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Smog and Dry Eye Disease: A Comprehensive Review (2019–2024)

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ABSTRACT

Background: *Smog, a complex mixture of particulate and gaseous pollutants, has become an increasingly prevalent environmental hazard due to industrialization, vehicular emissions, and urban expansion. While its respiratory and cardiovascular effects are well recognized, recent research highlights its emerging impact on ocular surface integrity and tear film stability. The eye, continuously exposed to environmental factors, is highly susceptible to the oxidative and inflammatory stress induced by airborne pollutants, positioning smog as a significant but modifiable risk factor for ocular disease.* ***Objective****: This review consolidates recent clinical, epidemiological, and experimental evidence (2019–2024) regarding the association between smog-related air pollution and dry eye disease (DED), with emphasis on pathophysiological mechanisms, environmental modifiers, and potential preventive approaches.* ***Methods****: A comprehensive literature review was performed using studies published between 2019 and 2024 that evaluated particulate matter ($PM_{2.5}$, PM_{10}) and gaseous pollutants (O_3, NO_2, SO_2, CO) in relation to ocular surface inflammation, tear film disruption, and meibomian gland function. Both human and experimental models were analyzed.* ***Results****: Recent multicenter and mechanistic studies consistently demonstrate that pollutant exposure leads to oxidative stress, mitochondrial dysfunction, cytokine activation (IL-6, IL-8, TNF-α), and tear film instability. Epidemiological data confirm higher DED incidence and symptom severity during high-smog episodes, often exacerbated by low humidity and temperature extremes.* ***Conclusion****: Smog-related air pollution contributes to DED through oxidative, inflammatory, and evaporative mechanisms. Standardized exposure metrics, longitudinal monitoring, and interventional trials are essential to mitigate this preventable cause of ocular morbidity.*

Keywords
Smog, Air Pollution, Dry Eye Disease, Ocular Surface, Particulate Matter, Inflammation, Oxidative Stress

INTRODUCTION

Smog represents a complex mixture of fog, smoke, and airborne pollutants that collectively form one of the most detrimental forms of urban air pollution. Over the past decade, its frequency and intensity have increased significantly, driven by accelerated industrialization, expanding vehicular traffic, and uncontrolled urbanization—often accompanied by large-scale deforestation and poor environmental regulation (1). The primary constituents of smog include particulate matter ($PM_{2.5}$ and PM_{10}) and gaseous pollutants such as nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon monoxide (CO), and ozone (O_3), along with various organic compounds, heavy metals, and allergens derived from combustion and industrial processes (2,3). These pollutants interact with atmospheric moisture to form dense, persistent layers of haze that reduce visibility and contribute to serious public-health crises during high-pollution seasons.

Although the respiratory and cardiovascular consequences of air pollution are well established, there is growing recognition of its adverse effects on ocular health. The human eye, constantly exposed to the external environment and lacking a keratinized protective barrier, is particularly vulnerable to the oxidative, inflammatory, and desiccating effects of airborne pollutants (4,5). Chronic exposure to smog and other air-quality hazards has been linked to ocular surface disorders including allergic conjunctivitis, pterygium, cataract formation, and most notably, dry eye disease (DED)—a multifactorial condition characterized by tear-film instability, hyperosmolarity, and ocular surface inflammation (6,7). These changes not only impair visual function but also reduce quality of life, productivity, and mental well-being, particularly in urban populations exposed to persistent environmental stressors.

Recent clinical, experimental, and epidemiological studies have highlighted the direct association between air-pollutant exposure and the incidence or exacerbation of DED. Mechanistic data suggest that particulate and gaseous pollutants induce oxidative stress, mitochondrial dysfunction, and inflammatory cytokine up-regulation within corneal and conjunctival epithelial cells, leading to epithelial barrier disruption, goblet-cell loss, and meibomian-gland dysfunction (8–10). Moreover, meteorological factors such as humidity, ambient temperature, and wind velocity appear to modulate pollutant toxicity and influence tear-film dynamics (11). These interactions underscore the complex environmental etiology of DED, in which smog-related exposure constitutes a modifiable risk factor of increasing global concern.

The present review aims to consolidate peer-reviewed evidence published between 2019 and 2024 concerning the relationship between smog-related air pollution and dry eye disease. It synthesizes findings from clinical, laboratory, and population-based research to elucidate the mechanistic pathways, epidemiological associations, and potential preventive strategies that link atmospheric pollution to ocular-surface pathology. By critically integrating recent insights, this review seeks to provide clinicians, researchers, and policymakers with an updated understanding of how urban air quality directly impacts ocular health and to highlight future directions for research and environmental intervention.

IMPACT OF SMOG ON HEALTH

Smog exerts a broad range of pathophysiological effects on the human body, primarily targeting the respiratory, cardiovascular, and immune systems. The fine and ultrafine particles that constitute smog—especially particulate matter less than 2.5 micrometers in diameter ($PM_{2.5}$)—are capable of penetrating deep into the bronchioles and alveoli, triggering local oxidative stress, epithelial injury, and systemic inflammation (12). Larger particles (PM_{10}) tend to deposit in the upper respiratory tract, causing irritation and allergic sensitization of the nasal and pharyngeal mucosa (13). The chemical constituents of smog, such as hydrocarbons, heavy metals, and polycyclic aromatic hydrocarbons (PAHs), further exacerbate tissue toxicity by generating reactive oxygen species (ROS) and impairing the antioxidant defense system (14).

These pollutants influence multiple organ systems beyond the lungs. The cardiovascular consequences include endothelial dysfunction, increased blood viscosity, and arrhythmogenic effects associated with autonomic imbalance and altered myocardial electrophysiology (15). Prolonged exposure to high pollutant levels has been correlated with elevated risks of ischemic heart disease, cerebrovascular accidents, and hypertension, highlighting the systemic impact of chronic smog exposure (16). Moreover, epidemiological evidence suggests that persistent exposure to NO_2 and sulfur compounds increases the risk of pulmonary malignancies, with mechanisms resembling those of tobacco-related carcinogenesis (17,18). The similarity in chemical composition between tobacco smoke and ambient air pollutants underscores their shared pathogenic potential through oxidative DNA damage and impaired immune surveillance (19).

Individuals with preexisting respiratory conditions such as asthma, chronic obstructive pulmonary disease (COPD), and previous tuberculosis infection are particularly vulnerable to smog exposure, as their impaired mucociliary clearance and compromised immune function amplify pollutant retention and inflammation (20,21). The cumulative impact of environmental exposure can lead to accelerated decline in pulmonary function, higher hospitalization rates, and increased mortality during high-smog episodes (22).

The immunological consequences of smog are equally profound. Exposure to airborne particulates induces systemic inflammation mediated by cytokines such as interleukin (IL)-6, tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP), contributing to a pro-inflammatory milieu implicated in chronic disease progression (23). This persistent low-grade inflammation may also influence extra-respiratory organs, including ocular tissues, by altering epithelial permeability and promoting vascular endothelial activation. Such systemic effects provide a mechanistic link between environmental air quality and the onset or exacerbation of ocular surface disorders like dry eye disease, discussed in the subsequent section.

In summary, smog acts as a multisystem toxicant whose effects extend far beyond the pulmonary system. Its fine particulate components, reactive gases, and secondary aerosols initiate oxidative, inflammatory, and immunologic cascades that not only threaten cardiopulmonary health but also predispose exposed populations to ocular morbidity. Understanding these mechanisms provides the foundation for exploring smog's specific influence on the eye and underscores the importance of integrated environmental health strategies.

IMPACT OF SMOG ON EYE HEALTH

Among the organs most vulnerable to environmental stressors, the human eye occupies a uniquely exposed position. The ocular surface—comprising the cornea, conjunctiva, tear film, and associated glands—is continuously in contact with the atmosphere and thus directly subjected to airborne pollutants. Recent epidemiological, clinical, and experimental evidence demonstrates that exposure to smog and air pollutants is a significant and modifiable risk factor for dry eye disease (DED), a multifactorial condition characterized by tear-film instability, hyperosmolarity, inflammation, and damage to the ocular surface (24,25).

EPIDEMIOLOGICAL AND CLINICAL EVIDENCE

A growing body of population-based and hospital-based studies from 2019–2024 has confirmed that both short- and long-term exposure to particulate ($PM_{2.5}$, PM_{10}) and gaseous pollutants (NO_2, SO_2, O_3, CO) significantly correlates with increased DED incidence and symptom severity. Investigations conducted in China, Taiwan, and other high-density urban centers consistently report that increased ambient concentrations of $PM_{2.5}$ and NO_2 are associated with elevated scores on the Ocular Surface Disease Index (OSDI), reduced Tear Break-Up Time (TBUT), and higher rates of meibomian gland dysfunction (MGD) (26–28). These findings are supported by time-series analyses showing a rise in outpatient visits for DED during pollution surges, confirming that smog-related exposure elicits acute symptomatic flares in susceptible individuals (29).

Longitudinal and multicenter studies further demonstrate that pollutant-induced ocular changes are not confined to transient irritation. Instead, they reflect chronic inflammatory processes that compromise tear secretion, lipid-layer stability, and corneal epithelial integrity. Elevated tear cytokine levels—particularly interleukin-6 (IL-6), interleukin-8 (IL-8), and tumor necrosis factor-alpha (TNF-α)—have been documented in residents of high-pollution zones, suggesting sustained activation of ocular innate immunity (30). These data emphasize that smog exposure influences not only the aqueous and mucin layers of the tear film but also the lipid layer through inflammatory meibomian gland obstruction, contributing to persistent ocular discomfort and visual disturbance.

EXPERIMENTAL AND MECHANISTIC INSIGHTS

Laboratory models provide compelling mechanistic support for these clinical observations. Animal and cell-based experiments have shown that particulate matter (particularly $PM_{2.5}$) induces corneal epithelial injury, mitochondrial dysfunction, and oxidative stress, culminating in apoptosis and goblet-cell loss (31). Mice exposed to $PM_{2.5}$ aerosols exhibit decreased TBUT, reduced tear secretion, and increased corneal fluorescein staining—findings that mirror human DED pathology (32). Mechanistically, reactive oxygen species (ROS) generated by pollutants activate nuclear factor-kappa B (NF-κB) signaling, promoting pro-inflammatory cytokine release and epithelial barrier disruption (33). These pathways converge to produce chronic ocular surface inflammation and microstructural changes resembling those observed in meibomian gland dysfunction.

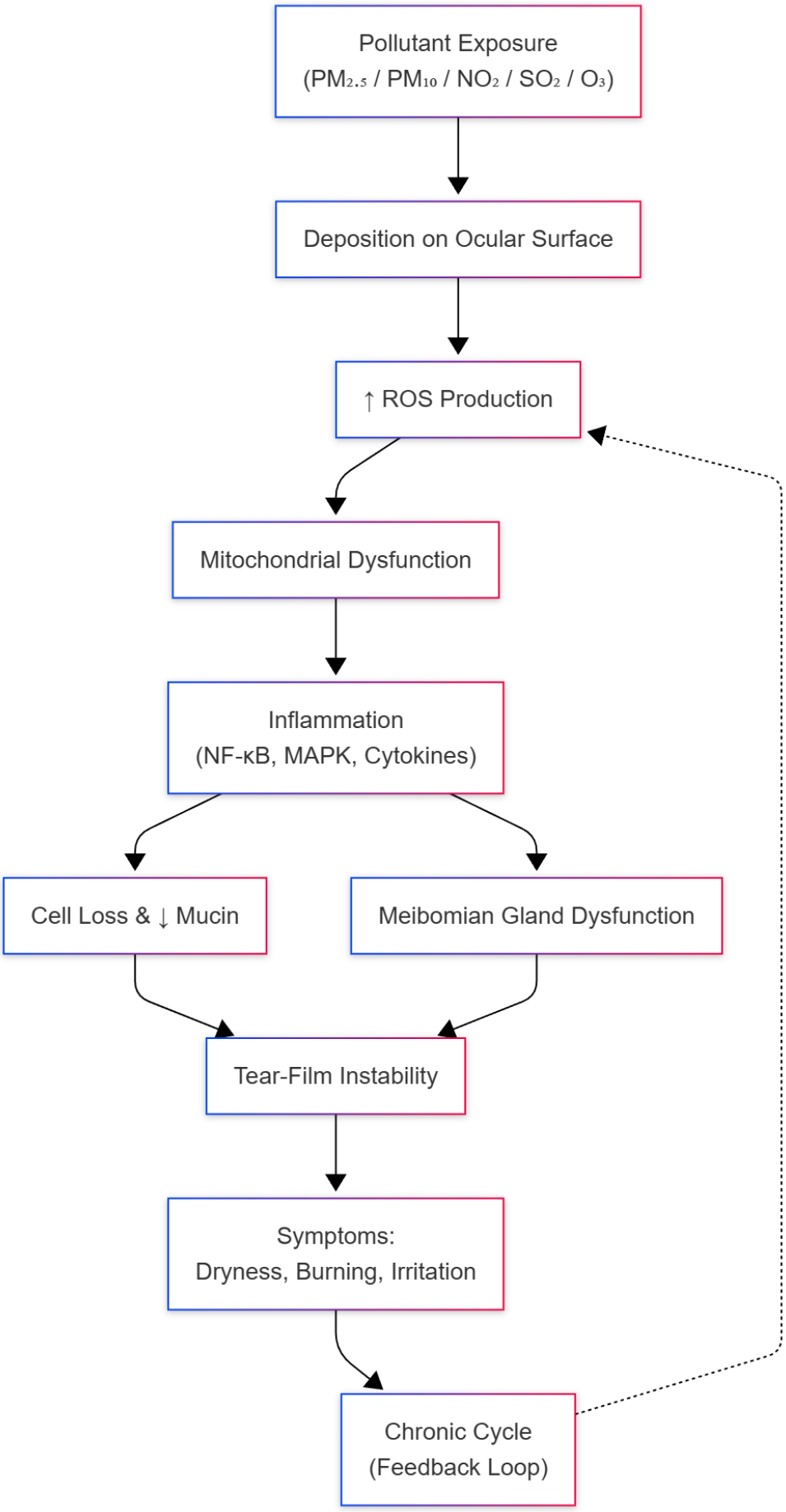

Figure 1 Flowchart of Mechanistic Insights

The synergistic effects of meteorological factors further modulate pollutant toxicity. Low ambient humidity, high temperature, and strong winds accelerate tear-film evaporation and exacerbate pollutant-induced desiccation stress (34). Conversely, temperature inversions during winter smog

episodes increase pollutant concentration near the surface, amplifying ocular exposure and inflammatory load. The cumulative outcome of these interactions is a cycle of oxidative stress, inflammation, and tear-film instability, establishing smog as a pivotal environmental determinant of ocular surface disease.

Summary of Key Recent Studies (2019–2024)

Year	Study / Title	Key Findings	Pollutants / Exposure	Ocular Parameters Affected
2023	Impact of Air Pollution on the Ocular Surface and Tear Cytokine Levels: A Multicenter Prospective Cohort Study	Elevated $PM_{2.5}$, O_3, NO_2, and SO_2 associated with higher OSDI, decreased TBUT, increased corneal staining, MGD, and elevated tear cytokines (IL-6, IL-8).	$PM_{2.5}$, PM_{10}, O_3, NO_2, SO_2	OSDI, TBUT, Schirmer's, corneal fluorescein staining (CFS), cytokine profile
2023	$PM_{2.5}$ Exposure Increases Dry Eye Disease Risks through Corneal Epithelial Inflammation and Mitochondrial Dysfunctions	Demonstrated that $PM_{2.5}$ causes mitochondrial injury and corneal epithelial inflammation in animal and cellular models.	$PM_{2.5}$ aerosol exposure	Corneal epithelial damage, mitochondrial dysfunction
2023	Association Between Air Pollution Exposure and Daily Outpatient Visits for DED: A Time-Series Study in Urumqi, China	Reported increased outpatient visits during high-pollution days, confirming acute symptomatic effects of smog exposure.	$PM_{2.5}$, PM_{10}	DED outpatient visit rates
2022–2024	Adverse Effects of Meteorological Factors and Air Pollutants on Dry Eye Disease: A Hospital-Based Retrospective Cohort Study	Found combined effects of pollutants and weather parameters (humidity, temperature) on tear secretion, osmolarity, and symptom severity.	$PM_{2.5}$, O_3, NO_2, CO	TBUT, osmolarity, SANDE score
2022	Association Between Ambient Air Pollution and Dry Eye Symptoms During the COVID-19 Pandemic	Survey data showed higher symptom prevalence and severity among residents of industrial regions during pandemic lockdowns.	$PM_{2.5}$, PM_{10}, SO_2, NO_2, O_3	OSDI-6, symptom severity
2019	Air Pollutant $PM_{2.5}$ Induces Dry Eye Syndrome in Mice	Demonstrated tear-film reduction, goblet-cell loss, and inflammatory activation (TNF-α, NF-κB).	$PM_{2.5}$ experimental exposure	TBUT, tear volume, epithelial histopathology

Collectively, findings from clinical, animal, and mechanistic studies converge to establish a causal link between air pollution and DED pathophysiology. Pollutant-induced oxidative stress and inflammation not only disrupt the tear film but also compromise epithelial integrity and meibomian gland function. These effects are further intensified by meteorological variables, creating a multifactorial interplay between environmental and host factors. Importantly, these studies identify smog exposure as a modifiable environmental determinant of ocular surface disease, offering opportunities for preventive strategies through environmental regulation, patient education, and ocular surface protection.

DISCUSSION AND MECHANISTIC SYNTHESIS

The collective body of evidence from 2019 to 2024 strongly supports a causal association between smog-related air pollution and the pathogenesis of dry eye disease (DED). Clinical and experimental studies converge on a shared pathophysiological framework in which chronic exposure to particulate and gaseous pollutants provokes oxidative stress, inflammatory signaling, and structural disruption of the ocular surface. This integrated mechanism explains the epidemiological trends observed in populations residing in high-pollution regions and underscores the biological plausibility of air pollution as a modifiable determinant of ocular morbidity.

At the molecular level, particulate matter—particularly $PM_{2.5}$—has been identified as a critical driver of oxidative stress and mitochondrial dysfunction within corneal and conjunctival epithelial cells (35). The generation of reactive oxygen species (ROS) leads to lipid peroxidation, protein oxidation, and DNA damage, which in turn activate nuclear factor kappa B (NF-κB) and mitogen-activated protein kinase (MAPK) signaling pathways (36). These pathways up-regulate pro-inflammatory cytokines such as interleukin-6 (IL-6), interleukin-8 (IL-8), and tumor necrosis factor-alpha (TNF-α), contributing to chronic subclinical inflammation that compromises tear-film stability and epithelial integrity. Experimental mouse models have replicated these findings, showing increased corneal fluorescein staining, goblet-cell loss, and decreased tear secretion following exposure to $PM_{2.5}$ aerosols (37).

Beyond oxidative injury, evidence suggests that pollutant-induced inflammation extends to the meibomian glands, disrupting lipid secretion and increasing tear-film evaporation (38). The resulting lipid-layer deficiency accelerates ocular surface desiccation, amplifying hyperosmolar stress and epithelial apoptosis. This sequence of events establishes a self-perpetuating cycle of inflammation and tear-film instability, characteristic of chronic DED. Clinical data corroborate these mechanisms; multicenter cohort studies have reported elevated tear cytokine concentrations and reduced meibomian gland expressibility among individuals with long-term exposure to urban air pollution (39). Such findings affirm the link between environmental exposure and meibomian gland dysfunction (MGD)—a major pathogenic contributor to evaporative dry eye.

Meteorological factors further modulate these pathological effects. High ambient temperatures and low humidity exacerbate tear evaporation, while temperature inversions and stagnant air during winter months intensify pollutant accumulation near ground level (40). This synergistic interaction between weather and pollution magnifies ocular exposure, explaining seasonal spikes in DED prevalence during smog episodes. Additionally, behavioral adaptations such as reduced outdoor activity and increased screen time during poor air-quality periods may indirectly worsen ocular dryness through decreased blink rate and prolonged visual strain (41).

From a public-health standpoint, the evidence underscores the need to recognize air pollution as a preventable environmental determinant of ocular disease. The ocular surface serves as both a sentinel and a target organ for environmental exposure, reflecting broader systemic inflammation

triggered by poor air quality. Given the growing urbanization and industrialization in low- and middle-income countries, the burden of DED linked to smog exposure is expected to rise substantially in coming decades unless effective environmental and clinical interventions are implemented.

Future studies should therefore emphasize longitudinal, multicenter, and mechanistic approaches. Standardized exposure assessments using personal air-quality monitors and satellite-based pollutant mapping could improve exposure quantification, while biomarker profiling (e.g., oxidative stress markers, tear cytokines, mitochondrial assays) would clarify individual susceptibility patterns. Furthermore, interventional trials evaluating the efficacy of protective measures—such as antioxidant-based artificial tears, humidified indoor environments, and barrier eyewear—are essential to translate these mechanistic insights into clinical prevention.

In summary, smog-related air pollution contributes to DED through interconnected mechanisms of oxidative stress, inflammation, and tear-film dysfunction. These findings not only reinforce the ocular surface's sensitivity to environmental stressors but also highlight air quality as a critical yet modifiable component of eye health policy. Addressing smog exposure through multidisciplinary collaboration among ophthalmologists, environmental scientists, and policymakers will be vital in mitigating the escalating public-health impact of pollution-induced ocular disease.

LIMITATIONS AND RECOMMENDATIONS

Despite substantial progress in understanding the relationship between air pollution and dry eye disease (DED), several methodological and conceptual limitations persist across current literature. The majority of available studies employ cross-sectional or short-term observational designs, limiting causal inference between pollutant exposure and DED onset or progression (42). The scarcity of longitudinal cohort data hinders the evaluation of temporal relationships and cumulative exposure effects, while confounding factors—such as indoor air quality, occupational exposure, screen time, and use of protective measures—are often underreported or insufficiently controlled (43).

Another critical limitation lies in exposure assessment methodology. Most studies rely on regional air-monitoring data or governmental air-quality indices, which may not accurately represent individual exposure variability. Such indirect estimation introduces exposure misclassification, potentially diluting true associations between pollutant levels and ocular surface outcomes (44). Future research must incorporate personal exposure monitoring through wearable sensors and satellite-based modeling to achieve higher spatial and temporal precision in exposure measurement.

Heterogeneity in diagnostic criteria and outcome measures represents a further barrier to data synthesis. Variations in DED assessment—ranging from questionnaire-based symptom evaluation to diverse objective tests (OSDI, TBUT, Schirmer's, osmolarity)—complicate cross-study comparison and meta-analytic integration (45). To enhance reproducibility and comparability, the field urgently requires standardization of ocular surface diagnostic protocols and consistent application of validated tools across clinical and epidemiological research.

Experimental models have provided valuable mechanistic insights but often fail to mimic chronic, low-dose human exposures or the multifactorial nature of environmental DED. Most laboratory studies employ acute or high-intensity pollutant exposure paradigms, which, while elucidating mechanistic pathways, may overestimate physiological relevance (46). Translational models incorporating long-term exposure to mixed pollutants under variable climatic conditions would bridge this gap and strengthen external validity.

From a clinical and public-health perspective, the evidence base underscores the need for preventive and interventional strategies targeting both individual and environmental determinants. Prospective interventional trials should examine the efficacy of protective eyewear, indoor humidification, antioxidant-enriched artificial tears, and lifestyle modifications to mitigate pollutant-induced ocular damage (47). Parallel to clinical strategies, policy-level interventions—including stricter emission regulations, promotion of green urban infrastructure, and improved public awareness—are imperative to reduce population-level exposure. Integrating ocular health metrics into existing air-quality monitoring frameworks could provide a novel early-warning indicator for environmental stress and population well-being (48).

In conclusion, while mounting evidence implicates smog-related air pollution as a modifiable risk factor for DED, future research must progress toward multidimensional, standardized, and longitudinal paradigms. A unified approach that combines advanced exposure modeling, molecular biomarker profiling, and real-world clinical assessment will not only clarify causal mechanisms but also inform both preventive ophthalmology and environmental health policy on a global scale.

CONCLUSION

Emerging evidence from 2019 to 2024 unequivocally establishes smog-related air pollution as a critical and modifiable determinant of dry eye disease (DED). The convergence of epidemiological, clinical, and experimental findings demonstrates that chronic exposure to particulate and gaseous pollutants precipitates ocular surface inflammation, tear-film instability, and glandular dysfunction through oxidative and immune-mediated pathways. These effects are compounded by environmental and behavioral modifiers such as humidity, temperature, and visual screen exposure, amplifying the burden of ocular morbidity in polluted urban environments.

The ocular surface, continuously exposed to external stressors, serves not only as a sensitive biomarker of environmental health but also as an early sentinel of systemic oxidative injury. Recognizing DED within the broader context of environmental pathophysiology reframes it from a local ophthalmic condition to a global public-health concern. Mitigation requires an integrated approach combining clinical prevention, technological innovation, and environmental policy—including improved emission controls, public awareness campaigns, and urban air-quality monitoring with ocular health indicators.

Future directions should emphasize longitudinal exposure studies, standardized diagnostic frameworks, and translational research linking molecular biomarkers to real-world clinical outcomes. Such efforts will facilitate evidence-based interventions that protect visual health while advancing environmental justice and sustainability. In essence, combating smog-induced DED demands collaboration across ophthalmology, environmental science, and policy domains—reflecting the inseparable connection between planetary health and human vision.

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