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Air Pollution and Neurological and Respiratory Diseases: A Narrative Review with Emphasis on India

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ABSTRACT

Air pollution is the leading environmental cause of death worldwide, contributing to an estimated 7.9 million deaths in 2023, and India bears one of the heaviest burdens, with about 2 million attributable deaths. Its harm to the lungs is long established. Evidence that it also damages the brain is newer but has grown quickly, and the 2024 Lancet Commission on dementia added air pollution to its list of modifiable risk factors. This narrative review summarises the exposure situation in India, the mechanisms by which fine particles and gases injure the lung and brain, and the evidence for respiratory and neurological outcomes. In 2025 India's population-weighted mean PM2.5 was 48.9 µg/m³, nearly ten times the WHO guideline of 5 µg/m³. Long-term PM2.5 exposure is associated with impaired lung growth in children, childhood asthma, COPD, stroke (13% higher risk of incident stroke per 10 µg/m³), dementia and Parkinson's disease. These are mostly observational associations from North American and European cohorts, and effect sizes per unit of exposure are modest, although population attributable burdens are large. Intervention evidence is mixed: improved air quality was followed by better lung development in California, but a randomised trial that included an Indian site found that switching to LPG cooking did not significantly reduce severe infant pneumonia. India's National Clean Air Programme has not met its revised 2025–26 target in most cities. Priorities include Indian cohorts for neurological outcomes, sustained emission reduction and alignment with WHO guidelines.

Key words: Air pollution; PM2.5; COPD; asthma; stroke; dementia; Parkinson's disease; India; National Clean Air Programme.

1. Introduction

The World Health Organization tightened its air quality guidelines in 2021, setting the annual PM2.5 guideline at 5 µg/m³, half the 2005 value, because studies kept finding harm at lower concentrations than previously thought [1]. The Health Effects Institute's State of Global Air 2025 estimated that air pollution contributed to 7.9 million deaths in 2023, of which 6.8 million were from noncommunicable diseases, and that India and China each had more than 2 million attributable deaths [2]. In India, attributable deaths rose from about 1.4 million in 2000 to about 2 million in 2023 [2].

For decades, the lung has been the main concern. A newer literature suggests that the brain is also a target. For the first time, State of Global Air 2025 quantified the effect of air pollution on dementia, estimating about 626,000 dementia deaths and 11.6 million healthy years of life lost worldwide in 2023 [2]. This review asks how strong the evidence is for respiratory and neurological harm, how the mechanisms are understood, what Indian data show, and what interventions have worked. It is a narrative review without a systematic search; effect estimates are taken from major meta-analyses, cohorts and trials identified by the author, and Indian evidence is highlighted where it exists.

2. Pollutants, Sources and Guideline Values

Ambient air pollution is a mixture. The pollutants of greatest health concern are fine particulate matter (PM2.5 and PM10), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide [1]. Fine particles arise from combustion of fuels in vehicles, industry, power generation and household cooking, from open burning of biomass and waste, and from secondary formation in the atmosphere. Traffic also emits nitrogen oxides and black carbon, and brake wear emits iron-rich particles. Table 1 gives the 2021 WHO guideline values.

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Table 1. WHO 2021 air quality guideline values for key pollutants [1], with typical sources.

Pollutant	Typical sources	Annual guideline	Short-term guideline
PM2.5	Vehicles, industry, biomass and waste burning, household solid fuels, secondary formation	5 µg/m³	15 µg/m³ (24 h)
PM10	Road and construction dust, combustion, industry	15 µg/m³	45 µg/m³ (24 h)
Nitrogen dioxide (NO_2)	Vehicle exhaust, power plants	10 µg/m³	25 µg/m³ (24 h)
Ozone (O_3)	Formed from NO_x and volatile organic compounds in sunlight	60 µg/m³ (peak season)	100 µg/m³ (8 h)
Sulfur dioxide (SO_2)	Coal and oil combustion	–	40 µg/m³ (24 h)
Carbon monoxide (CO)	Incomplete combustion	–	4 mg/m³ (24 h)

3. Exposure and Burden in India

The 2025 IQAir World Air Quality Report, released in March 2026, ranked India sixth most polluted of 143 countries, with a population-weighted mean PM2.5 of 48.9 µg/m³ (50.6 in 2024), almost ten times the WHO guideline. Loni in Uttar Pradesh was the world's most polluted city at 112.5 µg/m³, Byrnihat in Meghalaya recorded 101.1, Delhi 99.6, Ghaziabad 89.2 and Ula in West Bengal 86.6; New Delhi again ranked as the most polluted capital at 82.2 µg/m³ [3]. A seasonal surge compounds the annual mean: in December 2025 PM2.5 levels rose by 44% in Delhi [3] (Figure 1).

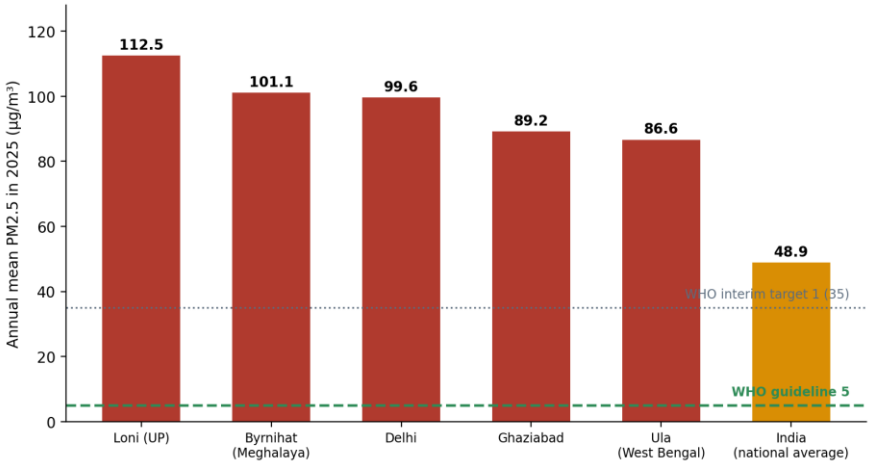


Figure 1. Annual mean PM2.5 in 2025 for the five most polluted Indian cities listed in the IQAir report and for India as a whole, against the WHO guideline (5 µg/m³) and WHO interim target 1 (35 µg/m³) [1,3]. Graph prepared from published values.

The Global Burden of Disease estimated that 1.67 million deaths in India in 2019 (17.8% of all deaths) were attributable to air pollution, of which 0.98 million were from ambient particulate matter and 0.61 million from household air pollution [4]. Per-capita economic loss was highest in Delhi, and the burden varied 5.4-fold across states [4]. By 2023, the estimate had risen to about 2 million deaths, of which about 89% were from non-communicable diseases; air pollution was reported to account for roughly seven in ten COPD deaths in India and more than one in three stroke deaths [2] (Figure 2). Because the 2023 figures were reported from the State of Global Air release, readers should consult the full report for state-level detail.

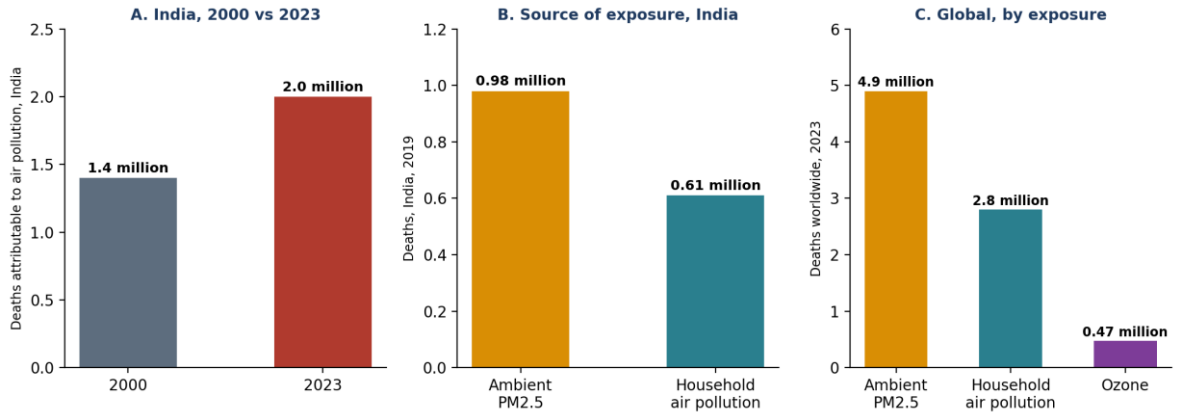


Figure 2. Burden of air pollution: (A). Deaths attributable to air pollution in India in 2000 and 2023 [2]. (B). Deaths in India in 2019 by source of exposure [4]. (C). Global deaths in 2023 by exposure; the groups overlap and do not sum to the total [2]. Graphs prepared from published values.

4. Mechanisms

Inhaled particles deposit along the airways and in the alveoli, where they provoke oxidative stress and inflammation. In the developing lung this can impair growth, and in adults it contributes to airway remodelling, COPD and exacerbations of asthma, while systemic spillover of inflammatory mediators affects the heart and blood vessels [5]. For the brain, two routes have been proposed (Figure 3). Systemic inflammation and endothelial dysfunction can injure cerebral vessels and the blood–brain barrier, and air pollution components can also reach the brain directly. Microglial activation and changes in the blood–brain barrier appear to be key steps, turning pollution exposure into a chronic source of pro-inflammatory factors and reactive oxygen species [6]. Magnetite nanoparticles with high-temperature, combustion-like morphology have been found abundantly in human brain tissue; particles smaller than about 200 nm may enter through the olfactory bulb [7]. Whether these particles cause disease in humans remains unproven [7]. The mechanistic picture is therefore plausible and partly supported by tissue and animal work but not yet confirmed as causal in people.

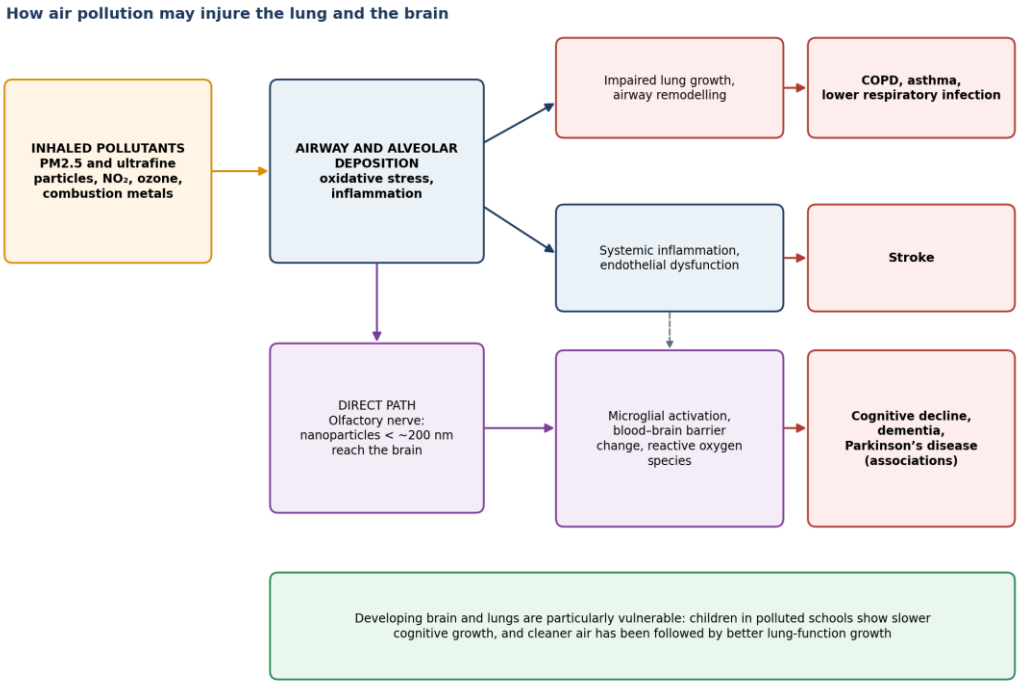


Figure 3. Proposed pathways by which air pollution may injure the lung and the brain, based on refs [5–9].

5. Respiratory Disease

Air pollution is a major driver of chronic respiratory disease. A review by the Forum of International Respiratory Societies estimated that about 1.6 million COPD deaths and 500,000 lung cancer deaths worldwide can be attributed to air pollution [5], and State of Global Air 2025 attributes roughly one in two COPD deaths worldwide to it [2]. India has 18% of the world's population but 32.0% of global disability-adjusted life-years from chronic respiratory diseases, and the number of people with COPD in India rose from 28.1 million in 1990 to 55.3 million in 2016 [10].

The most compelling evidence on lung development comes from southern California. Across three cohorts of 2,120 children followed from age 11 to 15, declining nitrogen dioxide and particulate levels over 13 years were associated with better growth of lung function, in children with and without asthma, and the proportion with clinically low FEV1 at age 15 fell from 7.9% to 6.3% to 3.6% [8]. For asthma, a meta-analysis of 41 studies found that traffic-related pollution was associated with childhood asthma incidence: relative risk 1.05 (95% CI 1.02–1.07) per 4 μg/m³ NO_2, 1.03 (1.01–1.05) per 1 μg/m³ PM2.5 and 1.08 (1.03–1.14) per unit of black carbon [11].

Household air pollution from solid fuels is a large part of India's burden [4], but interventions have had mixed results. In the Household Air Pollution Intervention Network (HAPIN) randomised trial, which included a site in Tamil Nadu, free LPG stoves and fuel did not significantly reduce severe pneumonia among 3,061 infants compared with continued biomass cooking [12], nor did they significantly change birth weight [13]. This does not show that household smoke is harmless, since exposure reductions may have been incomplete and ambient pollution persisted, but it cautions that fuel substitution alone may not deliver health gains. Table 2 summarises the respiratory evidence.

Table 2. Key evidence on air pollution and respiratory disease.

Outcome	Evidence	Key finding	Ref.
COPD (global)	Review of attributable burden	About 1.6 million COPD deaths attributed to air pollution; about 1 in 2 COPD deaths in 2023	[2,5]
Chronic respiratory disease (India)	GBD 1990–2016	COPD cases 28.1 → 55.3 million; India has 32.0% of global chronic respiratory DALYs	[10]
Lung growth in children	Three cohorts, 2,120 children	Lower NO_2 and PM associated with better FEV1 growth; low FEV1 at 15 years fell from 7.9% to 3.6%	[8]
Childhood asthma	Meta-analysis of 41 studies	RR 1.05 per 4 μg/m³ NO_2; 1.03 per 1 μg/m³ PM2.5; 1.08 for black carbon	[11]
Severe infant pneumonia	Randomised trial (HAPIN), 3,061 infants	LPG *vs* biomass cooking: no significant difference	[12]
Birth weight	Randomised trial (HAPIN)	LPG *vs* biomass cooking: no significant difference	[13]

6. Neurological Diseases

6.1 Stroke

Stroke is the best-established neurological outcome. A meta-analysis of 42 studies found that each 10 μg/m³ increase in long-term PM2.5 was associated with a 13% higher risk of incident stroke (95% CI 11%–15%) and a 24% higher risk of cerebrovascular mortality (13%–36%) [14]. Air pollution has been estimated to account for 21% of stroke deaths worldwide [5], and for more than one in three stroke deaths in India [2].

6.2 Dementia and Parkinson's disease

Evidence for dementia has strengthened. In a cohort of more than 63 million US Medicare beneficiaries, each 5 μg/m³ increase in annual PM2.5 was associated with a hazard ratio of about 1.13 for first hospital admission with Parkinson's disease and with Alzheimer's disease and related dementias, with a linear relationship below 16 μg/m³ [15]. In Ontario, people living within 50 m of a major road had a 7% higher incidence of dementia than those living farther away, but no higher incidence of Parkinson's disease or

multiple sclerosis [16], which illustrates that findings are not uniform across disorders. The 2024 Lancet Commission included air pollution among 14 modifiable risk factors that together may account for about 45% of dementia, and urged policymakers to improve air quality [17].

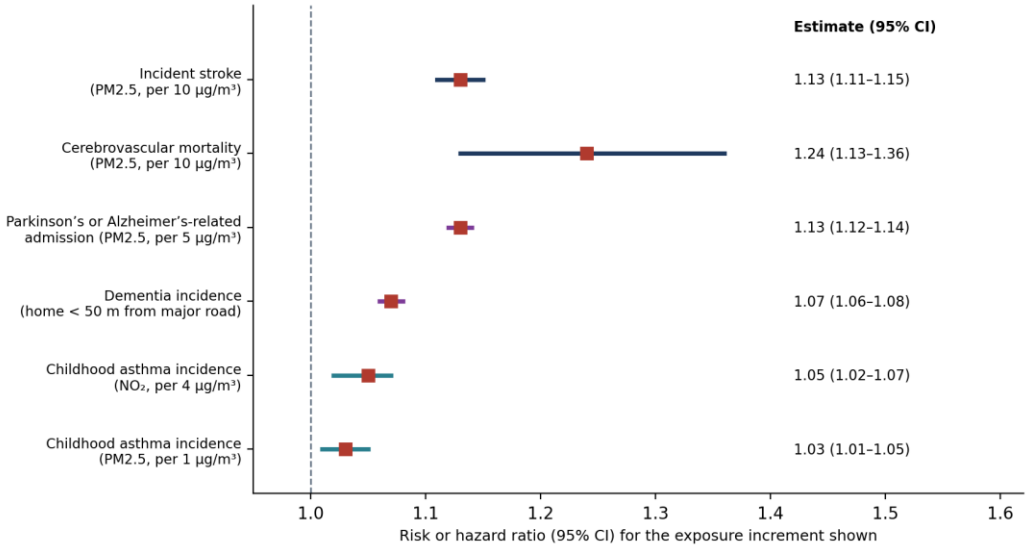


Figure 4. Selected effect estimates for stroke, dementia and childhood asthma from refs [11,14–16]. Squares are point estimates and lines 95% confidence intervals. Exposure increments differ between rows, so effect sizes are not directly comparable. Graph prepared from published values.

6.3 Neurodevelopment

The developing brain may be especially vulnerable. In a prospective study of 2,715 children aged 7 to 10 years in 39 Barcelona schools, children in schools with high traffic-related pollution gained less in working memory over 12 months (7.4% versus 11.5%) than children in paired low-pollution schools [9]. The authors stressed that such findings do not prove causation. Table 3 summarises the neurological evidence.

Table 3. Key evidence on air pollution and neurological outcomes.

Outcome	Evidence	Key finding	Ref.
Stroke	Meta-analysis of 42 studies	Incident stroke +13% and cerebrovascular mortality +24% per 10 µg/m³ PM2.5	[14]
Parkinson's disease and Alzheimer's-related dementias	US Medicare cohort, 63 million	HR about 1.13 per 5 µg/m³ PM2.5 for first hospital admission	[15]
Dementia (road proximity)	Ontario cohort, 243,611 cases	HR 1.07 within 50 m of a major road; no association with Parkinson's disease or multiple sclerosis	[16]
Dementia (population burden)	Lancet Commission; State of Global Air 2025	Air pollution is one of 14 modifiable risk factors; about 626,000 dementia deaths and 11.6 million healthy life-years lost in 2023	[2,17]
Child cognition	Prospective cohort, 2,715 children	Working-memory gain 7.4% vs 11.5% in high vs low pollution schools	[9]
Brain deposition of particles	Tissue analysis	Combustion-derived magnetite nanoparticles found in human brain	[7]

6.4 Interpreting the evidence

Three points limit confidence. First, most estimates come from observational studies in North America and Europe, where concentrations are far lower than in India, so extrapolating the exposure–response curve to Indian levels is uncertain; for instance, linearity was documented only below 16 µg/m³ in the Medicare cohort [15]. Second, residual confounding by socioeconomic status, smoking and noise is

hard to exclude, and the pattern differs between outcomes [16]. Third, we did not identify comparable large Indian cohorts for neurological outcomes, although this review did not use a systematic search. The relative risks are modest, but because exposure is universal and baseline disease is common, the attributable burden is substantial [2].

7. Interventions and Policy

India launched the National Clean Air Programme (NCAP) in 2019 to cover 130 cities. The target was revised to a reduction of up to 40% in PM10, or attainment of the 60 µg/m³ standard, by 2025–26. An independent assessment published in January 2026 found that of 100 NCAP cities with data, 23 recorded an increase in PM10, 51 achieved the initial 20–30% reduction target and only 23 achieved the 40% target; in 2025, 190 of 229 Indian cities with adequate data exceeded the national PM10 standard (Figure 5). In addition, 28 NCAP cities still lacked continuous monitoring and only 90 of 130 had completed source apportionment studies [18]. Government data for 2025–26 were released later and may show somewhat better progress, so readers should consult the latest official figures.

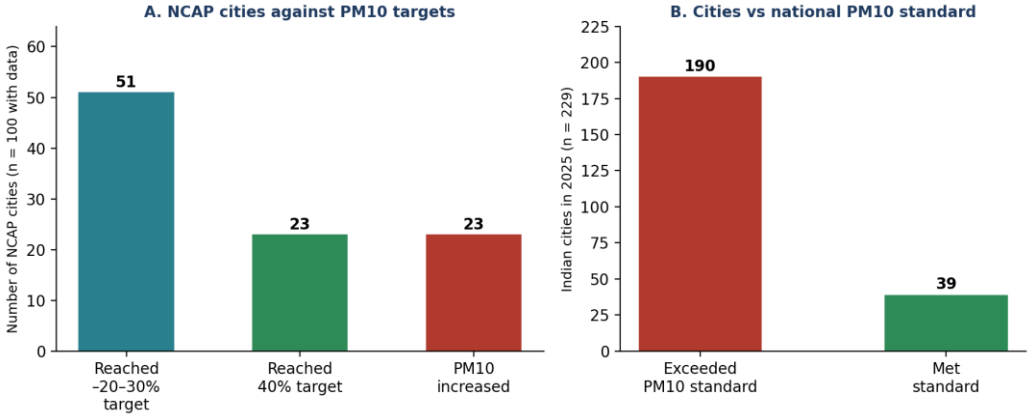


Figure 5. Progress of India's National Clean Air Programme according to the CREA 2026 assessment. (A) NCAP cities against PM10 reduction targets. (B) Indian cities relative to the national PM10 standard in 2025 [18]. Graphs prepared from published values.

International experience shows that policy can work. In southern California, sustained air-quality control was followed by improved lung development in children [8]. For India, the evidence points to a layered response (Table 4, Figure 6): source-specific emission cuts guided by local source apportionment, continuous monitoring and enforcement, household energy change coupled with other measures, protective measures during seasonal peaks, and research. For brain health specifically, the Lancet Commission recommends that policymakers improve air quality, particularly in areas with higher pollution [17].

Table 4. Policy levers and current position in India.

Lever	Position in India	Comment	Ref.
Guideline alignment	National mean PM2.5 48.9 µg/m³ versus WHO guideline of 5 µg/m³	Nearly tenfold gap	[1,3]
National Clean Air Programme	130 cities; revised target of 40% PM10 reduction by 2025–26; 23 cities met it in 2025 data; 190 of 229 cities exceeded PM10 standard	Targets largely unmet; monitoring and source apportionment incomplete	[18]
Household air pollution	0.61 million of 1.67 million attributable deaths in 2019; LPG switch did not significantly reduce infant pneumonia or change birth weight in HAPIN	Fuel switch alone may be insufficient	[4,12,13]
Dementia prevention	Air pollution one of 14 modifiable risk factors	Policy action on air quality recommended	[17]
Seasonal risk	PM2.5 in Delhi rose 44% in December 2025	Case for seasonal protective measures	[3]

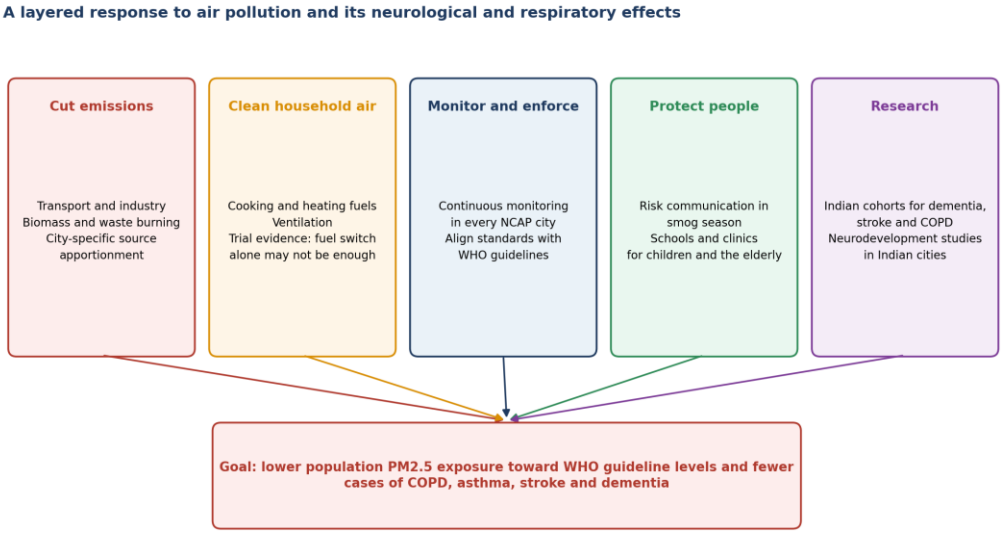


Figure 6. A layered response to air pollution and its respiratory and neurological effects in India, based on refs [3,12,17,18].

8. Research Gaps

Several gaps remain. Indian prospective cohorts are needed to measure the exposure–response relationship for stroke, dementia and COPD at the high concentrations that Indians experience. Neurodevelopmental studies in Indian cities are needed because children are exposed from before birth. The reasons for the null HAPIN results need to be understood, including how much exposure was actually reduced [12]. The mechanisms by which particles reach and affect the brain need confirmation in humans [6,7]. Finally, evaluation of clean-air policy should include health outcomes and not only pollutant concentrations [18].

9. Conclusions

Air pollution is a major and modifiable cause of respiratory disease and an increasingly credible contributor to stroke and dementia. India's exposure is among the highest in the world and the attributable burden is correspondingly large. The respiratory and stroke evidence is strong; the evidence for dementia, Parkinson's disease and child neurodevelopment is consistent but largely observational and drawn from lower-exposure settings. Interventions need to address sources at scale rather than rely on single measures, and India's current programme has not delivered the expected reductions in most cities. Cleaner air is a plausible way to protect both lungs and brains, and it should be pursued alongside research to measure the benefit in Indian populations.

Declaration, Conflict of Interest, Funding, and Other Statements

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Author Note

This manuscript is a narrative literature review intended for scholarly, educational, and research purposes in Environmental Sciences. The information presented is intended to support scientific understanding, critical evaluation, and further research on the subject. The review should not be interpreted as a substitute for site-specific environmental assessments, professional scientific judgment, regulatory requirements, or established environmental management practices. Any recommendations or conclusions concerning environmental monitoring, pollution control, ecological protection, or environmental remediation should be evaluated in the context of relevant scientific evidence, local environmental conditions, and applicable regulations.

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