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MODERN LIFESTYLE AND DRY EYE DISEASE: A NARRATIVE REVIEW OF MODIFIABLE RISKS AND PREVENTIVE STRATEGIES

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ABSTRACT

Background: Dry eye disease (DED) is a multifactorial ocular-surface disorder characterized by loss of tear-film homeostasis, resulting in ocular discomfort, visual disturbances, and progressive surface damage. While intrinsic factors such as age, sex, autoimmune disease, and systemic medications influence susceptibility, lifestyle and environmental determinants—including prolonged screen time, poor sleep, contact-lens wear, smoking, and suboptimal diet—have emerged as important, modifiable contributors to disease onset and severity. Addressing these factors is critical for effective prevention and management.
Methods: This narrative review synthesizes evidence from PubMed, Scopus, and Web of Science (1990–2025) on associations between lifestyle behaviors and DED. Eligible studies included randomized controlled trials, cohort and cross-sectional studies, and systematic reviews published in English. Emphasis was placed on identifying modifiable behaviors that impact tear-film stability, ocular-surface inflammation, and symptom burden.
Results: Evidence indicates that extended digital-screen use, disrupted sleep, contact-lens wear, and smoking increase DED risk and exacerbate symptoms. Conversely, interventions targeting modifiable behaviors—such as scheduled visual breaks, hydration, improved sleep hygiene, eyelid hygiene, smoking cessation, and nutritional optimization—are associated with improved tear-film stability, reduced inflammation, and symptomatic relief.
Conclusions: Lifestyle behaviors play a critical role in DED pathogenesis and progression. Integrating behavioral counseling into clinical practice and public-health strategies provides a low-cost, patient-empowering approach to reduce disease burden and improve ocular-surface health.

KEYWORDS

Dry Eye Disease, Lifestyle Factors, Screen Time, Sleep Quality, Meibomian Gland Dysfunction, Public Health

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1. Introduction

Dry eye disease (DED) is a multifactorial disease of the ocular surface, characterized by a loss of tear-film homeostasis and the presence of ocular symptoms. Central elements in its pathophysiology include tear-film instability, hyperosmolarity, ocular-surface inflammation, and epithelial damage, which form a self-perpetuating vicious cycle that amplifies disease severity and symptom burden [1]. Clinically, DED manifests as ocular discomfort and visual disturbance, which can fluctuate over time and substantially impact daily activities and visual comfort. The intensity of these manifestations may be influenced by individual, environmental, and behavioral factors, although the precise triggers vary among patients [2]. Recognizing the interplay of these mechanisms is essential for understanding the heterogeneous presentation of DED and for guiding effective management strategies that aim to restore tear-film homeostasis and reduce ocular-surface damage. From a pathophysiological standpoint, DED is subclassified into aqueous-deficient and evaporative types, though mixed forms are most commonly observed in clinical practice. Evaporative DED is frequently driven by meibomian gland dysfunction (MGD), which disrupts the lipid layer of the tear film, accelerates tear evaporation, and contributes to ocular-surface stress. The interplay between aqueous insufficiency and evaporative mechanisms generates a vicious cycle of tear-film instability, epithelial damage, and chronic inflammation, emphasizing the multifactorial nature of the disease [3]. Understanding these mechanistic underpinnings is critical for designing both pharmacologic and non-pharmacologic interventions that address the root causes rather than merely alleviating symptoms.

DED constitutes a major public-health burden on a global scale. Systematic reviews estimate that 5% to 50% of the world's population is affected by DED, although prevalence varies according to age, diagnostic criteria, geographic region, and population characteristics [2]. In Poland, population-based studies have reported

symptomatic prevalence ranging widely from 10–18% and up to 30%, reflecting differences in patient demographics and environmental exposures [4]. Multiple intrinsic and extrinsic risk factors have been identified, including female sex, advancing age, autoimmune disorders, diabetes mellitus, previous ocular surgery, and the use of systemic medications such as antihistamines, diuretics, or antidepressants, all of which can alter tear production or ocular-surface homeostasis [5]. These epidemiologic insights underscore the widespread impact of DED on both individual health and healthcare systems.

Lifestyle and environmental exposures have emerged as particularly important, modifiable determinants of DED risk. Prolonged digital-screen use, which is associated with decreased blink frequency and incomplete blinking, is consistently correlated with tear-film instability and ocular-surface desiccation. Additional factors, including low ambient humidity, exposure to airflow from air conditioning or heating, cigarette smoking, poor sleep quality, and suboptimal dietary habits, further increase the likelihood of DED onset or symptom exacerbation [6]. These lifestyle determinants interact with underlying physiologic susceptibilities, amplifying ocular-surface stress and accelerating disease progression. Notably, the COVID-19 pandemic has intensified many of these risk factors, as widespread shifts to remote work, increased screen exposure, and changes in indoor environmental conditions have been linked to heightened DED prevalence and symptom burden [7].

If left unmanaged, chronic DED can result in progressive ocular-surface damage, including corneal epithelial defects, increased susceptibility to infection, chronic inflammation, and structural changes that may impair visual acuity. Beyond objective ocular morbidity, DED has substantial implications for quality of life, contributing to discomfort, reduced reading or work efficiency, and limitations in daily activities [2]. Given the high prevalence of DED and the significant role of modifiable lifestyle and environmental factors, a comprehensive understanding of these determinants is of high clinical relevance.

This review aims to synthesize the current scientific evidence regarding behavioral and environmental influences on DED, highlighting factors that contribute to its development and progression. In addition, it seeks to identify preventive and mitigating lifestyle interventions—including screen-break strategies, environmental modifications, nutritional optimization, sleep improvement, and behavioral counseling—that may reduce the incidence, severity, or progression of DED. By integrating mechanistic understanding with practical lifestyle recommendations, this work emphasizes the potential for proactive, patient-centered approaches to prevent or alleviate the burden of this increasingly prevalent ocular disorder.

2. Methodology

This review is based on a narrative synthesis of scientific literature examining the relationship between lifestyle factors and dry eye disease (DED), emphasizing behavioral, environmental, and occupational influences on disease onset, severity, and management outcomes.

The literature search was conducted using major biomedical databases, including PubMed, Scopus, and Web of Science, covering publications from 1990 to 2025. The search strategy combined the following keywords: “dry eye disease,” “ocular surface,” “meibomian gland dysfunction,” “screen time,” “digital eye strain,” “sleep,” “diet,” “environmental exposure,” “lifestyle,” “physical activity,” “smoking,” and “risk factors.”

Eligible studies included randomized controlled trials, cohort and case-control studies, cross-sectional surveys, and systematic reviews or meta-analyses that investigated the association between modifiable lifestyle behaviors and DED. Only peer-reviewed articles published in English were considered. Additional references were identified through manual searches of bibliographies from key review articles.

Studies were screened for methodological quality, clinical relevance, and alignment with review objectives. Extracted data focused on the impact of lifestyle determinants—such as screen exposure, smoking, dietary habits, hydration, physical activity, and sleep quality—on DED incidence, symptom severity, and progression. Proposed biological mechanisms included alterations in tear-film stability, meibomian gland function, and ocular-surface inflammation.

This narrative approach enables a comprehensive integration of epidemiological and mechanistic evidence, highlighting how lifestyle behaviors contribute to DED development and clinical course.

3. Pathogenesis of Dry Eye Disease and Its Consequences

The pathogenesis of Dry Eye Disease (DED) is complex and revolves around a self-perpetuating cycle involving tear-film instability, hyperosmolarity, ocular-surface inflammation, and neurosensory dysfunction, which collectively contribute to both the onset and progression of the disease [8]. Under normal physiological conditions, the tear film serves multiple essential functions for ocular-surface homeostasis: it provides lubrication to reduce friction during blinking, supplies oxygen and nutrients to the avascular corneal epithelium, protects against microbial invasion, removes cellular debris, and maintains optical smoothness for clear vision. Disruption of any component of the tear film, whether aqueous, lipid, or mucin layers, can destabilize this delicate homeostatic balance and trigger a cascade of pathological events that reinforce and amplify ocular-surface damage.

DED can be broadly classified into aqueous-deficient and evaporative subtypes, although mixed forms are frequently observed in clinical practice. In the aqueous-deficient subtype, lacrimal gland dysfunction or autoimmune processes reduce tear secretion, resulting in insufficient tear volume and early tear-film break-up. In contrast, evaporative DED is most commonly driven by meibomian gland dysfunction (MGD), wherein meibum hyposecretion, obstruction, or altered lipid composition leads to disruption of the lipid layer. This increases tear evaporation and destabilizes the tear film, producing localized areas of hyperosmolarity [9,11]. Both subtypes ultimately converge on a common pathogenic endpoint: elevated tear-film osmolarity, which exerts deleterious effects on corneal and conjunctival epithelial cells. Hyperosmolar stress triggers osmotic imbalance, inducing cellular apoptosis and activating signaling pathways that upregulate pro-inflammatory cytokines, including interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) [10]. These inflammatory mediators not only contribute to epithelial damage but also amplify ocular-surface immune activation, creating a self-perpetuating cycle that perpetuates tissue injury and symptomatic discomfort.

Inflammation plays a central and sustained role in DED pathogenesis. Hyperosmolarity and epithelial damage activate resident antigen-presenting dendritic cells, which, in turn, stimulate adaptive immune responses through recruitment and activation of Th1 and Th17 lymphocytes. These lymphocytes release interferon-gamma (IFN-γ) and interleukin-17 (IL-17), potent inflammatory mediators that disrupt epithelial barrier integrity, reduce the density and function of conjunctival goblet cells, and damage meibomian gland acini [10]. The resulting reduction in mucin and lipid secretion exacerbates tear-film instability and evaporation, thereby reinforcing hyperosmolar stress and inflammation. In parallel, ocular-surface nerves are profoundly affected: corneal nerve fiber density is reduced, tortuosity increases, and sensory sensitivity becomes abnormal, contributing to altered neurosensory feedback. These changes link directly to symptom generation, such as burning, stinging, and foreign-body sensation, and may impair reflex tear production, further worsening ocular-surface desiccation [11].

Meibomian gland dysfunction remains the predominant factor driving the evaporative subtype of dry eye in clinic-based populations. In a retrospective study, approximately 86% of patients exhibited clinical signs of MGD, indicating its major contribution compared with pure aqueous-deficient forms. Dysfunctional meibomian glands, identified through elevated Foulks-Bron scores and normal or slightly reduced Schirmer test values, result in weakening of the tear-film lipid layer, which can increase tear evaporation and destabilize the tear film. While the study primarily focused on the distribution of subtypes rather than mechanistic measurements, these findings underscore the predominance of MGD as a key clinical feature of evaporative dry eye[12]. Beyond the mechanical and inflammatory components, DED pathogenesis encompasses complex interactions among tear-film biomechanics, immune dysregulation, glandular dysfunction, and neurosensory abnormalities, emphasizing that the disease is not merely a deficiency in tear quantity but a systemic dysfunction of ocular-surface homeostasis.

Clinically, patients with DED present with a spectrum of symptoms, including burning, foreign-body sensation, photophobia, dryness, and fluctuating vision, which often interfere with daily activities such as reading, computer use, and driving [5,13]. Objective structural changes observed on slit-lamp or imaging evaluation include corneal epithelial staining, goblet-cell loss, meibomian-gland dropout, and alterations in sub-basal corneal nerves. Functional consequences of DED extend beyond subjective discomfort, as the disease has been shown to significantly impair visual performance and quality of life. Population-based studies indicate that individuals with DED increase the odds of reduced health-related quality of life compared to unaffected individuals, highlighting the substantial burden imposed by this condition [14].

If left unrecognized or untreated, chronic DED may progress to severe and potentially irreversible ocular-surface damage. Complications include persistent epithelial defects, corneal ulceration, and, in extreme

cases, scarring. The cumulative effects of ongoing inflammation, tear-film instability, and neurosensory impairment can create a state of chronic ocular-surface dysfunction that is difficult to reverse. [1,2] Therefore, early recognition of the pathogenic cascade—ranging from tear-film disruption and hyperosmolar stress to immune activation and nerve dysfunction—is critical for timely intervention, prevention of irreversible complications, and the implementation of both pharmacologic and non-pharmacologic management strategies. By addressing the multifactorial drivers of DED early, clinicians can stabilize tear-film homeostasis, reduce inflammation, preserve corneal and conjunctival integrity, and improve patient quality of life.

Table 1. Pathophysiologic Mechanisms and Clinical Consequences of DED

Pathophysiologic Mechanism	Clinical Consequence
Tear-film instability & hyperosmolarity	Epithelial stress, inflammation, ocular discomfort
Ocular-surface inflammation	Goblet-cell loss, epithelial barrier disruption
Meibomian gland dysfunction	Increased evaporation, evaporative DED
Neurosensory abnormalities	Altered pain perception, reflex tear failure
Chronic epithelial damage	Corneal staining, infection risk, scarring

4. Lifestyle Factors and Dry Eye Disease (DED)

Lifestyle factors exert a profound influence on the onset, severity, symptom burden, and overall clinical course of dry eye disease (DED). A wide array of behavioral, environmental, and systemic exposures—including prolonged use of digital screens, contact-lens wear, meibomian gland dysfunction (MGD), cigarette smoking, insufficient or poor-quality sleep, and suboptimal dietary habits—have been consistently associated with the pathophysiological hallmarks of DED, namely tear-film instability, ocular-surface inflammation, epithelial stress, and neurosensory dysregulation [15,16]. Recognition of these factors provides an opportunity to implement targeted lifestyle interventions that can mitigate disease progression, improve patient-reported outcomes, and complement pharmacologic therapies.

Screen use

Prolonged exposure to visual display terminals (VDTs) is among the most consistently reported behavioral correlates of DED. During extended screen use, blink frequency typically decreases, and incomplete blinking becomes more common, accelerating tear-film break-up, increasing tear evaporation, and predisposing to ocular-surface desiccation [15]. These alterations contribute to symptoms collectively referred to as digital eye strain (DES), which frequently overlap with those of DED. Prevalence estimates for DES and associated dry-eye symptoms vary widely, ranging from 9.5% to 87.5%, depending on the diagnostic criteria applied, the study population, and occupational or lifestyle factors [15,16]. Prolonged visual focus during screen use reduces blink frequency and increases the incidence of incomplete blinking, which disrupts tear-film distribution across the corneal surface and contributes to ocular-surface discomfort. These effects underscore the importance of implementing structured visual breaks and conscious blink training to mitigate symptoms in affected individuals[15].

Contact-lens wear

Contact-lens wear is a well-established and clinically significant risk factor for ocular-surface discomfort and dryness. A Portuguese cross-sectional study reported that 21.5% of soft contact-lens wearers experience dry-eye symptoms, illustrating the substantial burden of lens-related ocular irritation [16]. Higher symptom prevalence is observed among users of reusable or extended-wear lenses, reflecting differences in lens material, oxygen permeability, surface wettability, and hygiene practices [17]. Contact lenses alter the natural tear film by increasing friction between the lens and the ocular surface, promoting localized inflammation, and predisposing to epithelial microtrauma. Additionally, lens deposits and microbial contamination may exacerbate ocular-surface stress, further contributing to symptomatic DED [17,18]. Consequently, lens selection, wear schedules, and proper hygiene represent critical modifiable factors in lifestyle-based management.

Meibomian gland dysfunction (MGD)

MGD is recognized as the most prevalent cause of evaporative DED, and its epidemiology is highly variable, with prevalence estimates ranging from 3.5% to almost 70%, depending on population characteristics, diagnostic criteria, and environmental exposures [19]. MGD disrupts the tear-film lipid layer, accelerating tear evaporation, destabilizing the tear film, and increasing ocular-surface stress. Patients with MGD often experience chronic irritation, burning, and fluctuating vision, reflecting the central role of lipid-layer insufficiency in evaporative dry eye [18,19]. Regular eyelid hygiene, warm compresses, and gland expression can help restore lipid-layer function, reduce tear evaporation, and alleviate symptom burden, underscoring the value of targeted lifestyle interventions in this context[19].

Smoking

The relationship between cigarette smoking and DED has been extensively investigated, with meta-analyses producing mixed results. While some studies indicate an increased risk of DED among smokers, others report inconsistent associations, likely due to differences in exposure assessment, study design, and confounding factors [20,21]. Nevertheless, biological plausibility strongly supports smoking as a contributing factor: tobacco smoke induces oxidative stress, promotes local inflammation, and impairs epithelial barrier function, all of which can destabilize the tear film and exacerbate DED symptoms [20,21]. Therefore, smoking cessation may be a practical, low-risk lifestyle measure that could confer meaningful symptomatic relief and promote ocular-surface health.

Sleep and circadian health

Poor sleep quality and circadian misalignment are reproducibly associated with increased DED prevalence. Cross-sectional and cohort studies demonstrate higher odds of dry-eye symptoms among individuals with sleep disorders, likely mediated through multiple mechanisms, including altered lacrimal-autonomic regulation, systemic inflammatory activation, and increased nocturnal screen exposure [22,23]. Poor sleep quality was significantly associated with a higher risk of DED and greater symptom severity, suggesting that improving sleep may potentially benefit ocular surface health and reduce symptoms, although direct interventional evidence is not yet available[23]. These findings highlight the interplay between systemic physiology, neurosensory function, and ocular-surface homeostasis, reinforcing the importance of holistic lifestyle management.

Dietary factors

Dietary composition, particularly intake of omega-3 fatty acids, has been extensively studied for its potential protective role in DED. Some randomized controlled trials report modest improvements in symptom burden and tear-film parameters, whereas others find no statistically significant effect [24,25]. Low dietary omega-3 intake, combined with high consumption of processed foods rich in pro-inflammatory nutrients, may exacerbate ocular-surface inflammation, suggesting that nutritional optimization can serve as a complementary intervention. Personalized dietary counseling, emphasizing adequate intake of omega-3–rich foods and reduction of pro-inflammatory dietary elements, can be a pragmatic component of lifestyle-based DED management [24,26].

Despite variability in study outcomes, the aggregated evidence supports the incorporation of pragmatic, low-risk lifestyle interventions into DED management. These include structured screen breaks, blink-rate training, environmental optimization such as improved indoor humidity, eyelid hygiene for MGD, smoking cessation, sleep improvement, and nutritional optimization. When combined with pharmacologic therapy, these interventions represent a safe, cost-effective, and patient-empowering strategy for reducing symptoms, preserving ocular-surface integrity, and potentially slowing disease progression [15-25].

Table 2. Prevalence of Dry Eye Disease Across Lifestyle-Related Risk Factors

Lifestyle-Related Factor	Reported Prevalence / Proportion of Affected Individuals
General population	11.6% (global estimate)
Digital screen users	9.5% to 87.5% symptomatic prevalence
Soft contact-lens wearers	21.5% symptomatic prevalence
Meibomian gland dysfunction (MGD)	3.5–70% prevalence (population-dependent)
Current or former smokers	Variable association; not consistently quantified
Individuals with poor sleep quality	Increased odds ratio (varies by study)
Low omega-3 dietary intake	Mixed evidence; variable effect sizes

5. Preventive and Therapeutic Implications of Lifestyle Modification in DED

Lifestyle modification represents a fundamental component of the contemporary management paradigm for dry eye disease (DED), serving as a vital complement to pharmacologic interventions, procedural therapies, and other conventional treatment modalities. A growing corpus of clinical and experimental evidence highlights that appropriately targeted lifestyle interventions can positively influence multiple pathophysiological mechanisms underlying DED, including tear-film instability, ocular-surface inflammation, epithelial disruption, meibomian gland dysfunction, and aberrant neurosensory signaling. Consequently, lifestyle modifications not only mitigate subjective symptoms such as dryness, irritation, burning, or visual fatigue but may also slow the progression of structural and functional ocular-surface changes associated with chronic disease[24].

Behavioral interventions, particularly in the context of prolonged exposure to visual display terminals (VDTs), constitute a cornerstone of lifestyle-based DED management. Structured strategies, such as the widely advocated 20-20-20 rule—whereby patients are instructed to interrupt their gaze every 20 minutes to focus on an object at least 20 feet away for 20 seconds—have been consistently shown to enhance blink frequency and completeness, promote uniform tear distribution, and prolong tear-film break-up time [27]. These effects are mechanistically linked to the prevention of localized tear-film thinning, reduction of epithelial microtrauma, and preservation of corneal and conjunctival surface integrity. In addition to visual behavior modifications, environmental factors play a critical role in tear-film homeostasis. Maintaining higher indoor relative humidity and avoiding direct exposure to air currents from air conditioners, fans, or heating vents significantly stabilizes the tear film, reduces evaporative stress, and enhances both objective clinical metrics and subjective symptom scores [28,29]. Such modifications are particularly relevant in office settings and dry indoor environments, where low humidity and air turbulence exacerbate tear evaporation and ocular discomfort[29].

In patients who use contact lenses, tailored lifestyle strategies targeting lens hygiene and wear patterns are highly effective. Transitioning from reusable lenses to daily disposable lenses, coupled with strict avoidance of overnight wear, has been associated with reductions in corneal epithelial staining, diminished ocular irritation, and enhanced overall comfort [30]. These benefits are thought to arise from decreased accumulation of protein and lipid deposits, lower microbial contamination, and reduced mechanical friction on the corneal epithelium. For individuals with meibomian gland dysfunction (MGD), regular lid hygiene practices—including the application of warm compresses to melt obstructed meibum and systematic lid massage to express gland contents—have been demonstrated to improve lipid-layer quality, reduce tear evaporation, and alleviate ocular surface inflammation [31]. By restoring the integrity and functionality of the tear-film lipid layer, these interventions help maintain a stable ocular surface and mitigate the cyclical exacerbation of DED symptoms.

Lifestyle modifications targeting systemic factors are equally important. Smoking cessation, for example, has been shown to confer symptomatic relief and objective improvement in ocular-surface health[32]. Tobacco use generates oxidative stress and pro-inflammatory mediators, which directly disrupt epithelial barrier function and destabilize the tear film. Discontinuation of smoking may reduce these detrimental effects, supporting ocular-surface healing and decreasing inflammatory burden. Similarly, optimizing sleep quality through interventions such as cognitive-behavioral therapy, structured sleep hygiene programs, and consistent sleep schedules has been associated with decreased tear osmolarity and improved ocular-surface integrity [33].

Sleep deficiency can exacerbate systemic inflammation, alter autonomic regulation of tear production, and impair corneal epithelial repair, so restoring adequate and high-quality sleep provides a mechanistic route for improving tear-film homeostasis and neurosensory feedback.

Nutritional interventions, while more variable in efficacy, represent another potentially beneficial dimension of lifestyle-based management. Omega-3 fatty acids, for instance, have been extensively investigated for their anti-inflammatory and lipid-stabilizing properties. Supplementation with omega-3s may enhance meibomian gland secretion, reduce inflammatory cytokine levels, and improve tear-film stability, although clinical trial results have been mixed. Some randomized controlled trials demonstrate significant reductions in patient-reported symptoms and improvements in objective tear-film metrics, whereas others show minimal effect, suggesting that factors such as baseline dietary intake, bioavailability, dosing, and patient-specific metabolic responses influence clinical outcomes [34,35].

Taken together, the integration of lifestyle counseling into routine DED management is not only evidence-based but also highly practical. These interventions are generally safe, cost-effective, and empower patients to take an active role in the control of their disease. When implemented alongside pharmacologic therapy—such as artificial tears, anti-inflammatory drops, or other targeted treatments—lifestyle modifications form the foundation of a holistic, patient-centered approach to DED care. By simultaneously addressing environmental, behavioral, systemic, and nutritional contributors, clinicians can optimize both symptom relief and long-term ocular-surface health, creating a comprehensive strategy that targets the multifactorial nature of this chronic and often debilitating condition [27-35].

6. Public Health Perspective and Future Directions

Dry eye disease (DED) has emerged as a significant and increasingly pressing public-health concern, not only because of its high and rising prevalence but also due to its substantial socioeconomic impact. Globally, approximately 11.6% of the population is affected by DED, a figure that is projected to increase in parallel with trends in digitalization, aging populations, urbanization, and environmental stressors such as low indoor humidity, air pollution, and prolonged exposure to visual display terminals (VDTs)[36,37]. The combination of these demographic, behavioral, and environmental factors has amplified both the direct healthcare costs associated with DED management and the indirect costs related to lost productivity, decreased work performance, and reduced quality of life.

Public health strategies targeting modifiable behavioral risk factors offer a promising avenue for reducing the overall burden of DED. Educational campaigns designed to promote ergonomic screen use, encourage regular visual breaks, ensure adequate systemic hydration, and optimize indoor humidity have the potential to lower DED incidence, particularly in high-risk populations such as office workers, students, and older adults [28,38]. Such programs not only aim to prevent the onset of symptoms but also to reinforce early recognition and self-management of DED, thereby reducing progression to more severe or chronic stages.

Despite growing recognition of the public-health relevance of DED, there remains a need for longitudinal, population-based studies to more precisely quantify the impact of lifestyle factors—such as screen exposure, sleep quality, and contact-lens habits—on the risk of developing DED over time. Data from interventional trials indicate that community-level programs can be effective in both raising awareness and improving symptom control. Examples include workplace implementations of blink-reminder software, educational modules focused on eyelid hygiene, and training on proper visual ergonomics, all of which have been associated with measurable improvements in ocular comfort and patient-reported outcomes[38].

Digital health technologies represent an emerging frontier in public-health approaches to DED prevention. Smartphone applications, wearable monitors, and automated reminders can facilitate adherence to preventive behaviors by tracking screen time, prompting regular breaks, and guiding eyelid care routines. These tools may be particularly useful in occupational and educational settings, where sustained digital engagement is unavoidable. Integration of such technologies into broader workplace health programs could enhance compliance, provide individualized feedback, and ultimately reduce the prevalence and severity of DED [36].

Policymakers are encouraged to consider ocular-surface health as an integral component of occupational health guidelines, particularly for professions with prolonged digital exposure. Incorporating recommendations on screen ergonomics, scheduled visual breaks, hydration, and environmental controls into workplace standards could prevent disease onset and mitigate the economic impact of DED. Additionally, cost-effectiveness analyses are warranted to evaluate the economic benefits of preventive interventions, including

reductions in healthcare utilization, medication use, and productivity losses, thereby providing evidence for resource allocation and policy prioritization [36].

Addressing DED within a public-health framework requires translating clinical evidence into scalable, population-level strategies that bridge ophthalmologic care with preventive medicine. By implementing comprehensive education, leveraging digital health technologies, and integrating ocular-surface health into occupational and community health initiatives, it is possible to reduce the burden of DED, improve quality of life, and achieve long-term economic benefits. Such an approach emphasizes proactive prevention, rather than reactive treatment, and underscores the necessity of a multidisciplinary perspective encompassing clinical practice, public health policy, and health technology innovation [36,38].

Table 3. Public Health Strategies for Reducing Dry Eye Disease Burden

Strategy Type	Example Interventions	Expected Outcomes
Individual-Level Interventions	Screen-time management, adequate sleep, smoking cessation	Reduced DED symptoms, improved tear-film stability
Workplace/Occupational Measures	Blink-reminder software, ergonomic workstation setup, humidity control	Decreased digital eye strain, fewer symptomatic employees
Public Health Campaigns	Education on ocular hygiene, hydration, and environmental awareness	Improved population-level awareness and early detection
Policy & Healthcare Integration	Occupational health guidelines, cost-effectiveness analyses	Systemic reduction in DED prevalence and productivity loss

7. Conclusions

Dry eye disease (DED) is a multifactorial ocular-surface disorder driven by tear-film instability, inflammation, and neurosensory abnormalities. Increasing evidence highlights the role of lifestyle factors—screen exposure, contact-lens wear, sleep quality, diet, and smoking—as modifiable contributors to disease onset and severity.

The literature supports that behavioral interventions can significantly alleviate symptoms, improve tear-film stability, and reduce inflammation. Incorporating preventive measures—such as regular visual breaks, adequate hydration, sleep improvement, nutritional optimization, and smoking cessation—into daily life and clinical practice can mitigate disease burden.

Future research should focus on longitudinal analyses and interventional studies to strengthen causal inference between lifestyle behaviors and DED outcomes. Public health efforts integrating education, occupational policy, and technology-based interventions will be essential to reduce the growing prevalence of DED globally.

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