Geliş/Received: 04.01.2025 | Kabul/Accepted: 08.05.2025 | Erken Görünüm/Early View: 30.05.2025

KOCATEPEİİBFD

Research Article | Araştırma Makalesi

Air pollution and premature deaths: A panel analysis of $PM_{2.5}$ effects in Europe

Muhammed Tümay | Assist. Prof. Dr., Gümüşhane University, mtumay@gumushane.edu.tr, 0000-0002-3226-3898

Corresponding author/Sorumlu yazar: Muhammed Tümay

Abstract

This research investigates air pollution's health and economic implications, focusing on the effect of fine particulate matter (PM2.5) on premature death in the European Union (EU)-27 countries from 2007 to 2019. It employs a robust fixed-effect (FE) panel regression to analyze the data. The study considers various economic and demographic factors, including healthcare expenditure as a percentage of GDP, income distribution, healthy life expectancy at age 65, fertility rates, and total deaths. The analysis reveals that a 1% increase in PM2.5 causes a significant 1.9% rise in premature deaths. These findings urge policymakers to limit air pollution and its destructive impact on public health. The results highlight the need to integrate environmental health interventions into economic policies to protect and improve population well-being. It also adds to the existing economic literature on environmental degradation and its impact on health.

Keywords: Air Pollution, Premature Mortality, Fixed Effects Model, Environmental Policy **JEL Codes:** Q53, I15, I18, Q58

Hava kirliliği ve erken ölümler: Avrupa'da $PM_{2.5}$ etkilerinin panel analizi

Öz

Bu araştırma, 2007'den 2019'a kadar Avrupa Birliği (AB)-27 ülkelerinde ince partikül maddenin (PM2.5) erken ölümler üzerindeki etkisine odaklanarak hava kirliliğinin sağlık ve ekonomik etkilerini araştırmaktadır. Verileri analiz etmek için sağlam sabit etkili (FE) panel regresyonu kullanmaktadır. Çalışma, GSYH'nin yüzdesi olarak sağlık harcamaları, gelir dağılımı, 65 yaşında sağlıklı yaşam beklentisi, doğurganlık oranları ve toplam ölümler dahil olmak üzere çeşitli ekonomik ve demografik faktörleri dikkate almaktadır. Analiz, PM2.5'teki %1'lik artışın erken ölümlerde %1,9 oranında önemli bir artışa neden olduğunu ortaya koymaktadır. Bu bulgular, politika yapıcılarını hava kirliliğini ve bunun halk sağlığı üzerindeki yıkıcı etkisini sınırlandırmaya teşvik etmektedir. Sonuçlar, nüfusun refahını korumak ve iyileştirmek için çevre sağlığı müdahalelerini ekonomi politikalarına entegre etme ihtiyacını vurgulamaktadır. Ayrıca, çevresel bozulma ve bunun sağlık üzerindeki etkisine ilişkin mevcut ekonomik literatüre de katkıda bulunmaktadır.

Anahtar Kelimeler: Hava Kirliliği, Erken Ölüm, Sabit Etkiler Modeli, Çevre Politikası **JEL Kodları:** Q53, I15, I18, Q58

Introduction

Air pollution poses a critical threat to both the environment and public health globally. It leads to high mortality and morbidity rates, economic losses, and increased pressure on healthcare systems. For example, the European Environment Agency ([EEA], 2023) reports that while there are reductions in emissions levels in 2021, ambient air pollution (primarily from $PM_{2.5}$, NO_2, and O_3) still exceeds safe limits, especially in urban areas of Central and Eastern Europe. One of them, $PM_{2.5}$, is particularly harmful due to its small size (diameter of 2.5 µm or less). It deeply penetrates the lungs and bloodstream, causing respiratory, cardiovascular, and metabolic diseases (World Health Organization [WHO], 2021). These health issues lead to the loss of healthy years of life and, in severe cases, result in premature deaths. The welfare losses due to reduced quality of life from air pollution are considerable, extending beyond direct healthcare costs to encompass broader societal impacts (EEA, 2023). $PM_{2.5}$ exposure is hazardous to disadvantaged groups, such as children, older people, and individuals with preexisting health issues. It leads to considerable premature deaths and declines in the quality of life across the EU (Organisation for Economic Co-operation and Development [OECD], 2016).

The EU has various environmental policies to reduce air pollution emissions and protect public health. One of them is the European Green Deal (EGD), which is a broad policy to achieve climate neutrality by 2050 and significantly enhance environmental health across member states. As part of the EGD, the Zero Pollution Action Plan has set goals to decrease premature deaths attributed to $PM_{2.5}$ by at least 55% from 2005 to 2030 (European Commission [EC], 2021). To reach these goals, the EU has revised its Ambient

How to cite this article / Bu makaleye atıf vermek için:
Tümay, M. (2025). Air pollution and premature deaths: A panel analysis of PM2.5 effects in Europe. *KOCATEPEİİBFD*, *27*(2), 228-237.
https://doi.org/10.33707/akuiibfd.1613250

Air Quality Directives to follow WHO guidelines, which impose stricter pollutant limits for member states (EC, 2022). The EU plans to reduce health problems caused by air pollution and improve air quality following these directives.

European Environment Agency reports that in 2020 alone, $PM_{2.5}$ caused potential additional premature deaths between 174,000 and 412,000 in the EU-27. These statistics emphasize the fatal effects of air pollution on people. Furthermore, the economic costs of mortality and illness from air pollution are substantial, including lost productivity, increased healthcare expenditures, and welfare losses. For example, health issues caused by air pollution are equivalent to €330 to €940 billion per year, indicating a significant financial loss to the EU (EC, 2021). This economic strain is even more significant in regions with high pollution levels and limited healthcare resources, which worsens socioeconomic disparities.

Another problem caused by air pollution is socioeconomic inequality, which requires urgent attention. Governments' precautions need to be revised to distribute the pollution burden among different socioeconomic groups because disadvantaged populations often experience higher levels of pollution exposure (EEA, 2024). This inequity worsens health and socioeconomic disparities. Several studies indicate that air pollution ($PM_{2.5}$) negatively affects productivity, reduces labor efficiency, and increases health issues, which further restricts economic growth in regions that are already facing financial challenges (Deschenes, 2010; Zivin & Neidell, 2012; Currie et al., 2014; Hansen-Lewis, 2018).

The study uses an FE panel regression model to add to the literature on the association between $PM_{2.5}$ and premature deaths across EU-27 countries from 2007 to 2019. The research includes essential economic and demographic variables, such as healthcare expenditure, income distribution, healthy life expectancy, fertility rates, and deaths. The analysis reveals a highly significant positive effect of increased $PM_{2.5}$ on premature deaths. These findings urge policymakers to limit air pollution and its harmful impact on public health. The results highlight the need to integrate environmental health interventions into economic policies to protect and improve population well-being.

1. Literature

Air pollution has a direct negative impact on health and economic outcomes and an indirect impact on socioeconomic factors such as GDP, labor productivity, and cognitive performance. $PM_{2.5}$ is identified as one of the most harmful air pollution due to its ability to penetrate deep into lung tissue and affect systemic health (WHO, 2024). Numerous studies estimated its direct and indirect effects.

Some studies show its direct and indirect effects on air pollution's economic and financial burdens. For instance, Matus et al. (2012) estimate that $PM_{2.5}$ led to a GDP loss of US$64 billion in China in 1995 from the economic impact of pollution-induced health costs. Zhou & Zhang (2023) estimate firm-level data in China and show that an increase (1 μg/m³) in $PM_{2.5}$ reduces labor share by 0.17 percentage points, indicating the indirect air pollution effect, such as labor dynamics and productivity. Further, Fu et al. (2021) use an instrumental variable (thermal inversion) and estimate that a decrease (1 μg/m³) in $PM_{2.5}$ increases productivity by approximately 0.82% in China's manufacturing sector, demonstrating that the improved air quality has a positive impact on potential economic benefits. Another study finds that acute exposure to $PM_{2.5}$ among older people increases mortality and substantial healthcare costs in the U.S. (Deryugina et al., 2019).

Several studies show that $PM_{2.5}$ significantly increases mortality rates and reduces life expectancy. Ebenstein et al. (2017) indicate that an increase (10 μg/m³) in $PM_{2.5}$ decreases life expectancy by approximately 0.64 years. Similarly, Wong et al. (2015) show that $PM_{2.5}$ significantly correlates with increased mortality in Hong Kong's elderly population. Dockery et al. (1993) found a positive significant relationship between air pollution and deaths across six U.S. cities. They show increased death rates from lung cancer and cardiopulmonary disease in areas with high particulate matter concentrations. Lelieveld et al. (2019) estimate that $PM_{2.5}$ leads to 790,000 deaths annually across Europe, with cardiovascular events accounting for the majority of these fatalities. Yin et al. (2017) similarly show that an increase (each 10 μg/m³) in $PM_{2.5}$ raises mortality hazard ratios for cardiovascular and respiratory diseases, especially in high-pollution areas. Jerrett et al. (2009) demonstrate that increasing $PM_{2.5}$ concentrations are significantly related to raising the death risk from cardiovascular causes.

Other indirect effects of $PM_{2.5}$ are on educational attainment and cognitive performance, which demonstrates its negative societal impacts. Ebenstein et al. (2016) investigated air pollution effects on students in Israel, finding that $PM_{2.5}$ exposure during high-stakes exams leads to declines in academic performance, which later correlate with lower educational attainment and earnings. Similarly, La Nauze & Severnini (2024) provide evidence that $PM_{2.5}$ exposure negatively affects adult cognition, with productivity implications for prime-age workers. Bedi et al. (2021) indicate the cognitive effects of $PM_{2.5}$ exposure in Brazil, observing that short-term exposure impairs fluid reasoning and working memory, both critical for occupational and educational success. Chang et al. (2019) extend these findings to the workplace, showing that $PM_{2.5}$ exposure negatively impacts worker productivity at a call center in China, even at pollution levels standard in many urban areas worldwide.

Other research emphasizes air pollution exacerbates socioeconomic inequalities. Persico et al. (2020) examined prenatal environmental pollutants exposure and found that children exposed to toxic waste sites experience worse behavioral and cognitive outcomes, which affect later-life labor market outcomes. Gourley (2020) analyzes the long-term effect of air pollution in the UK, finding that prenatal exposure is associated with lower wages, higher disability rates, and poorer overall health among adults. This highlights the multi-generational impact of pollution exposure.

Some studies also highlight potential premature mortality and morbidity reductions with stricter air quality guidelines. Khomenko et al. (2021) assess the potential for preventable mortality in European cities by evaluating compliance with WHO air quality standards. Their findings suggest that adherence to these guidelines could prevent tens of thousands of deaths in high-exposure urban areas, indicating the health benefits of stringent air quality standards.

Long-term studies estimate the effects of climate change on $PM_{2.5}$ levels, predicting increased premature deaths without intervention. Climate change increases in $PM_{2.5}$ will result in approximately 100,000 additional deaths annually by the end of the 21st century unless proactive measures are taken (Fang et al., 2013). Similar results from Cohen et al. (2017) analyze that $PM_{2.5}$ and ozone exposure caused 4.2 million deaths worldwide in 2015. They highlight that ambient air pollution causes an extensive global burden of disease.

Overall, the existing literature clearly shows that $PM_{2.5}$ not only causes direct health risks but also negatively impacts economic productivity, educational outcomes, and social equity. This study aims to contribute to the literature by examining the impact of $PM_{2.5}$ on premature mortality across 27 EU countries while controlling for important socio-economic factors. The findings aim to inform policymakers by demonstrating the economic and health benefits of improving air quality.

2. Data and Variables

The study uses data from EU-27 countries between 2007 and 2019, focusing on premature death as a dependent variable and air pollution as the primary independent variable. Additional control variables include health expenditure, income distribution, healthy life years, fertility rates, and total deaths. Data were primarily sourced from EEA and Eurostat, providing a comprehensive overview of environmental and socioeconomic factors impacting premature deaths. Table 1 presents definitions and sources for each variable, and Table 2 offers descriptive statistics.

Table 2 indicates descriptive statistics for each variable in the study. The average log value for Premature Deaths due to $PM_{2.5}$ is 8.315, with a standard deviation (sd) of 1.707, ranging from 4.248 to 11.137, indicating considerable variation in premature deaths across countries and over time. These variations align with the differences in environmental and socioeconomic conditions. $PM_{2.5}$ has a mean log value of 2.435 (sd = 0.392), with values spanning from 1.163 to 3.091, reflecting the diverse levels of particulate matter exposure. Countries with higher average $PM_{2.5}$ concentrations are expected to exhibit higher premature mortality rates due to the well-established health risks associated with fine particulate matter exposure. Current Health Expenditure has a log mean of 2.082 (sd = 0.227), varying between 1.5 and 2.461, showing differences in health spending across countries. Higher health expenditure is hypothesized to mitigate the impact of PM2.5 on mortality by enhancing healthcare systems' ability to address pollution-related illnesses. The Income Distribution variable, which captures inequality, shows a mean log value of 1.553 (sd = 0.230), with a minimum of 1.109 and a maximum of 2.119, suggesting considerable disparity in income distribution among the countries in the sample. Disparities in income distribution might exacerbate premature deaths due to unequal access to healthcare and vulnerability to environmental hazards. Healthy Life Years at Age 65 averages 2.1 (sd = 0.329), with a wide range from 1.03 to 2.785, implying variability in health outcomes for elderly populations. This indicator reflects the broader health outcomes and quality of life of elderly populations, which could influence the effect of air pollution on mortality rates. Fertility Rates have a mean log of 0.433 (sd = 0.125) and range from 0.135 to 0.722. While not directly linked to premature deaths, fertility rates could provide insights into demographic structures influencing health outcomes. The Number of Deaths shows a mean log value of 11.187 (sd = 1.431), with a range from 8.01 to 13.769. This variable captures general mortality trends, serving as a control for baseline differences in death rates across countries. These descriptive statistics illustrate diverse health and socioeconomic conditions across the countries analyzed and provide information on the $PM_{2.5}$ impact on premature deaths.

Table 1: Variables, Definitions, and Sources

Variables	Definition	Source
Premature Deaths $PM_{2.5}$	Estimated premature deaths due to exposure to fine particulate matter ($PM_{2.5}$).	European Environment Agency (EEA), 2022 *
Air Pollution Average $PM_{2.5}$ [μg/m³]	Average $PM_{2.5}$ concentration levels in μg/m³.	European Environment Agency (EEA), 2022 *
Current Health Expenditure (% of GDP)	Health expenditure as a percentage of GDP, covering healthcare goods and services.	WHO Global Health Expenditure Database, 2024
Income Distribution	the ratio between the top and bottom quintiles.	Eurostat, 2024
Healthy Life Years at Age 65	Expected years of life in a healthy condition at age 65, combining mortality and morbidity data.	Eurostat, 2024
Fertility Rates	Total fertility rate.	Eurostat, 2024
Number of Deaths	The total annual number of deaths.	Eurostat, 2024

Source: *European Environment Agency (EEA), "Air Quality Health Risk Assessments"

Table 2: Descriptive Statistics

Variables	Mean	Std. Dev	Min	Max
Log Premature Deaths $PM_{2.5}$	8.315	1.707	4.248	11.137
Log Air Pollution Average $PM_{2.5}$	2.435	0.392	1.163	3.091
Log Current Health Expenditure (% GDP)	2.082	0.227	1.5	2.461
Log Income Distribution	1.553	0.230	1.109	2.119
Log Healthy Life Years at Age 65	2.1	0.329	1.03	2.785
Log Fertility Rates	0.433	0.125	0.135	0.722
Log Number of Deaths	11.187	1.431	8.01	13.769

3. Estimation Strategy

The study exploits an FE panel regression model to analyze the impact of $PM_{2.5}$ on premature deaths across EU-27 countries from 2007 to 2019. This model controls time-invariant country-specific characteristics across countries for unobserved heterogeneity, allowing us to find the unbiased impact of $PM_{2.5}$ on premature mortality. Also, the suitability of this estimation method is determined, and several diagnostic tests are conducted to ensure robust results.

The Hausman test is used to distinguish whether a FE or RE model is more suitable. The null hypothesis of the Hausman test assumes that the random disturbance term is not associated with the regressors, suggesting that the RE model would be consistent and efficient (Hausman, 1978). However, the test results indicated that this assumption does not hold (p-value<0.05), leading us to reject the null hypothesis that RE is consistent (Wooldridge, 2010). Consequently, the FE model is considered more appropriate, as it controls for unobserved heterogeneity and provides consistent estimates when individual effects correlate with the regressors. Test statistic:

$$H = (\hat{\beta}_{RE} - \hat{\beta}_{FE})' [Var(\hat{\beta}_{RE}) - Var(\hat{\beta}_{FE})]^{-1} (\hat{\beta}_{RE} - \hat{\beta}_{FE}) \quad (1)$$

Where, $\hat{\beta}_{RE}$ and $\hat{\beta}_{FE}$ are coefficients from the random effects models and the FE models, respectively.

The study tests for the necessity of including time-fixed effects due to potential common shocks or time-specific factors that may simultaneously affect all countries in the sample (e.g., economic downturns, EU-wide policy changes). The test results indicate that time-fixed effects are incorporated, improving the model's capacity to control these annual fluctuations. Test statistic:

$$Log\ (Y_{it}) = \beta_0 + \beta_1 X_{it} + \alpha_i + \sum_{t=1}^{T-1} \lambda_t D_t + \epsilon_{it} \quad (2)$$

Where, D_t are dummy variables for each year. Joint significance of λ_t determines the necessity of time-fixed effects. α_i denotes country-fixed effects, and ϵ_{it} is the error term.

Cross-sectional dependence can bias the standard errors in panel data, implying that shocks in one country may correlate with outcomes in others (Baltagi, 2021). So, the Breusch-Pagan Lagrange Multiplier (B-P/LM) test is conducted for cross-sectional dependence (Breusch & Pagan, 1980). The test yielded a p-value (0.0000) lower than 0.05, indicating that we reject the null hypothesis of no cross-sectional dependence. Additionally, the Pesaran CD test has a p-value of 0.0814, suggesting weak evidence of cross-sectional dependence. Since at least one test confirms cross-sectional dependence, standard errors need correction. The Breusch-Pagan test statistic:

$$LM_{BP} = N(N-1)/2 \sum_{i=1}^{N-1} \sum_{j=i+1}^{N} \rho_{ij}^2 \quad (3)$$

Where, N is the number of cross-sectional units (27 countries). ρ_{ij}^2 is the correlation between residuals of country i and country j. Given the presence of cross-sectional dependence, standard errors are corrected using Driscoll-Kraay standard errors (Driscoll & Kraay, 1998), which account for heteroskedasticity, serial correlation, and cross-sectional dependence.

Heteroskedasticity in panel data can lead to inefficiency and biased standard errors, potentially affecting the reliability of statistical inference (Wooldridge, 2010; Greene, 2018; Baltagi, 2021). The Breusch-Pagan test for heteroskedasticity confirms its presence (p-value < 0.05). Consequently, the Driscoll-Kraay variance-covariance matrix is used to correct both heteroskedasticity and cross-sectional dependence.

To correct heteroskedasticity, the robust variance-covariance matrix for the FE model is:

$$Var\,(\hat{\beta})_{FE,DK} = (\tilde{X}'\tilde{X})^{-1}(\sum_{t=1}^{T} \tilde{X}'_t\,\hat{\hat{\Omega}}_t\tilde{X}_t)(\tilde{X}'\tilde{X})^{-1} \quad (4)$$

Where, $\hat{\hat{\Omega}}_t$ is the Driscoll-Kraay HAC estimator applied to demeaned residuals. $\tilde{X}_t$is the demeaned regressor matrix (within transformation applied).

Multicollinearity can inflate the variance of coefficient estimates, potentially leading to unreliable and unstable estimates. Each regressor's Variance Inflation Factor (VIF) is calculated to detect potential multicollinearity among the independent variables. All VIF values were below 10, suggesting no severe multicollinearity concerns (Greene, 2018). Therefore, all independent variables were retained in the model. Test:

$$VIF\,(X_k) = \frac{1}{1-\,R_k^2} \quad (5)$$

Where, R_k^2 is coefficient of determination from regressing X_k on all other independent variables.

The final model specification, incorporating both country-fixed and time-fixed effects and Driscoll-Kraay standard errors, is expressed as follows: 232

$$\begin{aligned} log(PrematureDeath_{it}) &= \alpha + \beta_1 log\,(PM_{2.5it}) + \beta_2 log\,(HealthExpenditureof\%GDP_{it}) \\ &+ \beta_3 log\,(IncomeDistribution_{it}) + \beta_4 log\,(HealthLifeYears_{it}) \\ &+ \beta_5 log\,(FertiliyRate_{it}) + \beta_6 log\,(Deaths_{it}) + \gamma_t + \eta_i + \varepsilon_{it} \end{aligned} \quad (5)$$

Where, $PrematureDeath_{it}$ represents the premature mortality in country i at time t, $PM_{2.5it}$ is the measure of air pollution (fine particulate matter), γ_t denotes time-fixed effects, η_i denotes country-fixed effects, and ε_{it} is the error term.

This estimation strategy ensures the model robustly captures air pollution's impact on premature deaths while addressing unobserved heterogeneity, serial correlation, heteroskedasticity, cross-sectional dependence, and multicollinearity issues. The fixed effects approach, combined with time-fixed effects and Driscoll-Kraay standard errors with lag(1), improves the model's reliability in estimating the relationship between $PM_{2.5}$ and premature deaths across EU-27 countries.

4. Results and Discussion

The study exploits FE estimators to analyze the $PM_{2.5}$ impact on premature death. Table 3 shows the estimation results for three models examining the relationship between $PM_{2.5}$ and premature deaths. These models are the FE model, the FE model with time-fixed effects, and the FE model with time-fixed effects and Driscoll-Kraay standard errors (lag=1), showing the process of eliminating heteroskedasticity and other potential biases.

The coefficient for $PM_{2.5}$, the primary independent variable, is highly statistically significant and positive across all models, with values of 1.785, 1.865, and 1.865, respectively. This high significance level suggests that increased $PM_{2.5}$ is related to increased premature mortality, causing severe public health risks. Specifically, a 1% increase in $PM_{2.5}$ is associated with an approximate 1.9% increase in premature deaths. This result aligns with previous findings in the literature, reinforcing that fine particulate matter poses a critical threat to public health (Dockery et al., 1993; Ebenstein et al., 2017; Lelieveld et al., 2019).

Health Expenditure coefficient indicates a negative and significant relationship with premature deaths across all models, with values of -0.284, -0.423, and -0.423. This finding implies that higher health expenditure, as a percentage of GDP, reduces premature deaths, likely due to improved healthcare access and quality. Some studies show that efficient health spending reduces the mortality rate (increasing life expectancy), which leads to fewer premature deaths (Adeboya et al., 2024; Plümper, T., & Neumayer, E., 2013). On the other hand, health expenditures may not be very effective if the diseases caused by $PM_{2.5}$, such as lung cancer, are insidious and their diagnosis is delayed (Gildea et al., 2017; Jeon et al., 2019).

Table 3. Estimation Results

Variables	**(1)** FE	**(2)** FE with Time-Fixed Effect	**(3)** FE with Time-Fixed Effect and Driscoll-Kraay standard error (lag=1)
Log ($PM_{2.5}$)	1.785***	1.865***	1.865***
	(0.059)	(0.096)	(0.257)
Log (HealthExp)	-0.284**	-0.423***	-0.423**
	(0.120)	(0.134)	(0.163)
Log (IncomeDist)	0.128	0.116	0.116
	(0.128)	(0.133)	(0.120)
Log (HealthLife)	-0.135*	-0.152**	-0.152**
	(0.074)	(0.074)	(0.052)
Log (FertiliyRate)	0.702***	0.730***	0.730***
	(0.150)	(0.160)	(0.175)
Log (Deaths)	1.620***	1.679***	1.679***
	(0.237)	(0.275)	(0.372)
Constant	-13.786***	-14.363***	-14.363***
	(2.760)	(3.121)	(4.635)
R-Squared	0.784	0.796	0.796
No. of Observation	345	345	345

Note: *Significance levels are * p<0.1, ** p<0.05, *** p<0.010. ( ) are standard error terms.*

The income distribution variable, a measure of income inequality, does not exhibit statistical significance across any of the models, indicating that income inequality may not have a direct or immediate effect on premature mortality in this context. This finding could suggest that income distribution influences mortality outcomes through more indirect pathways not captured in these models, although previous literature emphasizes a link between socioeconomic status and health outcomes (Gourley, 2020; Persico et al., 2020).

Healthy Life Years at age 65 has a negative coefficient in all models, with statistical significance across all models, highlighting that longer healthy life expectancy is associated with reduced premature deaths. This relationship underscores the importance of healthy aging and suggests that improvements in elderly health may contribute to lower premature mortality rates. For example, May et al. (2015) investigated that people who followed all four healthy lifestyle habits (Avoid smoking, low body mass index [BMI], regular physical activity, Mediterranean diet) lived at least two years longer in good health compared to those who did not follow any.

Fertility Rate is positively associated with premature deaths across all models, with coefficients of 0.702, 0.730, and 0.730, respectively, and statistical significance at conventional levels. This positive association could indicate that countries with higher fertility rates may experience different healthcare resource allocation dynamics or demographic pressures that indirectly impact premature mortality. Moreover, Le Bourg (2007) indicates that increased fertility could reduce longevity due to biological trade-offs between reproduction and survival. Although fertility does not consistently reduce longevity under natural fertility conditions, modern populations with more than approximately five children might experience slightly elevated mortality risks.

The death variable also shows a positive and highly significant association with premature mortality, with coefficients ranging from 1.620 to 1.679 across the models. This variable captures the general death rate, suggesting that higher mortality levels may correlate with elevated risks of premature death, perhaps due to broader systemic health challenges within certain countries. Furthermore, the estimation results correspond to studies from other regions, such as the U.S. and China, which show a robust link between $PM_{2.5}$ and increased death rates (Dockery et al., 1993; Ebenstein et al., 2017; Zhou & Zhang, 2023).

Overall, the high R-squared values of around 0.784 to 0.796 indicate that the models demonstrate a substantial proportion of the variation in premature deaths, supporting the relevance of these variables in understanding the determinants of premature mortality. The consistency and significance of $PM_{2.5}$'s impact across models underline the urgent need for policies addressing air quality to improve public health outcomes.

Table 4 provides the results from a series of diagnostic tests to ensure the selected models' reliability and validity. These tests assess multicollinearity, the suitability of the model, the need for time-fixed effects, and potential issues such as serial correlation, cross-sectional dependence, and heteroskedasticity, which could affect the accuracy of the estimations.

The Variance Inflation Factor (VIF) values for each variable are well below the threshold of 10, indicating no significant multicollinearity in the model. Low VIF values indicate that the independent variables do not exhibit problematic levels of correlation, meaning each variable contributes uniquely to explaining the variation in premature deaths without redundancy or overlap.

B-P/LM test results indicate that the RE model is preferable over a simple OLS model. This outcome (Prob > chibar2 = 0.0000) suggests the presence of unobserved panel effects that vary across countries, which would be unaccounted for in a standard OLS regression. Using either Random or Fixed Effects is more appropriate for this data structure. Furthermore, an F-test was conducted to compare FE and OLS. The results (F test that all u_i=0: F(26, 300) = 12.40 Prob > F = 0.0000) indicate that the FE model should be preferred over the OLS model.

The Hausman Test result supports the FE model over the RE model, with a highly significant result (Prob > chi2 = 0.0001). The Hausman Test evaluates whether the individual-specific effects are associated with the independent variables. The significant result indicates these correlations exist, and the FE model is more suitable. It controls these unobserved effects and reduces potential bias.

The Time-Fixed Effects Test (Prob > F = 0.0157) indicates the necessity of including time-fixed effects in the model to control time-specific factors influencing premature deaths across the sample period. The test result suggests that the factors (such as global economic changes, health trends, or international policy developments) vary over time and impact the entire sample similarly. By including time-fixed effects, the model more accurately isolates the impact of the variables on premature deaths, independent of annual fluctuations.

Table 4. Test Results

Variables	(1) VIF	(2) OLS vs RE LM Test	(3) FE vs RE Hausman Test	(4) Time-Fixed Effect	(5) Serial Correlation	(6) Cross-sectional Dependence	(7) Heteroskedasticity
Log ($PM_{2.5}$)	2.17	chibar2(01) = 243.46 Prob > chibar2 = 0.0000	chi2(01) = 29.26 Prob > chi2 = 0.0001	F(12, 26) = 2.72 Prob > F = 0.0157	F(1, 26) = 0.008 Prob > F = 0.9293	B-P/LM P-value=0.0000 Pesaran CD P-value=0.0814	chi2 (27) = 10957.56 Prob > chi2 = 0.0000
Log (HealthExp)	1.55						
Log (IncomeDist)	1.49						
Log (HealthLife)	1.47						
Log (FertiliyRate)	1.37						
Log (Deaths)	1.28						
Test Results	No Multicollinearity	RE are needed (panel effect)	FE are more appropriate	Time-Fixed effects are needed	No serial correlation	Weak Cross-sectional dependence	No Homoskedasticity

The Wooldridge Test for serial correlation (Prob > F = 0.9293) shows no evidence of serial correlation in the panel data. Serial correlation implies that observations are correlated across time within the same panel, which can cause standard errors to look smaller than they are and lead to inefficient estimates. The absence of serial correlation improves the accuracy of the observations in each period, which are sufficiently independent for reliable estimation.

The Breusch-Pagan Lagrange Multiplier (B-P/LM) test is conducted for cross-sectional dependence (Breusch & Pagan, 1980). The test yielded a p-value (0.0000) lower than 0.05, indicating that we reject the null hypothesis of no cross-sectional dependence. Additionally, the Pesaran CD test has a p-value of 0.0814, suggesting weak evidence of cross-sectional dependence. That is why standard errors are corrected using Driscoll-Kraay standard errors (Driscoll & Kraay, 1998), which account for heteroskedasticity, serial correlation, and cross-sectional dependence.

The Modified Wald Test (Baum, 2000) for heteroskedasticity (Prob > chi2 = 0.0000) indicates heteroskedasticity's existence in that the residuals' variance is not constant across observations. The result required the application of robust standard errors to address heteroskedasticity, which improves the accuracy and reliability of the coefficient estimates.

In conclusion, the diagnostic results in Table 4 demonstrate that the FE model with time-fixed effects and Driscoll-Kraay standard errors (Table 3, column 3) is the most appropriate model for this study. Using the robust model, the model addresses potential heterogeneity, serial correlation, cross-sectional dependence, and heteroskedasticity issues and provides robust estimates of the relationship between $PM_{2.5}$ and premature deaths. In this way, the estimation results reported in Table 3 are reliable in terms of the methodological rigor of the analysis. The robust analysis (Table 3, column 3) reveals that a 1% increase in $PM_{2.5}$ causes a significant 1.9% rise in premature deaths. These findings urge policymakers to limit air pollution and its destructive impact on public health. The results highlight the need to integrate environmental health interventions into economic policies to protect and improve population well-being. Strict air quality standards will mitigate the harmful effects of pollution on death and improve overall quality of life across Europe. Reducing air pollution will reduce healthcare expenditures, enhance labor productivity, and support sustainable development goals.

Conclusion

Air pollution poses a critical threat to both the environment and public health globally. $PM_{2.5}$ is particularly harmful because its small size allows it to penetrate deeply into the respiratory system, leading to or exacerbating cardiovascular and respiratory diseases (WHO, 2021). The study examines the impacts of $PM_{2.5}$ on premature deaths across EU-27 countries from 2007 to 2019. The result demonstrates a highly significant positive relationship between $PM_{2.5}$ and premature death by exploiting FE panel regression models. Specifically, the valid result, shown in the *FE with Time-Fixed Effect and Driscoll-Kraay standard error* model in Table 3, column 3, indicates a highly significant positive impact of a 1% increase in $PM_{2.5}$, causing a 1.9% increase in premature deaths. These findings urge policymakers to limit air pollution and its harmful effects on public health.

In light of these findings, reducing $PM_{2.5}$ pollution should be a fundamental public health priority for policymakers. This requires strengthening the enforcement of air quality regulations, such as stricter emission standards for vehicles, power generation, and industry, to control fine particulate emissions at their sources. Investments in cleaner energy sources and sustainable urban transport, such as expanded public transit and electric mobility options, will further help lower ambient $PM_{2.5}$ levels. Such evidence-based interventions can provide significant health benefits, as this study finds that even minor improvements in air quality can lead to notable reductions in premature mortality. While increased healthcare spending can reduce premature mortality, it cannot replace the need for pollution prevention. Moreover, preventing pollution is still essential for protecting societies from hazardous pollutant exposure, saving lives, and reducing long-term healthcare costs.

Despite the robust evidence established by this study, there are still many opportunities for future research. For example, analyses at spatial scales (such as city-level data) or within specific demographic groups (low-income communities or older people) can identify vulnerable populations and explain any health disparities associated with air pollution. In addition, investigating the effects of multiple pollutants (for example, examining $PM_{2.5}$ alongside ozone or nitrogen dioxide) and assessing the influence of climate change on air quality will provide more information on long-term environmental health risks. Another important opportunity is to evaluate the outcomes of policy interventions or natural experiments, such as low-emission zones or periods of significant emission reduction, to establish causal evidence of the health benefits from pollution control measures. Pursuing these research directions will deepen scientific knowledge and help policymakers design more effective, targeted strategies to reduce pollution-related premature mortality.

Author Contributions

The author has not declared any other contributors.

Acknowledgments

The author(s) did not provide acknowledgment.

Funding and Support

The author(s) did not report any funding or support information.

Conflict of Interests

The author(s) did not report any conflict of interest.

Ethics Statement

The author(s) did not report ethical committee approval as the research content does not require.

Kaynakça/References

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