Lung Cancer Mortality and Incidence Worldwide, 1990-2010: The Impact of PM2.5 and GDP

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Abstract. Lung cancer incidence and mortality vary widely across countries and time, and both air pollution and economic development may change the shape of these patterns. This study investigates the relationship between fine particulate air pollution (PM2.5) exposure and lung cancer rates worldwide from 1990 to 2010 and assess how gross domestic product (GDP) per capita modifies this relationship. By compiling a panel dataset of 20 countries and employ fixed-effect regression models with lagged exposures to account for the delayed carcinogenic impact. The author finds that higher long-term PM2.5 levels are associated with significantly elevated lung cancer incidence and mortality, whereas rising GDP (an indicator of development and health resources) corresponds to lower lung cancer rates, after controlling for latent factors. These results suggest that improving air quality could reduce lung cancer burden, especially in developing regions, while economic development may facilitate health improvements. The study fills gaps in global analyses of pollution-related cancer risk and highlights policy implications for air pollution control and tobacco mitigation in reducing future lung cancer deaths.

Keywords: $PM_{2.5}$, Lung cancer incidence and Mortality, Fixed effect regression, Latency analysis.

1. Introduction

PM2.5 is a particulate matter with a diameter of less than 2.5 micrometers, it is a critical air pollutant that will infiltrates the respiratory system and bloodstream, posing severe health risks, including asthma, cardiovascular diseases, and lung cancer. Epidemiological studies confirms that long-term exposure to PM2.5 will not only increase the incidence of lung diseases but also increases lung cancer mortality rates, particularly in regions with high pollution levels (Chen et al., 2018). The Global Burden of Disease Study estimates that PM2.5 has contributed to 4.1 million premature deaths globally in 2019, with lung cancer accounting for 21% of these fatalities (Chan, et al., 2021). Although the size-based definition is uniform, the toxicity of PM2.5 varies significantly depending on its chemical composition (e.g., sulfate from coal combustion vs. nitrate from vehicle emissions), which is often overlooked in public discourse (Hamra, 2014; Lelieveld et al., 2015). For instance, in Beijing, coal burning and traffic emissions contribute hugely to PM2.5-driven haze, with sulfate and ammonium being the dominant toxic components (Li et al., 2018). Chemical speciation studies reveal that coal-derived PM2.5 may be 2 to 3 times more carcinogenic than particles produce from traffic pollution due to higher polycyclic aromatic hydrocarbon (PAH) content (Neal et al., 2019).

The relationship between PM2.5 and lung cancer still have some debate due to three key complexities. Firstly, the dose-response curve shape is contested. While the Harvard Six Cities Study has established that there is a linear relationship down to 10μg/m³ (Pope et al., 2002). Chinese cohort data suggest a supra linear trend, where the risk will escalate exponentially above 35μg/m³ (China's 2015 national average). This difference may reflect regional differences in the composition of PM2.5 or population susceptibility (Raaschou-Nielsen et al., 2013). Secondly, confounding by smoking is pervasive. Although studies adjusted for smoking rates, but due to smoking synergies with PM2.5, residual confounding factors will still exist, a 2019 analysis showed smokers in high- PM2.5 areas face 5 times greater lung cancer risk than the non-smokers in clean areas. Chinese studies report there are shorter lags (5-7 years), possibly due to the higher baseline exposures accelerating disease onset (Perraud et al., 2015).

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Existing research emphasis 3 major points. The first one is some studies suggested that there is a linear dose response relationship between exposure on PM2.5 and lung cancer risk, other articles highlighted the threshold effect where morality plateaus at high concentrations (Pope et al., 2002). One example is a lancet article suggested that participants that is over 65 will have a decreasing HR for all cause mortality as PM2.5 concentration is higher than 50μg/m³, suggesting a nonlinear dynamic, this might suggest the competing mortality risk in elder population (Yin et al., 2020). The second one is the rural populations face a higher PM2.5 risk than urban populations. Possibly because of the uneven distributed medical resource and the exposure to solid fuel like coal used in the rural areas. The biomass burning for heating generates high carbon content. That can induce stronger oxidative stress in lung tissue. The third one is the latency between the exposure and the cancer diagnosis, most studies fail to account this hysteresis effect. The latency also varies by the subtype of the cancer, for example adenocarcinomas (PM2.5) developed much slower than squamous cell carcinomas (smoking). The aim of this article is to find the correlation between PM2.5 and lung cancer rate while considering the time delay and the developing index between different countries. To achieve that, this study uses linear regression setting the PM2.5 rate as the independent variable and the cancer rate as the dependent variable.

2. Methods

2.1. Data Source

This study has constructed a panel dataset of 20 countries observed annually from 1990 to 2010 (inclusive). The countries were selected based on data availability and quality, with an emphasis on those having reliable cancer registries and vital statistics over the 21-year period. The sample includes Australia, Austria, Belgium, Canada, China, Denmark, Finland, France, Germany, Israel, Italy, Japan, Netherlands, Norway, Singapore, South Korea, Sweden, Switzerland, the United Kingdom (UK), and the United States of America (USA). These nations span multiple regions (North America, Europe, East Asia, and Oceania) and different levels of economic development, although the majority are high-income economies. It has included China as a key upper-middle-income country experiencing rapid industrialization, to contrast with the high-income group. This purposive sampling captures a broad range of initial lung cancer rates and pollution levels, while focusing on countries with trustworthy longitudinal data (many low-income countries lacked continuous or reliable cancer statistics for this period). The rationale is that by selecting countries with strong medical reporting systems and consistent definitions, this paper reduces measurement error and ensure that observed changes are real and not artifacts of data quality. The variable definition is as Table 1 shows.

Table 1. Variable definitions

Lung Cancer Mortality	The annual number of new lung cancer cases diagnosed per 100,000 people
Lung Cancer incidence	The annual number of deaths due to lung cancer per 100,000 people
PM2.5	The population-weighted average concentration of fine particulate matter in ambient air.
GDP	Gross domestic product per person, adjusted according to purchasing power parity and inflation.

2.2. Data Description

From Table 2, several patterns can be observed. In 1990, the highest lung cancer incidence was in the USA (~113 per 100,000) and UK (~96), with many Western European countries in the 60–90 range, whereas much lower incidences appeared in Asia (e.g. China ~26, South Korea ~22). By 2010, incidence increased in most countries – notably, Japan's incidence nearly tripled (72 to 177) making it the highest in 2010, and China's more than doubled (26 to 57). Many European countries saw

sizable increases as well (France, Germany, Denmark all rose to ~100+). A few had more modest changes.

Mortality trends differed from incidence. Between 1990 and 2010, lung cancer mortality declined in several high-income countries despite the rising incidence. For instance, the UK's mortality dropped from 75 to 61, reflecting improvements in treatment and earlier detection, and reductions in male smoking. The USA and Denmark also had slightly lower mortality in 2010 than 1990, even as incidence rose, indicating progress in outcomes. Differently, countries like France and Germany experienced increased mortality over the period (e.g. France 44.5 to 58.2) due to rising cases (especially among women) outpacing any survival gains. In Asia, both incidence and mortality climbed steeply. China's mortality nearly doubled (23.6 to 43.5) and South Korea's exactly doubled (17.7 to 34.7) as smoking and aging took effect. Japan saw mortality rise from 33.9 to 60.2 (around 78% increase). These diverse trajectories show the importance of factors like smoking trends and healthcare improvements varying by country and sex. This analysis will control for these country-specific baseline differences via fixed effects, focusing on within-country changes over time and their association with pollution and GDP.

Table 2. Lung Cancer Mortality and Incidence in Selected Countries, 1990 and 2010 (per 100,000)

Country	Incidence 1990	Incidence 2010	Mortality 1990	Mortality 2010
Australia	63.0	96.5	38.4	39.0
Austria	55.4	99.1	44.4	48.0
Belgium	90.7	101.7	77.5	68.1
Canada	88.2	146.0	56.4	62.1
China	25.7	56.7	23.6	43.5
Denmark	94.5	137.1	78.6	73.8
Finland	62.7	86.9	41.3	42.6
France	56.0	135.0	44.5	58.2
Germany	75.6	121.1	52.0	59.4
Israel	25.7	36.6	23.8	26.4
Italy	67.9	99.9	57.6	61.2
Japan	72.0	177.6	33.9	60.2
Netherlands	80.5	120.0	62.3	67.5
Norway	55.3	107.6	38.8	46.0
Singapore	31.3	48.1	23.5	18.5
South Korea	21.8	86.5	17.7	34.7
Sweden	35.9	50.9	35.0	41.3
Switzerland	63.3	96.7	41.6	42.5
UK	95.8	115.4	75.1	61.3
USA	112.6	132.9	62.2	57.3

3. Regression Analysis Results

3.1. Model Results

Model A uses current year $PM_{2.5}$ and GDP. Model B uses a 1-year lag for both predictors. All models include country and year fixed effects. Robust t-statistics are in parentheses. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

In Model A, which examines within-country changes from 1990 to 2010, $PM_{2.5}$ has a positive coefficient of 0.79, statistically significant at the 5% level. Holding constant any broader trends and country-specific factors. This positive association aligns with the known carcinogenic effect of particulates (Table 3).

Table 3. Fixed-effects panel regression results for Lung Cancer Incidence (per 100k)

Predictor	Incidence Model A (FE)	Incidence Model B (FE, 1-year lag)
$PM_{2.5}$ (μg/m³)	+0.79 (2.3)**	+0.91 (1.7) *
GDP per capita ($1000)	-0.001 (–3.0)***	-0.002 (–13.3)***
Constant	21.81 (1.2)	–19.45 (–1.8)
Fixed effects (Country, Year)	Yes	Yes
Observations	420	400
R^2 (within)	0.943	0.938

Note: ** 0.01 significance, ***0.001 significance.

GDP per capita in Model A shows a coefficient of –0.001, significant at 1%. This means an increase of 1,000 in GDP per person is associated with a reduction of about 1.0 in the incidence rate. This negative relationship, conditional on country and year effects, likely reflects improvements in public health with development, for instance, better smoking cessation programs, early screening, and healthcare access that come with wealth. It might also capture an epidemiologic shift: as countries become wealthier beyond a point, smoking prevalence typically declines (due to education and regulations), and environmental quality may improve after initial industrial phases. The findings support this: within a given country, years of higher-than-expected GDP saw slightly lower lung cancer incidence than one would otherwise anticipate. Interestingly, the effect size here is small but significant. Without fixed effects, GDP's coefficient was positive, whereas with fixed effects it turns significantly negative – a clear indication of bias in the naive model. The fixed effects control for the fact that richer countries had higher baseline incidence; now looking at the changes over time, where rising GDP corresponds to declining incidence rates (after accounting for pollution and global trends).

Model B for incidence yields a very similar $PM_{2.5}$ coefficient (0.91) but with a slightly larger standard error ($p \approx 0.084$), marginally insignificant at 5% but significant at 10%). The GDP effect in Model B is –0.002, still highly significant. The increase in magnitude for GDP (–0.002 vs –0.001) might suggest that improvements in wealth take some time to manifest in lower incidence, or it could be capturing longer-run economic growth effects. The $PM_{2.5}$ coefficient being a bit higher with a lag could indicate a slight latency: pollution exposures might impact cancer incidence with a short delay. However, given a 1-year lag is likely too short for new tumors to develop, this probably reflects noise or the fact that using lagged values loses the first year (1990) where $PM_{2.5}$ was generally higher in some places. The main takeaway is that the positive association between $PM_{2.5}$ and incidence persists whether using concurrent or lagged pollution levels, consistent with a causal interpretation that sustained pollution leads to more cancer.

The R^2 values are around 0.94 in these FE models, meaning they explain ~94% of the variance in incidence within countries over time. This very high R^2 is expected because the model includes fixed effects that soak up a lot of variances. By comparison, the R^2 in the pooled model without fixed effects was only ~0.23, and adding country FE boosted it above 0.90. This underscores that most variation in lung cancer incidence is due to cross-country differences and overall time trends (like global smoking trends), while the within-country changes explained by $PM_{2.5}$ and GDP, though significant, are a smaller fraction. Still, the inclusion of $PM_{2.5}$ and GDP does add explanatory power: for incidence, the within-country R^2 rose from 0.216 with no FE to 0.943 with FE, indicating the full model fits the data very well. The F-tests for fixed effects (not shown) confirm they are jointly significant.

Model C uses current predictors; Model D uses 1-year lag. Country and year FE included in both. Robust t-statistics in parentheses.

In the mortality models (Table 4), the coefficients tell a consistent story. PM2.5 is positively and significantly associated with lung cancer mortality, with a somewhat larger magnitude than for incidence. Model C estimates that a 1μg/m³ increase in $PM_{2.5}$ raises the mortality rate by 1.07 per 100k ($p<0.001$). Put differently, a 10μg/m³ pollution increase would eventually translate into about 10.7 extra lung cancer deaths per 100k annually in that country. This is a substantial effect, considering that many countries' overall mortality rates are in the range of 40–70 per 100k (so a 10μg

change could shift mortality by on the order of ~15% of that range). It aligns with epidemiological estimates that long-term $PM_{2.5}$ exposure significantly elevates lung cancer mortality risk. The fact that the coefficient for mortality is slightly higher than for incidence could reflect the influence of pollution on both causing cancer and perhaps making cancer more fatal (for instance, pollution could exacerbate co-morbid respiratory conditions, reducing survival in lung cancer patients). It reflects that in countries where pollution decreased (Western nations), not only did fewer cases occur, but survival improved due to healthcare improvements – whereas in countries with pollution increases, more cases occurred and also survival gains may have been slower. The FE model's pollution coefficient captures net mortality outcomes.

Table 4. Fixed-effects regression results for Lung Cancer Mortality (per 100k)

Predictor	Mortality Model C (FE)	Mortality Model D (FE, 1-year lag)
$PM_{2.5}$ (μg/m³)	+1.07 (5.2)***	+0.57 (4.2)***
GDP per capita ($1000)	-0.0003 (–2.6)***	-0.0003 (–6.8)***
Constant	-16.16 (–1.6)	22.55 (8.4)***
Fixed effects (Country, Year)	Yes	Yes
Observations	420	400
R^2 (within)	0.971	0.967

GDP per capita in Model C has a small negative coefficient (–0.0003, $p<0.01$). which can be a sizeable portion of an average 60 per 100k mortality rate (about 5% reduction), this suggests that economic development has aided in reducing lung cancer mortality, likely via better treatment and early detection reducing case-fatality. It may also reflect cohort effects: as GDP rose, younger birth cohorts smoked less and had lower subsequent lung cancer mortality, an effect not fully captured by incidence changes yet within this period. It is notable that in the non-FE model, GDP's effect on mortality was positive (though very small), again showing how failing to account for baseline differences would mislead (wealthy countries had high past mortality, but changes in wealth reduce mortality).

In the lagged mortality Model D, $PM_{2.5}$ retains a positive coefficient of +0.57 ($p<0.001$). It roughly halved compared to the no-lag model, which could indicate that concurrent pollution has some immediate correlation with mortality, but after a year the effect is slightly attenuated. However, it remains highly significant and certainly not reversed – even a 1-year lag of pollution still shows that higher past $PM_{2.5}$ leads to more lung cancer deaths. The GDP effect in Model D is virtually the same (–0.0003) with strong significance, reinforcing that rising income per capita consistently correlates with improved lung cancer survival or lower mortality, even with a lag. The constant term difference (Model C's constant is negative and not significant, Model D's is positive and significant) is just an artifact of how the fixed effects absorb different grand means when lagging – not of substantive interest.

The R^2 for mortality models is extremely high (~0.97). Lung cancer mortality trends are slightly easier to predict than incidence because they change more slowly. The fixed effects plus the two covariates explain 96–97% of within-country variance in mortality. By contrast, a model without FE explained under 7% – again highlighting how much of the variance is cross-sectional and time-trend driven. The inclusion of fixed effects increased R^2 to ~0.95, and adding $PM_{2.5}$ and GDP nudged it to 0.971. Even if $PM_{2.5}$ and GDP effects seem numerically modest, they are systematically related to the portion of lung cancer trends not explained by generic time effects or country constants.

3.2. Key Findings and Interpretation

This study provides empirical evidence on how changes in ambient air pollution and economic conditions relate to lung cancer outcomes worldwide. The key statistical findings are: $PM_{2.5}$ air pollution has a positive, significant impact on lung cancer incidence and mortality in a global panel context, after controlling for country-specific characteristics and time trends; GDP per capita has a negative, significant association with lung cancer rates in recent decades, suggesting that

development (and its concomitant improvements in healthcare and risk factor control) helps reduce the lung cancer burden; Models that ignore fixed effects can be highly misleading – once this paper properly accounts for the long-term differences between countries and global secular trends, the true relationships emerge ($PM_{2.5}$ harmful, GDP beneficial). Additionally, this paper observed that incorporating even a short lag for pollution did not diminish its effect, implying that the rise in lung cancer from increased pollution exposure can begin to materialize in population statistics within a few years.

Quantitatively, the estimated pollution effect was on the order of 1 additional lung cancer death per 100k for each $\mu g/m^3$ increase in $PM_{2.5}$. This study also found that improvements in GDP per capita are linked to reductions in lung cancer incidence (~1 fewer case per 100k per $1k GDP). This likely reflects multiple indirect effects of development: better education and tobacco control, more resources for pollution regulation, and better medical care. In essence, as countries become wealthier, they can afford to tackle both primary prevention (reducing smoking, improving air quality) and secondary prevention (screening for early lung cancer in high-risk groups, as the US and some high-income countries have started to do).

This analysis also provides insight into the causal puzzle of why lung cancer trends differ between countries. This paper notes that even controlling for smoking implicitly via fixed effects, pollution independently contributes to risk. This suggests that the worldwide decline in male lung cancer mortality since 1990 in many high-income countries is not only due to reduced smoking, but also partly due to cleaner air. Conversely, in places where air quality worsened (e.g. parts of East Asia), it likely amplified the lung cancer rise driven by tobacco. This interplay might help explain why, for example, female lung cancer rates have risen even in never-smoking women in some urban areas, with second-hand smoke and pollution as probable contributors. The GDP finding similarly sheds light on an encouraging aspect: development brings a "protective" effect, likely through strong public health measures. Many Western countries have passed the peak of the tobacco epidemic and are reaping benefits; many developing countries are now at the inflection point (increasing rates), but if they continue to grow economically and implement public health interventions, they could similarly see a downturn in lung cancer in the future.

3.3. Strengths and Limitations

The strength of this study includes a comprehensive dataset covering 20 countries over two decades; fixed-effects models that robustly control for unobserved heterogeneity; lagged analyses revealing long-term PM2.5 effects. However, there are limitations for this study which includes: regional differences in its chemical composition are not taken into account (e.g. PM2.5 produced by coal burning contains higher levels of carcinogens PAHs, while traffic emissions are mainly nitrates). Differences in toxicity of PM2.5 from different sources may affect the dose-response relationship of lung cancer risk, resulting in biased regression coefficients. The absence of individual data including age, gender, smoking status which will affect the cancer rate.

4. Conclusion

Lung cancer incidence and mortality worldwide from 1990 to 2010 have been shaped by two forces: air pollution and economic development. Using a panel of 20 diverse countries, found that reductions in PM2.5 pollution are associated with significant decreases in lung cancer rates, whereas increases in pollution are associated with more cancer cases and deaths, highlighting ambient PM2.5 as an important modifiable risk factor on a population scale. At the same time, this paper observed that rising GDP per capita tends to coincide with lower lung cancer incidence and mortality, likely through improved healthcare systems and effective tobacco control that often accompany development. The fixed-effects regression approach demonstrated these effects while controlling for the long-standing differences between countries and addressing the time-lag challenge of carcinogenesis.

Looking ahead, future research should incorporate longer follow-up and more granular data (e.g., city-level analysis, cohort studies in emerging economies) to refine the understanding of exposure-response relationships and latency periods. It will also be important to monitor how emerging risk factors (such as electronic cigarette use or new environmental toxins) play into lung cancer trends.

References

[1] Chan, P.T.J., Dung, C.H., Jia, W., et al., 2021. Analysis of efficacy of intervention strategies for COVID-19 transmission: A case study of Hong Kong. Environment International, 156, 106723.

[2] Chen, H., Kinney, P. L., Liu, Y., Luo, J., Lv, Y., Shi, X., Wang, J., Xu, D., Yin, Z., Zeng, Y., Zhang, Y., 2018. Risk of all-cause mortality associated with long-term exposure to PM2.5. Environmental Health Perspectives, 470-477.

[3] Hamra, G.B., 2014. Outdoor particulate matter exposure and lung cancer: a systematic review and meta-analysis. Environmental Health Perspectives, 122 (9), 906–911.

[4] Lelieveld, J., Evans, J. S., Fnais, M., Giannadaki, D., Pozzer, A., 2015. The contribution of outdoor air pollution sources to premature mortality on a global scale. Nature, 525, 367-371.

[5] Li, T., Zhang, Y., Chan, P.T. J., et al., 2018. All-cause mortality risk associated with long-term exposure to ambient $PM_{2.5}$ in China: a cohort study. The Lancet Public Health, 3 (10), 470-477.

[6] Neal, R.D., Sun, F., Emery, J.D., Callister, M.E., 2019. Lung cancer. BMJ, 365, l1725.

[7] Perraud, V., Horne, J. R., Martinez, A.S., et al., 2015. The future of airborne sulfur-containing particles in the absence of fossil fuel sulfur dioxide emissions. Proceedings of the National Academy of Sciences, 112 (44), 13514-13519.

[8] Pope, C.A., Burnett, R.T., Thun, M.J., et al., 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA, 287 (9), 1132-1141.

[9] Raaschou-Nielsen, O., Andersen, Z.J., Beelen, R., et al., 2013. Air pollution and lung cancer incidence in 17 European cohorts: prospective analyzes from the ESCAPE study. Lancet Oncology, 14 (9), 813-822.

[10] Yin, P., Brauer, M., Cohen, A., et al., 2020. The effect of air pollution on deaths, disease burden, and life expectancy across China and its provinces, 1990–2017: an analysis for the Global Burden of Disease Study 2017. Lancet Planetary Health, 4 (9), e386-e398.