This is the Author's [accepted] manuscript version of the following contribution: Massidda C., The Role of Air Pollution and Income in Public Health in OECD Countries, Applied Economics Quarterly, vol. 27, N° 3, 2021, pagg. 225-254

The publisher's version is available at: https://doi.org/10.3790/aeq.67.3.225

The Role of Air Pollution and Income in Public Health in OECD Countries

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Abstract

This paper investigates the impact of particulate matter pollution and the level of development on mortality rates for a panel of 26 OECD countries during the 1990-2017 time span. First, our investigation involves the fixed effect estimation of a polynomial equation model where mortality rates represent the dependent variable. Then, to overcome potential endogeneity biases due to bilateral causality, we define a simultaneous equation model. Finally, we test whether different economic conditions affect the sensitiveness of mortality rates to pollution. The overall results show a positive impact of particulate matter on the number of deaths and a prevailing bidirectional negative relationship between particulate matter and per capita GDP. As far as the relationship between per capita GDP and mortality rates is concerned, results are less homogeneous across causes of mortality.

Keywords: particulate matter, mortality rates, panel data analysis, simultaneous equation model, OECD countries

JEL Classifications: Q53; I00; I15

1. Introduction

Quantifying the impact of air pollution on public health has become an essential component of the policy debate (Yazdi and Khanalizadeh, 2017). The economic aspects under consideration are highly relevant, but theoretical mechanisms are not completely clear, and empirical evidence is very scant. This paper aims to contribute to the above debate by investigating the relationship between environmental pollution, level of development, and health quality. As suggested by extant theoretical and empirical literature, this investigation addresses the following three hypotheses: Rising

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environmental pollution increases the mortality rate (H-1); increasing per capita income levels lower the mortality rate (H-2); increasing per capita income levels reduce environmental pollution (H-3)

The scope of this investigation implies putting health, pollution, and economic growth into the same framework to explore the complex interactions among these variables. Theoretical mechanisms underlining these relationships are complex because of potential nonlinearities and bidirectional causal relationships between each pair of variables. Thus, traditional econometric methods might suffer from potential endogeneity problems and omitted variable bias with a single regression equation. To address this kind of problem, we propose the following research strategy.

First, we define a multivariate model for a panel of 26 OECD countries during the 1990-2017 time span. In this model, mortality rates, expressed as the number of deaths per 100,000 inhabitants, represent the dependent variable, whereas the particulate matter of the two size ranges PM_{10} and $PM_{2.5}$ and per capita GDP are among the covariates (see among others Di et al., 2017; Raaschou-Nielsen et al., 2013; Dockery et al., 1993). Other covariates included in the model are the ratio between public health expenditure and total GDP, the share of the urban population (Mayer, 2001), and daily consumption of cigarettes and alcohol (Pope et al., 2002). As for the mortality rates, our choice concerns those causes of death for which particulate matter represents the main risk factor. Accordingly, we will consider the percentage of deaths attributable to ischaemic heart disease, lower respiratory infection, chronic obstructive pulmonary disease, malignant neoplasms, and, in particular, malignant neoplasms of the trachea, bronchus, and lung. Moreover, acute myocardial infarction, cerebrovascular diseases, and total death are also included to check for the robustness of our results. For each cause of death, we define two models using either PM_{10} or $PM_{2.5}$ among the regressors.

This polynomial model allows us to control the role of many covariates and, therefore, partially reduce the risk of estimation biases due to omitted variables. To estimate this model, we develop a panel fixed effect analysis with the advantage of controlling heterogeneity within the panel. However, as previously anticipated, this approach might suffer from potential endogeneity due to bidirectional causality between dependent and explanatory variables.

Following Lu et al. (2017), we define and estimate a simultaneous equation model (SEM) that includes a health equation, an environment equation, and an income equation to cope with this bias. The advantage of this approach is that it allows us to investigate the internal feedback mechanisms among the three main variables involved. To define the SEM, we add a proxy of human capital among the exogenous variables. It is given by the average total years of schooling for the adult population. We adopt the two stage least square method (2SLQ) to estimate the model.

Finally, we propose an exercise to test whether different economic conditions may affect the vulnerability of mortality to income, pollution, and other causes of cancer included in the model. To do this, we divide the sample of countries into four groups using the income quartiles as a dividing criterion, and then we estimate the polynomial equation for each subgroup. For the sake of synthesis, the results of this exercise will be reported only for total malignant neoplasms and PM_{10} among its determinants.

The proposed study contributes to increasing the scant number of panel data analyses on the relationship between environmental pollution, economic development, and public health and, compared to our main reference study (Lu et al., 2017), takes some significant steps forward.

First of all, to the best of our knowledge, this study is the first to investigate the relationship between pollution, economic development, and public health for a panel of 26 OECD countries. Second, thanks to available official data, this study can incorporate both PM_{10} and $PM_{2.5}$ as pollutants. This aspect of the analysis represents an important contribution given the growing concern about the

impact of particulate matter on health worldwide. Third, when it comes to mortality rates, the analysis includes eight major causes of death (Ritchie and Rose, 2018) that recognize particulate matter as one of their main risk factors.

Furthermore, the overall contribution of the analysis is emphasized by the recent international health crisis caused by the coronavirus pandemic (COVID-19). As addressed by the contemporary debate, long-term exposure to particulate matter may have worsened the health scenario in some geographic regions. According to the study by Coker et al. (2020), a positive association between the environmental concentration of $PM_{2.5}$ and excess deaths may be what happened in the first quarter of 2020 in the municipalities of Northern Italy.

The other contents of this paper are organized as follows. In Section 2, we discuss the relevant literature, and in Section 3, we present our research strategy. The results are discussed in Section 4. Section 5 draws the main conclusions of the analysis.

2. Literature

During the last decades, the relationship between environmental quality and health has been at the core of many epidemiological studies. Increasing environmental concern is motivated, among other things, by global warming, extreme weather conditions, and persistent chemical pollutants potentially harmful to human health and general well-being (O'neill et al., 2007). After Grossman's (1972) contribution, theoretically, the relationship between environmental quality and health has been discussed within the health production function model (HPFM). Accordingly, health is a function of different factors such as lifestyle, health care, environmental quality, level of development, heredity, and other inputs (see also Cropper, 1981; Gerking and Stanley, 1986).

Among the variables considered to measure environmental quality, air pollution has received significant attention as one of the primary sources of environmental costs, health status, and health expenditures (Gerdtham and Jonsson, 1991; Jerrett et al., 2003). In particular, the literature contains numerous attempts at quantifying a direct relationship between the mortality rate and exposure to air pollution. In this respect, empirical evidence is almost unanimous in favor of a positive correlation between these two phenomena (Stieb et al., 2002; Lu et al., 2017). However, according to Lu et al. (2017), most of the research consists of cross-sectional studies, time series analyses, and cohort-based studies, while panel data analyses are relatively scant.

This increasing collective awareness of the potential threats of environmental quality to health has made the relationship between air pollution and public health a central part of the current policy debate (Yazdi and Khanalizadeh, 2017). Economists have made noteworthy contributions to this debate following several lines of research. For instance, economists have focused on the evaluation of economic costs of health damage caused by environmental pollution (Xie et al., 2016), the measurement of the environmental value based on health damage costs (Pautrel 2008, 2009; Matus et al., 2012), the estimation of specific impacts such as crop yield losses (Miao et at., 2017), and the quantification of public health expenditure (Yazdi and Khanalizadeh, 2017).

Despite the wide variety of investigations,[2] little effort has been devoted to analyzing the relationship between pollution and health while considering the complex interactions with economic growth. Theoretical mechanisms underlining these relationships are complex because of potential nonlinearities and two-way relationships between each pair of variables. With the help of Figure 1, we create a framework that considers the main channels (CH) linking the three variables.

[2]An exhaustive discussion of the literature goes beyond the scope of the present contribution.

As we can see, Figure 1 reports the channel (CH 1) that runs from air pollution to health according to the HPFM previously discussed. According to H-1, the sign of this relationship is expected to be negative since an increase in air pollution lessens health and vice versa.

Additionally, Figure 1 introduces a direct channel running from growth to health (CH 2) that supports what was assumed in H-2. The HPFM is again the theoretical context that justifies this channel because it assumes that income is one of the primary inputs of the health production function. This link works through at least two mechanisms. On the one hand, economic growth leads to a higher quality of life and improved health conditions (Fogel, 1997). On the other hand, economic growth and technical progress lead to medical research advancement, which means improving the health level (Morand, 2005). However, regarding the relationship between economic growth and public health, CH 2 does not complete the theoretical framework. The endogenous growth model and human capital accumulation theories (HCAT) assume that good health pushes higher labor productivity and, consequently, improves the general state of the economy (Mayer, 2001; Stenberg, 2014). This relationship corresponds to CH 3 in Figure 1.

Therefore, CH 2 and CH 3 mean that economic growth and health can reinforce each other through a virtuous circle of feedback effects. The main consequence is that each factor adversely affecting growth impacts health and vice versa. In Figure 1, this can occur in two directions. As said above, if air pollution increases, it negatively affects human health (CH 1) and, consequently, reduces the value of human capital (labor productivity), depressing the economy (CH 3). This last effect, in turn, produces a negative backward effect on health, emphasizing environmental damage (CH 2).

The second direction is represented by the relationship between air pollution and growth (CH 4) that necessarily affects the intensity of the other channels already discussed. However, the mechanisms linking growth to the environment are not univocally defined. As a matter of fact, under the auspices of sustainability, these two phenomena might be characterized by a bidirectional relationship. In this respect, while growth theories can offer several tools to address this issue, theoretical literature linking pollution to income levels is relatively new. Brock and Taylor (2004) provide an interesting review in which four simple growth and environment models are developed to highlight the different ways the environment may or may not represent a constraint in the face of ongoing growth in per capita income. They underline scenarios where growth and pollution move in opposite directions and scenarios where they decrease together. The discussion mainly focuses on the incidence of abatement costs and the role of technological progress. However, when all mechanisms work together, nonlinearities and bi-directional causality cannot be excluded (Grossman and Krueger, 1995). Empirically, the well-known environmental Kuznets curve (EKC) represents the most common approach to investigating the link between growth and the environment (Bella et al., 2013; Shahiduzzaman and Khorshed, 2017). The central hypothesis is that income and pollution move along an inverted U-shaped curve. Accordingly, growth increases pollution until it reaches a turning point, then it starts to decrease pollution, improving environmental quality. This empirical framework offers a noteworthy scenario where causal relationships can also be investigated (for a recent comprehensive review of EKC literature, see Stern, 2017).

Figure 1 about here

Considering the scenario drawn in Figure 1, it emerges that there are several theoretical and empirical obstacles to a thorough investigation. As written above, the relationships between environmental quality, economic development, and public health have received little attention for this key reason.

Most of the research deals with this issue only indirectly, such as in the study of Arceo et al. (2016), where the authors find a negative relationship between pollution and infant mortality in Mexico, a typical developing country. However, some interesting exceptions consider environmental quality, economic development, and public health together. These are the works of Katrakilidis et al. (2016), Lu et al. (2017), Yazdi and Khanalizadeh (2017).

The contribution of Katrakilidis et al. (2017) analyzes the dynamic linkages between economic performance, health conditions, and environmental pollution in Greece. The authors investigate the two-way relationship between growth and environmental quality, health quality and environment, and the causal dynamic interdependencies between growth, environmental quality, and health simultaneously. The index of infant mortality per thousand approximates health quality. Results show that health is affected positively by growth and negatively by environmental degradation. Economic activity and environmental degradation systematically affect health in the long run when the three variables are analyzed.

Lu et al. (2017) focus on the relationship between China's economic performance, health conditions, and environmental pollution. The authors define a multivariate empirical framework with three measures of health quality (total mortality rates, perinatal mortality rates, average visits to pediatrics outpatient departments), together with real per capita GDP, per capita soot and dust emissions, per capita sulfur dioxide emissions, per capita wastewater emissions and other relevant covariates. The analysis utilizes panel data from 30 Chinese provinces from 2002 to 2014, and estimation is carried out first in a single equation context. Then, a three-simultaneous equations model (SEM) is estimated to address potential endogeneity. The main findings support the negative impact of pollution and income on health.

Finally, in Yazdi and Khanalizadeh's (2017) article, the impact of environmental quality and economic growth on health conditions is investigated by considering health expenditure instead of the number of deaths. The analysis is performed for the Middle East and North Africa region (MENA) countries from 1995 to 2014, and the method applied is an autoregressive distributed lag (ARDL). The results show cointegration between health expenditure, income, and emissions and statistically significant positive long-run effects of income and emissions on health expenditure.

3. Research Strategy

3.1 Empirical Model

As previously discussed in Figure 1, the complex relationships connecting public health, environmental pollution, and economic development could result from direct and indirect channels linking each pair of variables that are susceptible to nonlinearities and not necessarily unidirectional. Transferring this theoretical complexity into a single model is not a simple task for empirical studies. That is why many contributions from existing literature tend to sacrifice the general picture to focus on specific issues that are easier to model.

This research aims to contribute to the literature by investigating the relationships between environmental pollution, level of development, and health quality in the same empirical framework. In more detail, our investigation focuses on the impact of fine particulate matter of the two size ranges PM_{10} and $PM_{2.5}$ on mortality rates associated with some of the main causes of death worldwide. The analysis aims at addressing the following research hypotheses:

H-1 Increasing environmental pollution raises the mortality rate;

H-2 Increasing per capita income levels lower the mortality rate;

H-3 Increasing per capita income levels reduce environmental pollution.

The increasing concern around this pollutant motivates the choice of particulate matter. As well documented in the Global Burden of Disease Study (GBD) of the Institute for Health Metrics (IHME), outdoor air pollution (OAP) is the sixth leading risk factor for the number of deaths (3.41 million in 2017) worldwide, after high blood pressure, smoking, high blood sugar, total air pollution (outdoor and indoor) and obesity (Babatola 2018). Particulate matter produced by combustion processes is one of the two main components of OAP, the other being ozone. The mortality rates (number of deaths per 100,000 inhabitants) from particulate pollution (52.10) are significantly higher than those of ozone (4.87).[3] This difference means that when the risk of mortality from OPA is highlighted, actually the main concern is represented by exposure to PM_{10} and $PM_{2.5}$. This pollutant represents the main risk factor for the three leading causes of death that, according to GBD-IHME data, are cancer, cardiovascular diseases, and respiratory diseases (Ritchie and Rose, 2018). More in detail, WHO (2016) estimates a considerable impact of particulate matter on the percentage of deaths attributable to ischaemic heart disease (IHD), lower respiratory infection (LRI), chronic obstructive pulmonary disease (COPD), malignant neoplasms (MN), and, in particular, malignant neoplasms of trachea, bronchus and lung (TBL). Therefore, these five causes of death will be the subject of our investigation, plus acute myocardial infarction (AMI), cerebrovascular diseases (CD), and total deaths (TD) that we include to check for the robustness of our results.

Besides international statistics, previous empirical literature supports a significant correlation between particulate matter and mortality. Raaschou-Nielsen et al. (2013), for instance, find a positive relationship between PM_{10} and lung cancer. Similarly, Samet et al. (2000) find that an increase in PM_{10} is associated with increased mortality and cardiovascular diseases. Restricting attention to $PM_{2.5}$, Pope et al. (2002) and Di et al. (2017) show strong evidence of a positive relationship between this particulate and mortality risk in general. Dockery et al. (1993) estimate a positive correlation between mortality rates (associated with different causes of death) and several types of pollutants, including fine particulate, for six cities. They also test whether fine particulate produces different effects among subjects with different smoking statuses and genders. According to their results, the differences were not statistically significant.

Concerning the previous literature, we contribute by focusing on a panel (unbalanced) of 26 OECD countries during the 1990-2017 time span. This choice has the advantage of allowing the collection of a large amount of homogenous information for many countries necessary to grasp the impact of regional development differences on the relationship between pollution and health. The disadvantage is that, with country-year-level data, we cannot capture pollution variability over time and across areas within countries. However, we are confident that the advantage of the international comparison can compensate for the lack of microdata, which remains a crucial field for further investigation.

Our research strategy consists of three main steps.

As the first step, we define a single polynomial equation model where the dependent variable is the mortality rate, D, expressed as the number of deaths per 100,000 inhabitants, and particulate matter, PM, expressed in total per capita emissions (kg), is among its determinants. The model includes five more covariates. Following Lu et al. (2017), we consider constant GDP per capita (GDP) to measure the level of development (PPP in 2010 US dollars), the share of annual public health expenditure on

[3]Latest data downloaded from "Our World in Data" website (https://ourworldindata.org/outdoor-air-pollution).

total GDP (*HEXP*) to capture the role of the public policy on health care, and the share of urban population to measure the urbanization ratio (*URB*). Furthermore, following Pope et al. (2002), we also consider the number of cigarettes per adult per day (*CIG*) and daily alcohol consumption in liters per capita (*AL*) to measure smoking and alcohol addiction, respectively. Estimated coefficients of this polynomial model allow us to offer a piece of new evidence on the impact of all covariates on mortality rates. In particular, estimated coefficients for the variable *PM* and *GDP* provide a first test for the hypotheses defined in H-1 and H-2. We will consider these results as a reference for the rest of the investigation.

As an estimation method, we apply a fixed effect (FE) model to control the heterogeneity across countries. By imposing one time-invariant intercept for each panel unit, the FE model prevents unobservable values from becoming part of the error term. Other advantages of the model are that it is robust against the omission of time-invariant explanatory variables; it is suitable for estimating specific effects of a sample of countries; it is consistent even when the individual characteristics are correlated with the explanatory variables and when the Random Effect model is valid. In this case, we only lose efficiency (Abowd et al., 2008). Furthermore, besides cross-section fixed effects, given the time dimension of the panel, we introduce a time trend to control for the time change in the outcome variables.[4]

The empirical model is specified in the log-log form, and it comes as follows:

$$(1)\ lnD_{s,t} = \beta_1 lnPM_{s,t} + \beta_2 lnGDP_{s,t} + \beta_3 lnURB_{s,t} + \beta_4 lnHEXP_{s,t} + \beta_5 lnAL_{s,t} + \beta_6 lnCIG_{s,t} + \beta_s + \beta_t time + \varepsilon_{s,t}$$

where the subscripts *s* and *t* indicate countries and years, β_s is the unobservable time-invariant individual (country) effect, *time* is the time trend, and $\varepsilon_{s,t}$ is the error term. All estimated parameters represent elasticities.

As previously anticipated, the model is estimated considering eight indicators of mortality rate and using PM_{10} and $PM_{2.5}$ alternatively among regressors. This choice implies running 16 regressions, allowing us to compare the impact of PM_{10} and $PM_{2.5}$ on each cause of death and the impact of PM_{10} and $PM_{2.5}$ between different causes. Comparing the impacts of the other regressors can also provide interesting insights.

Despite the potential relevance of these results, the interpretation of estimated coefficients, however, requires caution. The traditional approach proposed to estimate Eq. (1) does not control the potential endogeneity due to bidirectional causality between dependent and explanatory variables. Moreover, possible relationships between regressors remain undetected.

Thus, the investigation moves from the single polynomial framework to a simultaneous equation model (SEM), where all three hypotheses in this study considered are tested simultaneously. This choice allows us to control the potential endogeneity due to bilateral causalities and investigate possible feedback effects among our investigation's three main variables. To define the model, we start from the theoretical framework outlined in Figure (1). Accordingly, we interpret health, pollution, and income as three potential endogenous variables and define the following system of three equations:

[4] The presence of a time trend assumes that the estimated effect is not specific to any given year. We estimate the model considering both trend and year dummies to control for time-fixed effects as a robustness check. All the results remained almost unchanged (results are available upon request).

$$\begin{cases} lnGDP_{s,t} = \gamma_0 + \gamma_1 lnD_{s,t} + \gamma_2 lnPM_{s,t} + \gamma_3 lnHC_{s,t} + \varphi_{s,t} \\ lnPM_{s,t} = \rho_0 + \rho_1 lnGDP_{s,t} + \rho_2 lnURB_{s,t} + \omega_{s,t} \\ lnD_{s,t} = \sigma_0 + \sigma_1 lnPM_{s,t} + \sigma_2 lnGDP_{s,t} + \sigma_3 lnAL_{s,t} + \sigma_4 lnCIG_{s,t} + \sigma_5 lnHEXP_{s,t} + \mu_{s,t} \end{cases}$$

The first equation considers $lnGDP_{s,t}$ as an endogenous variable and is defined according to CH. 3 and CH. 4 in Figure 1. As it appears, in addition to the mortality rate ($lnD_{s,t}$) and particulate matter ($lnPM_{s,t}$), it includes the human capital ($lnHC_{s,t}$) proxied by the level of education given by the number of average total years of schooling per the adult population. As is well known, according to endogenous growth theories, human capital is one of the crucial determinants of GDP growth.

The second equation's endogenous variable is particulate matter and includes $lnGDP_{s,t}$ as one of its main determinants. CH. 4 in Figure 1 supports this choice. The log-linearity of the relationship between these two variables is supported by the simple correlation analysis reported in the following sub-section 3.2. This equation also includes $lnURB_{s,t}$ as an exogenous regressor because urbanization is a phenomenon closely related to air quality. The high population concentration in urban areas implies intensive human activities and huge energy consumption that produce enormous pollution emissions. Furthermore, urbanization can deteriorate air quality by compromising the ecosystem's air purification capacity.

Finally, the third equation considers mortality rates ($lnD_{s,t}$) as an endogenous variable and is an expression of CH. 1 and CH. 2 of Figure 1. It replicates Eq. (1) with a difference in the exogenous variables: It does not include $lnURB_{s,t}$, which was used to define the second equation of the SEM.

In the system, each equation has at least one exogenous variable not in common with the other equations; therefore, the model can be identified, and two stage least square (2SLS) can be used as an estimation method (Lu et al., 2017). The SEM specification is estimated for the eight mortality indicators for both PM_{10} and $PM_{2.5}$. We will test our model for both under-identification and weak identification using the Anderson (1951) canonical correlations test (*A- canon. corr. LM test*) and the F version of the Cragg-Donald Wald test (*C-D Wald F test*) under the nulls of under-identification and weak identification. Since even a result of a rejection of the null does not totally avoid the weak instrument problems, the first stage allows us to test for both under-identification and weak identification for each endogenous regressor separately, using the method of Sanderson-Windmeijer (2015). The Sanderson-Windmeijer first-stage chi-squared (*SW Chi-sq*) and F statistics (*SW F*) are tests of under-identification and weak identification of individual endogenous regressors. First-stage testing also allows us to run the Anderson-Rubin (1949) test (*A-R Wald test*) that provides weak-instrument robust inference for testing the significance of the endogenous regressors in the structural equation under the null hypothesis that the coefficients of the endogenous regressors are jointly equal to zero. This test is equivalent to regressing each endogenous regressor on the complete set of instruments and testing that the coefficients of the excluded instruments are jointly equal to zero.

The third and last step concerns the intent to test whether different economic conditions may affect the vulnerability of mortality rates to pollution, income, and the other covariates in our model. To this end, we develop a subsample analysis. Technically, we divide our sample into four groups using income quartiles. The first quartile includes income levels up to 27954 dollars (25%), the second quartile includes income levels up to 36667 dollars (50%), the third includes income levels up to 45235 dollars (75%), and the fourth includes income levels up to 64905 dollars. Then we run separate regressions for each income group (lower, low-medium, high-medium, higher) with mortality rates as the dependent variable. For space, we will report commented results only for the model with malignant neoplasms as the dependent variable and PM_{10} among its determinants.

3.2 Dataset Description

This investigation considers a panel of 26 OECD countries during the 1990-2017 time span. The empirical analysis employs eight dependent variables and seven covariates. In Table 1, we provide descriptions and sources for each variable.

Table 1 about here

We also provide the main descriptive statistics for all the eight mortality indicators and the three main covariates of the empirical model*:* *lnGDP*, $lnPM_{10}$, and $lnPM_{2.5}$. Table 2 reports the main figures.

Table 2 about here

Finally, to complete the description of the primary data, we propose some correlation analysis. In particular, for the same variables reported in Table 2, we show scattered graphs with regression lines matching the observations regarding each mortality indicator with *lnGDP*, $lnPM_{10}$, and $lnPM_{2.5}$.[5] As we can see, there is a clear dominance of positive correlations between the number of deaths and particulate matter of both size ranges. Conversely, there is less clear-cut evidence about the sign correlating the number of deaths with *lnGDP*. Finally, the last row of Table 2 reports the correlation between *lnGDP* and the two pollutants $lnPM_{10}$ and $lnPM_{2.5}$. A downward sloping regression line appears in both graphs.

Figure 2 about here

4. Results

4.1 General results

As described above, the first step of our empirical analysis involves the fixed effects estimation of the model specified in Eq. (1) for the full sample. Table 3a reports the main findings concerning the causes of death for which particulate matter is considered one of the main risk factors, namely ischaemic heart disease (IHD), lower respiratory infection (LRI), chronic obstructive pulmonary disease (COPD), malignant neoplasms (MN), and, in particular, malignant neoplasms of trachea, bronchus, and lung (TBL). Table 3b in the Appendix reports the results for acute myocardial

[5] Since the USA and Canada behave like outliers, they were excluded from the sample to improve the representation of the regression lines. This choice only concerns the graphical analysis. Indeed, the USA and Canada are considered in the dataset used to develop the regression analysis.

infarction (AMI), cerebrovascular diseases (CD), and total deaths (TD) that we have considered for completeness and to check for the robustness of our main outcomes.

Let us start the discussion on the results by focusing on the *lnMN* equation. Regarding Model 1, the general picture is the following. All estimated coefficients for the main regressors have positive signs, but only three of them report statistical significance. They are $lnPM_{10}$, *lnGDP*, and *lnUrb*. Going deeper into details, we can see that the estimated coefficient for $lnPM_{10}$ fulfills our expectations implying that a 1% increase in emissions leads to a 0.07% rise in cancer deaths. The same considerations hold for *lnUrb*. Regarding *lnGDP*, the result leaves more room for discussion. Contrary to our initial assumption, the elasticity of 0.11 seems to indicate that, ceteris paribus, an increase in well-being while allowing people to improve their living conditions, is nevertheless positively correlated with mortality rates.

Turning our attention to Model 2 in Table 3a, we can see that when $lnPM_{2.5}$ measures pollution, results do not change significantly: signs and magnitude of statistically significant coefficients are almost confirmed.

When we consider the other mortality indicators from the rest of Table 3a, we can see that $lnPM_{10}$ and $lnPM_{2.5}$, with the only exclusion of the *lnCOPD* equation, always report positive and statistically significant coefficients. Conversely, regressions reveal a less clear-cut picture when the rest of the variables are considered. As we can see, the coefficients of *lnGDP*, *lnAl*, *lnUrb* show opposite signs depending on the mortality indicator. On the other hand, when significant, the coefficients of *lnHEXP* and *lnCIG* always maintain the same negative and positive signs, respectively.

Table 3a about here

This general picture is confirmed when we estimate the model for the remaining causes of mortality. Results are reported in Table 3b in the Appendix.

4.2 SEM Estimates

We move now to the second step of our analysis. As anticipated, it consists in estimating the SEM for the eight mortality indicators using either $lnPM_{10}$ or $lnPM_{2.5}$. Accordingly, we run 16 regressions in total. As the methodological section explains, the 2SLS approach allows us to estimate parameters robust to endogeneity biases. Statistical significance and signs of these parameters will allow us to derive robust relationships between each pair of endogenous variables and drive conclusions on these relationship directions. A detailed discussion of the estimated coefficients is provided only for the malignant neoplasms model reported in Table 4a. Results regarding all the other mortality indicators, reported in Tables 4b and 4c in the Appendix, will be commented on in general terms.

Table 4a about here

As we can see in Table 4a, the comparison between the two models defined alternatively with $lnPM_{10}$ e $lnPM_{2.5}$ does not highlight significant differences. In more detail, the income equation regression results show a significant negative coefficient for both mortality rates and particulate matter, whereas the impact of human capital on per capita GDP is positive. As for the environmental equation, per capita GDP negatively affects pollution, while the urbanization ratio reports a positive sign. Finally, in the health equation, we find that increasing pollution, alcohol consumption, and smoking cigarettes raise mortality rates, whereas health expenditure decreases *lnMN*. Per capita GDP is the only variable that does not report a statistically significant estimated elasticity. Matching together the information provided by the three equations, we can summarize the relationships between the endogenous variables as follows:

1. positive unidirectional relationship running from particulate matter to mortality rates;
2. negative unidirectional relationship running from mortality rates to per capita GDP;
3. negative bidirectional relationship between particulate matter and per capita GDP.

Instruments post-estimation tests are reported in Table 5. As we can see, both *SW Chi-sq* and *SW F* suggest the null rejection in all equations. We can also reject the null for both the *AR Wald test* and the *A. canon. corr. LM test*. As for the *CD Wald test*, according to the Stock-Yogo weak ID test critical values, the statistic is low only in the *lnMN* equation. However, given the general picture, we are confident that this fact does not threaten the general goodness of the model. Tests are reported in Table 5 in the Appendix.

Shifting attention to the other mortality indicators (Tables 4b, 4c), we find that the general results are almost confirmed for the relationships between particulate matter and mortality rates and the relationship between particulate matter and per capita GDP (with three cases of unidirectionality from per capita GDP to particulate matter). Conversely, signs and directions of the relationship between per capita GDP and mortality rates exhibit mixed evidence: In some cases, the sign is negative (CD, MN, IHD, TBL, TD); in others, the sign is positive (AMI, COPD, LRI).[6] Bidirectional relationships prevail. To summarize this general picture, in Table 6, the relationships between each pair of variables are shown with arrows. Double-headed arrows indicate bi-directional relationships, while single-headed arrows support relationships running in one direction.

The overall results reported in Tables 4a, 4b, 4c, and 5 show:

1. positive relationship running from particulate matter to the number of deaths;
2. heterogeneous relationship between per capita GDP and mortality rates;
3. a prevailing bidirectional negative relationship between particulate matter and per capita GDP.

According to these general results, while we confirm H-1, regarding H-3 we can confirm the sign of the relationship between income per capita and pollution, but not the causality relationship (our evidence favors a bidirectional causality instead of unidirectional causality). Finally, as for H-2, we lack a piece of clear-cut evidence, meaning that results are strictly dependent on the mortality indicator considered.

Table 6 about here

4.3 Results by Income Quartiles

In this section, we run separate Eq. (1) regressions for each income group. Table 7 reports the results. As we can see, two main outcomes emerge. On the one hand, when statistically significant, the estimated elasticities exhibit the same signs obtained at the full sample level. On the other hand, the magnitudes of these elasticities show some differences across quartiles.

Going into more in-depth detail, from Table 7, we can see that the impact of $lnPM_{10}$ is significantly higher for the fourth group (0.09). This result might suggest that cancer mortality in the wealthiest countries is more vulnerable to increasing pollution levels. Regarding the variable *lnGDP*, estimated

[6]In all models, post-estimation testing confirms the rejection of under-identification and weak identification problems. Results are available upon request.

coefficients report positive signs and show the lowest value for the first group. Therefore, it seems that the variations in per capita GDP have less impact on increasing cancer mortality when income is low.

As for the other covariates, we can see that they are rarely significantly different from zero, but when significant, they report expected signs. This is the case for alcohol consumption, the urbanization ratio, and health expenditure.

Table 7 about here

We can conclude that, although some differences between groups are evident, these differences do not show a definite trend. In other words, the analysis by income quartiles does not allow definitive conclusions on how economic conditions may affect mortality vulnerability to income, pollution, and other causes of cancer included in the model. These general results are almost entirely confirmed by estimates run with all the other mortality indicators of our dataset that we do not report here for synthesis.

5. Summary of the Main Findings and Policy Implications

This paper investigates the relationship between mortality rates, the concentration of particulate matter of the two size ranges PM_{10} and $PM_{2.5}$, and the development level for a panel of 26 OECD countries during the 1990-2017 time span. The analysis uses eight indicators to measure mortality rates and applies two estimation methods.

First, we provide fixed effect estimations of a polynomial equation model with mortality rates as the dependent variable. Then, to overcome potential endogeneity biases due to bilateral causality, we define a simultaneous equation model of three equations with dependents mortality rates, income, and pollution. As the last step of the analysis, we test whether different economic conditions affect the sensitivity of mortality rates to pollution. For this purpose, we propose partitioning our panel into four sub-samples using income quartiles as the criterion. Then we run separate regressions for each sub-sample. For space reasons, results are provided only for the malignant neoplasms model case.

The polynomial equation estimation provides a fairly homogeneous picture regarding the positive impact of both particulate matter and cigarette consumption. The negative impact of health expenditure on mortality rates is also widespread. Conversely, income per capita, alcohol consumption, and the urbanization ratio report contrasting results. After controlling for the endogeneity bias, the SEM analysis provides interesting insights into the relationships between the variables. In particular, the most frequently found relationships indicate: a positive unidirectional relationship running from particulate matter to mortality rates; a prevailing negative bidirectional relationship between particulate matter and per capita GDP; heterogeneous results across causes of death as far as the relationship between per capita GDP and mortality rates is concerned. The analysis by income groups delivers heterogeneity in the results that would deserve further investigation.

The political implications of these findings are worthy of attention. The most relevant outcome for decision-makers is that air pollution significantly determines mortality rates. This result is important

information under normal conditions, but it becomes fundamental in the presence of a health emergency such as the recent coronavirus pandemic (COVID-19). The other significant result concerns the negative relationship between particulate matter and per capita GDP, which confirms that OECD countries move along a virtuous circle where development means improved environmental conditions. Further important information from our study is that the estimated relationships between economic conditions and mortality rates are heterogeneous in both sign and direction. In particular, the estimated sign for three causes of death is positive instead of negative as expected (lower respiratory infection, chronic obstructive pulmonary disease, and acute myocardial infarction). For two of them, this negative relationship is bidirectional. This result deserves serious consideration because it would say that better economic conditions impact the onset and severity of some types of diseases and vice versa. Therefore, policymakers who decide on national treatment and prevention policies can find helpful insights in our results. Closely connected with this issue, a further result is worth mentioning: The negative elasticity estimated for healthcare expenditure means that allocating public resources in support of the national health system effectively reduces mortality and social and economic costs associated with this type of disease.

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APPENDIX

Table 3b about here

Table 4b about here

Table 4c about here

Table 5 about here