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Pm 2.5 Exposure: Fueling Chronic Disease Burden And Surging Urban Healthcare Costs

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ABSTRACT

Urban exposure to fine particulate matter (PM2.5) is a major environmental health concern with consequences that extend from chronic disease and premature mortality to healthcare utilization and economic productivity. This systematic review synthesizes peer-reviewed evidence reported in the source manuscript for 2010–2025, with emphasis on urban exposure, chronic disease, population-attributable burden, DALYs, healthcare expenditure and economic losses. The manuscript reports searches of PubMed, Scopus and Web of Science and prioritizes longitudinal cohorts, meta-analyses and health-economic models, integrating Global Burden of Disease estimates with satellite and ground-based observations. Reported concentration–response estimates include cardiovascular relative risks of 1.12–1.20 and respiratory relative risks of 1.10–1.18 per 10 µg/m³ increase in PM2.5. Major urban sources described include vehicular emissions, industrial activities, construction and road dust, biomass burning and secondary aerosol formation. Reported exposure examples include an annual mean of approximately 101 µg/m³ for Delhi, 25 µg/m³ for Puducherry and a monthly value of 123 µg/m³ for Dharuhera in 2025. The manuscript further reports substantial health and economic burdens in India and globally and describes a Shanghai case in which reported PM2.5 control costs were 0.76% of GDP compared with reported gains of about 1.01% of GDP. Overall, the evidence summarized supports integrated emission-control strategies, high-resolution exposure assessment and health-economic evaluation. Because the source manuscript does not provide complete database search strings, search dates, screening counts, exclusion reasons or a formal risk-of-bias procedure, these methodological details are explicitly identified rather than fabricated in this final draft.

Key words: PM2.5; urban air pollution; cardiovascular disease; respiratory disease; healthcare expenditure; economic burden; DALYs; systematic review

1. INTRODUCTION

Rapid urbanization and associated growth in transport, industry, construction and biomass burning have increased the importance of fine particulate matter (PM2.5) as an environmental health risk. PM2.5 is a heterogeneous mixture containing inorganic ions, organic matter, black carbon, nitrates, ammonium, sulfates and polycyclic aromatic hydrocarbons. Its concentration and chemical characteristics vary with emission sources, atmospheric chemistry, meteorology and urban form.

The source manuscript describes ambient PM2.5 as an important contributor to premature mortality and chronic disease, including ischemic heart disease, stroke, chronic obstructive pulmonary disease and lung cancer. The burden is not limited to clinical outcomes: hospital admissions, outpatient care, long-term treatment and productivity losses can increase healthcare and socioeconomic costs.

Exposure is spatially heterogeneous. Traffic corridors, industrial areas and construction zones can form localized hotspots, while meteorological conditions such as atmospheric inversions can trap pollutants near the surface. Indoor exposure is also relevant because ambient PM2.5 can penetrate buildings, making personal exposure different from outdoor concentrations.

The health effects described in the literature include oxidative stress, systemic inflammation, endothelial dysfunction and other biological pathways. These mechanisms provide a plausible link between inhaled particles and cardiovascular, respiratory, neurological and reproductive outcomes. Vulnerable populations, including children and older adults, may experience disproportionate impacts.

The objective of this review is to synthesize the evidence presented in the source manuscript concerning urban PM2.5 exposure, major sources, spatial and temporal patterns, chronic health outcomes, healthcare expenditure, economic losses and policy implications, with particular attention to India and selected urban case studies.

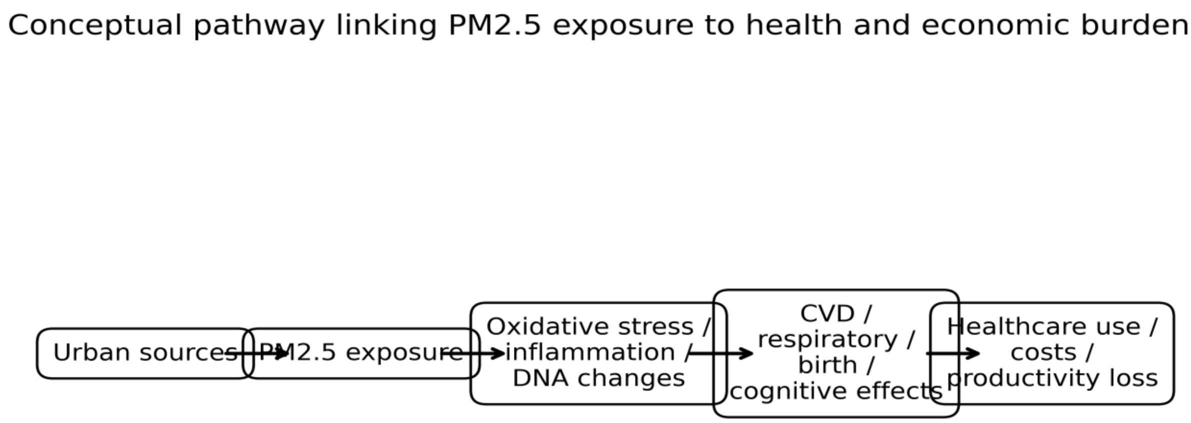

Figure 1. Conceptual framework linking urban PM2.5 sources and exposure with biological pathways, health outcomes and socioeconomic burden.

2. METHODS

2.1 Review design and information sources

The source manuscript describes the work as a systematic review of peer-reviewed literature published during 2010–2025. It reports searches of PubMed, Scopus and Web of Science and states that longitudinal cohorts, meta-analyses and health-economic models from urban settings were prioritized. The review also integrates Global Burden of Disease estimates and spatiotemporal observations derived from satellite and ground-based measurements.

2.2 Eligibility and evidence focus

The reported evidence focus includes urban PM2.5 exposure; chronic cardiovascular and respiratory outcomes; reproductive and neurological outcomes; population-attributable fractions; DALYs; healthcare utilization and expenditure; economic losses; source contributions; and policy or mitigation impacts. The source manuscript does not specify complete inclusion/exclusion criteria, database search strings, last-search dates, duplicate-removal procedures, screening counts, full-text exclusion reasons or a formal risk-of-bias instrument. These elements are required for a fully reproducible systematic review and should be completed from the author's search and screening records before journal submission.

2.3 Data extraction and synthesis

Quantitative values were extracted from the source manuscript without inventing new observations. Because the reported studies differ in geography, outcome, study design, year and metric, the quantitative results are presented descriptively rather than combined into a new meta-analysis. Reported ranges are displayed as ranges, and case-study values are retained in their original units.

2.4 Reporting framework

The manuscript states that PRISMA guidance was followed. PRISMA 2020 recommends reporting eligibility criteria, information sources, search strategies, study-selection processes, data collection, risk of bias and synthesis methods, together with a transparent flow diagram. (PRISMA 2020)

2.5 Methodological transparency

A numerical PRISMA flow diagram is not included in this final manuscript because the source document does not contain the necessary counts of records identified, duplicates removed, records screened, full texts assessed and studies included. Supplying invented counts would make the review methodologically unreliable. The author should insert the actual counts from the literature-search record before submission.

3. RESULTS

3.1 Urban PM2.5 sources

Vehicular emissions are described as a major urban source, contributing primary organic and elemental carbon and other traffic-related components. Near-road concentrations can be affected by exhaust, brake wear, tire wear and resuspended road dust.

Industrial activities contribute particles containing metals and crustal elements, while construction and road dust add mineral components such as Si, Al, Ca and Fe.

Biomass burning contributes organic matter, water-soluble organic carbon and polycyclic aromatic hydrocarbons, with seasonal variation associated with colder periods and regional burning events.

Secondary aerosol formation is described as an important contributor through atmospheric reactions involving SO2, NOx and volatile organic compounds, producing sulfate, nitrate, ammonium and secondary organic aerosol. The manuscript reports secondary aerosols at 30% to more than half of total PM2.5 mass in some urban settings.

Table 1. Major PM2.5 source categories described in the review

Source category	Reported characteristic	Potential relevance
Vehicular emissions	20–40% contribution reported in the manuscript	Major urban combustion/traffic source
Secondary aerosols	30% to >50% of PM2.5 mass in some settings	Major chemically formed fraction
Industrial activity	Metals, crustal elements and combustion products	Important localized urban source
Construction/road dust	Resuspended mineral/crustal material	Important near construction and unpaved/traffic areas
Biomass burning	Organic matter, WSOC and PAHs; seasonal	Important seasonal/regional contributor

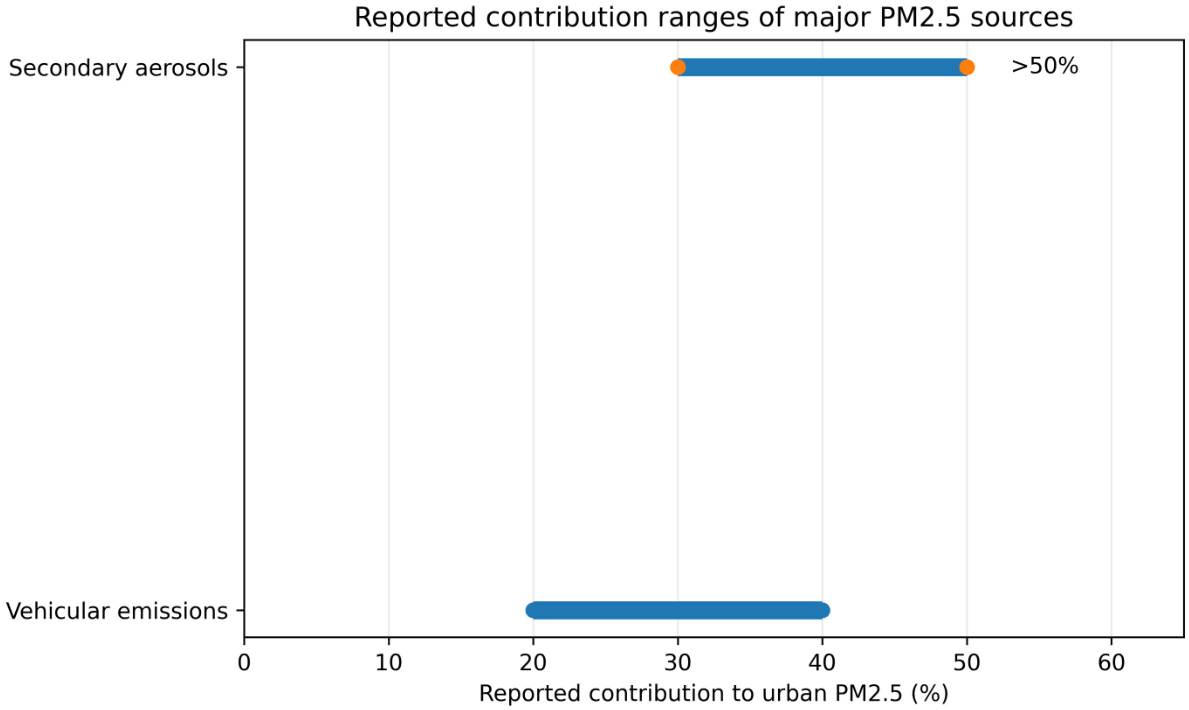

Figure 2. Reported contribution ranges for major PM2.5 sources. The manuscript reports secondary aerosols at 30% to more than half of total PM2.5 mass; no upper bound is invented.

3.2 Spatial and temporal exposure patterns

The review describes strong urban–rural gradients and localized hotspots near traffic corridors. High-resolution dispersion modeling indicates steep declines in concentrations with increasing distance from major roads. Meteorology influences these gradients, with atmospheric inversions favoring pollutant accumulation during colder periods and episodic spikes occurring during extreme events.

For India, the source manuscript reports higher concentrations in northern regions, particularly the Indo-Gangetic Plain. Delhi is reported at approximately 101 µg/m³ annual mean, while Puducherry is reported at 25 µg/m³. The manuscript also reports a 123 µg/m³ monthly PM2.5 value for Dharuhera during an October 2025 example.

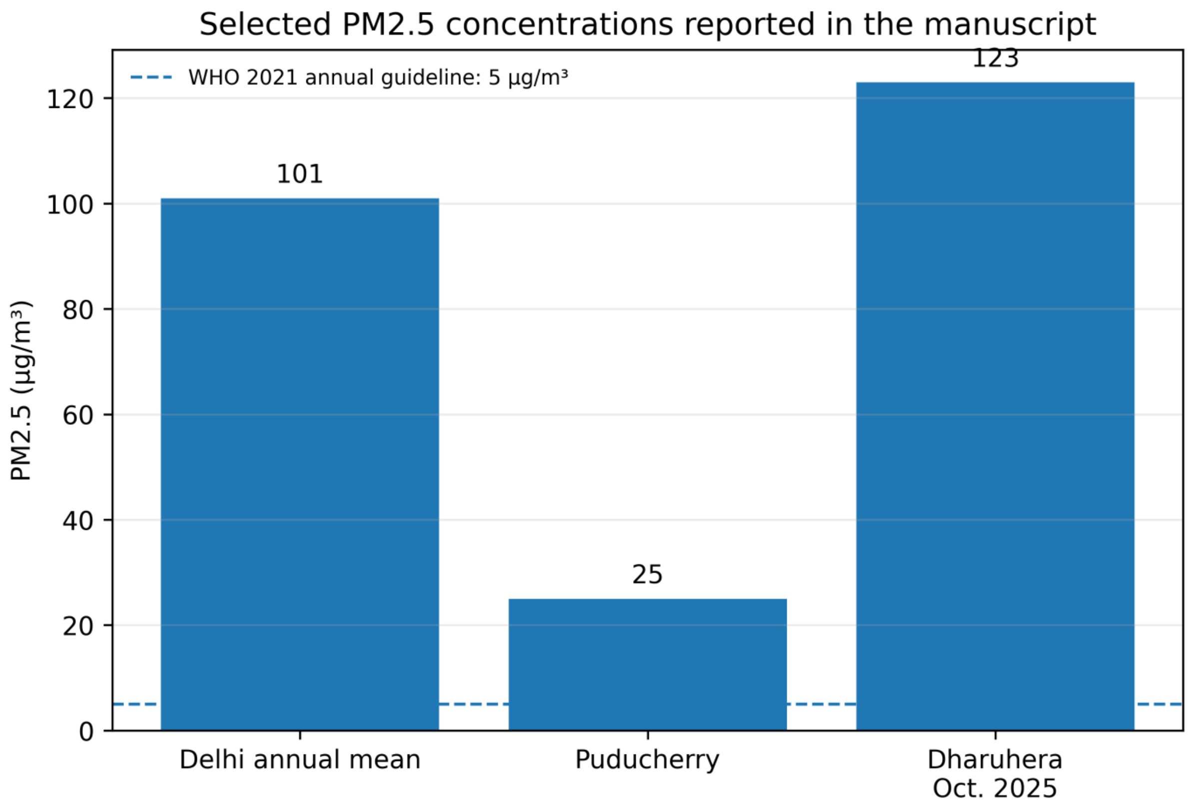

Figure 3. Selected PM2.5 concentrations explicitly reported in the manuscript for Delhi, Puducherry and Dharuhera.

Table 2. Selected exposure values reported in the manuscript

Location/context	Reported PM2.5	Unit / period
Delhi	~101	µg/m³ annual mean
Puducherry	25	µg/m³
Dharuhera	123	µg/m³ monthly value, October 2025 example
Indian cities in cited analysis	28–113	µg/m³ average levels across 10 cities

3.3 Health effects and biological pathways

The review describes pulmonary oxidative stress, immune activation and release of pro-inflammatory cytokines as pathways through which PM2.5 exposure can contribute to systemic inflammation and endothelial injury. These mechanisms are linked in the reviewed literature to cardiovascular disease, respiratory disease, reproductive outcomes and neurological effects.

For cardiovascular outcomes, the manuscript reports pro-atherosclerotic processes including plaque formation and instability. For respiratory outcomes, it discusses asthma, chronic obstructive pulmonary disease and lung cancer. Maternal exposure is associated in the reviewed evidence with low birth weight, preterm birth and small-for-gestational-age outcomes, particularly during early and middle pregnancy. Among older adults, chronic inflammation and oxidative stress are discussed in relation to cognitive decline and age-associated vulnerability.

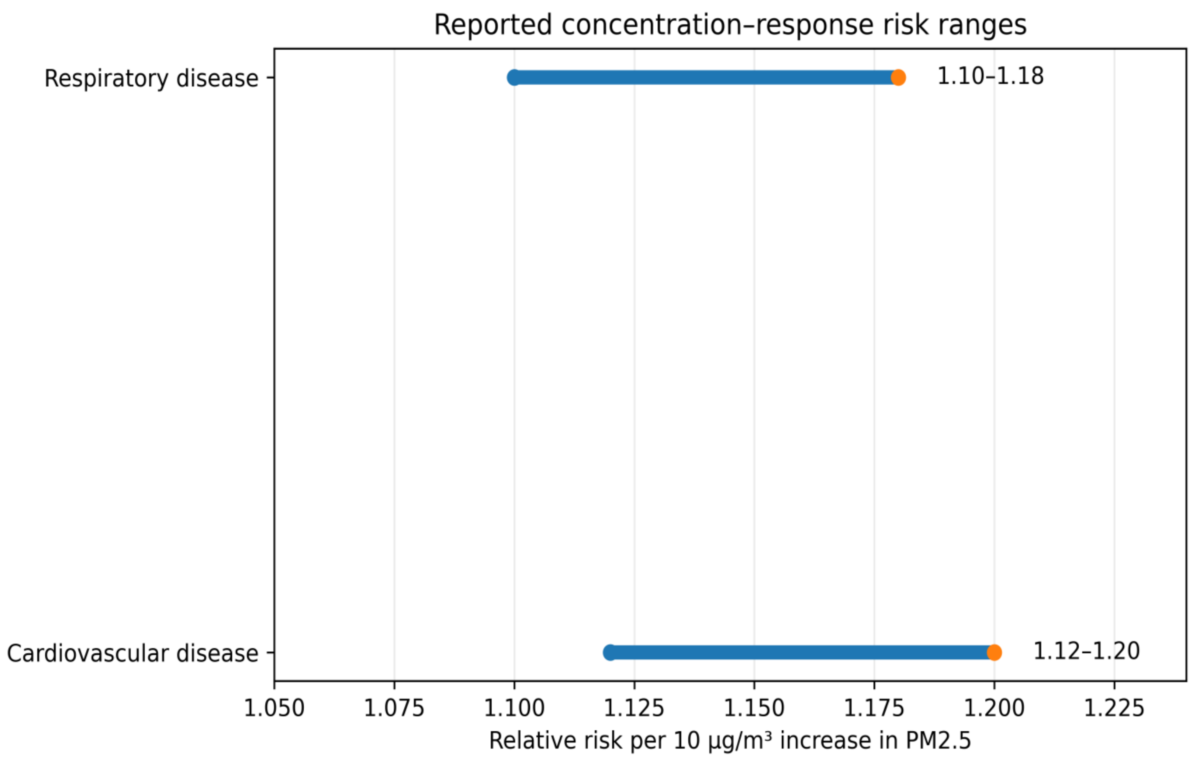

Figure 4. Reported concentration–response relative-risk ranges for cardiovascular and respiratory outcomes per 10 µg/m³ increase in PM2.5.

Table 3. Reported concentration–response and health-outcome evidence

Outcome/domain	Reported quantitative finding	Interpretation
Overall elevated risk	10–25% per 10 µg/m³ increment	Range stated in source manuscript

Cardiovascular disease	RR 1.12–1.20 per 10 μg/m³	Reported concentration–response range
Respiratory disease	RR 1.10–1.18 per 10 μg/m³	Reported concentration–response range
Birth outcomes	Higher PM2.5 associated with LBW, preterm birth and SGA	Risk described as nonlinear with exposure
Cognitive/age-related outcomes	Association described in older adults	Mechanisms include inflammation and oxidative stress

3.4 Healthcare expenditure and disease burden

The review links long-term PM2.5 exposure with increased hospital admissions, outpatient visits and treatment requirements. It reports a positive relationship between PM2.5 concentration and disease burden across Indian cities, using attributable deaths as a proxy for relative healthcare burden.

The source manuscript reports that PM2.5 exposure accounts for 3.7–11.5% of deaths in 10 Indian cities, with a correlation coefficient of 0.98 between average PM2.5 levels and the mortality measure described. Delhi is reported at 11.5% and 11,964 annual deaths, while Shimla is reported as the lowest-burden example.

For India in 2019, the cited Global Burden of Disease analysis estimated 1.67 million deaths attributable to air pollution overall, with 0.98 million attributable to ambient particulate matter pollution. The total economic loss from air pollution was US$36.8 billion, equivalent to 1.36% of GDP. The original source distinguishes total air-pollution burden from the ambient-particulate component; this distinction should be retained in interpretation. (India State-Level Disease Burden Initiative Air Pollution Collaborators, 2021)

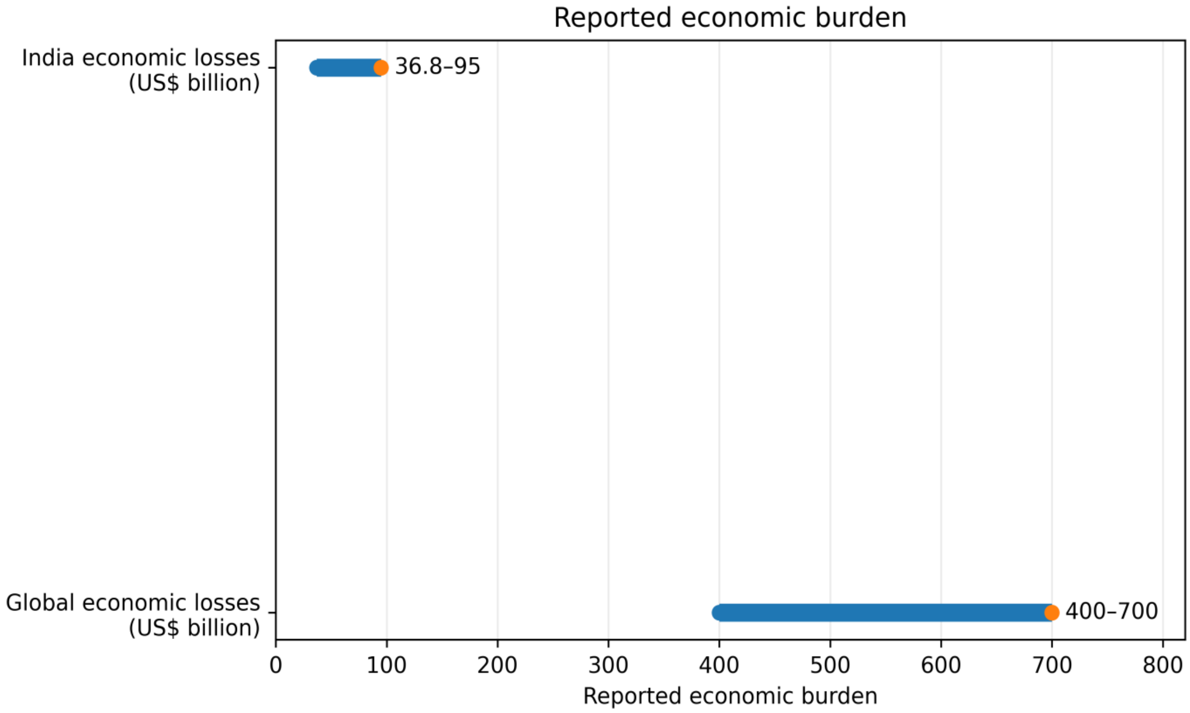

Figure 5. Reported economic-burden ranges associated with air pollution/PM2.5 at global and India scales.

Table 4. Key health and economic burden values reported in the review

Indicator	Reported value	Context
Global premature deaths attributable to ambient PM2.5	4.2 million	GBD 2015 estimate cited in the review
Global DALYs	25–60 million	Range reported in the source manuscript
Global economic losses	US$400–700 billion	Range reported in the source manuscript
India air-pollution-attributable deaths, 2019	1.67 million	All air pollution; not PM2.5 alone
India ambient particulate matter deaths, 2019	0.98 million	GBD 2019 component
India total air-pollution	US$36.8 billion (1.36%	Lost output from

economic loss, 2019	GDP)	premature death and morbidity
State GDP loss range	Up to 2.15%	Highest state-level total air-pollution loss reported
Direct healthcare spending	~US$11.9 billion	Value reported in the source manuscript; source-level verification recommended

3.5 Urban inequalities and vulnerable populations

The review emphasizes that pollution-related health burdens are unevenly distributed. Children are highlighted in relation to adverse birth outcomes, while older adults are described as particularly susceptible to cognitive and age-related effects. Outdoor workers and low-income communities may experience greater exposure and face constraints in accessing healthcare.

The manuscript reports differences in ICU-bed availability between Delhi and Kanpur and describes higher exposure among slum populations. These values are retained as reported but should be checked against the original cited sources before submission because the underlying data are not reproduced in the review.

3.6 Policy implications and economic benefits

The review argues that PM2.5 mitigation can reduce premature mortality, morbidity, healthcare demand and productivity losses. Proposed measures include traffic-emission controls, industrial and power-sector controls, cleaner production, urban planning and low-emission transport.

The Shanghai case study cited in the manuscript reports control costs equivalent to 0.76% of GDP and gains of about 1.01% of GDP. The original study is a peer-reviewed Environmental Science & Technology article and evaluates health and economic impacts under alternative air-pollution control and climate-policy scenarios. (Wu et al., 2017)

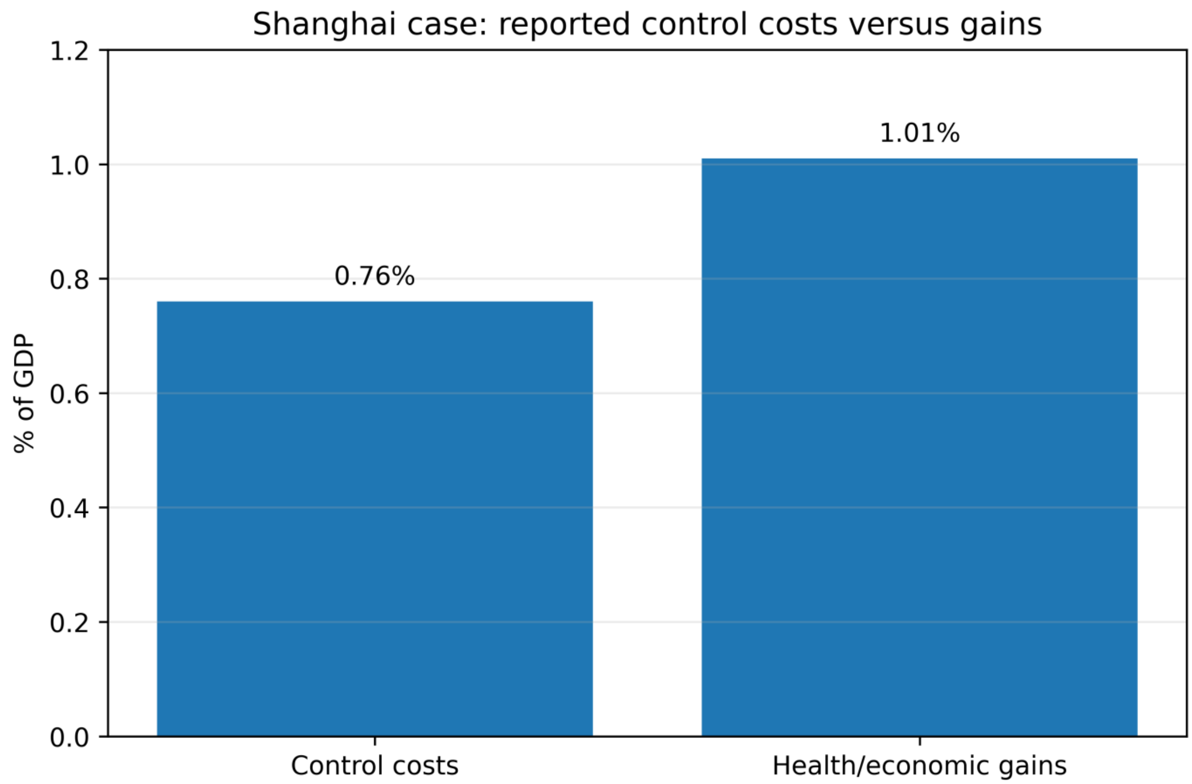

Figure 6. Shanghai case study: reported PM2.5 control costs and reported health/economic gains, expressed as percentages of GDP.

4. DISCUSSION

The evidence synthesized in this review indicates that urban PM2.5 is not simply an air-quality indicator but a multi-dimensional environmental health and economic risk. Exposure varies strongly across space and time, and source composition affects both concentration and toxicological potential.

The concentration–response findings reported in the manuscript support a measurable increase in cardiovascular and respiratory risk with increasing long-term PM2.5 exposure. These estimates should, however, be interpreted in the context of the designs and populations of the original studies rather than treated as a single universal effect size.

India represents a particularly important setting because high exposure in northern urban regions coincides with substantial population density and socioeconomic vulnerability. The cited GBD 2019 analysis demonstrates the scale of the national burden, including 1.67 million air-pollution-attributable deaths and US$36.8 billion in lost output in 2019. Importantly, that estimate covers air pollution broadly; the study separately attributes 0.98 million deaths to ambient particulate matter. (India State-Level Disease Burden Initiative Air Pollution Collaborators, 2021)

The economic evidence suggests that PM2.5 control can generate benefits beyond mortality reduction, including reduced healthcare utilization and productivity losses. The Shanghai case illustrates how integrated modeling can connect pollution control with economic outcomes. □cite□turn1search1□

The review also highlights a major methodological challenge: urban PM2.5 is heterogeneous in source, composition and exposure pattern. Consequently, combining estimates from different locations and study designs without accounting for these differences can produce misleading pooled effects. Future work should therefore prioritize high-resolution exposure assessment, longitudinal health cohorts, transparent uncertainty analysis and reproducible health-economic modeling.

5. CONCLUSION AND FUTURE RESEARCH

Urban PM2.5 exposure is associated in the evidence synthesized here with substantial chronic disease, mortality, healthcare and economic burdens. The review identifies vehicular emissions, industrial activity, construction and road dust, biomass burning and secondary aerosol formation as important contributors to urban exposure. Spatial and temporal heterogeneity is substantial, with traffic-related hotspots and meteorologically driven episodes complicating exposure assessment.

The review reports cardiovascular and respiratory relative-risk ranges of 1.12–1.20 and 1.10–1.18 per 10 $\mu g/m^3$ increase in PM2.5, respectively, alongside substantial national and global economic burdens. For India, the cited GBD 2019 study provides particularly strong evidence of the national scale of air-pollution impacts. (India State-Level Disease Burden Initiative Air Pollution Collaborators, 2021)

Future research should prioritize high-resolution spatiotemporal modeling, satellite–ground data integration, longitudinal cohorts with biomarkers, transparent uncertainty quantification and health-economic models. Policy evaluations should assess not only reductions in concentration and mortality but also healthcare expenditure, productivity and distributional effects across vulnerable populations.

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