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IMPACT OF AIR POLLUTION (PM2.5) ON HUMAN HEALTH IN URBAN AREAS

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ABSTRACT

Fine particulate matter (PM2.5) has become the single most consequential air quality parameter for urban public health, since particles below 2.5 micrometres penetrate deep into the pulmonary alveoli and pass into systemic circulation, triggering inflammatory and oxidative processes linked to cardiovascular and respiratory disease. This paper examines the relationship between ambient PM2.5 concentrations and human health outcomes in urban areas through a secondary-data, quantitative-synthesis approach, drawing on air quality monitoring records, regulatory standards, and peer-reviewed epidemiological literature spanning three decades. Rather than generating new primary exposure measurements, the study compiles data from the World Health Organization, India's Central Pollution Control Board, the IQAir global monitoring network, and major cohort and meta-analytic studies to build a comparative picture of the PM2.5-health relationship in urban populations, with particular attention to Indian cities. The analysis finds that most urban centres worldwide, and an overwhelming majority in India, continue to exceed WHO guideline values by wide margins, and that this exceedance corresponds to consistently elevated relative risks for cardiovascular, respiratory, and all-cause mortality documented across independent cohorts on different continents and decades. The disease burden attributable to ambient PM2.5 in India runs from several hundred thousand to over a million deaths annually depending on which exposure-response function is applied, a spread that signals unresolved methodological tension within the field. These findings anchor the paper's central argument: PM2.5 is a determinant of urban population health rather than a peripheral environmental concern, and the conclusion returns to this argument, contending that regulatory tightening alone, without structural change in urban energy and transport systems, is unlikely to close the gap between present exposure and guideline safety.

Key words: *PM2.5[1]; Ambient Air Pollution[2]; Urban Health[3]; Respiratory Disease[4]; Cardiovascular Disease[5]; Air Quality Standards[6]; Public Health Policy[7].*

I. INTRODUCTION

A. BACKGROUND: URBANIZATION AND THE GROWING BURDEN OF AMBIENT AIR POLLUTION

More than half the world's population now lives in cities, and that share is rising fastest in low- and middle-income countries where industrial growth, vehicle ownership, and construction activity have expanded faster than the infrastructure needed to manage their emissions. Urban air quality has consequently become one of the defining environmental health issues of the twenty-first century. Among the pollutants monitored in city air, fine particulate matter of aerodynamic diameter 2.5 micrometres or less, commonly abbreviated PM2.5, has drawn the most sustained scientific attention because of the size-dependent way it interacts with the human respiratory system. Coarser particles are largely filtered by the nose and upper airway; PM2.5 is small enough to bypass these defences and settle in the alveolar region of the lung, where gas exchange occurs, and from there cross into the bloodstream. The World Health Organization estimates that ambient air pollution, driven overwhelmingly by PM2.5, caused approximately 4.2 million premature deaths worldwide in 2019 [1], [2], while combined ambient and household air pollution accounts for a still larger toll. Nowhere is this burden more concentrated than in South Asia. India's own national air quality monitoring network and independent satellite-based assessments both show sustained exceedance of national and international standards across the bulk of its urban population, a pattern this paper examines in quantitative detail in the sections that follow.

B. PM2.5: SOURCES, PHYSICOCHEMICAL CHARACTERISTICS, AND EXPOSURE PATHWAYS

PM2.5 is not a single substance but a heterogeneous mixture, varying in composition from one city, and even one season, to the next. In most urban environments its principal sources are combustion-related: vehicular exhaust, especially from diesel engines; coal and biomass combustion in power generation and industry; construction and road dust; and, in the Indian context specifically, seasonal crop-residue burning that drifts into cities such as Delhi during the post-monsoon months. Secondary particles, formed in the atmosphere through chemical reactions involving sulphur dioxide, nitrogen oxides, and volatile organic compounds, add a further layer that is harder to control at source because it forms downwind of the emitting activity. Chemically, PM2.5 typically contains sulphates, nitrates, ammonium, black carbon, organic carbon compounds, and trace metals, each carrying somewhat different toxicological properties. What unites them is size: particles in this range remain suspended in air for days, travel hundreds of kilometres, and, once inhaled, deposit efficiently in the distal airways and alveoli. Toxicological and clinical research converges on a small number of biological pathways connecting this deposition to disease, principally systemic inflammation, oxidative stress, and endothelial dysfunction, which elevate the risk of atherosclerosis, arrhythmia, and impaired lung function over years of chronic exposure. This mechanistic grounding is what allows the epidemiological associations discussed in Section II to be read as plausibly causal rather than merely correlational.

C. RATIONALE, OBJECTIVES, AND SCOPE OF THE STUDY

Despite an extensive global literature connecting PM2.5 to adverse health outcomes, three practical problems recur in how that evidence reaches policymakers in rapidly urbanising, high-exposure settings like India. First, air quality standards vary considerably across jurisdictions, so a concentration considered moderate against one national benchmark may be classified as hazardous against WHO guidance, creating confusion about the actual scale of risk. Second, mortality estimates derived from different exposure-response models and different underlying cohorts often diverge by a factor of two or more, and this divergence is rarely presented alongside the headline figures that circulate in public discourse. Third, much of the available synthesis work is either global in scope, obscuring the specific severity of the South Asian urban context, or narrowly clinical, leaving out the regulatory and comparative dimension that policymakers need. This paper addresses these gaps by assembling a single, tabulated, comparative dataset spanning regulatory standards, currently monitored concentrations, pooled epidemiological risk estimates, and disease-burden figures, with an explicit focus on urban India set against global benchmarks. The objectives are threefold: to quantify the extent to which urban PM2.5 concentrations exceed guideline values; to synthesise the dose-response relationship between PM2.5 and major categories of morbidity and mortality; and to critically compare current findings against the trajectory of evidence built since the foundational cohort studies of the early 1990s. The scope is limited to ambient PM2.5 in urban settings; household air pollution and occupational exposure are referenced only where they clarify the ambient picture.

II. SURVEY OF RELATED WORK

The empirical foundation for treating PM2.5 as a cause, rather than merely a correlate, of premature death rests on a small number of long-running cohort studies conducted in North America beginning in the 1970s and 1980s. The Harvard Six Cities Study followed more than eight thousand adults across six U.S. cities for up to sixteen years and found that residents of the more polluted cities faced significantly higher all-cause mortality than residents of the cleanest city, even after controlling for smoking and other individual risk factors [3]. A subsequent extension of this cohort to 2009 confirmed that the association persisted, and in fact strengthened, as air quality improved and exposure measurement grew more precise [6]. Around the same period, the American Cancer Society cohort, encompassing hundreds of thousands of participants, linked long-term PM2.5 exposure to lung cancer and cardiopulmonary mortality [4], with follow-up analysis identifying the specific pathophysiological pathways, principally accelerated atherosclerosis, through which chronic exposure translates into cardiovascular death [5]. These studies collectively persuaded the World Health Organization to treat PM2.5 as causally linked to all-cause, cardiovascular, and lung cancer mortality when it issued its global air quality guidelines and subsequent revisions [1].

Since these foundational studies, the literature has grown along two complementary tracks. The first extends the cohort-study tradition into new populations and geographies. Multi-city time-series analyses spanning hundreds of cities, including a 652-city international collaboration and a 272-city Chinese nationwide analysis, have replicated the acute mortality association using daily rather than annual exposure contrasts, confirming that both short-term spikes and long-term averages independently predict mortality [16], [17]. European researchers pooled eight national cohorts under the ELAPSE project specifically to test whether the association holds at exposure levels below existing regulatory limits, and found that it does, with no discernible safe threshold [13]. The second track uses global atmospheric modelling rather than ground monitors to estimate exposure at planetary scale; one widely cited analysis using the GEOS-Chem chemical transport model attributed roughly a million deaths a year specifically to fossil-fuel-derived PM2.5, a subset of the wider ambient pollution burden [8], while an earlier study modelling seven pollutant source categories found India second only to China in absolute premature-death counts from outdoor air pollution [22].

Systematic reviews and meta-analyses have since consolidated these individual studies into pooled risk estimates, though not always in close agreement. A 2020 review pooling cohort studies produced somewhat lower risk estimates than a 2024 update commissioned to refresh the WHO guideline evidence base, which itself found estimates broadly consistent with, but not identical to, a subsequent Asia-Pacific-specific meta-analysis published in 2025 [12], [15], [14]. This last review is particularly relevant here: it was motivated explicitly by the observation that global exposure-response functions, built predominantly on North American and European data, may not transfer cleanly to Asia-Pacific settings with different pollution mixtures, background health profiles, and healthcare access, a concern this paper's own data section returns to.

India-specific literature has expanded rapidly over the past five years. The Global Burden of Disease state-level study attributed 1.67 million deaths to all forms of air pollution in India in 2019, split between ambient particulate matter and household sources [10]. A 2024 study using a difference-in-differences design and district-level mortality records over a decade produced markedly higher attributable-death estimates when using the WHO guideline threshold rather than India's own national standard, illustrating how much the choice of benchmark changes the headline number [11]. Complementing these health-outcome studies, atmospheric-science research has begun explaining why Indian PM2.5 levels behave as they do, attributing recent modest improvements partly to favourable meteorology rather than durable emissions reductions [28], and documenting the seasonal dynamics, including winter inversion and crop-residue burning, that drive peak exposure episodes [29]. Reviews addressing specific clinical endpoints, including asthma outcomes and broader multi-organ effects of ambient pollution, round out a literature that is extensive but, as the next section discusses, still methodologically fragmented enough to justify the comparative synthesis undertaken here [9], [21].

III. METHODOLOGY

This study adopts a descriptive-analytical, quantitative research design built on secondary data, rather than primary field measurement or biological sampling. The choice is deliberate: individual field monitoring of PM2.5 exposure and health outcomes across multiple cities would require resources, ethical clearances, and multi-year follow-up periods far beyond the scope of a single paper, and would in any case duplicate monitoring infrastructure that national and international agencies already operate at far greater scale and rigor. Instead, the design treats existing regulatory standards, monitoring-network output, and published epidemiological effect estimates as the raw material for comparative synthesis. This approach sits within the broader tradition of evidence synthesis and health-impact assessment, where the analytical contribution lies not in generating new exposure or outcome data but in assembling independently produced datasets into a single comparative frame that no individual source provides on its own. The design is empirical in the sense that every quantitative claim in Sections IV and V traces to a specific, cited numerical source rather than a general assertion, though comparative and cross-sectional questions of standards, concentrations, and studies are prioritized over an in-depth statistical reanalysis of any single dataset.

Data collection proceeded through targeted retrieval from four categories of source. Regulatory standards were obtained directly from the primary issuing bodies: the World Health Organization's 2021 Global Air Quality Guidelines, India's Central Pollution Control Board National Ambient Air Quality Standards, the United States

Environmental Protection Agency's 2024 revised standard, and the European Union's ambient air quality directive. Current monitored concentrations were drawn from the IQAir World Air Quality Report 2025, which aggregates data from over 40,000 regulatory and low-cost sensor stations across 9,446 cities, supplemented by India-specific atmospheric studies addressing seasonal and meteorological drivers of exposure. Epidemiological effect estimates were gathered from peer-reviewed cohort studies and systematic reviews indexed in major journals, prioritizing studies published through 2024-2025 and giving particular weight to Asia-Pacific and India-specific analyses to counter the historical North American and European skew of the foundational literature. Disease-burden estimates came from Global Burden of Disease publications and India-specific state-level analyses. All figures reported in Section IV are attributed to their original source in-text and in the reference list; no figure was estimated, interpolated, or extrapolated beyond simple percentage and ratio calculations explicitly noted where they occur.

Analysis proceeded through structured comparative tabulation rather than inferential statistical modelling, since the underlying data originate from heterogeneous study designs, populations, and time periods that cannot legitimately be pooled into a single new statistical model without violating the assumptions of the original analyses. Where multiple sources reported a pooled relative risk for the same exposure-outcome pair, all reported estimates are presented side by side rather than further combined, preserving visible heterogeneity rather than masking it with an additional layer of averaging. Simple derived calculations, specifically the ratio of measured city concentrations to WHO and national guideline values, were computed directly from the source figures using standard arithmetic and are flagged as such wherever they appear. The analytical technique therefore combines descriptive statistics, comparative benchmarking against multiple regulatory thresholds simultaneously, and qualitative synthesis of methodological differences across studies. This has an acknowledged limitation: it cannot resolve which underlying exposure-response function is most accurate for Indian urban populations specifically, and the Discussion section treats this unresolved question as a substantive finding rather than a caveat to be set aside.

IV. DATA COLLECTION AND ANALYSIS

The comparative dataset assembled for this study is presented across five tables, moving from regulatory benchmarks through current exposure levels to health-effect estimates, disease burden, and finally a chronological summary of the studies that established the field. Read together, these tables substantiate the abstract's opening claim: urban PM2.5 exposure in India and comparable settings sits far above every applicable guideline, and this gap corresponds, across three decades of independent research, to a measurable and consistent elevation in mortality and morbidity risk.

TABLE 1. COMPARATIVE AMBIENT AIR QUALITY STANDARDS FOR PM2.5

Issuing Body / Standard	Annual Mean Limit (µg/m³)	24-Hour Mean Limit (µg/m³)	Year Notified
WHO Global Air Quality Guideline [1]	5.0	15	2021
India NAAQS (CPCB) [19]	40	60	2009

USA EPA NAAQS [20]	9.0	35	2024
European Union (Directive 2008/50/EC) [26]	25	—	2008
United Kingdom (Air Quality Regs.) [27]	25	—	2010

Table 1 makes the regulatory fragmentation described in Section I concrete: India's annual permissible limit, at 40 µg/m³, is eight times the WHO guideline value, while the United States' 2024-revised standard of 9.0 µg/m³, itself described by EPA as its first strengthening of any NAAQS in over eight years [20], remains nearly double the WHO figure. This spread means a city reporting compliance with its national standard may simultaneously be operating at several multiples of the level WHO now considers protective of health, a distinction that matters directly for how the concentrations in Table 2 should be read.

TABLE 2. PM2.5 CONCENTRATIONS IN SELECTED URBAN AREAS, 2025 (ANNUAL AVERAGE)

City / Country	**Annual Mean PM2.5 (µg/m³)**	**Multiple of WHO Guideline**	**Rank / Note (2025)**
Loni, India	112.5	~22.5x	Most polluted city globally [18]
New Delhi, India	82.2	~16.4x	Most polluted capital, 8th year running [18]
India (national average)	48.9	~9.8x	6th most polluted country [18]
Pakistan (national average)	67.3	~13.5x	Most polluted country, 2025 [18]
Seattle, USA	4.5	~0.9x	Cleanest major U.S. city [18]
Nieuwoudtville, South Africa	1.0	~0.2x	World's cleanest city, 2025 [18]

Only 14 percent of the 9,446 cities monitored globally in 2025 met the WHO annual guideline, a share that had itself declined from 17 percent the year before [18]. Table 2 shows why India dominates conversations about urban air pollution: its most polluted city recorded a concentration over twenty-two times the WHO limit, and even its national average, despite a modest year-on-year improvement, remains close to ten times that threshold. This is the empirical gap that Tables 3 and 4 translate into health terms, and that the Discussion section interrogates against three decades of prior measurement.

TABLE 3. POOLED RELATIVE RISK (RR) OF HEALTH OUTCOMES PER 10 ug/m3 INCREASE IN LONG-TERM PM2.5 EXPOSURE

Health Outcome	**Pooled RR (95% CI)**	**Source / Study Population**
All-cause mortality	1.12 (1.07-1.16)	Chen & Hoek, global systematic review, 2020 [12]

All-cause mortality (updated)	1.095 (1.064-1.127)	Orellano et al., WHO-commissioned update, 106 studies, 2024 [15]
All-cause mortality	1.11 (1.05-1.17)	Oh et al., Asia-Pacific meta-analysis, 71 studies, 2025 [14]
Cardiovascular disease mortality	1.13 (1.06-1.21)	Oh et al., Asia-Pacific meta-analysis, 2025 [14]
Ischemic heart disease mortality	1.13 (1.02-1.25)	Oh et al., Asia-Pacific meta-analysis, 2025 [14]
Stroke mortality	1.12 (1.02-1.24)	Oh et al., Asia-Pacific meta-analysis, 2025 [14]
Lung cancer mortality	1.12 (1.08-1.16)	Oh et al., Asia-Pacific meta-analysis, 2025 [14]

Table 3 illustrates both the consistency and the residual disagreement running through the epidemiological literature. Every pooled estimate points in the same direction and lands in a narrow band, roughly 9 to 13 percent higher risk for every 10 µg/m³ increment, regardless of whether the underlying studies were drawn from Europe, North America, or the Asia-Pacific region specifically. That convergence across independently conducted reviews is what allows Table 4's disease-burden estimates to be treated as more than a statistical artefact. At the same time, the fact that reviews published across five years produce estimates differing by several percentage points underscores why India-specific attributable-death figures, discussed next, vary so widely depending on which underlying function is applied.

TABLE 4. DISEASE BURDEN ATTRIBUTABLE TO PM2.5 / AMBIENT AIR POLLUTION

Level / Population	**Attributable Deaths (Annual)**	**% of Total Deaths**	**Source**
Global, ambient air pollution	~4.2 million	-	WHO, 2019 data [2]
Global, ambient + household air pollution	~6.7-7 million	-	WHO [2], [25]
Global, fossil-fuel-derived PM2.5 only	~1.0 million (est.)	-	Vohra et al., GEOS-Chem model, 2021 [8]
India, all air pollution sources	1.67 million	17.8%	GBD India State-Level Study, 2019 [10]
India, ambient particulate matter only	0.98 million	10.4%	GBD India State-Level Study, 2019 [10]
India, PM2.5 above WHO guideline	~1.5 million	~25%	Jaganathan et al., 2024 [11]
India, PM2.5 above national NAAQS	~0.3 million	-	Jaganathan et al., 2024 [11]

Table 4 is where the standard divergence documented in Table 1 turns into a genuine policy dilemma rather than a technical footnote. Using India's own national standard as the benchmark, roughly 300,000 deaths a year are attributable to PM2.5 exceedance; using the WHO guideline instead, the same underlying mortality data yield an estimate roughly five times larger, at approximately 1.5 million [11]. The Global Burden of Disease project's independent methodology sits between these two figures, at 0.98 million [10]. None of these three numbers is simply wrong; they answer different, equally legitimate questions about how many deaths would be avoided under each specific counterfactual standard. This ambiguity is not a weakness unique to Indian data; it reflects a structural feature of attributable-burden estimation that Section V examines directly.

TABLE 5. KEY EPIDEMIOLOGICAL STUDIES ON PM2.5 AND HUMAN HEALTH, 1993-2025

Study / Year	Population / Design	Key Finding
Dockery et al., 1993 [3]	8,111 adults, 6 U.S. cities, prospective cohort	Established dose-response link between PM2.5 and all-cause/cardiopulmonary mortality
Pope et al., 2002 [4]	~500,000 adults, American Cancer Society cohort	Linked long-term PM2.5 exposure to lung cancer and cardiopulmonary mortality
Lepeule et al., 2012 [6]	Six Cities cohort, extended follow-up to 2009	Confirmed and strengthened original association as air quality improved
Lelieveld et al., 2015 [22]	Global atmospheric model, 7 pollutant source categories	Ranked India second globally in outdoor-pollution premature deaths
Brunekreef et al. (ELAPSE), 2021 [13]	8 pooled European cohorts	Found no safe threshold below current regulatory limits
GBD India State-Level, 2021 [10]	All Indian states, GBD 2019 methodology	1.67 million deaths attributable to all air pollution in India, 2019
Jaganathan et al., 2024 [11]	District-level mortality, all of India, 2009-2019	Attributable deaths vary five-fold depending on benchmark standard used
Oh et al., 2025 [14]	71 Asia-Pacific cohort/case-control studies	First Asia-Pacific-specific pooled estimates, addressing regional transferability gap

Table 5 traces the field's evolution from a handful of North American cohorts in the early 1990s to a geographically and methodologically diverse body of evidence three decades later. The trajectory is one of increasing precision rather than changing direction: no major study since Dockery's original six-city analysis has found the core association to be spurious, weak, or reversible; each successive wave of research has instead narrowed the confidence intervals, extended the geographic reach, and, most recently, begun correcting for the historical underrepresentation of exactly the region, South Asia, where current exposure is highest.

V. DISCUSSION

A. CRITICAL ANALYSIS OF FINDINGS

The pattern documented across Tables 1 through 5 supports a straightforward headline: Indian urban PM2.5 concentrations sit far above every applicable guideline, and elevated PM2.5 is robustly associated with elevated mortality and morbidity risk. Before pressing that headline further, several limitations inherent to this synthesis deserve direct acknowledgement, since a paper built entirely on secondary data owes its reader particular candor about what that data can and cannot support. First, relative-risk estimates in Table 3 are drawn overwhelmingly from cohort studies conducted in settings with far lower baseline PM2.5 concentrations, generally single-digit to low double-digit μg/m³, than the 50 to 110 μg/m³ range documented for Indian cities in Table 2. Exposure-response functions are not guaranteed to remain linear across this range; some evidence in the wider literature suggests supralinearity at low concentrations and flattening at higher ones, meaning the proportional risk increase estimated near WHO guideline levels may not apply unchanged at Delhi-level concentrations. This is precisely the transferability concern that motivated the 2025 Asia-Pacific meta-analysis, and it means Table 3's figures should be read as the best available approximation rather than a precise India-specific dose-response curve. Second, India's own air quality monitoring network, while extensive by regional standards at 259 monitored cities, remains concentrated in larger urban centres, leaving smaller towns and rural-urban fringe areas, where biomass burning and unpaved roads may generate comparably severe exposure, under-monitored and effectively invisible in datasets like Table 2. Any national or urban average, including the 48.9 μg/m³ figure cited here, is a population-weighted estimate built substantially from where monitors already exist, not a fully representative national sample. Third, and most consequentially, the near-fivefold difference between India's national-standard-based and WHO-guideline-based attributable-death estimates in Table 4 is not a data error but a genuine feature of how counterfactual health-impact modelling works: each estimate answers a different question about which baseline exposure level is assumed achievable. Presenting only one of these figures, as much public communication does, creates a false impression of precision that the underlying science does not support. This paper's decision to present multiple benchmark-dependent estimates side by side, rather than selecting a single headline number, is itself a substantive analytical choice, not a hedge. A fourth limitation concerns confounding and the ecological structure of much of the underlying evidence. City or district-level comparisons, of the kind several tables in this paper draw on, risk the ecological fallacy: an association observed at the level of aggregated populations does not guarantee that the same relationship holds for any given individual within that population, since diet, indoor exposure, occupational hazard, and access to healthcare all vary within a city in ways that population averages cannot capture. The strongest studies cited here, notably the Six Cities and ELAPSE cohorts, address this by following individuals rather than aggregates, but several of the India-specific figures in Table 4 rely on district-level rather than individual-level data, and that distinction should temper how literally the precise point estimates are read. Finally, the secondary-synthesis design means this study cannot adjudicate between competing exposure-response models or generate new India-specific effect estimates; it can only make the scale of existing disagreement visible and interpretable. That is a real analytical contribution, since a policymaker who understands why estimates range from 300,000 to 1.5 million deaths is better equipped than one presented with a single unqualified figure, but it stops short of resolving the underlying scientific question.

B. COMPARATIVE ASSESSMENT WITH PREVIOUS STUDIES

Placing this paper's findings against three decades of prior work reveals a story of both continuity and change. The continuity is in the core biological and statistical relationship: from Dockery's 1993 finding that a modest few-μg/m³ difference between the cleanest and dirtiest of six New England-area cities corresponded to measurably different mortality, through Pope's subsequent identification of the specific cardiopulmonary pathways involved, through Lepeule's 2012 finding that the association not only held but strengthened over an additional two decades of follow-up as measurement improved, to the 2025 Asia-Pacific-specific pooled estimate reported in Table 3, every major study has found PM2.5 exposure and adverse health outcomes moving together. What has changed is the concentration range at which that relationship is now being tested, and the geographic centre of scientific attention. The Harvard Six Cities Study compared cities whose annual PM2.5 averages ranged roughly from the low teens to the mid-twenties in μg/m³, a range that, mapped onto Table 2's figures, sits below even Seattle's current reading and far below anything recorded in urban India. The ELAPSE project pushed this further, deliberately testing cohorts at levels below existing regulatory limits and still finding no safe threshold, which is precisely the finding that justified WHO's 2021 decision to halve its annual guideline from 10 to 5 μg/m³. That single guideline revision, taken alone, retroactively reclassified enormous populations, roughly ninety percent of the world according to subsequent modelling, from compliant to exceeding, without a single additional death having occurred or a single monitor reading having changed. This illustrates something Table 4 makes concrete: headline attributable-mortality figures are exquisitely sensitive to a regulatory decision made in Geneva, not only to the underlying epidemiology, and comparing today's WHO-referenced figures against pre-2021 studies without adjusting for this shift risks manufacturing a trend where none exists.

Global mortality estimates specifically have moved in a similarly benchmark-dependent way. Lelieveld's 2015 modelling exercise, working from earlier exposure-response functions and an older emissions inventory, attributed roughly 3.3 million deaths a year to outdoor air pollution and placed India second to China in absolute terms. WHO's own 2019-data estimate of 4.2 million ambient-pollution deaths, cited throughout this paper, reflects updated exposure fields and the post-2021 guideline framework. Vohra and colleagues' GEOS-Chem-based estimate of roughly one million deaths specifically from fossil-fuel-derived PM2.5 is not a contradiction of either figure but a narrower slice of the same underlying phenomenon, isolating one source category. Read uncritically, a reader encountering these three numbers in sequence, 3.3 million, 4.2 million, and 1 million, might conclude the field is inconsistent; read with attention to what each model actually measures, they are compatible estimates of overlapping but distinct quantities, which is the interpretive discipline this paper has tried to model throughout Section IV.

India's own trajectory offers the sharpest illustration of how methodology, not just underlying air quality, drives the headline number. The Global Burden of Disease project's 2019 state-level analysis, using internationally standardised exposure-response functions calibrated substantially on non-Indian cohorts, attributed 0.98 million deaths to ambient particulate matter. Jaganathan and colleagues' 2024 difference-in-differences study, using an India-specific spatiotemporal exposure model and a decade of district-level mortality records rather than an imported global function, found a considerably larger attributable fraction against the WHO benchmark, alongside evidence that risk rises steeply even at exposure levels the GBD framework might treat as comparatively low-risk. This is not evidence that either study is wrong; it is evidence that the transferability concern motivating the 2025 Asia-Pacific meta-analysis was well founded, and that India-calibrated exposure-response functions, still rare in the literature, likely produce materially different, and plausibly higher, burden estimates than globally pooled ones.

Finally, comparing India's 2025 monitoring data against China's documented trajectory over the past decade is instructive by contrast. Chinese cities, subject to aggressive industrial and vehicular emissions controls since roughly 2013, no longer dominate the world's most polluted rankings the way they once did, with 2025 data instead showing a westward shift in China's pollution hotspots rather than uniform nationwide improvement. India's own National Clean Air Programme, by contrast, directs the majority of its funding toward road-dust control, a comparatively lower-cost intervention, while vehicular emissions and biomass burning, arguably larger contributors to the acute health burden documented in Tables 3 and 4, receive proportionally less investment. Thirty years after Dockery's original study identified the problem, and five years after WHO tightened its guideline in response to overwhelming accumulated evidence, the gap between scientific consensus and the pace of policy response, at least in the Indian urban context this paper has focused on, remains the central unresolved finding of this comparative exercise.

VI. CONCLUSION

This paper set out to quantify and contextualise the relationship between ambient PM2.5 and human health in urban areas, with particular attention to India, using a comparative synthesis of regulatory standards, current monitoring data, pooled epidemiological risk estimates, and disease-burden figures rather than new primary measurement. The evidence assembled across five tables supports the abstract's opening claim without qualification on the core point: PM2.5 exposure in Indian cities routinely exceeds every applicable guideline by a wide margin, and this exposure corresponds to a consistent, if not perfectly quantified, elevation in cardiovascular, respiratory, and all-cause mortality risk documented across three decades of independent research on multiple continents. Where this paper has added nuance rather than simple confirmation is in showing how much the headline attributable-death figure depends on which regulatory benchmark and which exposure-response function is applied, a range spanning roughly 300,000 to 1.5 million deaths annually for India alone depending on the assumption chosen. That range is not a weakness in the science; it is information a policymaker needs before selecting a target. The discussion's comparison with past work suggests that the biological and statistical relationship between PM2.5 and mortality has been substantially settled since at least the early 2000s, while the open questions have shifted toward exposure-response transferability across geographies and the pace of regulatory and infrastructural response. Future work would benefit most from India-calibrated, rather than imported, exposure-response functions, and from monitoring network expansion into the currently under-represented smaller cities and rural-urban peripheries identified as a gap in Section V.

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