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REVIEW ARTICLE

Air Pollution in Dhaka City: A Review of Biochemical Markers of Environmental Stress

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ABSTRACT

Dhaka, one of the world's fastest-growing megacities, experiences persistently high levels of air pollution, including fine particulate matter ($PM_{2.5}$, PM_{10}), black carbon, and episodic peaks of toxic gases such as NO_2, SO_2, O_3, and CO. These pollutants arise from diverse sources, including vehicular emissions, industrial activities, brick kilns, construction, and open waste burning, and exhibit strong spatial and seasonal variability. Emerging biomarker studies in Dhaka reveal that exposure to these pollutants induces measurable physiological and molecular alterations, including oxidative stress, reduced antioxidant defenses, systemic inflammation, impaired oxygenation, and early genotoxic effects. Children, pregnant women, and occupationally exposed adults demonstrate heightened susceptibility, with short-term exposures linked to acute lung function decline and long-term exposure associated with cumulative biochemical dysregulation. Integration of ambient and indoor pollution data with biochemical outcomes highlights mechanistic pathways connecting environmental exposure to adverse health effects. This review consolidates Bangladesh-specific evidence on air pollution and biochemical markers of environmental stress, emphasizing the biological plausibility of pollution-induced health risks and the utility of biomarkers in environmental health assessment.

Keywords: Dhaka; Air pollution; Biomarkers; Oxidative stress; Inflammation; PM2.5

1. Introduction

Dhaka, the capital of Bangladesh and one of the world's fastest-growing megacities, consistently experiences some of the highest levels of ambient air pollution globally. Rapid and largely unplanned urbanization, population density, industrial expansion, and traffic congestion have created a complex environmental crisis centered around deteriorating air quality[1]. Major air pollutants in Dhaka include fine and coarse particulate matter ($PM_{2.5}$ and PM_{10}), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), carbon monoxide (CO), and black carbon, which originate mainly from vehicular emissions, brick kilns, industrial activities, construction dust, and open waste burning[2]. Seasonal meteorological conditions, such as temperature inversions and low wind speeds during winter, further intensify pollutant accumulation, leading to prolonged periods of hazardous air quality[3].

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Chronic and acute exposure to these air pollutants has been shown globally to contribute to a wide range of adverse health outcomes, including respiratory diseases, cardiovascular disorders, metabolic dysfunction, neurodevelopmental impairments, and increased mortality. Vulnerable populations such as children, the elderly, pregnant women, and individuals with pre-existing illnesses are particularly at risk[4]. In Dhaka, multiple local studies and national assessments have reported persistent exceedance of both national and World Health Organization (WHO) air quality guidelines, highlighting an alarming public health burden. However, while epidemiological evidence links air pollution exposure to disease outcomes, there remains a crucial need to better understand the underlying biological and molecular mechanisms through which these pollutants exert their harmful effects, particularly in the Bangladeshi population[5].

Biochemical markers of environmental stress provide valuable mechanistic insight into how air pollutants disrupt physiological homeostasis. These biomarkers include indicators of oxidative stress such as malondialdehyde (MDA), reduced glutathione (GSH), and antioxidant enzymes (superoxide dismutase, catalase); inflammatory markers including C-reactive protein (CRP), interleukins (IL-6), and tumor necrosis factor-α (TNF-α); hypoxia-related indicators such as peripheral oxygen saturation (SpO_2); and genotoxicity markers like 8-hydroxy-2′-deoxyguanosine (8-OHdG)[6,7]. Changes in these parameters reflect cellular responses to pollutant-induced oxidative damage, systemic inflammation, impaired oxygen transport, and DNA damage, which may precede the development of chronic diseases. Therefore, integrating biochemical markers with environmental exposure data serves as a critical bridge between air pollution and disease pathogenesis.

Although global research on air pollution and biomarkers has expanded substantially, studies focusing specifically on Dhaka and the Bangladeshi population remain relatively limited and fragmented. Most existing research in Bangladesh has focused on ambient pollutant monitoring and health symptom surveillance, with fewer studies systematically exploring biochemical or molecular endpoints in exposed populations. Given Dhaka's unique environmental context, emission sources, socio-economic conditions, and population vulnerability, findings from other countries may not fully capture the local health risks and biological responses. This review aims to synthesize and critically evaluate existing Bangladeshi and Dhaka-focused studies that link air pollution exposure with biochemical markers of environmental stress.

2. Methods

This review employed a focused literature-based approach to synthesize current knowledge on air pollution and associated biochemical markers in Dhaka, Bangladesh. The primary objective was to identify studies examining human biomarker responses to particulate matter (PM) and co-pollutants, providing insight into mechanistic pathways of pollution-induced health effects. A systematic search of relevant literature was conducted covering the period from 2020 to 2025, with inclusion of earlier foundational Dhaka-based studies when necessary to contextualize recent findings. Electronic databases including PubMed, Google Scholar, and ResearchGate were searched, alongside institutional reports and national assessments, such as those produced by government agencies, academic institutions, and policy think tanks.

Search terms combined air pollution and biomarker-related keywords with location-specific descriptors, including "Dhaka air pollution biomarkers," "PM2.5 Dhaka oxidative stress," "children Dhaka air pollution biomarkers," "indoor PM Dhaka SpO_2," and "black carbon Dhaka." The search strategy prioritized studies that measured biochemical or physiological markers in human populations, including schoolchildren, adult residents, occupationally exposed workers, and pregnant women. Additionally, indoor monitoring studies conducted in Dhaka homes and schools were included to provide insight into personal exposure profiles and their association with biochemical outcomes. Air monitoring and source apportionment studies reporting

ambient $PM_{2.5}$, PM_{10}, black carbon, and gaseous pollutant levels were also considered to contextualize biomarker findings within the broader exposure environment.

From this comprehensive search, 10–12 representative studies and reports were selected for synthesis based on relevance, quality, and the inclusion of measurable biomarker outcomes. Data were systematically extracted from each study, including population characteristics, exposure metrics, biochemical markers assessed, key findings, and study limitations. Where specific Dhaka-focused biomarker data were limited, nationally representative studies and reports including Dhaka measurements were incorporated to provide environmental context. Both cross-sectional and panel-based studies were considered to capture acute and chronic exposure effects, and information on indoor versus outdoor exposures was highlighted where available.

The extracted data were synthesized narratively to identify patterns of exposure, biomarker responses, and population vulnerability. Particular attention was paid to oxidative stress markers (e.g., malondialdehyde, 8-OHdG), antioxidant enzymes (e.g., superoxide dismutase, catalase, glutathione), inflammatory markers (e.g., CRP, IL-6, TNF-α), and hypoxia indicators (SpO_2), as these were the most commonly assessed endpoints in Dhaka studies. This approach allowed the integration of environmental measurements, human biomarker responses, and mechanistic insights to develop a cohesive understanding of how air pollution in Dhaka impacts human health at the biochemical level. The methods and data synthesis provided the foundation for the results, discussion, and conclusions presented in this review.

3. Results

Table 1 summarizes the key Dhaka-focused and Bangladesh-relevant studies linking air pollution exposure to biochemical and physiological outcomes. These studies provide the foundational context for understanding pollution–biomarker relationships in urban Bangladesh.

Table 1. Dhaka Air Pollution and Biomarker Studies

No.	Study (Authors, Year)	Population / Matrix	Pollutants Measured	Biomarkers / Outcomes	Key Findings
1	Impact of fine particulate matter and toxic gases on the health of schoolchildren in Dhaka (2023)[8]	250 schoolchildren, Dhaka	$PM_{2.5}$, PM_{10}, NO_2, CO	Spirometry (FEV_1, FVC), respiratory symptoms	Short-term PM & gas exposure significantly reduced lung function and increased respiratory symptoms.
2	Indoor particulate matter exposure and correlation of $PM_{2.5}$ with lung efficacy and SpO_2 (2024)[9]	Urban households, Dhaka	Indoor/outdoor $PM_{1.0}$, $PM_{2.5}$, PM_{10}	SpO_2, lung capacity indicators	Indoor $PM_{2.5}$ showed strong negative correlation with SpO_2 and lung efficacy.
3	Wintertime black carbon assessment in Dhaka (2025)[10]	Ambient monitoring (winter season)	Black carbon, $PM_{2.5}$	Exposure burden mapping	Winter BC levels extremely high; BC major contributor to toxic $PM_{2.5}$.
4	Spatiotemporal dynamics of air pollution in Dhaka (2025)[10]	Satellite & ground-based data	$PM_{2.5}$, NO_2, SO_2, O_3	Exposure distribution & mapping	Strong seasonal and spatial variation; winter worst, central Dhaka most

No.	Study (Authors, Year)	Population / Matrix	Pollutants Measured	Biomarkers / Outcomes	Key Findings
					affected.
5	Short-term exposure to ambient particulate matter and health in Dhaka schoolchildren (2019)[11]	Panel study (children)	$PM_{2.5}$, PM_{10}	Respiratory morbidity, airway inflammation	Short-term PM peaks triggered acute respiratory symptoms and reduced lung function.
6	Air pollution and dysregulated blood biomarkers in pregnant women (2024 review)[12]	Pregnant women cohorts (Bangladesh included)	$PM_{2.5}$ & O_3 proxies	CRP, IL-6, TNF-α, pregnancy outcomes	Positive association between PM exposure and systemic inflammation; higher risk of adverse birth outcomes.
7	Atmospheric particulate matter and trace metals in Bangladesh (2024 review)[8]	National data (Dhaka emphasized)	$PM_{2.5}$/PM_{10}, metals (Pb, Cd, As)	Exposure toxicological framework	PM-bound toxic metals identified as critical drivers of oxidative stress.
8	Indoor $PM_{2.5}$ heavy metals in Dhaka households (2025)[7]	Indoor air PM filters	$PM_{2.5}$-bound metals	Metal load index, oxidative stress relevance	Significant heavy metal contamination observed in indoor air PM samples.
9	Long-term air pollution and cardiometabolic biomarkers (contextual global study applied to Dhaka) (2024)[6]	Population cohort (modeled for Dhaka)	$PM_{2.5}$	CRP, MPO, GDF-15, oxidative stress markers	Long-term PM exposure linked with chronic inflammation and systemic oxidative stress.
10	National air quality assessments (CPD/IGC reports) (2023)[5]	Dhaka and national level	$PM_{2.5}$/PM_{10}, source apportionment	Policy and exposure assessment	Chronic exceedance of air quality limits confirmed; brick kilns & traffic major sources.

Table 1. (*Continued*)

3.1. Biomarkers used in dhaka air pollution studies

Studies conducted in Bangladesh, along with international evidence adapted to the Dhaka context, consistently highlight a panel of biochemical and physiological markers that respond to air pollution exposure.

Table 2. Key biomarker categories and air pollution-linked alterations[10,11,13]

Biomarker Type	Specific Biomarkers	Observed Changes	Health Implications
Oxidative stress markers	MDA, 8-OHdG, GSH, SOD, CAT, GPx	↑ MDA & 8-OHdG, ↓ GSH, ↓ antioxidant enzymes	Indicates increased lipid peroxidation and oxidative DNA damage.
Inflammatory markers	CRP, IL-6, TNF-α, MPO	Elevated levels with higher PM exposure	Suggests systemic inflammation and cardiovascular risk.

Biomarker Type	Specific Biomarkers	Observed Changes	Health Implications
Hypoxia / oxygenation	SpO_2	Reduced SpO_2 in residents exposed to high indoor/outdoor PM	Indicates impaired oxygen delivery and pulmonary compromise.
Respiratory function markers	FEV_1, FVC, PEFR	Decline during high pollution episodes	Suggests airway inflammation and obstruction.
Genotoxicity markers	8-OHdG, comet assay	Increased DNA damage (limited Bangladesh studies, strong regional evidence)	Potential risk for long-term mutagenic and carcinogenic effects.

Table 2. (*Continued*)

3.2. Pollutant-specific biochemical impacts in Dhaka

Different pollutants have different toxicological profiles. Their effects on biochemical parameters can be summarized as follows:

Table 3. Pollutant-wise biochemical and physiological effects[9,13,14]

Pollutant	Source in Dhaka	Associated Biomarkers	Reported Biological Effects
$PM_{2.5}$	Brick kilns, traffic, construction	MDA, CRP, IL-6, 8-OHdG	Oxidative stress, systemic inflammation, reduced lung function
PM_{10}	Road dust, construction	FEV_1, FVC, SpO_2	Obstructive lung changes, hypoxia
Black carbon	Diesel vehicles, brick kilns	MPO, CRP, oxidative stress markers	High cardiopulmonary toxicity
NO_2	Traffic emissions	CRP, lung function indices	Respiratory irritation, airway inflammation
O_3	Photochemical reactions	IL-6, oxidative markers	Oxidative lung damage
PM-bound heavy metals (Pb, Cd, As)	Industrial emission, dust	8-OHdG, MDA	Neurotoxicity, genotoxicity, cancer risk

3.3. Exposure Context: Dhaka pollutant patterns

Recent ground-based air quality monitoring and satellite observations clearly indicate that Dhaka persistently exceeds both Bangladesh National Ambient Air Quality Standards and the World Health Organization (WHO) Air Quality Guidelines. Throughout most of the year, fine particulate matter ($PM_{2.5}$) levels remain above safe thresholds, but the situation becomes critically severe during the winter months (November–February)[15]. This chronic exceedance reflects rapid urbanization, uncontrolled industrial growth, and high vehicular density, making Dhaka one of the most polluted megacities globally. The extreme elevation of $PM_{2.5}$ concentrations during the winter season can be attributed to a combination of meteorological and anthropogenic factors. Temperature inversion traps pollutants close to the ground by preventing vertical air mixing, while low wind speed limits horizontal dispersion of pollutants. At the same time, emissions from thousands of brick kilns operating around Dhaka significantly increase during winter. Heavy traffic congestion, arising from increasing vehicle numbers and inefficient road management, also contributes a large volume of particulate emissions. Additionally, long-range transboundary pollution from neighboring regions further worsens the air quality situation during this period[16].

Beyond particulate mass concentration, the toxic nature of Dhaka's air pollution is intensified by high levels of black carbon and $PM_{2.5}$-bound heavy metals such as lead, cadmium, and arsenic. Black carbon particles, mainly emitted from diesel engines and brick kilns, have strong oxidative properties and are associated with cardiovascular and respiratory diseases. PM-bound heavy metals contribute to increased oxidative stress and DNA damage in exposed individuals, disrupting normal cellular functions and increasing the risk of chronic diseases, including cancer. The biochemical and physiological impacts of these pollutants are not uniform across all population groups. Vulnerable populations, particularly children, pregnant women, and the elderly, experience stronger and more harmful biomarker responses. In children, developing lungs are more sensitive to pollutants, leading to reduced lung function and increased asthma risk. In pregnant women, exposure to high levels of $PM_{2.5}$ is associated with elevated inflammatory markers and adverse pregnancy outcomes such as low birth weight and preterm birth. Among the elderly, chronic exposure accelerates oxidative stress and systemic inflammation, aggravating cardiovascular and respiratory diseases[17]. These findings emphasize the urgent need for targeted public health interventions to protect high-risk populations.

3.4. Vulnerable population groups and biomarker outcomes

Table 4. Vulnerable Groups and Air Pollution Biomarkers[17,18]

Population Group	Major Exposure	Biomarker Response	Potential Health Outcomes
School children	High traffic exposure near schools	↓ FEV_1, ↑ respiratory symptoms	Impaired lung growth, asthma risk
Pregnant women	Chronic $PM_{2.5}$ exposure	↑ CRP, IL-6, altered oxidative markers	Preterm birth, low birth weight
Elderly	Long-term urban exposure	↑ oxidative stress & inflammation	Increased cardiovascular morbidity
Outdoor workers	Construction dust, traffic emissions	↓ SpO_2, ↑ MDA	Chronic respiratory & cardiovascular damage

3.5. Integration of exposure patterns and biomarker responses

The clear temporal overlap between Dhaka's severe winter pollution episodes and the observed increases in oxidative stress, systemic inflammation, and impaired lung function provides strong biological plausibility for a causal relationship. During winter, when $PM_{2.5}$, black carbon, and toxic metal levels peak, several studies have reported parallel elevations in oxidative stress markers such as malondialdehyde (MDA) and 8-hydroxy-2′-deoxyguanosine (8-OHdG), alongside reductions in antioxidant defenses like glutathione (GSH). These shifts indicate that air pollutants induce an imbalance between reactive oxygen species production and antioxidant protection, leading to cellular damage and physiological stress in exposed populations. Short-term exposure to high concentrations of particulate matter and traffic-related pollutants has been shown to trigger immediate biological and functional responses, including increased airway inflammation, reduced spirometric indices (e.g., FEV_1 and FVC), decreased peripheral oxygen saturation (SpO_2), and elevated inflammatory markers such as C-reactive protein (CRP) and interleukin-6 (IL-6). These acute responses are particularly evident among schoolchildren, outdoor workers, and individuals living near major roads or industrial zones. In contrast, chronic long-term exposure results in cumulative biochemical dysregulation, characterized by persistent low-grade inflammation, chronic oxidative stress, endothelial

dysfunction, and DNA damage, which are key mechanisms underlying the development of cardiovascular diseases, chronic respiratory disorders, metabolic syndrome, and adverse reproductive outcomes[14,18].

The integration of environmental exposure data with biochemical and clinical marker findings strengthens the mechanistic understanding of how air pollution contributes to disease development in Dhaka. Instead of relying solely on self-reported symptoms or hospital records, incorporating biomarker-based evidence allows for the detection of subclinical effects before overt disease manifests. This approach provides a more sensitive and mechanistic framework for assessing health risk and identifying vulnerable populations. The concordance between seasonal pollution peaks, biomarker alterations, and clinical outcomes strongly supports the role of air pollution as a key driver of environmental stress and disease burden in urban Bangladesh. This integrated body of evidence underscores the urgent need for multi-level interventions in Dhaka and other rapidly urbanizing Bangladeshi cities[19]. Effective strategies must combine strict air quality control policies with population-level biomarker-based health surveillance systems. Regular monitoring of oxidative stress, inflammatory markers, and respiratory function in high-risk groups such as traffic police, garment workers, schoolchildren, and pregnant women could help in early detection and prevention of pollution-related health damage. Such integration of environmental and biomedical data will be crucial for developing targeted public health interventions and strengthening national policies aimed at reducing the long-term health burden of air pollution[16].

4. Discussion

4.1. Overview of air pollution in Dhaka

Air pollution in Dhaka is a persistent environmental and public health challenge, characterized by extremely high concentrations of particulate matter ($PM_{2.5}$ and PM_{10}), black carbon, and intermittent peaks of toxic gases, including nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), and carbon monoxide (CO). The sources of pollution are diverse and largely anthropogenic. Vehicular emissions from a rapidly growing number of motor vehicles contribute substantially to nitrogen oxides, carbon monoxide, and fine particulates. Brick kilns, which proliferate around Dhaka, emit high levels of $PM_{2.5}$, PM_{10}, black carbon, and trace metals such as lead (Pb), cadmium (Cd), and arsenic (As). Industrial zones release sulfur dioxide, nitrogen oxides, volatile organic compounds, and particulate matter, while construction activities and road dust contribute coarse particulates. Open waste burning further introduces toxic gases and fine particulates into the urban atmosphere[10]. Spatial distribution of pollutants is highly heterogeneous. Areas with dense traffic corridors, brick kiln clusters, or industrial activity exhibit significantly higher pollutant concentrations compared to suburban or low-traffic zones. Temporally, pollutant concentrations fluctuate seasonally. During winter, atmospheric inversions, low wind speeds, and higher energy consumption exacerbate pollution accumulation, resulting in peaks in $PM_{2.5}$ and PM_{10}. Satellite-based studies corroborate ground-level monitoring, showing consistent exceedances of both Bangladesh national air quality standards and WHO guidelines. The persistent nature of pollution, combined with seasonal peaks, underscores chronic and episodic exposure for residents across the city[13].

4.2. Biochemical and physiological responses

Exposure to elevated air pollution in Dhaka is associated with distinct biochemical and physiological alterations. Oxidative stress is a primary response to particulate and gaseous pollutants. Malondialdehyde (MDA), a marker of lipid peroxidation, is consistently elevated in exposed populations, indicating cellular membrane damage. DNA damage, measured through 8-hydroxy-2′-deoxyguanosine (8-OHdG), reflects oxidative assault on nucleic acids, suggesting that environmental stress may have long-term genotoxic implications. Antioxidant defenses are compromised following exposure. Reduced glutathione (GSH),

superoxide dismutase (SOD), and catalase (CAT) activity have been reported as diminished, reflecting a weakened ability to neutralize reactive oxygen species[18]. This imbalance between pro-oxidants and antioxidants contributes to systemic oxidative stress, which is a recognized mechanism for the development of cardiovascular, respiratory, and metabolic diseases. Systemic inflammation represents another key biological response. Biomarkers such as C-reactive protein (CRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and myeloperoxidase (MPO) are elevated in exposed cohorts. These findings indicate activation of pro-inflammatory pathways and chronic low-grade inflammation, which may predispose individuals to endothelial dysfunction, insulin resistance, and increased risk of atherosclerosis. Hypoxic stress, demonstrated by reduced peripheral oxygen saturation (SpO_2), reflects impaired gas exchange and decreased oxygen availability, particularly in populations experiencing high indoor or outdoor particulate exposures. Children, pregnant women, and occupationally exposed adults exhibit the most pronounced biomarker changes, highlighting the interaction between developmental stage, physiological vulnerability, and pollutant susceptibility[20].

4.3. Respiratory function and clinical implications

Respiratory endpoints are consistently affected in Dhaka populations exposed to air pollution. Short-term exposure panels, including school-based studies and indoor assessments, report transient reductions in lung function indices such as forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC). These functional impairments often coincide with acute peaks in $PM_{2.5}$, PM_{10}, and toxic gases, resulting in increased prevalence of respiratory symptoms including coughing, wheezing, and dyspnea[20]. Long-term exposure appears to produce cumulative effects. Cross-sectional biomarker studies indicate sustained oxidative stress, chronic inflammation, and genotoxic damage, which likely contribute to the city's high burden of chronic respiratory diseases, cardiovascular disorders, and metabolic dysregulation. Indoor air pollution, particularly in poorly ventilated households with biomass cooking or proximity to traffic corridors, contributes substantially to personal exposure, suggesting that total pollutant burden is a combination of both outdoor and indoor sources. These findings demonstrate that both acute and chronic exposures have measurable impacts on respiratory and systemic health[9].

4.4. Source contributions and seasonal patterns

Understanding pollutant sources is critical for interpreting biomarker outcomes. Vehicular traffic remains a dominant source of NO_2, CO, and fine PM, while brick kilns contribute high levels of $PM_{2.5}$, black carbon, and trace metals. Industrial discharges, including sulfur dioxide and volatile organic compounds, further amplify chemical exposure. Construction activities and road dust contribute coarse particulate matter, and open waste burning releases multiple toxic compounds including dioxins and PAHs (polycyclic aromatic hydrocarbons)[21]. Seasonal patterns reveal winter peaks as the most critical period for exposure. During winter, temperature inversions trap pollutants close to the ground, resulting in higher particulate and gaseous concentrations. Biomarker studies consistently show that winter peaks coincide with maximal oxidative stress, inflammation, and hypoxia in exposed individuals. Furthermore, $PM_{2.5}$-bound heavy metals and black carbon particles amplify toxicity, exacerbating oxidative and inflammatory responses. Seasonal and source-specific variations underscore the complexity of exposure-response relationships and the necessity of considering both temporal and spatial pollutant patterns[10].

4.5. Integration of exposure and biomarker data

Integration of air pollution measurements with biochemical markers illustrates a clear mechanistic pathway linking environmental exposure to molecular and physiological outcomes. Ambient and indoor pollutant levels drive oxidative stress, inflammatory cascades, hypoxia, and DNA damage. Short-term

exposures provoke acute changes in biomarkers, often reversible but clinically relevant, while chronic exposure results in cumulative biochemical dysregulation with potential long-term health consequences. The concordance between exposure peaks and biomarker elevations demonstrates a dose-response relationship. Acute $PM_{2.5}$ spikes correlate with transient lung function decline, increased oxidative stress, and inflammatory activation, while chronic high exposure contributes to persistent biochemical alterations, endothelial dysfunction, and potential progression to chronic disease. This integrated evidence confirms the utility of biochemical markers as sensitive indicators of environmental stress and early warning signals of health risk in urban populations[22].

4.6. Vulnerable populations and differential susceptibility

Biochemical and physiological responses are particularly pronounced in vulnerable groups. Children exhibit impaired lung function and heightened oxidative stress, reflecting developmental sensitivity. Pregnant women show increased inflammatory and oxidative biomarkers, which may have implications for fetal development and birth outcomes. Elderly individuals and occupational cohorts exposed to high particulate loads demonstrate cumulative oxidative and inflammatory damage, reflecting long-term susceptibility. The heterogeneity of response across populations emphasizes that exposure effects are influenced not only by pollutant concentration and composition but also by age, physiological status, and activity patterns. Indoor exposure, socio-economic status, and housing conditions further modulate susceptibility, illustrating the multifactorial nature of pollutant effects. Collectively, the evidence underscores that air pollution in Dhaka acts as a major biochemical stressor with measurable acute and chronic health impacts across multiple physiological systems[23].

5. Conclusions

Air pollution in Dhaka presents a severe and continuing threat to public health. Biomarker studies — though limited — reveal plausible mechanistic pathways (oxidative stress, inflammation, hypoxia) through which PM and co-pollutants affect residents, particularly children and vulnerable groups. To translate mechanistic insight into effective mitigation, Bangladesh needs standardized biomarker protocols, longitudinal cohort investments, improved personal exposure assessment, and decisive policy measures to tackle major emission sources. This review consolidates current Bangladesh-based evidence and outlines practical research and policy steps to protect Dhaka's population from pollution-driven biochemical stress.

6. Recommendations

Based on the synthesis of current evidence regarding air pollution and biochemical responses in Dhaka, the following recommendations are proposed for research, public health practice, and policy interventions:

6.1. Research recommendations

1. **Standardized Biomarker Panels**: Establish uniform biomarker panels encompassing oxidative stress markers (MDA, 8-OHdG), antioxidant enzymes (GSH, SOD, CAT), inflammatory markers (CRP, IL-6, TNF-α, MPO), hypoxia indicators (SpO_2), and genotoxicity assays. This will improve comparability across studies and facilitate meta-analyses.
2. **Longitudinal Cohort Studies**: Initiate long-term cohort studies targeting children, pregnant women, elderly populations, and occupationally exposed groups. Repeated biomarker sampling over time will allow assessment of cumulative exposure effects, disease progression, and temporal trends in biochemical alterations.

3. **Personal Exposure Assessment**: Employ wearable or personal monitors for $PM_{2.5}$, black carbon, and gaseous pollutants to capture true individual-level exposure, reducing misclassification associated with ambient or short-term indoor measurements.
4. **Multi-Pollutant and Indoor–Outdoor Integration**: Investigate combined effects of multiple pollutants and integrate indoor and outdoor air quality measurements to better reflect real-world exposure scenarios.
5. **Omics and Molecular Approaches**: Incorporate genomic, transcriptomic, metabolomic, and epigenetic analyses to elucidate molecular mechanisms, identify early biomarkers of susceptibility, and explore population-specific responses.
6. **Linkage with Clinical Outcomes**: Integrate biomarker findings with health endpoints such as lung function tests, hospital admissions, cardiovascular events, and pregnancy outcomes to strengthen translational relevance.

6.2. Public health and policy recommendations

1. **Emission Reduction Strategies**: Implement stricter regulations for vehicular emissions, brick kiln operations, industrial discharges, and construction dust. Promote clean fuel alternatives and modernized technologies to reduce PM and black carbon emissions.
2. **Urban Planning and Traffic Management**: Develop urban planning initiatives to reduce traffic congestion in densely populated areas, create green buffers, and relocate high-emission industries away from residential zones.
3. **Indoor Air Quality Improvement**: Promote ventilation improvements, household-level air filtration, and reduction of indoor biomass burning to minimize indoor exposure, particularly in vulnerable populations.
4. **Health Surveillance and Biomonitoring**: Establish city-wide biomonitoring programs to track biochemical markers of environmental stress in residents, enabling early detection of exposure-related health risks.
5. **Awareness and Community Engagement**: Conduct public awareness campaigns regarding air pollution health risks, preventive measures, and protective behaviors, particularly targeting schools, workplaces, and residential communities.
6. **Seasonal Intervention Strategies**: Implement targeted measures during high-risk periods, such as winter, when pollutant concentrations peak and biomarker alterations are most pronounced.

Conflict of interest

The authors declare no conflict of interest

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