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Economic and technological aspects of the impact of $PM_{2.5}$ particles on human health and productivity

ABSTRACT

Over the past decades, technological and economic growth has significantly contributed to the improvement of the health care system through increased investment in technological research, training and the application of new technologies in health care. There is evidence that rapid technological and economic growth, contributing to the improvement of the health care system, also leads to a reduction in mortality rates, as well as to a longer life expectancy, which indirectly affects human activity in scientific and political spheres. At the same time, there is evidence that 2.5-micron particles (PM2.5 particles), as a result of industrial development and increased car use, have a negative impact on health outcomes around the world. This paper analyses the impact of increased concentration of PM2.5 particles in the air on economic and technological development, in order to explore whether there is an interdependent relationship between them. The paper aims to clarify the long-term effect of PM2.5 on health outcomes, control of technological and economic growth, as well as other important factors, based on the analysis of given panel data and the application of modern econometric methods. Accordingly, the purpose of this paper is to provide information on the negative impact of PM2.5 particles on human health, as an indirect impact on the development of new materials and technologies, taking into account socio-economic factors at the global level.

Keywords: *PM2.5 particles, technological & economic growth, human health, regression analysis, panel data analysis.*

1. INTRODUCTION

Exposure to high levels of $PM_{2.5}$ air pollution affects human respiratory and inflammatory systems and leads to heart disease and cancer [1]. This process is very complex and research in this area requires a multidisciplinary approach. Gakidou et al. (2017) [2] documented the global problem of disease from $PM_{2.5}$ and published the data of ~4.1 million deaths in 2016.The World Health Organization (WHO) [3] estimated that about 3 million deaths occur annually due to city-based outdoor and indoor air pollution in 2012 [3]. Air pollution affects all regions, settings, socioeconomic groups, and age groups.

Air pollution is responsible for about one in every nine deaths annually, with almost two-thirds of those deaths happening in the Western Pacific and Southeast Asia [3], namely 1.1 million and 799,000 deaths respectively. Moreover, due to global migration from rural to urban areas, more people will move to cities in the coming decades, which will increase the number of people exposed to dangerous $PM_{2.5}$ [4,5].

A number of scientific studies suggest that one of the largest gaps in our current knowledge relates to air pollutants [6-10].

It has been observed that technological and economic growth simultaneously improves health care and poses threats to the environment, while there is evidence that $PM_{2.5}$exposure has a negative consequence on health outcomes. Our first hypothesis proposes that the $PM_{2.5}$ concentration-response curve plotted against economic growth follows Kuznets' inverted "U" curve. We can

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presume that this relationship is affected by the governmental regulatory capacity to adopt and implement environmental regulations to control $PM_{2.5}$ concentration. Furthermore, we can assume that high $PM_{2.5}$ concentrations lead to higher mortality rates attributed to $PM_{2.5}$. In our attempt to clarify this relationship we investigate other social-economic and demographic factors that may influence this relation such as education, governance indicators, health risk factors, etc. Our second hypothesis is to assess the association and impact of $PM_{2.5}$ concentration on mortality rate attributed to $PM_{2.5}$. Our third hypothesis is to confirm potential positive effects of technological and economic development on health and to estimate the effects of GPD per capita (in purchasing power parity) on mortality rates attributed to $PM_{2.5}$.

We investigate the potential endogeneity in our model by including a "Polity" variable to instrument GDP. Our "polity" variable is an indicator whose components measure the autocratic and democratic parts of government. We believe the "Polity" variable is likely to be positively correlated with GDP and some studies provide evidence that globally more democratic governments tend to have higher technological and economic growth and higher health spending (Gregorio and Gregorio, 2013). Thus, the "þPolity" variable may have a direct impact on health outcomes. An additional reason for using the "polity" variable as an instrument is that we have never found this variable to be statistically significant in any of our model estimations. We also verify the results following the steps explained in the methodology section by performing an over-identification test and checking the validity of our instruments. We find that the "Polity" variable is valid, relevant and not a weak instrument. This variable is taken from the publicly available Polity IV project database [11,12]. We also re-scale the polity indicator in order to transform all data points to be on a positive scale. In this study, we aim to estimate the long-term effect of $PM_{2.5}$ on associated health outcomes using a panel data structure. It is well-known that socio-economic determinants of health have a strong influence on health outcomes and that technological and economic status of an individual affects his or her susceptibility to $PM_{2.5}$ pollution.

2. METHODS

2.1. Air-pollution Data

Many of the studies that analyze the effects of $PM_{2.5}$ on health outcomes ignore the possible endogeneity between GDP and the health outcomes variable in their regression models. Technological and economic growth positively affects health outcomes, but this relationship can also go in the opposite direction, with good health positively affecting economic growth.

The goal of our analysis is to estimate the marginal effects of $PM_{2.5}$ on $PM_{2.5}$-associated mortality while controlling for technological and economic growth and other control factors that may affect this relation. The selection of covariates and instruments for our estimation is based on theoretical concepts, literature reviews and past empirical research. The dependent variable in our analysis is mortality per 100,000 individuals from cardiovascular and respiratory diseases and lung cancer for individuals older than 35. This data is publicly available from the World Health Organization's vital death registration database, classified by the International Classification of Diseases (ICD). We obtained mortality data for cardiovascular disorders (including ischemic heart disease, ischemic stroke and hemorrhagic and other non-ischemic strokes), lung cancer (trachea, bronchus and lung cancer) and all chronic respiratory diseases from 1990 to 2015. There are several limitations to the mortality data that we use. Firstly, not all countries report mortality data to the World Health Organization [3,13], leading to a potential selection bias. Thus, the unbalanced panel of mortality data for 91 countries (1397 data points) we construct is composed mostly of data from high and upper-middle-income countries. Secondly, there is a lag in the reporting of mortality data for certain countries, mainly those that are low-income, so we have a limited data for 2015 (15 data points). In our attempt to strengthen our mortality dataset, we considered using the mortality data projected by WHO and IHME using the global burden of diseases methodology. However, this option was not implemented given the potential bias of the result, since the global burden of disease methodology [14] uses the same covariates in the regression model to project missing mortality data as used in our analysis (GDP, human capital and smoking prevalence).

Our main explanatory variable of interest in the model is mean annual exposure to particulate matter with a diameter size of 2.5 microns or less ($PM_{2.5}$), expressed in micrograms per cubic meter (ìg/m^3). This exposure data was obtained from the World Bank Group database. The exposure is determined by calculating to mean the yearly concentrations of PM25 in urban and rural populations. The information of contact of the population to air pollution is taken the Global Burden of Disease study and the Institute for Health Metrics and Evaluation at the University of Washington. Atmospheric chemistry transport models, satellite observations, and ground level

monitoring is used to obtain the information of yearly concentrations.

Potential covariates were selected based on an extensive review of the theoretical and empirical literature. These variables include: gross domestic product per capita (PPP) from the World Bank Group, human capital levels (expressed in average years of schooling for adults from the World Health Organization), health risk indicators, smoking prevalence from IHME, immunization coverage of Diphtheria-tetanus-pertussis (DTP) from the World Bank Group, Gini index from the World Bank Group and World Income Inequality Database (WIID3.4), governance indicators (regulatory quality from the World Governance Indicators (WGI) database), fossil fuel energy consumption (% of total consumption), and urban population (% of total population) from the World Bank Group, and a "polity indicator" from the Polity IV project.

The relationship between the technological and economic growth rate and population health status in the context of the $PM_{2.5}$ variable has been well elaborated. We hypothesize that countries with higher GDP will have a lower disease burden. We pay special attention to the potential for a reverse causality relationship between health and GDP, as we suspect that there is a high likelihood of endogeneity in the model. Many recent studies have found that human capital has a positive effect on economic growth and health. We thus proxy human capital (specifically its education component) with "average years of schooling" for adults. We also considered including the Human Development Index (HDI) in the model instead of average years of schooling, but this variable was not included due to the presence of life expectancy in the design of the HDI which would consequently cause endogeneity in the model and bias our results.

2.2. Panel Regression Estimators

2.2.1. Fixed Effects (FE) Models

Fixed effects analyses are used when there correlation exists between the observed explanatory variables and the unobserved effect: E (x_{it} á$_i$) = 0. The term fixed effect does not mean that á$_i$ are treated as non-random, but rather that we allow for it to be correlated with the observed explanatory variables. More precisely, the fixed-effects estimator needs the two following assumptions to be consistent:

Assumption FE.1. (Strict exogeneity) E (v_{it} |X_i) = 0.

Assumption FE.2. (Identification) All explanatory variables change over time

In order to now derive the Fixed Effects estimator, first take the average of the variables by the individual:

$$\bar{y}_i = \bar{\alpha}_i + \beta\bar{x}_i + \bar{v}_i$$

where $\bar{y}_i = \Sigma_{t=1}^{T} y_{it}$ (similarly for x and v). Note that $\bar{\alpha}_i = \alpha_i$. This estimator is called the between estimator: it eliminates the time dimension of the panel to produce the cross-sectional dimension only. Now, subtract the averaged variables from the previous equations to obtain the proper estimator (this is called time-demeaning):

$$y_{it} - \bar{y}_i = (\alpha_i - \bar{\alpha}_i) + \beta(x_{it} - \bar{x}_i) +$$
$$+ (v_{it} - \bar{v}_i) = \beta(x_{it} - \bar{x}_i) + (\varepsilon_{it} - \bar{\varepsilon}_i)$$

Therefore, the estimation equation for the fixed effect or within estimator is:

$$\tilde{y}_i = \beta\tilde{x}_i + \tilde{v}_i$$

Through the within-transformation, by subtracting the mean of all the variables, we eliminate the á$_i$, and therefore the initial omitted variable bias.

2.2.2. Arellano-Bond (AB) Estimator

When considering panel data, because the dependent variable is tracked through time, the dependent variable relies on lagged values, i.e. on its values in previous periods. Nevertheless, attention is needed due to OLS estimation with a lagged dependent variable and serially associated error terms which can lead to inconsistent parameter estimates.

What can be reflected on is the approximation of fixed-effects models for short panels (large N, small T) with one or more lags of the depended variable as regressors. Initially, the set properties are removed by first-differencing the model, $\Delta y_{i,t} = y_{i,t} - y_{i,t-1}$.

The model can be written as:

$$y_{i,t} = \gamma_1 y_{i,t-1} + \cdots + \gamma_p y_{i,t-p} + \beta x_{it} + \alpha_i + v_{it}$$

Where t = p + 1, ..., T and á$_i$ is the fixed effect. The regressors x_{it} are assumed to be uncorrelated with v_{it}.

The dynamic model delivers a few various causes for correlating in y over time: 1) true state dependence, i.e. directly through y in preceding periods 2) observed heterogeneity, i.e. directly through observables x and 3) unobserved heterogeneity, i.e. indirectly through the time-invariant individual effect a_i.

After first differencing, the model becomes:

$$\Delta y_{i,t} = \gamma_1 \Delta y_{i,t-1} + \cdots + \gamma_p \Delta y_{i,t-p} + \beta \Delta x_{it} + \Delta v_{it}$$

3. RESULTS

We employed several regression estimators to investigate the marginal effects of $PM_{2.5}$ concentration on mortality attributed to $PM_{2.5}$ such as: OLS, FE and RE, instrumental variables regression and Arellano-Bond estimators. We are presenting our results, focusing on two fixed effects models and two Arellano-Bond models. Our research can be replicated using the same methods described in this study. We first tested the stationarity of time series variables using a Fisher test for unbalanced panel data. Our Fisher's test confirmed a sufficient level of stationarity, and thus we proceeded with classical model specifications in levels as explained in the methodology section.

We first employ simple OLS models with the same covariates that were included in the original Global Burden of Diseases (GBD) methodology: GDP, smoking intensity, human capital and a time dummy variable (Mathers & Loncar, 2006).We differentiate the response by interacting: (i) $PM_{2.5}$ concentrations with dummy variables for different thresholds (we use dummies for 25, 50, 75 and above 100 ìg/m^3), and (ii) GDP with dummy variables using income group classifications (low middle, upper middle, and high income). We observe that the dummy variable for lower-middle income countries has a significant ($p<0.001$) and positive effect on mortality at a level of 149, while the dummy variable for upper-middle income is significant ($p<0.001$) and negatively associated with mortality at a level of -98. The presumption here is that lower-middle income countries experience more $PM_{2.5}$-related mortality. This is a major limitation of our study, since we have a limited number of countries from the lower-middle income group in our sample, thus potentially preventing us from observing positive effects of $PM_{2.5}$ on a global scale. In addition, the various levels of $PM_{2.5}$concentrations added through dummy variables in OLS model show a positive and significant association ($p<0.001$).

We employ fixed effects models, Table 1, to analyze the determinants of our dependent variable: the death rate attributable to $PM_{2.5}$ concentrations (per 100,000 individuals). Our main covariates of interest are the annual mean of $PM_{2.5}$ concentrations, expressed in ìg/m^3; GDP per capita (PPP in USD); and other health risks, socio-economic and governance control variables such as smoking prevalence and human capital levels. Comparing the within estimation with the generalized least squares (GLS) approach using a Hausman test, we employ Fixed Effects (FE) models for the interpretation of the results, as the Within estimation is known to be the best linear unbiased estimator in this case.

Table 1. Fixed effects regression results[1]

Tabela 1. Rezultati regresije fiksnih efekata[1]

Variable	FE1 (lin-lin)		FE2 (log-log)	
$PM_{2.5}$	-6.68	(6.23)	-0.230	(0.173)
Lag $PM_{2.5}$	-3.72	(5.11)	-0.251	(0.151)
GDP PPP per capita	-0.016***	(0.002)	0.446	(0.334)
Sq GDP PPP per capita	0.000***	(0.000)	-0.038*	(0.018)
I.T. $PM_{2.5}$* Gini	-2.22	(3.17)	0.096	(0.085)
Fossil Use	3.89***	(0.843)	0.217***	(0.053)
Smoking Prevalence	1040***	(190)	0.709***	(0.056)
Human Capital	-27.1**	(5.87)	-0.559***	(0.083)
Regulatory Quality	-81.8	(16.6)	-0.271***	(0.053)
Constant	1336***	(106)	8.69***	(1.56)
R-squared: N:	0.530 651		0.608 Log-likelihood: 651	

[1]Note: *, **, and *** is 10, 5 and 1% respectively

Table 1 presents the results of the fixed effects lin-lin and log-log models. Both models do not show statistically significant effects of $PM_{2.5}$ concentration on mortality. As expected from theory and past empirical studies, human capital and smoking prevalence are shown to be consistently statistically significant ($p<0.001$), with a negative effect of around -27 for the human capital variable and a positive association demonstrated for the “smoke prevalence” variable.

To control for the effects of indoor $PM_{2.5}$ pollution and government regulatory capacity, we also include in the model variables such as “percentage of use of fossil fuel” and “regulatory

quality." Both variables have a significant association ($p<0.001$) with $PM_{2.5}$-related mortality.

Finally, we turn to a dynamic panel model specification, which we estimate using the Arellano-Bond (ABOND) procedure, Table 2, for the endogenous covariate in the model. In the models ABOND1 and ABOND2, GDP was instrumented by polity variable. We first test H_0 (Wu-Hausman F and Durbin-Wu-Hausman tests) that GDP can be treated as exogenous. The H_0 was rejected (p=0.0000). The first stage regression statistics show that there is a strong correlation between our instrument and endogenous variable (Rsq=0.7) and F statistics is larger than any of critical values, thus we conclude that our instruments are not week. Finally, Sargan statistics and overidentification test of all instruments show that instruments are valid and model correctly specified as p values reported huge values (p=0.5). As expected, for all variants of the dynamic panel models that we investigated, the lag of death rate is found to be statistically significant at a high level ($p<0.001$), with a positive coefficient estimated for the lag of $PM_{2.5}$-related mortality.

Table 2. Arellano-Bond regression results[1]

Tabela 2. Rezultati Arellano-Bondo-ve regresije[1]

Variable	ABOND1 (lin-lin)		ABOND2 (lin-lin)	
Lag Death Rate	0.475***	(0.038)	0.534***	(0.033)
$PM_{2.5}$	-2.76	(4.00)	-4.44	(1.08)
Lag $PM_{2.5}$	1.24	(3.22)	2.38	(1.46)
GDP PPP per capita	-0.008***	(0.002)	-0.002***	(0.000)
Sq GDP PPP per capita	0.000*	(0.000)	-	-
I.T. $PM_{2.5}$* Gini	-7.02	(3.92)	146***	(30.5)
Smoking Prevalence	668**	(233)	927***	(150)
Fossil Use	3.55***	(0.716)	2.54***	(0.417)
Human Capital	-3.19	(3.77)	-1.86	(0.954)
Regulatory Quality	-19.5	(14.5)	-28.9**	(9.14)
Constant	336***	(99.4)	94.3	(56.5)
N:	408		408	

[1]Note: *, **, and *** represent 10, 5 and 1%

The $PM_{2.5}$ concentration again has a negative association with $PM_{2.5}$-related mortality at a level of - 2.8 (ABOND1 model) and- 4.4 (ABOND2 model). However, these relationships are not found to be statistically significant. The lag of $PM_{2.5}$ concentration has a positive association with $PM_{2.5}$-related mortality, but not at a statistically significant level. The GDP variable has a high statistical significance ($p<0.001$) across both models, with a negative sign on the estimated coefficient for both of -0.002 and -0.008, respectively. Smoking prevalence is found to have a significantly ($p<0.001$) positive effect on the $PM_{2.5}$-related mortality rate, with a regression coefficient of 668 and 927 for the two models, respectively.

The estimates for the socio-economic variables are consistent with the previous findings, showing a statistically significant association with mortality ($p<0.001$) and with a positive sign identified for the use of fossil fuels variable and coefficient estimates of 2.5 and 3.6, respectively. Human capital and regulatory quality are shown to have a negative association with mortality which is statistically significant only for the regulatory quality variable ($p<0.001$) with a coefficient estimate of –29 (ABOND2).

In addition, we observe from ABOND2 model that every increase in the inequality of wealth distribution has a positive relationship with mortality rates at a statistically significant level ($p<0.001$) and a coefficient estimate of 146.

The main limitation of this dataset is that pollutant concentrations are sensitive to local

conditions that are difficult to capture. In addition, $PM_{2.5}$ data are only available for five year periods from 1990 to 2010, so we interpolate the missing data to create a more robust dataset.

Finally, our unbalanced panel consists of data from 89 countries over the period 1990 to 2015 (1387 data points). Most of the mortality and $PM_{2.5}$ data in our dataset come from high and upper-middle-income countries (90%) - therefore, our analysis and results should be interpreted in this context. We have 935 data points from the high-income country, 314 countries/year data from the upper-middle-income group and just 138 data points coming from the lower-middle income group.

The log-transformed model does not yield findings that differ greatly from those seen so far from the other regression models. To summarize, $PM_{2.5}$ concentration and the lag of $PM_{2.5}$ concentration are not found to be statistically significant, showing a mixed association with mortality. Other control variables in the model give statistically significant results that are consistent with the previous models in this study.

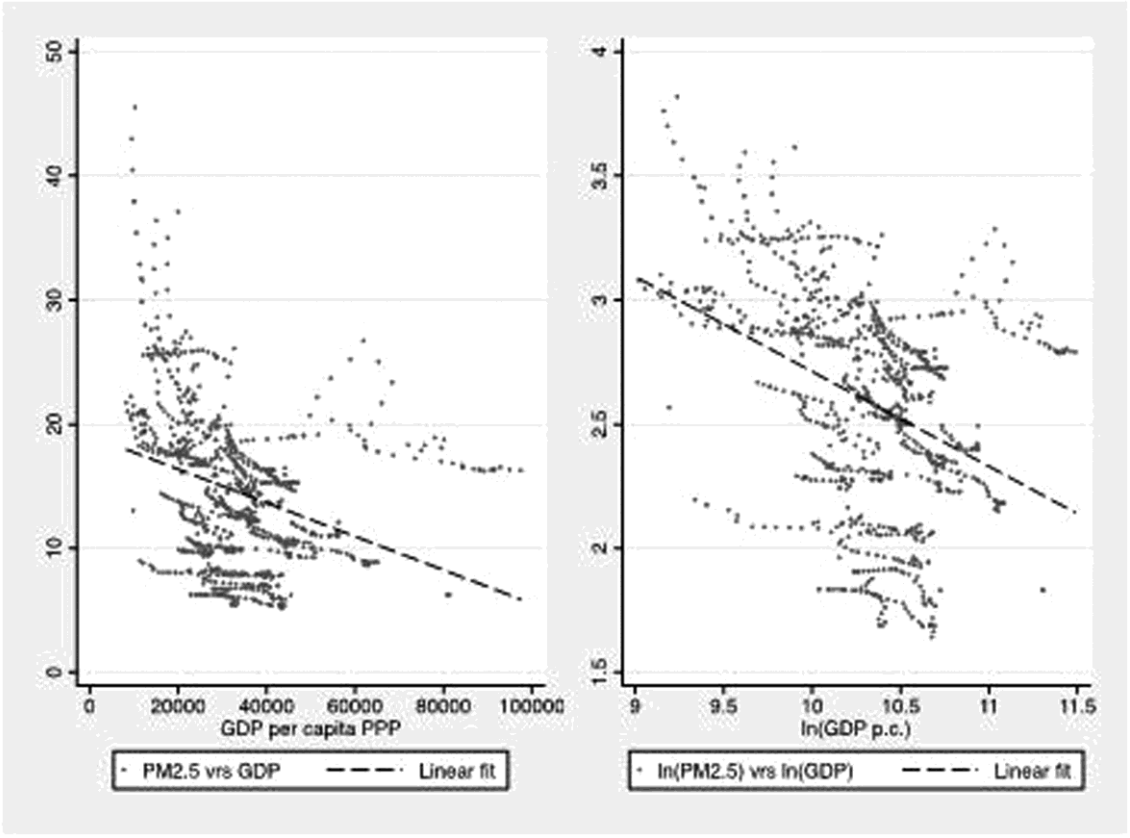
The magnitude of some of the coefficients is very small due to the different units of measurements of the various variables. In addition, the results for the linear models and linear models with log-transformed variables should be interpreted differently due to the log rescaling.

However, similar results have been reported from several other empirical studies, i.e. Venners et al. (2003) [15] lending support to our results. We can observe that mean annual $PM_{2.5}$ concentration has a different effect on health outcomes at various levels of GDP per capita; namely, the initial models report a positive and statistically significant ($p<0.001$) relationship between $PM_{2.5}$ concentrations on mortality for lower-middle income level countries. Since our dataset has a limited number of lower-middle income countries (10%) it is not surprising that we do not observe a positive effect of $PM_{2.5}$ concentrations on mortality at the global level.

Furthermore, it is observed that $PM_{2.5}$ concentrations have a positive and insignificant association with mortality rates. It is reasonable to believe that the lag of $PM_{2.5}$ concentration has a delayed effect on $PM_{2.5}$-related mortality and that deaths are likely to continue occurring in subsequent years. We therefore, include a lag of $PM_{2.5}$concentrations to measure this effect. The models show that the lag of $PM_{2.5}$ concentration has a statistically significant and positive association, while this relationship becomes insignificant with mixed results of the estimated regression coefficient for the ABOND models. Overall, these results should be interpreted with caution given the limitations of this study, as described in the Discussion section. Furthermore, as GDP per capita (PPP) expressed in international US dollars gives a consistently negative effect of GDP on the rate of mortality attributable to $PM_{2.5}$ pollution at a high level of statistical significance ($p<0.001$) across all models. Namely, we can estimate from the more advanced regression models that control for endogeneity and autocorrelation that for each increase of ten thousand USD in GDP per capita (PPP), we can expect on average a decrease in the $PM_{2.5}$-related mortality rate of between 20 and 110 deaths per 100,000 individuals.

Smoking prevalence is one of the main risk factors for mortality related to lung cancer and cardiovascular and respiratory disease [14,16]. Our study confirms that smoking prevalence is consistently and strongly associated with higher mortality rates at a high significance level ($p<0.001$). Our study estimates that a 1% increase in smoking prevalence in a country's population will on average lead to an increase of between 668 and 927 deaths per 100,000 individuals. Average years of schooling (our proxy for human capital) shows a negative association with mortality in most of the models (FE, ABOND), and based on the coefficient estimates we can expect that every 1-year increase in a country's average years of schooling reduces the $PM_{2.5}$-related mortality rate by on average between 3 and 26 deaths per 100,000 individuals.

Since most of our countries are from high and upper-middle income group (90%), the focus of this study is on the right segment of Environmental Kuznets' inverted "U" curve. After exclusion of outliers, we can observe Fig. 1, a trend between economic growth and $PM_{2.5}$ concentration for each income level. Fig.1 (i) shows, as expected a declining trend between GDP and $PM_{2.5}$ for high income counties. This trend corresponds to the right segment of inverted "U" curve. However, this relation in case of upper-middle income countries does not have a clear trend which might correspond to the middle segment in EKC. We speculate that a threshold to indicate turning point of EKC might be in this segment. Finally, our limited number of lower-middle income countries shows an increasing trend of relation between $PM_{2.5}$ and GDP which corresponds to the left segment of inverted "U" curve.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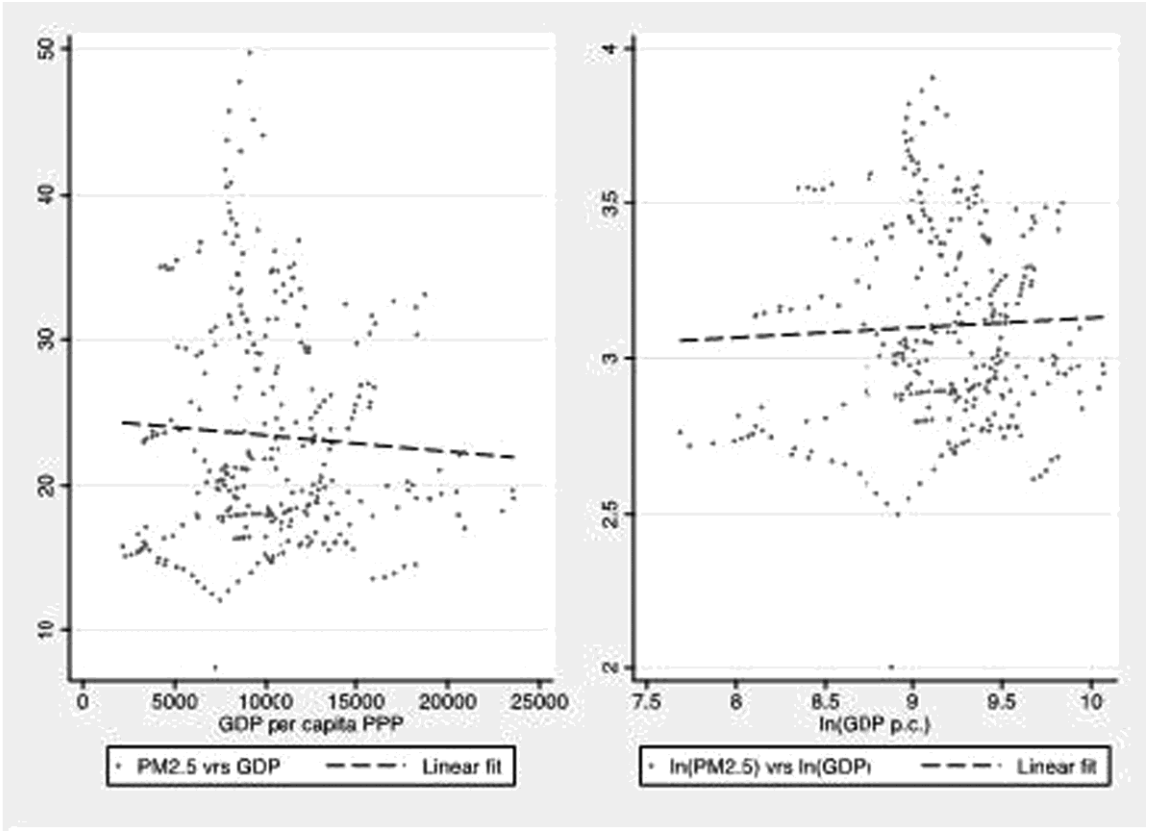
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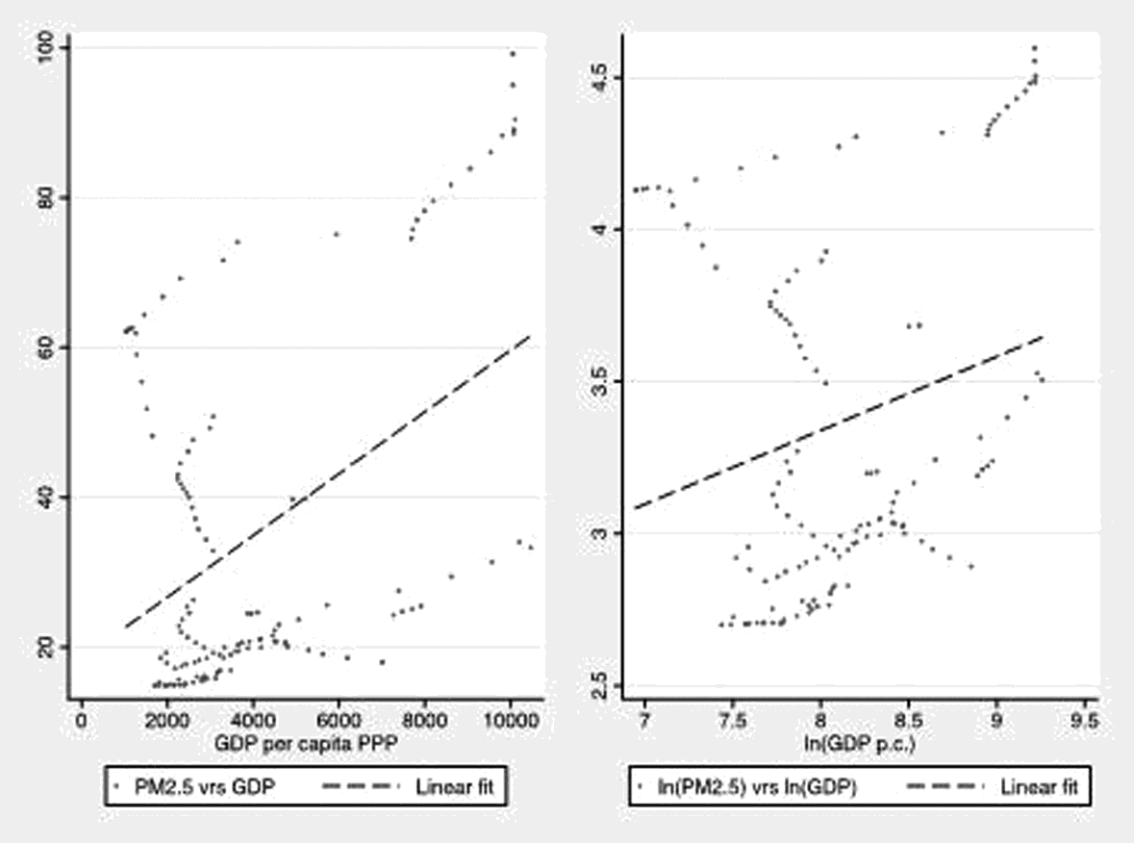

(iii)

Figure 1. Distribution of PM2.5 concentration and GDP per capita PPP for different income countries, (i) high-middle income, (ii) upper-middle-income and (iii) lower-middle-income countries.

Slika 1. Distribucija koncentracije PM2,5 čestica i BDP-a po glavi stanovnika PPP za različite zemlje sa (i) visoko-srednjim dohotkom, (ii) višim srednjim dohotkom i (iii) zemljama nižeg srednjeg dohotka.

Our limited number of lower-middle, Fig. 1 (iii), income countries shows an increasing trend of the relation between $PM_{2.5}$ and GDP which corresponds to the left segment of inverted ”U” curve.

4. DISCUSSION

It is clear that the relationship between $PM_{2.5}$ and health outcomes cannot be modeled without including technological and economic growth. Across the many empirical studies, including our own, economic growth yields a highly statistically significant and negative association with adverse health outcomes. The inverse relation between technological and economic growth and health deserves special attention, as the endogenous nature of economic growth has a potential biasing effect on the results. We can observe that many past and recent studies have ignored this endogeneity issue, compromising the results of their regression estimations.

Increased investments in production factors and intensified technological and economic growth in many countries compromise air quality and exposing the population to the harmful level of $PM_{2.5}$ concentrations. Many previous studies analyzed and showed negative short-term effects of PM on health outcomes controlling for the effects of other socio-economic and health risk variables that might affect this relationship [17-19]. The health outcomes attributed to $PM_{2.5}$ as externalities of technological and economic growth are affected by many biological, chemical, socio-economic and behavioral indicators that vary across countries and regions. Moreover, some of these individual factors have a very strong association with health outcomes attributed to $PM_{2.5}$, e.g. smokeing intensity or individual health status. The long-term effect of PM has been elaborated in several prominent studies where researchers found a positive association of PM concentration on PM-related mortality [20,21]. Most of these study designs were focused on limited urban areas, so researchers were able to measure daily or monthly PM concentration including PM concentration

peaks and record morbidity and mortality data attributed to PM. In these settings, a number of studies confirmed a positive association with mortality rate. However, some studies such as Venners et al. (2003) [15] observed a surprisingly negative association between $PM_{2.5}$ and daily mortality.

The complex relationship between $PM_{2.5}$, technological and economic growth and health outcomes is affected by health risk factors such as e.g. smoking intensity, blood pressure, nutrition and other socio-economic determinates: education, equity of distribution of wealth, government regulatory and health system capacity, etc. In addition, it is obvious that indoor and outdoor $PM_{2.5}$ pollution cannot be observed in isolation as both sources of pollution affect cardiovascular, respiratory and lung cancer-related morbidity and mortality.

The data available for this study consists of reported data mostly from upper-middle and high-income countries (90%), thus the findings from our empirical study should be interpreted in the context of countries included in the analysis. Our attempt to improve the robustness of the analysis and include projected mortality data, especially for lower-middle income countries from the Global of Burden of Disease (GBD) study has been discarded as the GBD methodology uses the same covariates to estimate missing mortality data (i.e. GDP, smoking intensity and human capital). Thus, to avoid clear endogeneity in the regression models we remain with a limited number of countries who reported mortality data through the WHO vital registration mortality system.

To our knowledge, this is the 1st study to investigate the long-term health effects of $PM_{2.5}$ concentration using publicly available cross-country and time-series data. Our panel consists of 89 countries observed during the period from 1990 to 2015. Many past and recent studies have analyzed the effects of ambient $PM_{2.5}$ on health outcomes using limited urban areas within a few cities or countries. However, our study design imposes a certain limitation that should be addressed onward. Firstly, our panel data structure captures the average value of $PM_{2.5}$ concentration without the ability to observe the scalar time of exposure to $PM_{2.5}$ concentration. Secondly, many recent studies reported that there is a presence of significant geospatial variation of $PM_{2.5}$ concentration across the areas within the cities and among various cities and countries. Since our main covariate of interest is expressed as the average value of $PM_{2.5}$ concentration we have a reason to be concerned about a potential variation of $PM_{2.5}$ concentration between urban and rural areas or even between the cities within the countries. Thirdly, our study design is not able to consider individual characteristics of the population exposed to $PM_{2.5}$ concentration such as gender, individual health status and some individual health risk factors. To overcome these limitations our strategy is to employ the proxy variables to control the unobserved factors mentioned above.

Since technological and economic growth might have positive or negative side effects on income inequality, we hypothesize that populations in a country with lower income, higher inequality of wealth distribution, and a higher percentage of urban populations are likely to be exposed to $PM_{2.5}$ emissions longer. Thus, we proxy the time of exposure variable with Gini index and share of urban population in the country. We presume that population with a various level of wealth distribution is exposed to the various level of $PM_{2.5}$ concentration, thus we employ iteration term as the product of Gini and $PM_{2.5}$ to control this factor. Also, it is reasonable to hypothesize that urban populations are closer to the sources of $PM_{2.5}$ emission and therefore exposed to higher level of $PM_{2.5}$ concentrations for longer. Loncar et al. (2019) [22] successfully applied panel data analysis for environmental and social responsibility of companies across EU countries and showed the socio-environmental dimension as a structural element of a strategy of successful companies.

Despite several limitations that were encountered, making it more difficult to identify the underlying robust effect of $PM_{2.5}$ concentrations on health, we observe some positive $PM_{2.5}$ effects on health using various proxy variables to control for unobserved factors. Although the relationship between technological and economic growth and health has been elaborated many times, such findings reiterate the importance of the many interconnections existing between various sectors (e.g., health, industry/technology and economy), and offer the opportunity to improve new approaches toward health-related sustainable development goals.

5. CONCLUSION AND FUTURE PERSPECTIVES

This study is expected to contribute to the debate on three core issues of global concern with an emphasis on the framework to support further empirical investigations and policy development. So far there are a limited number of studies that investigate the long-term effect of $PM_{2.5}$ on health using panel data. Thus, the current study is expected to fill this gap within the literature by

investigating these relationships as a nexus and aims to support the development of more robust and globally effective policy frameworks for both the developed and developing worlds.

Although the views vary on whether the Kuznets' curve exists in environmental settings, our analysis confirms the existence of the shape of the inverted "U" curve. Efforts to expand the boundaries of study design brought certain complexities and limitations in which proposed improvements are being introduced. In the future, the resources needed to deal with the consequences of air pollution will be significantly higher, if global efforts to control $PM_{2.5}$ emissions fail. It is imperative that technological and economic development, environment and health care are be studied within one single framework to provide new evidence to all stakeholders such as health care professionals, physics, chemists, technologists, environmentalists, economists, managers and politicians. The study methods provide a robust means to guide the implementation of similar analyses with more robust datasets and hopefully inspire further researchers to continue working with more advanced econometric methods in this area.

In recent years, the impact of $PM_{2.5}$ particles is mainly monitored on human health, however, future perspectives can be seen as the impact of dust with the controlled composition of $PM_{2.5}$ particles as a direct impact on material corrosion, since the external environment has a significant impact on a material surface. In the same way, in technological processes, the atmospheric composition at the level of $PM_{2.5}$ particles can be monitored in a targeted manner, which would lead to certain analytical data and adequate conclusions.

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IZVOD

EKONOMSKI I TEHNOLOŠKI ASPEKTI UTICAJA ČESTICA $PM_{2,5}$ NA ZDRAVLJE I PRODUKTIVNOST LJUDI

Tokom proteklih nekoliko decenija tehnološki i ekonomski rast su značajno doprineli poboljšanju sistema zdravstvene zaštite kroz povećana ulaganja u tehnološka istraživanja, obuku i primenu novih tehnologija u zdravstvu. Postoje podaci da brz tehnološki i ekonomski rast, doprinoseći poboljšanju sistema zdravstvene zaštite, takođe dovodi i do smanjenja stope smrtnosti, kao i do dužeg životnog veka ljudi, što indeirektno utiče na aktivnost čoveka u naučnim i političkim sferama. Istovremeno, postoje podaci da čestice veleičine 2,5 mikrona ($PM_{2,5}$ čestice), kao rezultat industrijskog razvoja i povećane upotrebe automobila, utiču negativno na zdravstvene ishode širom sveta. U ovom radu se analizira uticaj povećane koncentracije $PM_{2,5}$ čestica u vazduhu na ekonomski i tehnološki razvoj, kako bi se došlo do zaključka da li između njih postoji uzajamno zavisan odnos. Rad ima za cilj da razjasni dugoročni efekat $PM_{2,5}$ na zdravstvene ishode, na kontrolu tehnološkog i ekonomskog rasta, kao i na druge važne faktore, na osnovu analize datih panel podataka i na osnovu primene savremenih ekonometrijskih metoda, kako bi se potvrdile iznete tvrdnje. Shodno tome, svrha ovog rada je da pruži informacije o negativnom uticaju $PM_{2,5}$ čestica na zdravlje ljudi, kao indirektnog uticaja na razvoj novih materijala i tehnologija, uzimajući u obzir socio-ekonomske faktore na globalnom nivou.

Ključne reči: *$PM_{2,5}$ čestice, tehnološki & ekonomski rast, zdravlje ljudi, regresiona analiza, analiza panel podataka.*

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