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THE IMPACT OF AIR POLLUTION ON OUR HEALTH

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ABSTRACT

Air pollution is a mixture of gases and particles that enters the body mainly through the respiratory tract but affects many organ systems. This narrative review summarizes the sources and main pollutants of ambient air, the mechanisms by which they damage tissue, and the health outcomes most consistently linked to exposure. Fifteen sources were selected, including World Health Organization (WHO) guidelines, the State of Global Air 2025 report, The Lancet Commission on pollution and health, cohort studies, clinical reviews, and a World Bank assessment of air quality in Tashkent. Current estimates attribute about 7.9 million deaths in 2023 to air pollution, most of them from noncommunicable diseases such as ischaemic heart disease, stroke, chronic obstructive pulmonary disease (COPD), and lung cancer. Evidence also links exposure to impaired lung growth in children and to asthma, and emerging evidence links it to dementia.

Estimates of the burden differ between studies because of differences in exposure models and exposure–response functions, and population-level associations do not predict the outcome for an individual.

The principal conclusion is that air pollution is a preventable, modifiable risk factor whose control requires action at the level of emission sources as well as protective measures for vulnerable people. The review is an educational synthesis and reports no original patient observations or measurements.

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Introduction Air is the one environmental resource that a person cannot choose to avoid. An adult breathes thousands of litres of air every day, so any harmful substance suspended in it reaches the airways continuously. Air pollution is commonly described as the presence in the air of substances at concentrations that can harm human health; its most studied components are fine particulate matter (PM2.5, particles with a diameter of 2.5 micrometres or less), coarse particles (PM10), nitrogen dioxide (NO2), ozone (O3), sulphur dioxide (SO2), and carbon monoxide (CO) [1]. Because fine particles are small enough to reach the deep lung and some constituents can enter the circulation, their effects are not confined to the respiratory system [9].

The scale of the problem is large. The State of Global Air 2025 report estimates that air pollution contributed to 7.9 million deaths in 2023, about one in eight deaths worldwide, and that the largest burden falls on low- and middle-income countries [2]. Wider analyses of pollution of all kinds, including water and occupational exposures, attribute about nine million premature deaths a year to pollution, with air pollution as the largest component [3; 4]. These numbers explain why air quality is treated not only as an environmental topic but as a central public health issue.

The topic is also locally relevant. A World Bank assessment published in 2024 reported that the annual average concentration of PM2.5 in Tashkent was 38.8 µg/m3, several times the WHO annual guideline of 5 µg/m3, and that the health cost of this exposure corresponds to about 0.7% of Uzbekistan's gross domestic product [1; 14]. For a future physician, understanding how polluted air causes disease is therefore both a general medical topic and a practical one.

The purpose of this article is to explain how air pollution affects human health. The review addresses three questions: which pollutants matter most and how they are guided by current standards; through what biological mechanisms they cause harm; and which health outcomes are supported by the strongest evidence. The article is an educational synthesis of established evidence; it is not a risk-prediction tool and does not estimate the risk of any individual person.

Literature review Early evidence on long-term exposure came from cohort studies. In the Harvard Six Cities study, Dockery and colleagues followed adults in six US cities for fourteen to sixteen years and found that, after adjustment for smoking and other factors, mortality was higher in the most polluted city than in the least polluted one, with fine particles showing the strongest association [5]. Pope and Dockery later reviewed the accumulated evidence on fine particulate matter and concluded that both short-term and long-term exposures are associated with cardiopulmonary mortality, and that reductions in pollution have been accompanied by measurable health gains [6]. These studies are observational, so causal inference relies on consistency across populations, adjustment for confounders, and biological plausibility.

Mechanistic and clinical reviews supply that plausibility. The American Heart Association statement by Brook and colleagues concluded that exposure to PM2.5 over hours to weeks can trigger cardiovascular events, and that longer-term exposure increases cardiovascular risk and reduces life expectancy [7]. Rajagopalan and colleagues describe pathways in which inhaled

particles produce pulmonary and systemic inflammation, oxidative stress, and autonomic imbalance, which in turn promote atherosclerosis, thrombosis, and altered vascular tone [8]. Schraufnagel and colleagues of the Forum of International Respiratory Societies reviewed multisystem effects and noted that polluted air enters through the respiratory tract but can damage many other organs; they also stated that harm occurs on a continuum of dose, including levels below standards previously considered safe [9].

For the respiratory system, Guarnieri and Balmes reviewed the relationship between outdoor air pollution and asthma, covering both the exacerbation of existing disease and evidence that exposure contributes to the development of asthma [10]. For children, Gauderman and colleagues studied cohorts of schoolchildren in Southern California over two decades and reported that improved air quality was associated with less impairment of lung-function growth [11]. This finding is important because it connects a reduction in exposure with a measurable improvement, which supports the interpretation that the association is modifiable. Regarding cancer, the International Agency for Research on Cancer evaluated outdoor air pollution and particulate matter and classified them as carcinogenic to humans (Group 1) [12].

Burden estimates depend on method. Burnett and colleagues used a new exposure–response function that includes high-concentration settings and estimated 8.9 million deaths from outdoor PM2.5 in 2015, whereas the Global Burden of Disease analysis by Cohen and colleagues estimated 4.2 million for the same year with a different function [13; 15]. The two numbers are not contradictory evidence about whether pollution is harmful; they show how model assumptions change the size of an estimate.

In 2021, WHO revised its global air quality guidelines in light of expanded evidence, lowering the recommended annual PM2.5 level from 10 to 5 µg/m3 [1].

Research methodology A structured narrative review was conducted using targeted searches of PubMed-indexed literature, publisher and DOI records, and official publications of WHO, the Health Effects Institute, and the World Bank. Search concepts included air pollution, particulate matter, cardiovascular disease, respiratory disease, children's lung development, cancer, and air quality guidelines. Publication details and key figures were verified against online records on 22 October 2026. Fifteen references were selected for relevance, authority, and identifiable publication details. The selection was purposive and designed for an educational overview; it was not an exhaustive systematic review. The synthesis compared information across four levels: pollutant and source, biological mechanism, health outcome, and prevention. Evidence types were kept distinct: long-term cohort studies, clinical and mechanistic reviews, modelled burden estimates, and regulatory guidance answer different questions. Quantitative values are reported only where their source and meaning could be identified, and the original wording of estimates, such as the year and exposure model, is preserved. No patient records, surveys, measurements, or unpublished data were collected, and no pooled effect sizes or new prevalence estimates were

calculated. Some sources were consulted through abstracts and official summaries rather than full text, which is a limitation of the review.

Analysis and results Pollutants and guideline values. The first result of the synthesis is that different pollutants act through different exposures and are regulated by different metrics. PM2.5 is the pollutant most strongly linked to mortality in the reviewed literature, and the WHO guidelines set both annual and 24-hour limits for it. The 2021 WHO values are 5 µg/m3 (annual) and 15 µg/m3 (24 h) for PM2.5, 15 µg/m3 (annual) for PM10, 10 µg/m3 (annual) for NO2, 60 µg/m3 (peak season) for O3, 40 µg/m3 (24 h) for SO2, and 4 mg/m3 (24 h) for CO [1]. WHO analysis indicated that almost 80% of PM2.5-related deaths could be avoided if air pollution levels were reduced to those proposed in the updated guideline [1].

Mechanisms. The reviewed sources describe a common biological sequence. Particles and gases deposit in the airways and trigger local inflammation and oxidative stress. Inflammatory mediators and, for the smallest particles, some particle constituents reach the circulation, where they can alter endothelial function, coagulation, and autonomic control of the heart [8; 9]. This explains why inhalation of polluted air is linked not only to cough and asthma but also to myocardial infarction and stroke. The mechanisms are supported by experimental and clinical studies, but the relative contribution of each pathway differs between pollutants and individuals.

Health outcomes by organ system. The strongest evidence concerns the cardiovascular and respiratory systems. The State of Global Air 2025 report attributes 6.8 million of the 7.9 million deaths (86%) to noncommunicable diseases and reports that ambient PM2.5 accounted for 4.9 million deaths, household air pollution for 2.8 million, and ozone for 470,000 [2]. An earlier review cited estimates of about 500,000 lung cancer deaths and 1.6 million COPD deaths a year, and reported that air pollution accounts for a substantial share of cardiovascular and stroke deaths, while noting links to diabetes, reduced cognitive function, and effects on child development [9]. The 2025 report additionally estimated that air-pollution-related dementia caused about 626,000 deaths in 2023, although the causal interpretation of the dementia evidence is still developing [2]. These are population-level associations and cannot assign a cause to an individual illness.

Children and vulnerable groups. Children breathe more air relative to body weight and their lungs and brains are still developing, so exposure during growth may have lasting effects. The Southern California cohorts reported that children who grew up in cleaner air had less impairment of lung-function growth than children in more polluted conditions [11]. The respiratory societies' review likewise stated that people with other illnesses or less social support are more vulnerable [9]. Older adults, people with heart or lung disease, and those with high exposure are therefore priority groups for protection.

The situation in Uzbekistan. The World Bank assessment found that PM2.5 in Tashkent peaks in winter. Among human-caused sources, heating contributed 28%, transport 16%, and industry 13%, while windblown dust, a cross-boundary source, accounted for 36% in summer [14]. A report summary also noted that the Ministry of Health adopted a PM2.5 standard for residential

air in May 2024, aligned with WHO recommendations as an initial interim target [14]. These figures are for Tashkent and should not be assumed to apply equally to every region of the country.

Prevention. Because most sources are anthropogenic, prevention operates at two levels.

Population-level measures reduce emissions from heating, transport, and industry and extend monitoring and standards; the observed benefit of improved air quality for children's lungs supports this approach [11; 14]. Individual-level measures, such as limiting outdoor exertion during high-pollution days, improving indoor air, and seeking care for persistent respiratory or cardiac symptoms, reduce personal exposure but cannot replace cleaner air. The reviewed sources do not provide a single validated individual threshold, so recommendations should rely on local air quality information and medical advice.

Conclusion and recommendations Air pollution is a leading environmental risk factor for death and disease. Fine particulate matter enters through the lungs, causes inflammation and oxidative stress, and contributes to disease in the heart, vessels, lungs, and probably the brain. The strongest evidence concerns cardiovascular and respiratory disease, lung cancer, and impaired lung development in children, while evidence on dementia and metabolic disease is still maturing. Burden estimates differ between models, so numbers should always be quoted with their source and year, and population estimates should not be converted into individual predictions [2; 9; 13; 15].

Recommendations derived from the synthesis are as follows. First, public policy should aim at the 2021 WHO guideline values, using the interim targets as steps rather than as a safe level [1]. Second, for cities such as Tashkent, priority should go to the sectors identified as the largest sources, namely heating, transport, and industry, together with dust control [14]. Third, health workers should ask about exposure when assessing asthma, COPD, and cardiovascular risk and should advise vulnerable patients on protection during high-pollution periods. Fourth, children, older adults, and people with chronic disease should be treated as priority groups [9; 11]. These are recommendations drawn from published evidence, not measured outcomes of this review.

Future research should improve local exposure monitoring, estimate the burden in Uzbekistan with national data, and test which interventions produce the largest health gains at the lowest cost. Until such evidence is available, the central priority remains the reduction of emissions at the source, supported by clear public information and attention to the people most at risk [2; 4; 14].

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