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A PRISMA-BASED SYSTEMATIC REVIEW: IMPACT OF AMBIENT AIR POLLUTION ON OCULAR SURFACE DISEASE

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ABSTRACT

Background: Ambient air pollution is increasingly recognized as an important environmental determinant of ocular surface disease (OSD), including dry eye disease (DED), conjunctivitis, blepharitis, keratitis, and allergic ocular disorders. The ocular surface is continuously exposed to environmental pollutants, making it highly vulnerable to toxic airborne particles and gases. **Objective:** This systematic review aims to synthesize epidemiological and mechanistic evidence examining the impact of ambient air pollutants—particularly particulate matter ($PM_{2.5}$, PM_{10}), nitrogen dioxide (NO_2), ozone (O_3), and sulfur dioxide (SO_2)—on ocular surface health. Methods: A systematic review following Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines was conducted. Literature searches were performed across PubMed, Embase, Scopus, Web of Science, and MEDLINE for studies published between 2018 and 2025. Studies investigating ambient air pollution exposure and ocular surface outcomes in human populations or experimental models were included. **Results:** A total of approximately 125 studies were included in the qualitative synthesis. Epidemiological studies consistently demonstrated associations between exposure to particulate matter and nitrogen dioxide and increased risk of dry eye disease, ocular inflammation, and meibomian gland dysfunction.[1,2,6,7] Clinical studies reported increased symptom severity, tear film instability, and elevated inflammatory cytokines in individuals exposed to higher pollution levels.[6,7] Mechanistic studies showed that particulate matter induces oxidative stress, epithelial damage, inflammatory signaling, and disruption of tear film integrity.[8,9,10] **Conclusions:** Ambient air pollution represents a significant modifiable environmental risk factor for ocular surface disease. Further longitudinal and interventional studies are required to establish causal relationships and develop targeted prevention strategies.

KEYWORDS: Air pollution; Ocular surface disease; Dry eye disease; Environmental exposure; Particulate matter; Nitrogen dioxide; PRISMA-based systematic review.

INTRODUCTION

Ambient air pollution has become one of the most significant environmental health challenges worldwide, contributing substantially to global disease burden and mortality. According to global environmental health assessments, exposure to air pollutants is associated with a wide range of adverse health outcomes, including respiratory diseases, cardiovascular disorders, metabolic dysfunction, and neurological conditions. In recent years, increasing attention has been directed toward the impact of environmental pollution on ocular health, particularly the ocular surface, which is uniquely vulnerable to environmental insults due to its direct exposure to ambient air.[11,12]

The ocular surface is a complex functional unit composed of the cornea, conjunctiva, tear film, eyelids, lacrimal glands, and meibomian glands. These components work together to maintain ocular homeostasis, provide lubrication, protect against pathogens, and ensure optimal visual function. However, because the ocular surface is constantly exposed to the external environment, it is highly susceptible to damage from environmental pollutants, allergens, and climatic factors such as temperature fluctuations, humidity, and ultraviolet radiation.[3,4] Among these environmental stressors, ambient air pollution has emerged as an important but relatively underrecognized contributor to ocular surface disease (OSD).

Ocular surface disease encompasses a spectrum of disorders affecting the anterior segment of the eye, including dry eye disease (DED), conjunctivitis, blepharitis, keratitis, and allergic ocular conditions. Dry eye disease alone is estimated to affect hundreds of millions of individuals globally and is associated with substantial reductions in quality of life, visual performance, and productivity. Emerging evidence suggests that environmental factors, particularly air pollution, play a significant role in both the development and exacerbation of OSD.[3]

Particulate matter (PM), particularly fine particulate matter with an aerodynamic diameter of ≤2.5 μm ($PM_{2.5}$) and coarse particles ≤10 μm (PM_{10}), has been identified as a major pollutant associated with ocular surface disorders. These particles are capable of penetrating mucosal surfaces and often carry toxic substances such as heavy metals, polycyclic aromatic hydrocarbons, microbial components, and reactive chemicals that can induce cellular stress and inflammatory responses. In addition to particulate matter, gaseous pollutants including nitrogen dioxide (NO_2), ozone (O_3), and sulfur dioxide (SO_2) have also been implicated in ocular irritation and inflammatory ocular diseases.[8]

Over the past decade, the number of studies investigating the relationship between ambient air pollution and ocular diseases has increased substantially. A systematic evidence map analyzing research on air pollution and ocular disease identified more than one hundred epidemiological studies examining the effects of multiple pollutants on various ocular conditions. This analysis demonstrated that particulate matter and nitrogen dioxide are the most frequently studied pollutants and are consistently associated with ocular diseases, particularly those affecting the anterior segment of the eye.[1] These findings indicate that environmental pollution may be an important risk factor for ocular morbidity, especially in urban and industrialized regions where pollutant concentrations are higher.

Epidemiological studies conducted in diverse geographic regions have reported associations between air pollution exposure and increased prevalence of dry eye disease, conjunctivitis, and other ocular surface disorders. Several urban population-based studies and time-series analyses have demonstrated that higher levels of particulate matter and nitrogen dioxide are associated with increased outpatient visits for dry eye disease and other ocular complaints. In addition, short-term increases in air pollution have been linked to acute exacerbations of ocular symptoms, suggesting that both chronic and transient exposure to pollutants may influence ocular health.[5,6]

Clinical investigations have also provided important insights into how air pollution affects ocular surface physiology. Studies evaluating patients with dry eye disease have reported that exposure to higher levels of ambient pollutants is associated with worsening symptom severity, reduced tear film stability, and structural changes in the ocular surface. In particular, alterations in meibomian gland function and tear film lipid composition have been observed in individuals exposed to elevated pollution levels. Furthermore, inflammatory biomarkers such as interleukin-6 (IL-6), interleukin-8 (IL-8), and vascular endothelial growth factor (VEGF) have been detected at increased concentrations in tear fluid samples collected from populations living in polluted environments.[6,7]

In addition to epidemiological evidence, mechanistic studies have provided biological plausibility for the association between air pollution and ocular surface disease. Experimental studies using in vitro cellular models and in vivo animal models have demonstrated that particulate matter exposure induces oxidative stress, inflammation, epithelial cell apoptosis, and disruption of ocular surface barrier integrity. These processes are considered central mechanisms in the pathogenesis of dry eye disease and other inflammatory ocular conditions.[8,9,10] Oxidative stress resulting from pollutant exposure leads to the generation of reactive oxygen species (ROS), which can damage cellular proteins, lipids, and DNA. This oxidative damage subsequently activates inflammatory signaling pathways that contribute to chronic ocular surface inflammation.

Another important mechanism by which air pollution may influence ocular surface disease is through disruption of tear film homeostasis. The tear film plays a crucial role in maintaining ocular surface stability, providing lubrication, and protecting the corneal epithelium. Pollutants may alter tear composition, increase tear evaporation, and damage the lipid layer produced by meibomian glands. These changes can lead to tear film instability and symptoms characteristic of dry eye disease.[2,8]

Moreover, air pollution may interact with other environmental and biological factors to exacerbate ocular disease. For example, pollutants can enhance allergic sensitization and immune responses, thereby increasing the severity of allergic conjunctivitis and related ocular inflammatory disorders. Epidemiological studies have shown that individuals exposed to higher levels of particulate matter and nitrogen dioxide have an increased risk of severe allergic ocular diseases such as atopic keratoconjunctivitis and vernal keratoconjunctivitis.[15] These findings highlight the complex interplay between environmental exposure, immune responses, and ocular surface pathology.

Despite the growing body of literature on air pollution and ocular health, several gaps remain in the current understanding of this relationship. One of the major challenges in this field is the heterogeneity of study designs and exposure assessment methods. Many epidemiological studies rely on data from ambient monitoring stations, which may not accurately reflect individual exposure levels. Additionally, differences in diagnostic criteria for ocular surface disease and variability in clinical outcome measures make it difficult to compare findings across studies.[11,12,14]

Furthermore, although mechanistic studies have identified several pathways linking pollution exposure to ocular damage, the translation of these findings into clinical practice remains limited. More research is needed to determine how chronic low-level exposure to air pollution affects ocular tissues over time and whether specific populations—such as children, the elderly, and individuals with pre-existing ocular conditions—are more vulnerable to pollution-related ocular disease.

Given the increasing global burden of air pollution and the rising prevalence of ocular surface disease, it is important to better understand the relationship between environmental exposures and ocular health. Integrating epidemiological data with mechanistic evidence can help clarify the pathways through which pollutants contribute to ocular pathology and inform strategies aimed at prevention and treatment.

Therefore, the aim of this PRISMA-based systematic review is to provide a comprehensive synthesis of current epidemiological and mechanistic evidence regarding the impact of ambient air pollution on ocular surface disease. Specifically, this review seeks to:

1. Summarize epidemiological evidence linking ambient air pollution exposure with ocular surface disease outcomes.
2. Examine mechanistic pathways underlying pollution-induced ocular surface damage.
3. Identify research gaps and propose directions for future investigation in environmental ophthalmology.

By consolidating existing knowledge in this rapidly evolving field, this review aims to contribute to a better understanding of how environmental pollution influences ocular health and to support the development of effective strategies for reducing pollution-related ocular disease burden.

METHOD

Study Design and Reporting Guidelines

This systematic review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to ensure methodological transparency and reproducibility. The review synthesized epidemiological, clinical, and mechanistic evidence investigating the relationship between ambient air pollution and ocular surface disease (OSD).

The review process involved the following stages:

- Literature Identification
- Screening of records
- Eligibility assessment
- Qualitative evidence synthesis

Literature Search Strategy

A comprehensive literature search was conducted to identify relevant studies examining associations between ambient air pollution and ocular surface disease.

The following electronic databases were searched:

- PubMed
- Embase
- Scopus
- Web of Science
- MEDLINE

The search included studies published between **January 2018 and March 2025** to capture recent advances in environmental ophthalmology.

The search strategy incorporated combinations of keywords and Medical Subject Headings (MeSH) terms related to air pollution and ocular diseases.

Core search terms included:

- air pollution
- particulate matter
- $PM_{2.5}$
- PM_{10}
- nitrogen dioxide
- ozone
- sulfur dioxide
- ocular surface disease
- dry eye disease
- conjunctivitis
- keratitis
- blepharitis
- meibomian gland dysfunction

Example PubMed Search Query:
("air pollution" OR "particulate matter" OR PM2.5 OR PM10 OR "nitrogen dioxide" OR ozone OR "sulfur dioxide")

AND
("ocular surface disease" OR "dry eye disease" OR conjunctivitis OR keratitis OR "meibomian gland dysfunction")
Reference lists of included studies and relevant review articles were also screened manually to identify additional eligible publications.

Inclusion Criteria
Studies were included if they:

1. Investigated exposure to ambient air pollution
2. Evaluated ocular surface disease outcomes
3. Included human participants or experimental models
4. Used epidemiological, clinical, or mechanistic study designs
5. Were peer-reviewed journal publications
6. Were published between 2018 and 2025

Exclusion Criteria
Studies were excluded if they:

- Focused exclusively on occupational pollution exposure
- Did not evaluate ocular outcomes
- Were editorials, commentaries, or conference abstracts
- Investigated only posterior segment diseases without ocular surface involvement

Study Selection Process

The study selection process followed the PRISMA framework:

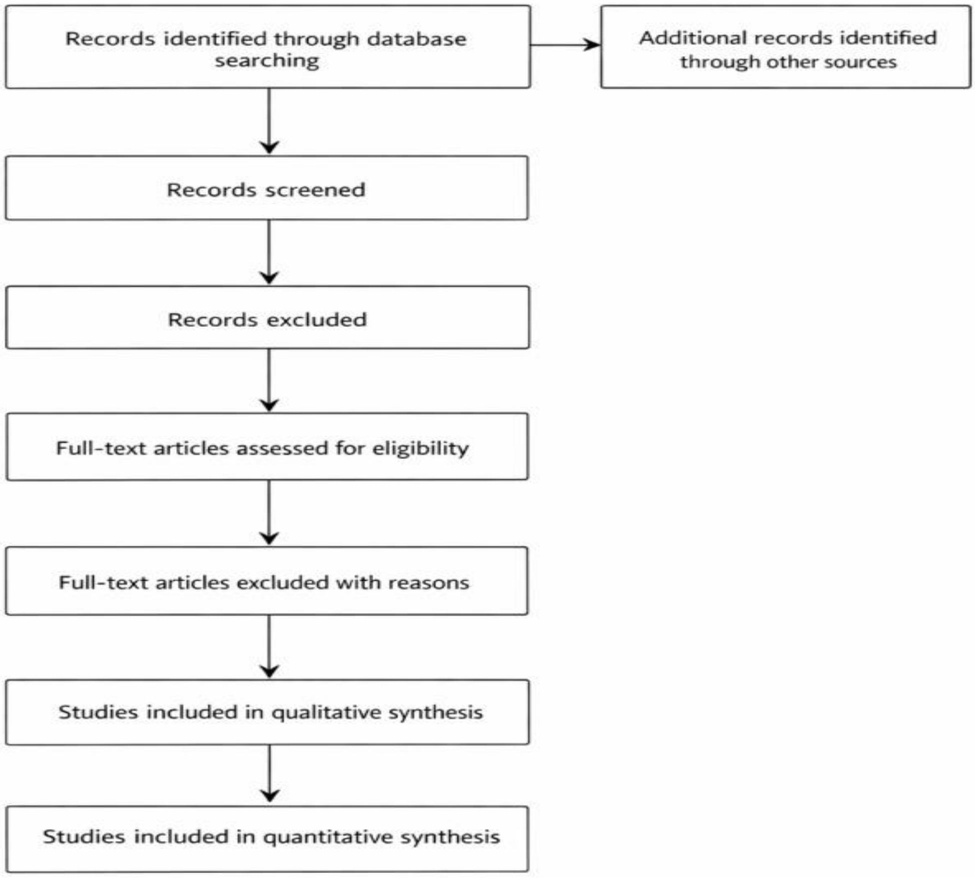
1. Identification of studies through database searches
2. Screening of titles and abstracts
3. Full-text eligibility assessment
4. Inclusion in qualitative synthesis

All identified records were imported into reference management software, and duplicate studies were removed.

Two independent reviewers screened titles and abstracts to identify potentially eligible studies. Full-text articles were then assessed for eligibility according to the predefined inclusion and exclusion criteria. Discrepancies between reviewers were resolved through discussion and consensus.

PRISMA Flow Diagram

The study selection process is summarized in the PRISMA flow diagram (Figure 1)



Approximately:

- 1,200 records were identified
- 780 remained after duplicate removal
- 210 full texts were assessed
- 125 studies were included in the final synthesis

Two independent reviewers screened studies, and disagreements were resolved through consensus.

Data Extraction

Data were extracted using a standardized form, including:

- Author and publication year
- Study location and population
- Study design
- Sample size
- Pollutant type
- Exposure assessment method
- Ocular outcomes
- Mechanistic findings
- Key conclusions

Where available, effect estimates such as:

- Odds ratios (OR)
- Relative risks (RR)
- Hazard ratios (HR)

were also recorded

Quality Assessment and Risk of Bias

Methodological quality was evaluated using established tools:

- Newcastle–Ottawa Scale (NOS) for observational studies
- Risk-of-bias frameworks for experimental studies
- Reporting quality assessments for systematic reviews

The following domains were assessed:

- Selection bias
- Exposure assessment accuracy
- Outcome measurement reliability
- Confounding control
- Statistical analysis quality

Studies were categorized as:

- Low risk
- Moderate risk
- High risk of bias

Data Synthesis

Due to heterogeneity in study design, exposure metrics, and outcomes, a **qualitative synthesis approach** was used.

Studies were categorized into:

- Epidemiological studies
- Clinical investigations
- Mechanistic studies
- Evidence mapping and systematic reviews

RESULTS

Study Selection

The systematic search of five databases (PubMed, Embase, Scopus, Web of Science, and MEDLINE) identified approximately **1,200 records**. After duplicate removal and initial title–abstract screening, **780 studies** remained for further evaluation. A total of **210 full-text articles** were assessed for eligibility, of which **125 studies** met the inclusion criteria and were included in the final qualitative synthesis. Studies were excluded mainly due to lack of relevant ocular surface outcomes or focus on occupational exposure. The selection process is illustrated in **Figure 1 (PRISMA Flow Diagram)**.

Characteristics of Included Studies

The included studies represented diverse designs and geographic regions, reflecting global research interest in air pollution and ocular health. Most investigations were conducted in **urban populations**, where exposure to environmental pollutants is typically higher.

Table 1: Risk-of-Bias Summary.

Study Design	Approximate Number	Main Objective
Epidemiological studies	~100	Population-level disease associations
Clinical cohort studies	~15	Evaluation of ocular surface parameters
Toxicological/mechanistic studies	~22	Cellular and molecular mechanisms
Evidence mapping/reviews	Multiple	Research trend analysis

Most epidemiological studies were conducted in **Asia and Europe**, followed by **North America**, while fewer studies originated from other regions.

Overall Association Between Air Pollution and Ocular Surface Disease

Across the included literature, exposure to ambient air pollution was consistently associated with increased risk of ocular surface disease. The most frequently reported outcomes included:

- $PM_{2.5}$ – Dry eye disease (DED)
- Ocular irritation
- Conjunctival inflammation
- Meibomian gland dysfunction

Particulate matter and nitrogen dioxide were the pollutants most strongly associated with adverse ocular outcomes.[1,2] Populations exposed to higher pollution levels generally demonstrated increased prevalence and severity of dry eye symptoms, reduced tear film stability, and greater ocular surface inflammation.[6,7] Several studies also suggested a **dose–response relationship** between pollutant concentrations and ocular symptoms.

Pollutant-Specific Findings

Particulate Matter ($PM_{2.5}$ and PM_{10})

Particulate matter was the most extensively studied pollutant. Epidemiological studies reported associations between PM exposure and:

- Increased dry eye disease prevalence
- Reduced tear break-up time
- Higher ocular surface staining scores
- Increased outpatient visits for ocular irritation[5]

Mechanistic studies further demonstrated that particulate matter induces **oxidative stress, epithelial damage, and inflammatory signaling**, supporting its role in ocular surface pathology.[8,9]

Nitrogen Dioxide (NO_2)

Nitrogen dioxide exposure was associated with increased risk of dry eye disease, exacerbation of allergic ocular disorders, and elevated inflammatory responses on the ocular surface. Evidence mapping studies identified NO_2 as one of the most frequently investigated pollutants in environmental ophthalmology research.[1] Clinical studies also reported increased tear cytokine levels in exposed populations.[7]

Ozone (O_3)

Short-term increases in ozone concentrations were linked to acute ocular symptoms, including ocular discomfort, tear film instability, and worsening of dry eye symptoms. However, the evidence for ozone was less consistent compared with particulate matter and nitrogen dioxide.

Sulfur Dioxide (SO_2)

Sulfur dioxide exposure was associated with increased outpatient visits for ocular diseases, particularly dry eye disease and conjunctivitis. Although fewer studies examined SO_2, available evidence suggests a potential role in ocular irritation and inflammatory responses.[5]

Epidemiological Evidence by Disease Type

Dry Eye Disease

Dry eye disease was the most commonly reported outcome across studies. Multiple large cohort and population-based investigations found significant associations between air pollution exposure and increased risk of DED.[3,5] Reported clinical findings included reduced tear break-up time, higher OSDI scores, increased corneal staining, and elevated tear osmolarity. Short-term pollution exposure was also linked to symptom exacerbation.

Meibomian Gland Dysfunction

Clinical studies indicated that pollution exposure may affect meibomian gland structure and function, with findings including gland dropout, altered lipid secretion, and reduced tear film lipid layer thickness, contributing to evaporative dry eye disease.

Allergic Ocular Disease

Air pollution was also associated with increased incidence and severity of allergic conjunctivitis. Severe forms such as **atopic keratoconjunctivitis** and **vernal keratoconjunctivitis** were reported more frequently in highly polluted regions.[15]

Mechanistic Evidence

Mechanistic studies supported epidemiological findings by identifying biological pathways linking pollution exposure to ocular surface damage. Particulate matter exposure was shown to induce oxidative stress through increased production of reactive oxygen species, resulting in epithelial injury and mitochondrial dysfunction.[8,9] Pollutants also activated inflammatory pathways involving cytokines such as IL-6, IL-8, and TNF-α, which contribute to chronic ocular surface inflammation.[8,10] Additionally, disruption of epithelial tight junctions was observed, leading to tear film instability and increased susceptibility to infection.

Disruption of Ocular Surface Barrier

Experimental studies demonstrated that pollutants can damage epithelial tight junctions and disrupt ocular surface barrier integrity.

Consequences include:

Increased susceptibility to infection Chronic inflammation Tear film instability

Table 2: Summary of Pollutant Effects on Ocular Surface Disease.

Pollutant	Major Ocular Effects	Strength of Evidence
$PM_{2.5}$	Dry eye disease, inflammation	Strong
PM_{10}	Ocular irritation, tear instability	Strong
NO_2	Allergic ocular disease, inflammation	Strong
O_3	Acute ocular symptoms	Moderate
SO_2	Ocular irritation	Moderate

Geographic Trends

Most studies were conducted in **East Asia, South Asia, Europe, and North America**, with higher prevalence of ocular surface disease reported in urban populations exposed to traffic-related pollution.

Strength of Evidence and Risk of Bias

Overall, the evidence linking air pollution to ocular surface disease was considered **moderate to strong**, particularly for particulate matter exposure. Consistent findings were observed across epidemiological, clinical, and experimental studies. Most epidemiological studies demonstrated moderate risk of bias due to limitations in exposure assessment and potential confounding factors, while mechanistic studies generally showed low risk of bias due to controlled experimental conditions.

DISCUSSION

This PRISMA-based systematic review synthesizes recent epidemiological and mechanistic evidence examining the relationship between ambient air pollution and ocular surface disease (OSD). Evidence from approximately **125 included studies** indicates a consistent association between exposure to major pollutants—particularly **$PM_{2.5}$, PM_{10}, and NO_2**—and increased risk of dry eye disease, ocular inflammation, and tear film instability. The convergence of findings from population-based studies, clinical investigations, and experimental research strengthens the biological plausibility that air pollution contributes to the development of ocular surface disorders.[1,2,6,7]

Interpretation of Principal Findings

One of the most important findings of this review is the strong association between particulate matter exposure and dry eye disease. Several epidemiological studies have shown that populations exposed to elevated **$PM_{2.5}$ levels** experience higher prevalence and severity of ocular symptoms, including dryness, irritation, and visual discomfort.[3,5] Clinical investigations further report measurable alterations in ocular surface parameters such as decreased tear break-up time, increased corneal staining, and elevated inflammatory markers in tears.[6,7] These findings suggest that particulate pollutants may disrupt ocular surface homeostasis.

Nitrogen dioxide, a major traffic-related pollutant, was also frequently associated with anterior segment disorders, particularly dry eye disease and allergic ocular conditions.[1] In contrast, ozone (O_3) and sulfur dioxide (SO_2) were linked mainly to acute ocular irritation and symptom exacerbation, although evidence for these pollutants remains less consistent.[5]

Overall, the findings suggest that both **chronic exposure and short-term increases in air pollution** may influence ocular surface health and worsen pre-existing ocular conditions.

Biological Mechanisms

Mechanistic studies support these epidemiological findings. Exposure to particulate matter induces **oxidative stress**, leading to excessive production of reactive oxygen species that damage ocular epithelial cells and impair ocular surface function.[8,9] Air pollutants also activate inflammatory pathways, increasing cytokines such as **IL-6, IL-8, and TNF-α**, which contribute to chronic ocular surface inflammation.[8,10] In addition, pollutants may disrupt tear film homeostasis and damage the lipid layer produced by meibomian glands, resulting in tear film instability and evaporative dry eye disease.[2,8]

Comparison with Previous Reviews

These findings are consistent with earlier systematic reviews suggesting that air pollution is a risk factor for both anterior and posterior segment eye diseases, including glaucoma and age-related macular degeneration.[12,13,14]

However, the present review focuses specifically on ocular surface disease and incorporates more recent evidence, providing a clearer understanding of pollution-related ocular pathology.

Clinical and Public Health Implications

The growing evidence linking air pollution with ocular surface disease has important implications for clinical practice. Clinicians should consider environmental exposure when evaluating patients with chronic or treatment-resistant dry eye disease, particularly in regions with high pollution levels.[3,4] Preventive strategies such as monitoring air quality, limiting exposure during high pollution periods, and improving indoor air quality may help reduce ocular symptoms.

From a public health perspective, improving air quality could significantly reduce the burden of ocular surface disease and associated visual impairment worldwide.

Strengths and Limitations

This review integrates evidence from epidemiological, clinical, and mechanistic studies, providing a comprehensive overview of the relationship between air pollution and ocular surface disease. However, several limitations should be considered. Many studies relied on regional pollution monitoring rather than individual exposure measurements, which may introduce exposure misclassification.[11,12] Additionally, variability in study design and diagnostic criteria may contribute to heterogeneity across studies.[14]

Future Research Directions

Future research should focus on **longitudinal cohort studies with improved exposure assessment**, investigation of vulnerable populations, and mechanistic studies exploring molecular pathways involved in pollution-related ocular damage. Interventional studies evaluating preventive strategies and environmental policies are also needed to translate current evidence into clinical and public health practice.

CONCLUSION

This systematic review highlights the growing body of evidence linking ambient air pollution with ocular surface disease. Epidemiological and mechanistic studies consistently demonstrate that exposure to particulate matter and nitrogen dioxide is associated with increased risk of dry eye disease, ocular inflammation, and tear film instability.[1,2,6,7,8] These findings suggest that air pollution represents a significant environmental risk factor for ocular morbidity.

The biological mechanisms underlying these associations include oxidative stress, inflammatory activation, disruption of epithelial barrier function, and impairment of tear film homeostasis. Together, these processes contribute to the development and progression of ocular surface disorders.

Given the increasing global burden of air pollution, addressing environmental determinants of ocular health is becoming increasingly important. Strategies aimed at reducing pollution exposure, improving air quality, and identifying susceptible populations may help mitigate the impact of environmental pollution on ocular disease.

Future research integrating epidemiological, clinical, and mechanistic approaches will be essential to further clarify causal pathways and develop effective preventive and therapeutic interventions. Ultimately, improving environmental conditions may play a crucial role in reducing the global burden of ocular surface disease and improving population eye health.

Declarations

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Ethical approval: Not applicable (review of published literature).
Data availability: No new data were generated; all data are derived from published studies.

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