

Investigating the Association between Environmental Degradation and Population Health: A Country-Level Study

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Abstract

Environmental degradation is widely held to harm human health, yet cross-national evidence is complicated by the fact that the same affluence that reduces pollution also funds the health systems that extend life, so simple comparisons confound the two. Using a country-year panel of 70 countries observed over 2009-2020 (721 observations, averaging 10.3 years per country and spanning all four World Bank income groups), we relate six environmental-degradation indicators—particulate pollution, per-capita greenhouse-gas emissions, and energy, mineral, and forest depletion, and freshwater withdrawals—to two population-health outcomes, life expectancy and the incidence of tuberculosis, and a composite, controlling for income, education, and health-system capacity (physicians and health expenditure). Environmental degradation is associated with worse health: higher particulate pollution, greenhouse-gas emissions, and energy and forest depletion are associated with lower life expectancy, and energy depletion with higher tuberculosis incidence. The central result is the particulate-pollution association with life expectancy, which is large and significant both across countries and—unlike most cross-national environmental associations—within countries over time, surviving country and year fixed effects and net of health-system capacity, and robust to measuring exposure with one- to three-year lags. Directional tests find no evidence that health outcomes predict subsequent environmental degradation, and no evidence of temporal precedence in either direction for the particulate-longevity pair, so the estimates are reported as associations consistent with an environment-to-health reading rather than as causal identification. We assess robustness with fixed-effects, Driscoll-Kraay, and system-GMM estimators and specification, reverse-causality, Granger, and lagged-exposure tests, and interpret the results through the environmental burden of disease and the development-health literature. The findings indicate that air pollution in particular carries a

measurable longevity cost that economic development and medical capacity do not fully offset.

Keywords

Environmental Degradation, Air Pollution, Particulate Matter, Life Expectancy, Tuberculosis, Population Health, Climate Change, Panel Data, Environmental Burden of Disease

1. Introduction

That a degraded environment is bad for human health is among the most consequential propositions in sustainability and public-health research. Air pollution, contaminated water, and the depletion of natural resources are all plausibly linked to disease and premature death, and quantifying these links is central to valuing environmental policy. Yet establishing the relationship across countries is harder than it appears, because the environment and health are both bound to economic development. Affluent countries tend to have cleaner air and longer lives, but they also fund the health systems—physicians, hospitals, medicines—that extend life independently of the environment. A simple comparison of polluted and clean countries therefore risks attributing to the environment what is in fact the work of income and medical capacity.

Three features of the problem make it more demanding than a single correlation can capture, and each shapes the design that follows. The first is that environmental degradation is not one thing but many. Airborne particulates, greenhouse-gas emissions, and the depletion of energy, mineral, and forest resources act on human health through distinct physical and economic channels, and they need not carry the same health consequence; a stressor that shortens lives through direct inhalation exposure operates quite differently from one that registers only as a national accounting entry for resource drawdown. Collapsing these into a single index of environmental quality would average away exactly the heterogeneity a health assessment needs, since the question is not whether the environment in general matters for health but which stressors do, and how much.

The second feature is that population health is likewise not a single quantity. Life expectancy summarizes mortality across all causes and ages, whereas the incidence of an infectious disease such as tuberculosis responds to a narrower and partly different set of forces—crowding, nutrition, immune competence, and the reach of case-finding and treatment. An environmental stressor may register in one of these outcomes and not the other, and only by relating each stressor to each outcome within a common framework can the pattern of association be observed rather than presumed. We therefore adopt a deliberately delineated design, relating six environmental stressors to two health outcomes and a composite, so that the reach of each stressor across the health outcomes is visible directly.

The third feature is that inference is fragile. Because environmental quality, in-

come, and health are jointly determined, a partial correlation between a country's pollution burden and its longevity is weak evidence of an environmental effect: it may reflect the affluence that lowers pollution and lengthens life through unrelated channels, the medical capacity that affluence funds, or reverse feedback in which longer-lived, wealthier populations demand and obtain cleaner environments. Credible inference therefore requires holding constant not only income and education but health-system capacity, asking whether any association survives within countries over time as well as across them, and testing the direction of the relationship. Our design does each of these and treats the surviving within-country association as the demanding test against which the headline claim is judged.

This study addresses that confound directly. We relate six measures of environmental degradation to two core population-health outcomes—life expectancy and the incidence of tuberculosis—across a large country-year panel spanning the full range of national income, and we hold constant not only income and education but health-system capacity, so that the estimated environmental associations are net of the medical resources that would otherwise drive them. We then ask whether any association survives within countries over time, the more demanding test, and whether health outcomes predict subsequent environmental change, as reverse feedback would imply.

The contribution is a delineated, country-level mapping of which environmental stressors are associated with which health outcomes, net of development and medical capacity; evidence that air pollution in particular carries a longevity cost that holds within countries and not merely across them; and directional tests that find no evidence of reverse feedback from health to the environment, which is consistent with, though not sufficient to establish, an environment-to-health reading.

The remainder of the paper is organized as follows. Section 2 reviews the relevant literatures—on environmental degradation and health, on particulate air pollution and mortality, on development, health systems, and the Preston curve, on resource depletion and the broader environmental burden of disease, and on the identification problems that motivate the design. Section 3 develops the conceptual framework and states the hypotheses. Section 4 describes the data and methods. Section 5 presents the results and the within-country robustness analysis. Section 6 reports the endogeneity and directional tests. Section 7 discusses the findings; Section 8 draws out their implications for policy and scholarship; Section 9 considers the scope and limitations of the study; and Section 10 concludes and outlines directions for future research.

2. Literature Review

2.1. Environmental Degradation and Health

Numerous literature documents the health consequences of environmental degradation, encompassing air and water pollution, resource depletion, and climate

change. The environmental burden of disease is substantial, with ambient air pollution alone implicated in millions of premature deaths annually. The challenge in cross-national work is separating the environmental signal from the developmental context in which it sits, since the wealth that lowers some forms of pollution also extends life through channels unrelated to the environment.

We therefore frame the analysis around population-health outcomes that are plausibly environment-sensitive—life expectancy and tuberculosis incidence—and treat health-system capacity as a control rather than as a competing explanation to be ignored.

The scale of the burden is what makes its cross-national measurement worth the difficulty. Ambient and household air pollution together rank among the leading environmental risk factors in global burden-of-disease accounting, implicated in millions of premature deaths each year and in a substantial share of the disease burden from cardiovascular and respiratory conditions (Cohen et al., 2017; Landrigan et al., 2018). Such estimates are built up from individual-level dose-response relationships applied to population exposures; the complementary question, and the one a country panel can address, is whether the burden they imply is visible in the movement of aggregate health outcomes—whether countries carrying heavier environmental burdens have measurably shorter lives, and whether reductions in that burden within a country track gains in longevity over time. The value of the cross-national view is not that it improves on the epidemiology but that it tests whether the epidemiological signal aggregates to the national scale at which environmental policy is set.

2.2. Air Pollution and Mortality

The most developed strand of this literature concerns particulate air pollution and mortality. Fine particulate matter is robustly associated with cardiovascular and respiratory disease and with elevated mortality in cohort and time-series studies (Pope III & Dockery, 2006), and air pollution is plausibly linked to respiratory infections including tuberculosis. The cross-national question is whether these micro-level associations aggregate to a measurable population-level relationship between a country's particulate burden and its life expectancy, net of income and medical care—and, more demanding, whether changes in pollution within a country track changes in longevity over time.

The mechanisms are well characterized at the individual level. Fine particulate matter penetrates deep into the respiratory tract and enters the bloodstream, contributing to systemic inflammation, atherosclerosis, and impaired pulmonary function, and it is associated with elevated cardiovascular and respiratory mortality in large cohort and time-series studies (Pope III & Dockery, 2006). Particulate exposure has also been linked to susceptibility to respiratory infection, which supplies a plausible pathway to tuberculosis incidence, although that population-level association is weaker and more heavily mediated by the social determinants that dominate infectious-disease transmission. The strength of the individual-level ev-

idence for the mortality channel is what makes particulate pollution the natural focal stressor for a longevity outcome, and it is why we treat the particulate-life-expectancy association as the study's central estimate rather than as one association among six.

What the micro-level evidence cannot settle is whether the relationship is identified within countries or only across them. Cross-sectional comparisons of polluted and clean nations are confounded by everything that distinguishes rich from poor countries, so a negative pollution-longevity correlation in the cross-section is compatible with a purely developmental explanation. The more demanding question is whether a country that reduces its particulate burden over time experiences a corresponding gain in life expectancy, net of the income and medical resources that also change. This is precisely the question our fixed-effects specification is designed to answer, and it is the margin on which most cross-national environmental associations weaken.

Our design speaks to exactly this question, and the particulate-pollution-life-expectancy association is the study's central estimate.

2.3. Development, Health Systems, and the Preston Curve

The relationship between income and health is summarized by the Preston curve, in which life expectancy rises steeply with income at low levels and flattens at high ones (Preston, 1975). Because income drives both environmental quality and health-system capacity, the Preston relationship is the principal confounder in any environment-health analysis. The environmental Kuznets curve adds a parallel income-degradation relationship (Grossman & Krueger, 1995; Stern, 2004), and the STIRPAT framework situates environmental impact within population, affluence, and technology (Dietz & Rosa, 1997; York et al., 2003). Together these motivate controlling for income, education, and—crucially—health-system capacity, so that environmental associations are not simply proxying for medical resources.

The Preston relationship is not merely a control to be included but the central inferential obstacle. Because income shifts the entire mapping from environment to health—cleaning the air, funding physicians and hospitals, and raising education simultaneously—any environmental coefficient estimated without holding development constant is liable to be development in disguise (Deaton, 2013). The response adopted here is to condition on income and its square, on education, and, unusually for cross-national environment-health work, on two direct measures of health-system capacity: physicians per capita and health expenditure. Including medical capacity as a control means the environmental associations are estimated net of the very resources through which affluence most plausibly lengthens life independently of the environment, so that what remains is less easily dismissed as a proxy for development. The environmental Kuznets curve and STIRPAT traditions (Dietz & Rosa, 1997; Dinda, 2004; Grossman & Krueger, 1995; Stern, 2004; York et al., 2003) supply the parallel logic on the environmental side, in which income drives degradation through offsetting scale, composition,

and technique effects, and they motivate treating income as a shape to be estimated rather than a linear nuisance.

2.4. Resource Depletion and the Broader Environmental Burden

Beyond air pollution, the depletion of energy, mineral, and forest resources and the withdrawal of freshwater constitute a broader environmental burden whose health consequences are more diffuse and less directly tied to human exposure. Resource depletion acts on health partly through the degradation of the ecosystems on which subsistence and livelihoods depend, partly through the pollution that extraction and combustion generate, and partly as a marker of an extractive development path with its own health profile. Forest loss, for example, is associated with altered disease ecologies and with the loss of provisioning services, while energy depletion often accompanies the fossil-intensive activity that also raises local pollution. These channels are real but indirect, and the expectation is that resource-depletion stressors carry a smaller and less robust health cost than direct-exposure pollutants.

Freshwater withdrawal occupies an ambiguous position in this scheme. Water is fundamental to health, yet national withdrawal volumes are governed largely by agriculture, industry, and climate rather than by any direct health-relevant exposure, and withdrawals tend to rise with the economic development that also lengthens life. A cross-national withdrawal measure therefore behaves less as a stressor than as a development indicator, a point that becomes important when interpreting its association with life expectancy. Including it alongside the other stressors is nonetheless useful, both because water is a central environmental dimension and because its anomalous behavior illustrates the more general hazard of reading a development-correlated environmental measure as a health effect. This multi-stressor treatment—relating each environmental dimension to each health outcome rather than summarizing them—parallels the multi-outcome logic increasingly adopted in cross-national environmental analysis and is what allows the direct-exposure and resource-accounting stressors to be distinguished rather than merged.

2.5. Confounding, Reverse Causation, and Identification

The methodological literature on cross-national environment-health analysis converges on a single caution: the relationship is confounded, dynamic, and potentially bidirectional, so that a partial correlation is weak evidence of an environmental effect. Confounding by income and institutions is the first-order concern, addressed here by conditioning on development and medical capacity. Persistence is the second: both health outcomes and environmental conditions adjust slowly, so contemporaneous associations may over- or understate the relationships that would obtain in equilibrium, which motivates dynamic estimation even where the resulting estimates are imprecise. Reverse causation is the third, and the most conceptually interesting: healthier, longer-lived populations are also wealthier and better organized and may demand and obtain cleaner environments, so an envi-

ronment-to-health reading cannot simply be assumed.

Our identification strategy responds to each of these in turn without overclaiming. We absorb unobserved country and year heterogeneity through two-way fixed effects and allow for cross-sectional dependence with Driscoll-Kraay standard errors (Driscoll & Kraay, 1998); we probe persistence with a system-GMM estimator (Roodman, 2009) that we report but do not lean upon, given the near-unit-root behavior of life expectancy; and we test direction directly, relating lagged health outcomes to current environmental stressors and running panel Granger tests in both directions. Neither device identifies causal direction: fixed effects absorb only heterogeneity that is constant within a country, leaving time-varying confounders untouched, and panel Granger tests bear on temporal precedence in the conditional mean rather than on causation. We deliberately do not report an external-instrument estimate, because no credible exclusion restriction is available for a national environmental stressor with respect to national health, and a weak or invalid instrument would purchase the appearance of causal identification at the cost of its substance. The coefficients are accordingly interpreted as conditional associations—strengthened where they survive within-country variation and the directional tests—and this graduated posture, claiming association, testing direction, and declining to manufacture an instrument, is central to how the results should be read.

2.6. The Gap

Two gaps motivate this study. First, cross-national environment-health work often examines a single stressor or a single outcome, so the comparative association of different environmental stressors with different health outcomes is rarely assessed within a common framework that also controls for health-system capacity; as a result, a carbon or air-pollution finding is easily reported as an environmental one, and the medical resources that confound the relationship are often left out. Second, much of the evidence is cross-sectional, leaving open whether environmental associations with health survive within countries over time—the margin on which the developmental confound is hardest to sustain. We address both by relating six environmental stressors to two health outcomes and a composite in a delineated design, controlling for income, education, and medical capacity, and by re-estimating every relationship within countries and testing its direction.

3. Conceptual Framework and Hypotheses

Figure 1 organizes the analysis around a single distinction: environmental degradation acts on population health through an environmental-burden channel, in which pollution and resource depletion raise disease and mortality, while development and health-system capacity act on health through separate channels that must be held constant if the environmental burden is to be isolated. The environmental-burden channel is strongest where human exposure is most direct, which locates the clearest expected effect in particulate air pollution, whose link to mortality is the best established at the individual level. Resource-depletion stressors sit

further from direct exposure and are expected to carry smaller and less robust effects, while freshwater withdrawal, governed largely by development, is expected to behave anomalously.

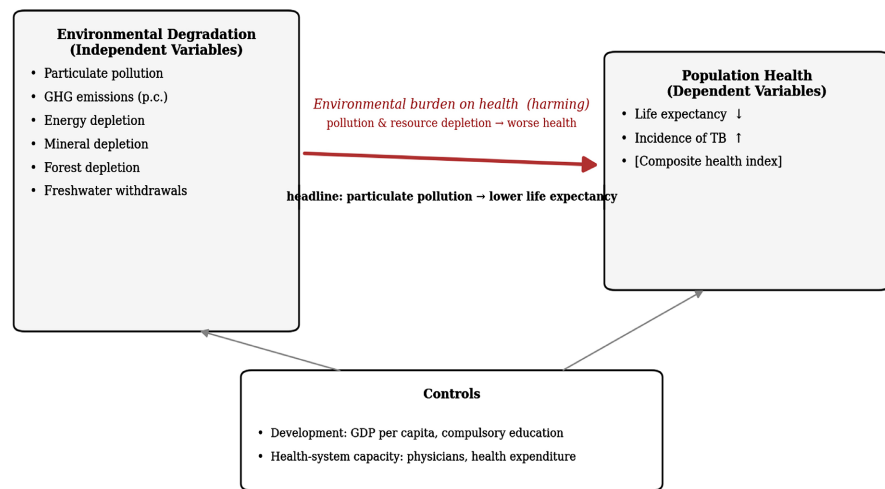


Figure 1. Conceptual framework: environmental degradation acts on population health, net of development and health-system capacity; particulate pollution is the headline channel.

The framework's inferential content lies in what it holds constant. Because affluence simultaneously cleans the environment, funds medical care, and lengthens life, the environmental associations of interest are defined only net of income, education, and health-system capacity; the coefficients reported below are conditional on these, and the framework treats the surviving within-country association—rather than the raw cross-country correlation—as the quantity that speaks to an environmental effect. The directional arrows run from environment to health, but the framework admits the possibility of feedback, which the analysis probes rather than assumes; the tests that follow bear on temporal precedence and on the presence of feedback, not on causal identification.

From this framework we derive five expectations, each tied to the logic above. First, environmental degradation as a whole will be associated with worse population health—lower life expectancy and higher tuberculosis incidence—so that the balance of stressor-outcome associations is harmful rather than neutral (H1). Second, particulate air pollution, as the stressor most directly tied to human exposure and best established as a cause of mortality, will be the strongest stressor and will be associated with lower life expectancy (H2). Third, and most demanding, this particulate association will hold not only across countries but within countries over time, surviving country and year fixed effects and the health-system controls—the test that distinguishes an environmental effect from its developmental correlates (H3). Fourth, resource depletion, particularly of energy and forests, will also be associated with worse health, though less strongly and less robustly than direct-exposure pollution, reflecting its more indirect channels (H4). Fifth, health outcomes will not predict subsequent environmental degradation, so that reverse feedback is not

the source of the association (H5). The freshwater-withdrawal measure, by contrast, is expected to behave as a development indicator rather than a stressor, a prediction we treat as a diagnostic on the design rather than as a substantive hypothesis.

4. Data and Methods

4.1. Data and Sample

We use a country-year panel of 70 countries observed from 2009 to 2020, comprising 721 observations and averaging 10.3 years per country, compiled from World Bank indicators. The sample spans all four World Bank income groups, providing variation across the full development range and supporting both cross-country and within-country estimation. Countries enter the panel if the World Bank reports all six environmental stressors, both health outcomes, and every control variable in at least one year of the window, and country-years with any missing analysis variable are dropped, so the estimation sample is complete by construction and no imputation is used. The 70 retained countries supply 721 of the 840 country-years the 2009-2020 window allows, or 85.8 per cent; coverage ranges from twelve years for 32 countries to two years for the least-covered one. Appendix A lists every country with its income group, region, and years observed.

The World Bank indicators offer internationally comparable environmental, economic, and health series with the broad country coverage needed to identify the relationships of interest, and the panel structure—repeated observation of the same countries—supports estimation both across countries and within them over time. The inclusion of all four income groups is deliberate: variation across the full development range is what makes it possible to separate the environmental associations from the developmental context, since a sample confined to affluent countries would offer little leverage on the confound at the heart of the analysis. The panel is close to but not perfectly balanced, averaging 10.3 of a possible twelve years per country, and all national-total variables are expressed in per-capita or intensity terms before analysis so that estimated relationships reflect environmental intensity rather than country size. Because the complete-case rule is applied to the same variable set for every country, coverage is uneven across the income range: the 36 high-income countries supply 92.4 per cent of their possible country-years, the 19 upper-middle-income countries 82.9 per cent, the 10 lower-middle-income countries 74.2 per cent, and the 5 low-income countries 73.3 per cent. The cross-income-group readings below therefore rest on denser data at the top of the income distribution than at the bottom, and the low-income group contributes 44 of the 721 observations. In counts, 119 of the 840 possible country-years are excluded: 33 of 432 in the high-income group, 39 of 228 in the upper-middle-income group, 31 of 120 in the lower-middle-income group, and 16 of 60 in the low-income group.

4.2. Variables

Each environmental stressor enters as a distinct independent variable and each health outcome as a distinct dependent variable ([Table 1](#) and [Table 2](#)). Life ex-

pectancy is oriented so that higher values denote better health and tuberculosis incidence so that higher values denote worse health; the composite Health Index is sign-oriented so that higher values denote better health. Emissions are expressed per capita and income as the natural log of GDP per capita, mean-centered, with a squared term for the EKC. Crucially, we control for health-system capacity—physicians per capita and health expenditure—so that environmental associations are estimated net of medical resources.

Table 1. Independent and control variables.

Variable	Definition	Unit	Source
Environmental degradation (independent variables)			
Particulate pollution	Adjusted savings: particulate emission damage (NY.ADJ.DPEM.GN.ZS)	% of GNI	WB
GHG emissions (per capita)	Greenhouse-gas emissions per capita (EN.ATM.GHGT.KT.CE ÷ SP.POP.TOTL × 10 ⁶)	kg CO ₂ e per capita	WB
Energy depletion	Adjusted savings: energy depletion (NY.ADJ.DNGY.GN.ZS)	% of GNI	WB
Mineral depletion	Adjusted savings: mineral depletion (NY.ADJ.DMIN.GN.ZS)	% of GNI	WB
Forest depletion	Adjusted savings: net forest depletion (NY.ADJ.DFOR.GN.ZS)	% of GNI	WB
Freshwater withdrawals	Annual freshwater withdrawals (ER.H2O.FWTL.ZS)	% of internal resources	WB
Control variables			
GDP per capita	GDP per capita, current US\$ (NY.GDP.PCAP.CD); natural log, mean-centred, with squared term for EKC	US\$	WB
Compulsory education	Duration of compulsory education (SE.COM.DURS)	years	WB
Physicians	Physicians per 1000 people (SH.MED.PHYS.ZS; health-system capacity)	per 1000	WB
Health expenditure	Current health expenditure (SH.XPD.CHEX.GD.ZS; health-system capacity)	% of GDP	WB
Population	Total population (SP.POP.TOTL; natural log)	persons	WB

Table 2. Dependent variables (population-health outcomes).

Variable	Definition	Unit	Source
Life expectancy	Life expectancy at birth (SP.DYN.LE00.IN; higher = better)	years	WB
Incidence of TB	Incidence of tuberculosis (SH.TBS.INCD; higher = worse)	per 100,000	WB
Health Index	Equal-weighted, sign-oriented standardized composite (higher = better)	std. index	Authors

The composite Health Index is formed by standardizing each health outcome, orienting each so that higher values denote better health, and averaging with equal weights; it is reported as a descriptive summary rather than as the primary outcome, since the delineated stressor-by-outcome estimates are where the analysis does its work. Two design choices warrant emphasis. First, health-system capacity enters as a control rather than as an outcome: physicians per capita and health expenditure are the principal channels through which affluence lengthens life independently of the environment, so conditioning on them is what makes the residual environmental associations interpretable. GDP per capita, health expenditure, and physicians are treated as confounders, on the argument that their levels are set by the development path rather than by environmental degradation. Where that argument fails—where a control responds to environmental degradation—the variable is post-exposure, and conditioning on it can move the environmental coefficient in either direction, through collider as well as mediator paths, rather than only toward zero (Montgomery et al., 2018). The estimates are accordingly not claimed as a lower bound on the environmental burden, and Section 5.4 reports the central estimate with and without the health-system controls. Second, income enters in logs with a squared term, following the environmental Kuznets and Preston logics, so that the strongly non-linear relationship between income and both environment and health is captured rather than forced into a line.

Every series is taken from the World Bank's World Development Indicators (World Bank, n.d.), and the indicator codes and transformations are as follows. Particulate pollution is adjusted savings: particulate emission damage (NY.ADJ.DPEM.GN.ZS), in per cent of GNI; the World Bank builds it from Global Burden of Disease estimates of mortality attributable to ambient PM_{2.5}, ambient ozone, and household PM_{2.5} from solid-fuel cooking, valued as foregone labour income, with non-benchmark years extrapolated from mortality trends. Energy, mineral, and net forest depletion are the companion adjusted-savings series (NY.ADJ.DNGY.GN.ZS, NY.ADJ.DMIN.GN.ZS, NY.ADJ.DFOR.GN.ZS), also in per cent of GNI. Greenhouse-gas emissions are total emissions in kilotonnes of CO₂ equivalent (EN.ATM.GHGT.KT.CE) divided by total population (SP.POP.TOTL) and multiplied by 10⁶, giving kilograms of CO₂ equivalent per capita. Freshwater withdrawals are annual total withdrawals as a per cent of internal renewable resources (ER.H2O.FWTL.ZS), a ratio that exceeds 100 where withdrawal draws on external or non-renewable sources. Life expectancy at birth (SP.DYN.LE00.IN) is in years and tuberculosis incidence (SH.TBS.INCD) per 100,000 people. Among the controls, GDP per capita is in current US dollars (NY.GDP.PCAP.CD), entered as its natural logarithm, mean-centred on the estimation sample, together with the square of that centred term; current health expenditure is in per cent of GDP (SH.XPD.CHEX.GD.ZS); physicians are per 1,000 people (SH.MED.PHYS.ZS); compulsory education is its duration in years (SE.COM.DURS); and population (SP.POP.TOTL) enters in natural logarithms. No price deflator is applied: the only monetary variable, GDP per capita, enters in

logarithms and mean-centred, every other variable is a ratio, rate, or physical intensity, and no aggregation is performed beyond the per-capita and per-cent-of-GNI normalisations carried by the source series themselves.

Coefficients are therefore read in the units of the source series. A within-country particulate coefficient of -2.849 on life expectancy says that a one-percentage-point increase in particulate emission damage as a share of GNI accompanies a fall of 2.849 years in life expectancy; a one-standard-deviation increase in the measure (0.67 percentage points) corresponds to about 1.9 years. An energy-depletion coefficient of -0.271 in the pooled model is per percentage point of GNI, and a greenhouse-gas coefficient is per kilogram of CO_2 equivalent per capita, so that a one-tonne increase in per-capita emissions corresponds to 1,000 times the tabulated value.

4.3. Estimation Strategy

We report descriptive statistics, a correlation matrix, and variance-inflation factors, then estimate for each health outcome a multiple linear regression on the full set of six environmental stressors and controls, with country-clustered standard errors. We re-estimate the full model with two-way fixed effects and Driscoll-Kraay standard errors to identify associations from within-country variation and to allow for cross-sectional dependence. To make the pattern explicit, we tally, for each estimator, the environmental associations that harm health versus those that appear protective, and we display the full sign pattern.

For robustness we retain all six stressors throughout and add a specification test of fixed against random effects in cluster-robust auxiliary-regression form (Hausman, 1978; Mundlak, 1978), a Blundell-Bond system-GMM estimator (Roodman, 2009) with the lagged dependent variable and the full stressor set (instruments collapsed; variables standardized), reverse-causality and panel-Granger tests for every stressor-outcome pair to assess temporal precedence in each direction, and a set of lagged and moving-average exposure specifications for the central particulate association. We do not report an external-instrument estimate, as a credible exclusion restriction is unavailable; coefficients are interpreted as conditional associations.

The logic of this graduated sequence is to let the reader see how the central associations behave as successive identifying assumptions are relaxed. The pooled regression establishes the conditional associations in the full cross-section and time series; the two-way fixed-effects specification removes all stable between-country differences and identifies the associations from within-country variation alone, the more demanding test; the Driscoll-Kraay standard errors guard against the cross-sectional dependence that national environmental and health series are likely to exhibit; and the dynamic and directional tests probe persistence and the direction of the relationship. Rather than resting the conclusions on any single estimator, we report the sign and significance pattern across estimators and tally, for each, the environmental associations that harm health against those that appear protective, so that the coherence—or fragility—of the pattern is visible directly rather than asserted.

5. Results and Analysis

5.1. Descriptive Statistics

Table 3 summarizes the sample, which spans the full range of national income, environmental quality, and population health.

The spread is wide on every dimension. Life expectancy ranges from the low fifties to the mid-eighties, tuberculosis incidence from a handful of cases per hundred thousand to several hundred, and GDP per capita across more than two orders of magnitude, so the sample contains the developmental heterogeneity on which identification depends. The environmental stressors are similarly dispersed, and their skewness—several are bounded below at zero and have long right tails—reflects the concentration of resource depletion and heavy pollution in particular countries and years.

Table 3. Descriptive statistics (721 country-year observations).

Variable	N	Mean	Std. Dev.	Min/Max
Life expectancy (years)	721	76.08	6.62	50.54/84.56
Incidence of TB (per 100k)	721	52.79	77.72	2.10/428.00
Health Index (composite)	721	0.00	0.90	−3.12/0.90
GHG emissions (per capita)	721	8,043	5,602	451/36,691
Energy depletion (% GNI)	721	1.29	3.20	0.00/24.42
Mineral depletion (% GNI)	721	0.45	1.17	0.00/7.99
Particulate pollution	721	0.38	0.67	0.01/3.63
Freshwater withdrawals	721	57.07	363.00	0.15/4748.41
Forest depletion (% GNI)	721	0.43	1.34	0.00/7.64
Physicians (per 1k)	721	2.85	1.52	0.01/7.95
Health expenditure	721	7.17	2.62	2.08/13.68
Compulsory education (yrs)	721	10.08	1.96	5.00/15.00
GDP per capita (US\$)	721	23,994	23,634	455/123,679

5.2. Correlation and Collinearity

Table 4 reports Pearson correlations. No variance-inflation factor exceeds six (maximum 5.1), so multicollinearity does not threaten the estimates.

Table 4. Correlation matrix.

	1	2	3	4	5	6	7	8	9	10	11
1. LifeExp	1										
2. TB	−.64	1									
3. GHG per capita	.42	−.24	1								
4. Energy depletion	−.23	.22	.26	1							

Continued

5. Mineral depletion	−.43	.37	−.08	.06	1						
6. Particulate pollution	−.85	.49	−.40	.06	.34	1					
7. Freshwater withdrawals	.03	−.01	.40	.48	−.05	.00	1				
8. Forest depletion	−.62	.35	−.31	.03	.22	.62	−.05	1			
9. loggdp_c	.90	−.65	.57	−.12	−.40	−.77	.03	−.55	1		
10. Physicians	.58	−.44	.30	−.06	−.28	−.61	−.12	−.52	.60	1	
11. HealthExp	.64	−.60	.18	−.34	−.27	−.45	−.12	−.32	.63	.60	1

Variables: 1 = Life expectancy (years); 2 = Incidence of TB (per 100k); 3 = GHG emissions (per capita); 4 = Energy depletion (% GNI); 5 = Mineral depletion (% GNI); 6 = Particulate pollution; 7 = Freshwater withdrawals; 8 = Forest depletion (% GNI); 9 = log GDP per capita; 10 = Physicians (per 1k); 11 = Health expenditure.

5.3. Multiple Linear Regression: Each Stressor on Each Health Outcome

Table 5 reports the coefficient of every environmental stressor and control on each health outcome, each from a separate country-clustered regression. Environmental degradation is associated with worse health: higher particulate pollution, greenhouse-gas emissions, and energy and forest depletion are each associated with lower life expectancy. The particulate-pollution coefficient is the largest and most precisely estimated, and the life-expectancy model fits closely ($R^2 = 0.93$), reflecting the strong role of the income and health-system controls against which the environmental associations are net. **Table 6** and **Table 7** report the full regressions for life expectancy and tuberculosis incidence.

Table 5. Multiple linear regression coefficients: each environmental stressor on each health outcome (pooled).

Predictor	LifeExp	TB	HealthIdx
GHG emissions (per capita)	-0.000*	0.002	-0.000
Energy depletion (% GNI)	-0.271***	1.747	-0.032*
Mineral depletion (% GNI)	-0.186	7.457	-0.062
Particulate pollution	-3.670***	-1.430	-0.268*
Freshwater withdrawals	0.002***	-0.018	0.000*
Forest depletion (% GNI)	-0.539*	-0.067	-0.040
log GDP per capita	2.818***	-28.536**	0.396***
Compulsory education (yrs)	-0.313**	-1.147	-0.016
Physicians (per 1k)	-0.261	1.471	-0.029
Health expenditure	0.352**	-8.209**	0.079***
log Population	0.262	5.023	-0.013
R²	0.93	0.53	0.83

Each column is a separate regression; country-clustered standard errors. *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$.

The signs are as the framework anticipates. Among the stressors, particulate

pollution carries by far the largest life-expectancy penalty, and greenhouse-gas emissions and energy and forest depletion each enter negatively, so that the direct-exposure and resource-accounting stressors align in harming longevity. The controls behave as the developmental logic predicts: income enters strongly and positively, and health expenditure positively, confirming that the environmental associations are estimated against a backdrop in which development and medical capacity are the dominant correlates of longevity. That the environmental coefficients remain significant net of these controls is the first indication that they are not simply proxying for development. The tuberculosis model fits less well and yields few significant stressor associations in the pooled specification, consistent with infectious-disease incidence being governed more heavily by social and programmatic factors than by the environmental stressors measured here.

Table 6. Full regression for life expectancy.

Predictor	Coef.	Std. err.	t	Sig.
Constant	75.956	2.450	31.00	<0.001***
GHG emissions (per capita)	−0.000	0.000	−2.15	0.031*
Energy depletion (% GNI)	−0.271	0.070	−3.86	<0.001***
Mineral depletion (% GNI)	−0.186	0.194	−0.96	0.337
Particulate pollution	−3.670	0.594	−6.18	<0.001***
Freshwater withdrawals	0.002	0.000	4.55	<0.001***
Forest depletion (% GNI)	−0.539	0.266	−2.03	0.043*
log GDP per capita	2.818	0.333	8.47	<0.001***
Compulsory education (yrs)	−0.313	0.109	−2.88	0.004**
Physicians (per 1k)	−0.261	0.196	−1.33	0.182
Health expenditure	0.352	0.120	2.94	0.003**
log Population	0.262	0.141	1.86	0.062

$R^2 = 0.925$; adj. $R^2 = 0.924$; $F = 100.7$; $N = 721$.

Table 7. Full regression for tuberculosis incidence.

Predictor	Coef.	Std. err.	t	Sig.
Constant	20.837	51.634	0.40	0.687
GHG emissions (per capita)	0.002	0.002	0.85	0.394
Energy depletion (% GNI)	1.747	2.139	0.82	0.414
Mineral depletion (% GNI)	7.457	10.387	0.72	0.473
Particulate pollution	−1.430	23.627	−0.06	0.952
Freshwater withdrawals	−0.018	0.019	−0.99	0.324
Forest depletion (% GNI)	−0.067	7.734	−0.01	0.993
log GDP per capita	−28.536	9.929	−2.87	0.004**
Compulsory education (yrs)	−1.147	3.524	−0.33	0.745

Continued

Physicians (per 1k)	1.471	4.591	0.32	0.749
Health expenditure	−8.209	2.980	−2.75	0.006**
log Population	5.023	4.683	1.07	0.283

$R^2 = 0.526$; adj. $R^2 = 0.518$; $F = 7.2$; $N = 721$.

5.4. Within-Country Robustness: Fixed Effects

Table 8 re-estimates the full model under two-way fixed effects. The central result survives: higher particulate pollution remains associated with lower life expectancy within countries over time, net of country and year effects and of health-system capacity, and energy depletion is associated with higher tuberculosis incidence. That the particulate-life-expectancy association holds within countries—where most cross-national environmental associations weaken—is the strongest evidence in the study. Driscoll-Kraay standard errors yield the same qualitative pattern.

Table 8. Fixed-effects coefficients: each environmental stressor on each health outcome (within-country).

Predictor	LifeExp	TB	HealthIdx
GHG emissions (per capita)	0.000	−0.001	0.000
Energy depletion (% GNI)	−0.043	2.333*	−0.018*
Mineral depletion (% GNI)	−0.040	0.182	−0.004
Particulate pollution	−2.849**	29.068	−0.402*
Freshwater withdrawals	0.001	−0.039*	0.000*
Forest depletion (% GNI)	0.022	2.429	−0.014
Within R²	0.19	0.25	0.27

Country and year effects; country-clustered standard errors. *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$.

The contrast between the pooled and within-country estimates is the analytical heart of the study. Removing all stable between-country differences strips out precisely the developmental correlates that make cross-sectional environmental associations difficult to interpret, so the associations that survive are identified from changes within countries over time. That the particulate-life-expectancy association is among the survivors—attenuated in magnitude, as within estimators typically are, but retaining its sign and significance—is far harder to attribute to confounding than a cross-sectional correlation would be. The appearance of an energy-depletion association with tuberculosis within countries, absent in the pooled model, further suggests that some resource-depletion channels operate through year-to-year variation that the cross-section obscures. The health-system controls do not drive the estimate, and they do not attenuate it uniformly. Dropping physicians and health expenditure moves the within-country particulate coefficient

from -2.849 to -3.092 , and removing all controls moves it to -3.523 ; in the pooled specification the same coefficient moves in the opposite direction, from -3.670 with the health-system controls to -3.472 without them. Conditioning on medical capacity is therefore not a device for producing a conservative estimate, and the coefficients are not interpreted as bounds.

5.5. The Pattern of Environmental Harm

The associations are predominantly harmful, as the environmental-burden framework predicts. Counting significant stressor-outcome cells across the three reported outcomes, the pooled model yields 6 harming against 2 apparently protective; the fixed-effects model 4 and 2; and the Driscoll-Kraay model 6 and 3. Restricted to the two primary outcomes, life expectancy and tuberculosis incidence, the same tallies are 4 and 1, 2 and 1, and 4 and 2; the difference is the composite index, which adds harming cells for energy depletion and particulate pollution and one apparently protective cell for freshwater withdrawals. The harming associations concentrate on life expectancy and are led by particulate pollution and resource depletion; the apparently protective associations are confined to freshwater withdrawals, which enters positively for life expectancy and the composite and negatively for tuberculosis incidence, and this reflects the fact that water withdrawals rise with the agricultural and industrial development that also extends life, and should not be read as a health benefit of water extraction. **Figure 2** displays the sign pattern for all three outcomes under pooled and fixed-effects estimation.

Read through the framework, the pattern is coherent: the environmental stressors most directly tied to human exposure—airborne particulates above all—carry the clearest health cost, and that cost is visible even within countries over time and net of the medical resources that might otherwise mask it.

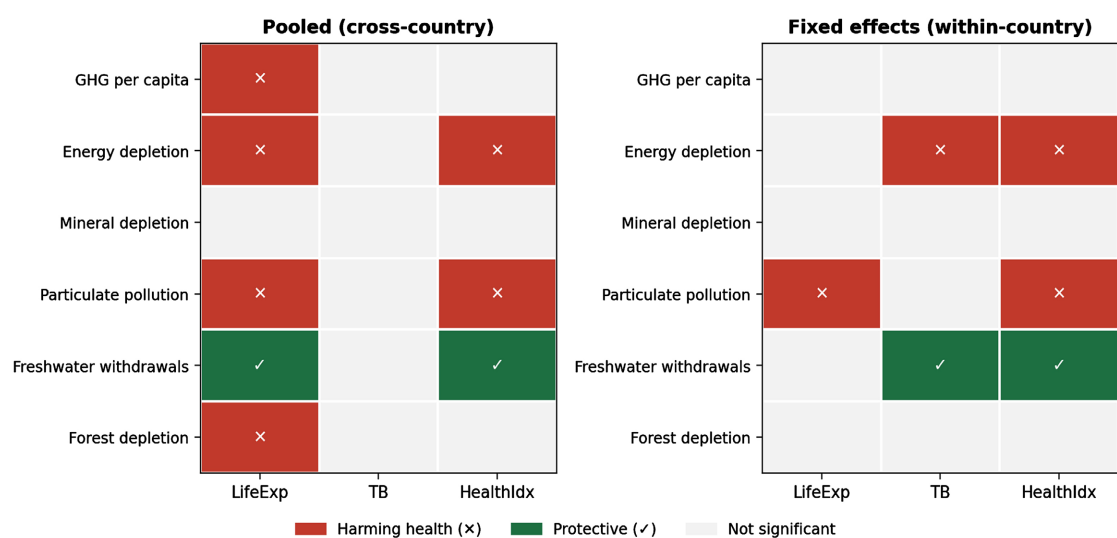


Figure 2. Sign map of environmental stressor-health associations for all three health outcomes under pooled and fixed-effects estimation. $\times$ = harming health; $\checkmark$ = protective.

The sign tally is therefore not merely a count but a summary of where the environmental-burden logic holds: the harmful associations cluster on the mortality outcome and on the stressors closest to human exposure, the apparently protective associations are confined to the development-correlated water measure, and the pattern is stable enough across estimators to be read as a coherent signal rather than as noise.

6. Endogeneity and Robustness

We probe robustness retaining the full stressor set. The specification test in **Table 9** is the auxiliary-regression form of the Hausman test: the within-country means of the time-varying regressors are added to the random-effects specification and tested jointly under a country-clustered covariance matrix (Mundlak, 1978; Wooldridge, 2010). This form is used because the classical Hausman statistic is not guaranteed to be non-negative when the difference between the two estimated covariance matrices is not positive definite, and because it does not require the random-effects estimator to be efficient. The test rejects random effects for all three outcomes, so the two-way fixed-effects estimates reported in Section 5.4 are the appropriate specification throughout. To address persistence we estimate a system-GMM model for each (standardized) outcome with the lagged dependent variable and the full stressor set, instruments collapsed (**Table 10**). The dynamic estimates are tentative—life expectancy is highly persistent, with an autoregressive parameter near unity, and the tuberculosis specification shows signs of instrument weakness—so we report them for completeness and do not lean on them; where the diagnostics are acceptable, the results do not overturn the static findings.

Table 9. Specification tests (fixed vs. random effects) by outcome, cluster-robust auxiliary-regression form.

Outcome	χ^2 (df = 11)	p-value	Decision
Life expectancy	126.1	<0.001	Fixed effects
Incidence of TB	41.7	<0.001	Fixed effects
Health Index	54.7	<0.001	Fixed effects

Table 10. System GMM coefficients: each stressor on each (standardized) outcome.

Predictor	LifeExp	TB	HealthIdx
GHG per capita	0.014	0.261	−0.017
Energy depletion	0.021	0.238	−0.026
Mineral depletion	0.004	0.099	−0.010
Particulate pollution	0.043	0.008	−0.033*
Freshwater withdrawals	−0.018	−0.183	0.018
Forest depletion	0.016	−0.017	−0.005
ρ (persistence)	1.11	−0.75	0.84

Continued

Hansen p	0.238	0.997	0.19
reliable	no	no	yes

Lagged DV; collapsed instruments; standardized. ρ = persistence; reliable where Hansen in (0.05, 0.99) and $|\rho| < 1$. *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$.

We also examine direction. **Table 11** reports the association of lagged health outcomes with current environmental stressors, and **Table 12** reports panel Granger tests in both directions. There is no evidence that health outcomes drive subsequent environmental degradation: none of the twelve cells in the health-to-environment direction is significant. In the environment-to-health direction only one of twelve cells is significant, greenhouse-gas emissions with tuberculosis incidence, and the particulate-life-expectancy pair is significant in neither direction. The Granger evidence therefore establishes the absence of reverse feedback rather than temporal precedence for the central association, and neither it nor the fixed-effects results exclude time-varying confounding. Taken together, the robustness evidence supports a cautious, association-based reading in which environmental degradation—particulate pollution most clearly—is associated with worse population health, within countries as well as across them, and without evidence of reverse feedback from health to the environment.

Table 11. Reverse causality: lagged health outcome $\rightarrow$ current stressor (two-way fixed effects).

Predictor	LifeExp	TB
GHG per capita	63.560	−2.230
Energy depletion	0.011	0.007
Mineral depletion	−0.057	0.005
Particulate pollution	−0.002	0.000
Freshwater withdrawals	0.130	0.022
Forest depletion	−0.054	0.003

Coefficient on the lagged outcome. *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$.

Table 12. Panel Granger non-causality (both directions) by stressor-outcome pair.

Predictor	LifeExp	TB
GHG per capita	./.	F*/.
Energy depletion	./.	./.
Mineral depletion	./.	./.
Particulate pollution	./.	./.
Freshwater withdrawals	./.	./.
Forest depletion	./.	./.

F = stressor $\rightarrow$ health significant; R = health $\rightarrow$ stressor significant ($p < 0.05$); . = not significant.

Life expectancy reflects cumulative rather than contemporaneous mortality

conditions, so the alignment of exposure and outcome timing matters for how the particulate association should be read. We therefore re-estimate the within-country specification with the particulate measure lagged one, two, and three years, and as a three-year mean covering t to $t - 2$, holding the remaining stressors and the controls at their contemporaneous values (Table 13). The association is stable across the set: -3.208 at one lag, -2.937 at two, and -2.736 at three, against -2.849 contemporaneously, each significant at conventional levels, with the composite index behaving the same way and tuberculosis incidence remaining insignificant throughout. The three-year mean yields the largest coefficient, -4.906 , as cumulative-exposure reasoning would suggest, and lagging all six stressors together by one year leaves the particulate coefficient at -3.129 . Same-year measurement is therefore not what produces the central association, and the falling sample size at longer lags—from 721 observations to 499 at three years—is the main cost of the exercise.

Table 13. Lagged particulate exposure: two-way fixed-effects coefficients on each health outcome.

Particulate exposure timing	LifeExp	TB	HealthIdx	N
Contemporaneous (t)	-2.849^{**}	29.068	-0.402^*	721
Lagged one year ($t - 1$)	-3.208^{***}	35.497	-0.471^*	617
Lagged two years ($t - 2$)	-2.937^{**}	34.553	-0.444^*	564
Lagged three years ($t - 3$)	-2.736^*	31.192	-0.407^*	499
Three-year mean (t to $t - 2$)	-4.906^{***}	45.778	-0.665^*	538

*Each cell is the particulate coefficient from a separate two-way fixed-effects regression with the remaining five stressors and all controls at their contemporaneous values, country-clustered standard errors. *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$.*

7. Discussion

Taken together, the diagnostic battery neither manufactures nor destroys the central result. The dynamic estimates are too weakly identified to adjudicate the associations and are reported only for completeness; the specification tests select the within-country estimator for all three outcomes; the directional tests find no trace of a health-to-environment pathway, though they also find no temporal precedence in the forward direction for the central pair; and the particulate association is unchanged when exposure is lagged. What survives is a conditional, association-based reading in which environmental degradation—particulate pollution most clearly—is linked to worse population health, within countries as well as across them, and with no evidence of reverse feedback, with the strength of the claim calibrated to the strength of the identification behind it.

The findings support, and sharpen, the proposition that environmental degradation harms population health. The relationship is not uniform across stressors and outcomes; it concentrates where human exposure is most direct. Particulate air pollution carries the clearest and most robust cost, associated with lower life

expectancy both across countries and within them over time, and net of the income and health-system capacity that might otherwise account for the pattern. Resource depletion, particularly of energy and forests, adds further harm, and energy depletion is associated with higher tuberculosis incidence within countries.

That the particulate-life-expectancy association survives fixed effects is the result's most important feature. Much cross-national environmental evidence reflects stable differences between rich and poor countries and cannot distinguish the environment from its developmental correlates; here, by contrast, changes in a country's particulate burden track changes in its life expectancy over time, which is far harder to attribute to confounding. The directional tests are consistent with this reading without confirming it: there is no evidence that health outcomes drive subsequent environmental degradation, which removes reverse feedback as an explanation, but the Granger tests show no temporal precedence in the forward direction for this pair either, and fixed effects leave time-varying confounders untouched. The association is therefore consistent with an environment-to-health interpretation rather than evidence for it.

The broader implication is that the health returns to environmental improvement—especially air-quality improvement—are real and are not subsumed by economic development or medical care. For policy, this strengthens the health case for pollution control as a complement to, not a substitute for, investment in health systems; for scholarship, it argues for treating the environment as a distinct determinant of population health, measured net of the medical resources with which it is so easily confounded.

These findings both corroborate and discipline the broader environment-health literature. The large individual-level evidence that particulate pollution shortens lives (Cohen et al., 2017; Pope III & Dockery, 2006) leads one to expect a population-level longevity penalty, and the analysis confirms that the penalty is visible in aggregate national outcomes and, more tellingly, in their movement within countries over time. At the same time, the result disciplines an over-broad reading of that literature: the health cost of environmental degradation is not uniform across stressors but concentrated where exposure is direct, so an aggregate “environmental-quality” claim would overstate the reach of the evidence. The burden-of-disease framing is vindicated for air pollution and only partially extended to the resource-depletion stressors.

The resource-depletion results occupy a middle ground consistent with their more indirect channels. Energy and forest depletion are associated with lower life expectancy in the cross-section, and energy depletion with higher tuberculosis incidence within countries, but these associations are smaller and less robust than the particulate result, as the framework anticipated. The tuberculosis outcome behaves largely as the epidemiology would predict: its incidence is governed more by social determinants, crowding, and the reach of treatment programs than by the environmental stressors measured here, so the environmental signal in tuberculosis is faint except where a resource-depletion channel plausibly operates. This

asymmetry between the mortality and infectious-disease outcomes is itself informative, underscoring that environmental stressors reach different health outcomes to sharply different degrees.

The freshwater-withdrawal result is the clearest illustration of why delineation matters. Read naively, higher withdrawals are associated with longer life, which would be a perverse finding were it a health effect of water extraction. It is not: withdrawals rise with the agricultural and industrial development that independently lengthens life, so the coefficient reflects the development-correlated character of the measure rather than any benefit of drawing down water. Had the environmental stressors been collapsed into a single index, this development-driven component would have contaminated the composite and obscured the genuine, exposure-driven signal in particulate pollution. The anomaly is thus a vindication of the delineated design: only by keeping the stressors separate can a development indicator masquerading as a stressor be identified as such.

A final feature of the evidence deserves emphasis. Because health-system capacity enters as a control, the environmental associations are estimated net of any effect that operates through medical resources, and because income enters flexibly, they are net of the developmental channel as well. What this conditioning delivers is an estimate net of the developmental and medical channels, not a bound on the total burden. If health expenditure, physicians, or income respond to environmental degradation, they are post-exposure variables, and conditioning on them can bias the environmental coefficient in either direction; the sensitivity reported in Section 5.4, in which the coefficient grows in the within-country specification and shrinks in the pooled one when the health-system controls are removed, is what that ambiguity looks like in these data. That a measurable longevity cost remains after such conditioning is the strongest form the claim can take within an observational design, and the claim is about association net of specified channels rather than about a lower bound on a causal effect.

8. Implications

The results carry implications for both policy and scholarship, and they follow from the study's central finding that air pollution imposes a measurable longevity cost that development and medical care do not fully offset. For health policy, this strengthens the case for air-quality improvement as a direct health intervention rather than as a co-benefit of climate or industrial policy. Because the particulate-longevity association survives within countries and net of health-system capacity, the health returns to reducing particulate exposure are not subsumed by rising incomes or expanding medical services; they are additional to them. Pollution control and health-system investment are, on this evidence, complements rather than substitutes—a country that reduces its particulate burden can expect longevity gains over and above those delivered by its physicians and hospitals.

The distributional reading reinforces the point. The environmental health burden falls most heavily on lower-income populations, which are both more exposed

and less shielded by medical capacity, yet these are precisely the settings under-represented in a sample weighted toward higher-income countries; the estimates here are therefore likely to understate the burden borne where it is greatest. This argues for prioritizing air-quality policy in developing settings, where the marginal longevity return to cleaner air is plausibly largest. The resource-depletion results counsel a more targeted response: because these stressors act through indirect and heterogeneous channels, they call for instruments aimed at the specific pathway—forest and energy governance, extraction regulation—rather than for treating resource depletion as a generic health hazard.

For scholarship, the implication is methodological as much as substantive. Because environmental stressors reach different health outcomes to sharply different degrees, and because the environment is so easily confounded with the medical capacity that development funds, studies that relate a single stressor to a single outcome without conditioning on health-system capacity will systematically misstate the environmental burden—reporting as an environmental effect what is partly medical, or missing a genuine effect masked by development. The delineated, capacity-controlled design adopted here makes both the heterogeneity and the confound visible, and the approach is portable: wherever a multidimensional environmental driver is related to a multidimensional health outcome, disaggregating both and conditioning on the resources that confound them reveals which stressors move which outcomes, and how much of the apparent effect survives once development is held constant. Treating the environment as a distinct determinant of population health, measured net of medical capacity, is the study's central methodological recommendation.

9. Scope and Limitations

Several limitations bound these conclusions. The analysis is association-based: environmental quality, income, and health are jointly determined, and although we control for income, education, and health-system capacity and test direction, time-varying confounders remain a live possibility that neither the fixed effects nor the Granger tests can exclude. The health outcomes are deliberately limited to two population-level measures; richer health vectors—cause-specific mortality, morbidity, and child health—would sharpen the channel analysis, and we treat health-system variables as controls rather than outcomes by design. Several environmental measures are imperfect proxies, and the freshwater-withdrawals variable in particular behaves as a development indicator rather than a pure stressor.

Two features of the design bear directly on how the central estimate should be read. The particulate measure is the World Bank's monetised damage series, built from modelled mortality attributable to PM_{2.5} and ozone exposure and extrapolated between benchmark years from mortality trends, so part of its association with life expectancy is carried by construction rather than by behaviour; the coefficient is best read as the association between a mortality-valued exposure index and aggregate longevity, and a concentration-based exposure measure would sup-

ply an independent test. The health-system controls, in turn, are treated as confounders, but if environmental degradation shifts health expenditure or physician supply they are post-exposure variables, and the estimates carry whatever bias that conditioning induces. Sample coverage is a third boundary: complete-case selection retains 85.8 per cent of possible country-years overall but only about three-quarters in the two lowest income groups, so the income-group comparisons rest on thinner data exactly where the environmental burden is expected to be heaviest.

The dynamic-panel evidence is tentative given the persistence of the outcomes, and is reported rather than relied upon. The sample, while spanning all income groups, is weighted toward higher-income countries, so the estimates may understate the environmental health burden borne disproportionately by lower-income populations. These limitations point to the value of cause-specific health outcomes, finer environmental exposure measures, and broader coverage of low-income settings.

Two further caveats concern measurement and mechanism. On measurement, the environmental stressors are national aggregates that mask within-country variation in exposure, so the country-level associations need not hold at finer geographic or individual scales, and the familiar caution against ecological inference applies: a relationship among national means is not a dose-response relationship for individuals. On mechanism, the design identifies associations but not the biological or social pathways through which they operate; it cannot say whether the particulate-longevity association works through cardiovascular mortality, respiratory disease, or some combination, because the health outcomes are aggregate rather than cause-specific. These limitations are not incidental but definitional of a cross-national panel design, and they mark the boundary between what this study can establish—that a conditional, within-country, environment-to-health association exists at the national scale—and what it must leave to finer-grained epidemiology.

10. Conclusions and Future Research

Across 70 countries spanning the full income range, environmental degradation is associated with worse population health, and the association is clearest and most robust for air pollution. Higher particulate pollution is associated with lower life expectancy both across and within countries, net of income, education, and health-system capacity, and the association holds when exposure is lagged by up to three years, with no evidence that health outcomes drive subsequent environmental degradation. Resource depletion adds further harm. Air pollution, in short, carries a measurable longevity cost that development and medical care do not fully offset.

The contribution is threefold. Substantively, the study provides a delineated, country-level mapping of which environmental stressors are associated with which health outcomes, net of development and medical capacity, and identifies

air pollution as the stressor carrying the clearest and most robust cost. Methodologically, it shows that the particulate-longevity association survives the within-country test that most cross-national environmental associations fail, and that it is robust to lagged measurement of exposure and to the removal of the health-system controls, so that the headline claim rests on the demanding rather than the permissive evidence. Conceptually, it demonstrates the value of treating both the environment and health as plural, and of conditioning environmental associations on the medical capacity with which they are so easily confounded—without which a genuine longevity cost of air pollution would be difficult to distinguish from the developmental backdrop against which it is set.

Future research should extend the health vector to cause-specific mortality and morbidity to identify the channels through which environmental stressors act; improve exposure measurement beyond national aggregates; broaden coverage of low-income countries, where the environmental health burden is likely greatest; and link the cross-national patterns documented here to the micro-level dose-response evidence on which the mechanisms ultimately rest.

Reproducibility. All statistics derive from `ds_build_panel.py`, `ds_full_analysis.py`, `ds_display_build.py`, `ds_validation.py`, and `ds_revision.py` applied to the frozen panel; figures from `ds_fig1.py` and `ds_figs.py`; results JSONs and a SHA-256 manifest accompany the manuscript.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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Appendix A. Countries Included in the Panel

The estimation sample comprises the 70 countries listed below, each observed in every year for which the World Bank reports all six environmental stressors, both health outcomes, and every control variable. Country-years with any missing analysis variable are excluded, giving 721 of the 840 country-years that the 2009-2020 window allows. Income group and region are the World Bank classifications carried in the source extract.

Country	Income group	Region	Years observed	N
Algeria	Lower middle income	Middle East & North Africa	2016, 2018	2
Argentina	Upper middle income	Latin America & Caribbean	2013, 2015-2017, 2019-2020	6
Armenia	Upper middle income	Europe & Central Asia	2009-2015, 2017	8
Australia	High income	East Asia & Pacific	2009-2020	12
Austria	High income	Europe & Central Asia	2009-2020	12
Azerbaijan	Upper middle income	Europe & Central Asia	2009-2019	11
Bahrain	High income	Middle East & North Africa	2012-2016	5
Bangladesh	Lower middle income	South Asia	2009, 2011-2020	11
Belarus	Upper middle income	Europe & Central Asia	2011-2019	9
Belgium	High income	Europe & Central Asia	2009-2020	12
Bulgaria	Upper middle income	Europe & Central Asia	2009-2015, 2018	8
Burkina Faso	Low income	Sub-Saharan Africa	2010-2015, 2017-2019	9
Cabo Verde	Lower middle income	Sub-Saharan Africa	2009-2013, 2015-2016, 2018	8
Canada	High income	North America	2010, 2012-2020	10
Chad	Low income	Sub-Saharan Africa	2012-2020	9
Chile	High income	Latin America & Caribbean	2009-2020	12
China	Upper middle income	East Asia & Pacific	2009-2019	11
Colombia	Upper middle income	Latin America & Caribbean	2009-2020	12
Costa Rica	Upper middle income	Latin America & Caribbean	2012-2020	9
Croatia	High income	Europe & Central Asia	2009-2019	11
Cuba	Upper middle income	Latin America & Caribbean	2009-2010, 2012-2016	7
Cyprus	High income	Europe & Central Asia	2009-2019	11
Czechia	High income	Europe & Central Asia	2009-2020	12
Denmark	High income	Europe & Central Asia	2009-2019	11
Estonia	High income	Europe & Central Asia	2009-2020	12
Finland	High income	Europe & Central Asia	2009-2020	12
France	High income	Europe & Central Asia	2009-2020	12

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Georgia	Upper middle income	Europe & Central Asia	2009-2020	12
Germany	High income	Europe & Central Asia	2009-2020	12
Ghana	Lower middle income	Sub-Saharan Africa	2009-2020	12
Greece	High income	Europe & Central Asia	2009-2020	12
Hungary	High income	Europe & Central Asia	2009-2020	12
Iceland	High income	Europe & Central Asia	2011-2019	9
Indonesia	Upper middle income	East Asia & Pacific	2009-2010, 2012, 2015, 2017-2020	8
Israel	High income	Middle East & North Africa	2009-2020	12
Italy	High income	Europe & Central Asia	2009-2020	12
Japan	High income	East Asia & Pacific	2010, 2012, 2014, 2016, 2018, 2020	6
Kazakhstan	Upper middle income	Europe & Central Asia	2009-2020	12
Korea, Rep.	High income	East Asia & Pacific	2009-2010, 2015-2020	8
Kyrgyz Republic	Lower middle income	Europe & Central Asia	2009-2014, 2019	7
Lao PDR	Lower middle income	East Asia & Pacific	2009-2010, 2012-2020	11
Latvia	High income	Europe & Central Asia	2009-2020	12
Lebanon	Lower middle income	Middle East & North Africa	2010-2011, 2013, 2016-2019	7
Lithuania	High income	Europe & Central Asia	2009-2020	12
Luxembourg	High income	Europe & Central Asia	2009-2017	9
Madagascar	Low income	Sub-Saharan Africa	2009-2014, 2018	7
Malaysia	Upper middle income	East Asia & Pacific	2009-2020	12
Maldives	Upper middle income	South Asia	2009-2010, 2014, 2016-2019	7
Mali	Low income	Sub-Saharan Africa	2009-2018	10
Mauritius	Upper middle income	Sub-Saharan Africa	2009-2020	12
Mexico	Upper middle income	Latin America & Caribbean	2009-2018, 2020	11
Moldova	Upper middle income	Europe & Central Asia	2009-2015, 2017, 2019-2020	10
Mongolia	Lower middle income	East Asia & Pacific	2009-2016, 2018	9
Netherlands	High income	Europe & Central Asia	2009-2020	12
New Zealand	High income	East Asia & Pacific	2009-2020	12
Norway	High income	Europe & Central Asia	2009-2020	12
Pakistan	Lower middle income	South Asia	2009-2019	11
Poland	High income	Europe & Central Asia	2009-2017, 2020	10

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Portugal	High income	Europe & Central Asia	2009-2020	12
Russian Federation	Upper middle income	Europe & Central Asia	2009-2020	12
Rwanda	Low income	Sub-Saharan Africa	2010-2011, 2013-2019	9
Singapore	High income	East Asia & Pacific	2009-2019	11
Slovak Republic	High income	Europe & Central Asia	2009-2020	12
Slovenia	High income	Europe & Central Asia	2009-2020	12
Spain	High income	Europe & Central Asia	2009-2020	12
Sri Lanka	Lower middle income	South Asia	2009-2018, 2020	11
Sweden	High income	Europe & Central Asia	2009-2020	12
Switzerland	High income	Europe & Central Asia	2009-2020	12
Türkiye	Upper middle income	Europe & Central Asia	2009-2020	12
United Kingdom	High income	Europe & Central Asia	2009-2018	10