutant for the distance interpolation approach, and columns 6-9 the results for the dispersion model approach. The heteroscedasticity-robust standard errors are provided in parenthesis, and are block-bootstrapped with replacement, drawing 200 times randomly from the entire history of each region. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent, respectively.

4.3 Emergency and elective hospital admissions

We now distinguish between channels through which patients enter the hospital. There is a claim in the epidemiological literature that ambient air pollution has different effects on emergency and elective hospital admissions (e.g., Perez et al., 2015). To investigate this issue, we create two datasets according to the type of admission and estimate two separate regressions on the relationship between pollution exposure and hospital admissions with IV methods. Again, we use spatial and temporal lags as instrumental variables.

Table 5 presents the parameter estimates of the second stage regressions. Our results show that the effect of air pollution is more pronounced for the emergency than for the elective admissions. Looking at the estimates of the treatment effect for PM10, which are not significant in the baseline specification, we find that the IV estimates of separate regressions convey the same picture. The relationship between NO2 exposure and admissions for cardiovascular diseases is highly significant only for emergency admissions. Turning to the treatment effect for SO2 exposure, we find that both emergency and elective hospital admissions for all cardiovascular causes are positively associated with SO2 exposure. Conversely, we find no evidence that SO2 exposure is associated with higher emergency and elective admissions for respiratory diseases. Although the distinction between emergency and elective hospital admissions provides additional insights, it has to be noted that the number of observations is low for some diagnoses of emergency admissions.[19] Therefore, we believe that our baseline regressions, where we consider the sum of both types of hospital admissions, provide more interesting and reliable findings.

[19] A low number of observations is associated with less variation in the count outcome and implies that there is a higher risk of spurious regression.

Table 5: IV estimates of the effect of ambient air pollution on hospital admissions (elective and emergency admissions)

Cause of hospital admissions	Elective admissions				Emergency admissions			
	PM10	NO2	SO2	O3	PM10	NO2	SO2	O3
All cardiovascular diseases	0.832	1.265	3.312*	0.091	1.945	5.610***	3.232*	0.147
	(1.597)	(1.817)	(1.807)	(0.228)	(1.480)	(1.595)	(1.664)	(0.180)
Coronary artery disease	1.052	1.154	0.316	0.242	2.239	9.697***	4.120*	0.175
	(2.089)	(2.195)	(2.104)	(0.343)	(2.004)	(2.278)	(2.125)	(0.273)
Cerebrovascular disease	−1.894	2.232	4.925**	−0.188	4.589	5.112	0.279	0.029
	(2.145)	(2.458)	(2.240)	(0.324)	(3.007)	(3.362)	(3.339)	(0.457)
All respiratory diseases	−2.337	−2.114	1.750	0.074	−1.498	1.807	0.301	−0.619**
	(1.727)	(1.834)	(2.064)	(0.231)	(1.927)	(1.907)	(1.622)	(0.256)
Pneumonia	−2.960	−3.007	−0.879	−0.373	−2.909	−1.877	−8.189*	−0.023
	(2.309)	(2.201)	(2.149)	(0.274)	(5.011)	(5.794)	(4.554)	(0.695)
COPD	−4.005	−4.204	−1.141	−0.246	−2.783	9.551**	4.265	−0.097
	(2.666)	(2.785)	(2.668)	(0.470)	(6.303)	(4.543)	(4.500)	(0.760)
Asthma	5.734	−7.764*	−2.939	0.819	−5.012	−11.762	−1.862	−1.229
	(4.944)	(4.589)	(4.574)	(0.610)	(10.507)	(9.177)	(8.509)	(1.581)
Diabetes	−3.553	−0.329	−0.846	−0.683	0.109	−2.743	−2.185	1.217**
	(4.421)	(3.286)	(3.371)	(0.586)	(3.816)	(3.995)	(3.185)	(0.547)
Diseases of middle ear and mastoid	7.366	0.114	−9.292	1.789	0.958	−0.661	1.315	0.030
	(9.503)	(8.036)	(7.264)	(1.269)	(5.320)	(4.731)	(4.078)	(0.742)

Note: This table reports the estimates of the treatment effect for the comparison between elective and emergency admissions (Poisson PML estimates) obtained by estimating different models for each pollutant. All estimates and standard errors are rescaled (x100). The sample consists of 604 MedStat regions for the period 2001 to 2013. We assume that the data generating process follows a Poisson distribution. All regressions include control variables and both year-by-canton and MedStat region fixed effects. The regression results are sorted by cause of hospital admissions. Columns 2-5 report the estimates of the treatment effect for elective admissions, and columns 6-9 the results for emergency admissions. The heteroscedasticity-robust standard errors are provided in parenthesis, and are adjusted for within cluster correlation at the MedStat region level. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent, respectively.

5. Conclusions

Ambient air pollution is the environmental factor with the greatest impact on human health. Several epidemiological studies provide evidence for a significant association between ambient air pollution and human health. However, the recent economic literature has challenged the identification strategy used in these studies. This paper explores the association between ambient air pollution and morbidity using hospital admission data from Switzerland. We try to strengthen the understanding of the impact of air pollutants on morbidity using geographically explicit air pollution measures derived from a dispersion model. This novel approach enables us to circumvent the measurement problem at the source and to construct a reliable measure of local pollution exposure. Our results suggest that the previous approach to measuring air pollution can induce significant estimation bias. We find a significant association between ambient air pollution and health outcomes. These results are robust to different distributional assumptions and non-linearity in the treatment effect. We also find substantial differences among the causes of hospital admissions. While SO2 and NO2 exposure appear to be strongly associated with admission rates for coronary artery and cerebrovascular diseases, the association between PM10 exposure and hospital admissions is not confirmed in all model specifications. Note that the transport sector, hit by the recent scandal on falsified emission performance data, are responsible for the largest part of these pollutants. Although exposure to air pollution has decreased significantly during the study period, our findings may indicate that there is still potential to further reduce the exposure to pollutants with the aim to mitigate the negative impact on health outcomes. These efforts should focus on reducing the exposure to SO2 and NO2 which show the strongest association with hospital admissions and, therefore, offer the largest benefits regarding human health. Thus, our results may contribute to a more accurate evaluation of future environmental policies that aim at a reduction of air pollution exposure.

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Online supplementary material

Table A1: Descriptive statistics of outcome, treatment and control variables

Variables	Mean	Standard deviation			Trend
		Overall	Between	Within	
Outcome					
All cardiovascular diseases	213.91	162.51	156.26	45.07	0.10
Coronary artery disease	60.31	45.93	43.29	15.47	0.02
Cerebrovascular disease	28.09	23.95	21.80	9.95	0.17
All respiratory diseases	104.62	86.13	82.28	25.64	0.11
Pneumonia	26.09	24.22	22.31	9.49	0.15
COPD	13.37	12.99	11.65	5.77	0.09
Asthma	3.75	5.02	4.40	2.42	−0.02
Negative control					
Diabetes	8.60	8.15	7.16	3.89	0.03
Diseases of middle ear and mastoid	3.18	3.40	2.66	2.12	−0.02
Inverse distance approach					
PM10	21.90	3.80	2.79	2.75	−0.51
NO2	27.46	5.92	5.55	2.18	−0.12
SO2	3.71	2.19	1.67	1.34	−0.46
O3	150.83	15.42	9.33	12.28	−0.37
Dispersion model approach					
PM10	19.58	3.68	2.90	2.26	−0.47
NO2	18.89	5.79	5.70	1.05	−0.13
SO2	2.33	1.44	1.26	0.69	−0.39
O3	156.05	14.95	7.50	12.93	−0.44
Control					
Population (in thousands)	12.68	9.04	9.02	0.65	0.05
Share of foreigners	0.19	0.10	0.10	0.01	0.11
Share of females	0.51	0.01	0.01	0.00	−0.13
Share of working-age population	0.68	0.02	0.02	0.01	0.03
Average household income (in thousands)	62.38	15.38	14.65	4.72	0.18
Income inequality measure	0.44	0.05	0.05	0.01	0.12
Unemployment rate	0.03	0.01	0.01	0.01	0.10
Number of ambulatory doctors	21.08	51.97	50.73	11.44	0.02
Number of stationary doctors	22.08	37.20	36.54	7.10	0.04

Note: This table summarizes statistics for outcome, treatment and control variables. The statistics is based on data for 604 MedStat regions and the period 2001 to 2013. We present the mean, and the standard deviation in terms of overall, between and within variation, and the time trend. The time trend is defined as the correlation between each variable and time.

Table A2: The effect of ambient air pollution on hospital admissions (Gaussian PML estimates)

Cause of hospital admissions	Inverse distance approach				Dispersion model approach			
	PM10	NO2	SO2	O3	PM10	NO2	SO2	O3
All cardiovascular diseases	0.109	−0.066	−0.009	−0.079	0.932	2.952***	3.303**	0.179
	(0.307)	(0.206)	(0.431)	(0.091)	(0.811)	(1.082)	(1.355)	(0.173)
Coronary artery disease	−0.556	−0.019	−1.409*	−0.045	0.512	5.646***	3.208*	0.404*
	(0.492)	(0.319)	(0.726)	(0.139)	(1.190)	(1.489)	(1.724)	(0.225)
Cerebrovascular disease	0.804	−0.309	−0.296	−0.363***	−0.655	4.853**	5.360***	−0.461
	(0.554)	(0.327)	(0.757)	(0.122)	(1.464)	(1.907)	(1.981)	(0.330)
All respiratory diseases	−0.016	−0.131	−0.937**	−0.169*	−0.816	0.187	1.897	−0.158
	(0.335)	(0.187)	(0.466)	(0.090)	(0.817)	(1.119)	(1.229)	(0.189)
Pneumonia	0.642	−0.087	−1.560***	−0.169	−1.063	−0.269	0.493	0.014
	(0.526)	(0.322)	(0.566)	(0.139)	(1.475)	(1.743)	(1.504)	(0.302)
COPD	0.222	−1.175**	−1.352	−0.266	−0.977	−0.079	2.666	−0.067
	(0.673)	(0.576)	(1.219)	(0.177)	(2.115)	(2.271)	(2.359)	(0.435)
Asthma	−1.993	−1.068	−0.640	−0.600*	−5.215	−8.960**	−0.330	−1.609**
	(1.239)	(0.965)	(2.099)	(0.332)	(5.290)	(4.408)	(4.156)	(0.668)
Diabetes	−1.135*	−0.892**	−0.233	0.185	−2.530	0.542	−4.352	−0.357
	(0.666)	(0.380)	(1.130)	(0.189)	(2.195)	(1.933)	(3.461)	(0.559)
Diseases of middle ear and mastoid	−4.570***	−0.133	−1.813	0.336	−9.927**	−4.977	1.153	0.004
	(0.854)	(0.786)	(.)	(0.228)	(4.174)	(4.073)	(3.249)	(0.768)

Note: This table reports the estimates of the treatment effect for the Gaussian PML estimator obtained by estimating different models for each pollutant. All estimates and standard errors are rescaled (x100). The sample consists of 604 MedStat regions for the period 2001 to 2013. All regressions include control variables and both year-by-canton and MedStat region fixed effects. The regression results are sorted by cause of hospital admissions. Columns 2-5 report the estimates of the treatment effect by pollutant for the inverse distance approach, and columns 6-9 the results for the dispersion model approach. The heteroscedasticity-robust standard errors are provided in parenthesis, and are adjusted for within cluster correlation at the MedStat region level. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent, respectively.

Table A3: The effect of ambient air pollution on hospital admissions (Negative Binomial PML estimates)

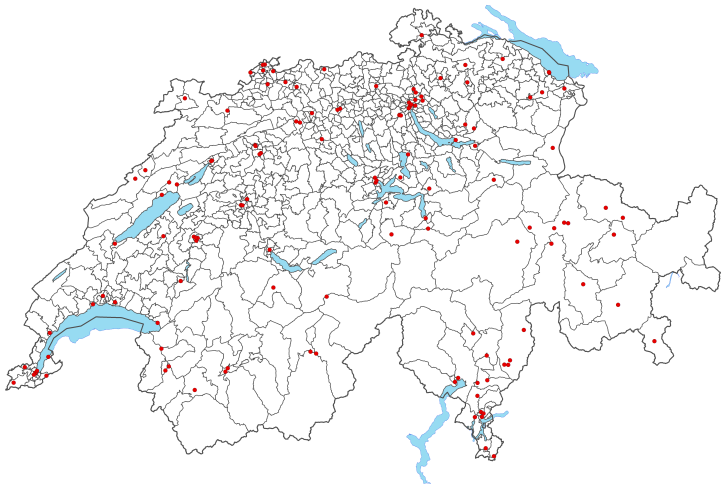
Cause of hospital admissions	Inverse distance approach				Dispersion model approach			
	PM10	NO2	SO2	O3	PM10	NO2	SO2	O3
All cardiovascular diseases	0.146	−0.253	−0.308	−0.058	1.268	3.194***	3.790***	0.053
	(0.233)	(0.165)	(0.349)	(0.065)	(0.883)	(1.042)	(1.039)	(0.134)
Coronary artery disease	−0.072	−0.444*	−1.301**	−0.185*	1.570	5.076***	3.751***	0.035
	(0.353)	(0.250)	(0.516)	(0.102)	(1.313)	(1.355)	(1.237)	(0.213)
Cerebrovascular disease	0.354	−0.696**	−1.011*	−0.055	1.202	3.085*	4.471***	0.203
	(0.417)	(0.278)	(0.590)	(0.095)	(1.643)	(1.670)	(1.610)	(0.291)
All respiratory diseases	−0.249	0.664***	−0.099	−0.202**	−0.196	0.298	2.361**	−0.054
	(0.298)	(0.213)	(0.549)	(0.084)	(1.038)	(1.149)	(1.174)	(0.155)
Pneumonia	0.428	0.420	−1.405**	−0.068	0.456	0.346	1.231	−0.026
	(0.462)	(0.284)	(0.667)	(0.113)	(1.587)	(1.697)	(1.481)	(0.242)
COPD	−0.685	−0.387	−1.967*	−0.152	−1.485	−0.052	2.298	−0.079
	(0.665)	(0.462)	(1.156)	(0.191)	(2.362)	(2.202)	(2.246)	(0.409)
Asthma	1.259	−0.769	−1.011	−0.338	8.158**	0.139	4.541	0.681
	(1.035)	(0.698)	(1.577)	(0.270)	(3.501)	(3.371)	(3.461)	(0.604)
Diabetes	−1.519**	0.309	0.625	−0.017	−1.992	1.099	3.069	−0.132
	(0.612)	(0.428)	(1.202)	(0.168)	(2.375)	(2.392)	(2.308)	(0.391)
Diseases of middle ear and mastoid	−2.822***	0.511	−2.392*	0.251	1.444	1.306	−0.088	0.164
	(0.995)	(0.664)	(1.393)	(0.232)	(3.679)	(3.499)	(3.172)	(0.592)

Note: This table reports the estimates of the treatment effect for the Negative Binomial PML estimator obtained by estimating different models for each pollutant. All estimates and standard errors are rescaled (x100). The sample consists of 604 MedStat regions for the period 2001 to 2013. All regressions include control variables and both year-by-canton and MedStat region fixed effects. The regression results are sorted by cause of hospital admissions. Columns 2-5 report the estimates of the treatment effect by pollutant for the inverse distance approach, and columns 6-9 the results for the dispersion model approach. The heteroscedasticity-robust standard errors are provided in parenthesis, and are adjusted for within cluster correlation at the MedStat region level. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent, respectively.

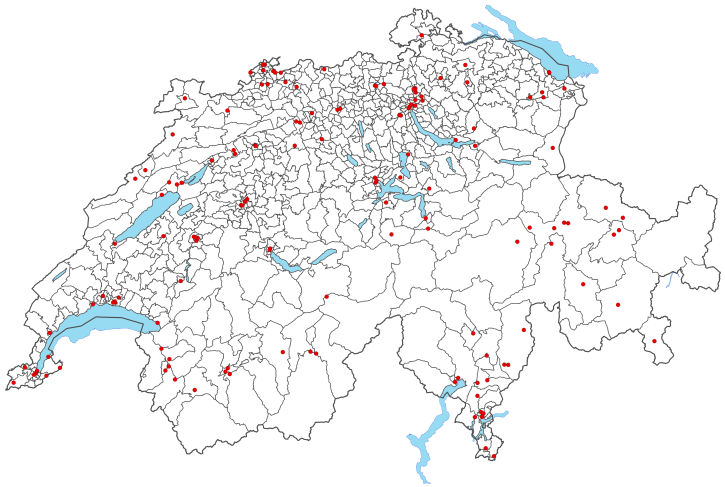
Table A4: First stage OLS regression results of spatial and temporal lags on interpolated pollution exposure (spatial and temporal lag instruments)

	PM10	NO2	SO2	O3
	Distance interpolated exposure			
Spatial lag	0.854***	0.779***	0.939***	1.020***
	(0.031)	(0.051)	(0.032)	(0.025)
Temporal lag	0.193***	0.324***	0.144***	0.125***
	(0.017)	(0.055)	(0.021)	(0.025)
	Dispersion model exposure			
Spatial lag	0.959***	0.949***	0.786***	1.015***
	(0.053)	(0.029)	(0.039)	(0.016)
Temporal lag	0.066**	0.179***	0.355***	0.027***
	(0.028)	(0.016)	(0.043)	(0.009)

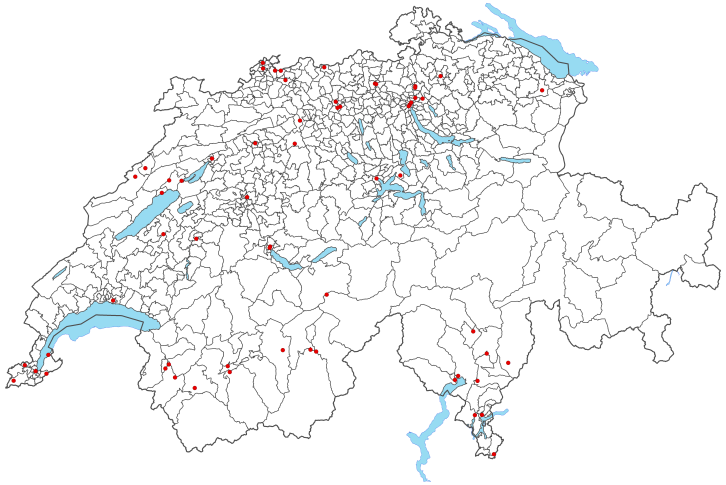
Note: This table reports the estimates of the first-stage OLS regression of spatial and temporal lags on interpolated pollution exposure. All regressions include year-by-canton fixed effects and region fixed effects. The standard errors are provided in parenthesis, and are adjusted for within cluster correlation at the region level. The heteroscedasticity-robust standard errors are provided in parenthesis, and are adjusted for within cluster correlation at the MedStat region level. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent, respectively.



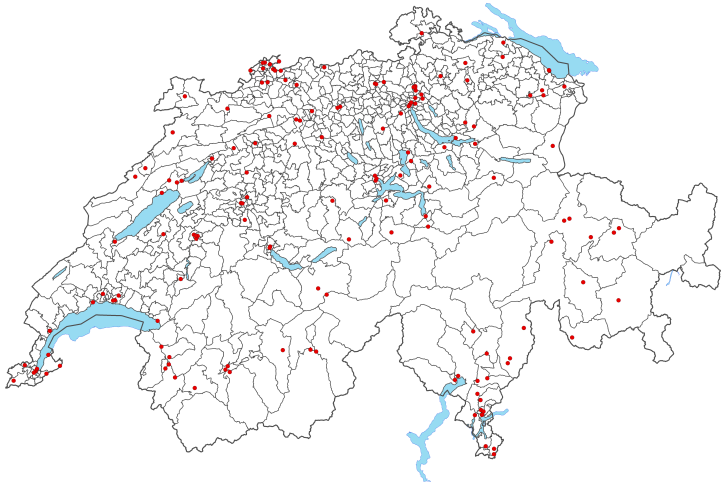
PM10



NO2



SO2



O3

Figure A1: Geographical distribution of pollution monitoring sites (red bullets) in the 604 MedStat regions during the period 2001 to 2013.

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