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DIGITAL SCREEN EXPOSURE AND DRY EYE DISEASE: A REVIEW OF CLINICAL AND PATHOPHYSIOLOGICAL PERSPECTIVES

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ABSTRACT

Background: Dry eye disease (DED) is one of the most common ocular surface diseases and is associated with disruption of tear film homeostasis, tear instability, and chronic inflammation. With the increasing use of digital devices in everyday life, their potential impact on the development and severity of DED symptoms is increasingly being analyzed.
Aim: The aim of this narrative review was to present current data on the relationship between digital device use and dry eye disease, with particular emphasis on pathophysiological mechanisms, epidemiological data, and possible prevention and treatment options.
Methods: A literature review was conducted using publications available in the PubMed, Scopus, and Google Scholar databases. Clinical and epidemiological studies, as well as review articles on ocular surface disorders associated with digital exposure, were included.
Results: The analyzed data indicate that prolonged screen use may contribute to tear film destabilization through decreased blinking frequency, increased tear evaporation, and meibomian gland dysfunction. Numerous studies have also observed a higher incidence of DED symptoms in individuals who use digital devices extensively.
Conclusion: Growing exposure to screens is making screen-related dry eye an increasingly common clinical problem in modern ophthalmology. Being a modifiable factor, it creates the opportunity to implement interventions that reduce the severity of DED symptoms and improve patient comfort.

KEYWORDS

Dry Eye Disease, Digital Screen Exposure, Digital Eye Strain, Tear Film Instability, Meibomian Gland Dysfunction, Ocular Surface Disorder

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1. Introduction

Several definitions of dry eye disease have been developed over the years. The current definition, based on the TFOS DEWS III, describes dry eye disease as a multifactorial, symptomatic disease characterized by a loss of tear film and/or ocular surface homeostasis. The etiological factors include tear film instability and hyperosmolarity, inflammation, and ocular surface damage, along with neurosensory abnormalities. The definition does not include the terms "progressive" or "chronic," although these terms are often used by clinicians in daily clinical practice (Wolffsohn et al., 2025). Given its chronic nature, impact on visual function, and quality of life in patients with dry eye disease (DED), this condition can be classified as a serious health problem worldwide.

Recent decades have seen a significant increase in the incidence of this disease, and epidemiological studies estimate that it may affect between 5% and 50% of people worldwide, depending on the diagnostic criteria used and the demographic characteristics of the groups studied (Stapleton et al., 2017). Women, the elderly, contact lens wearers and those exposed to environmental or occupational risk factors are particularly at increased risk (Sengo et al., 2023; Meyer et al., 2021).

Ocular discomfort is not the only problem faced by people with DED. Research indicates its potential impact on reduced work performance and daily functioning, negatively impacting patients' psychological well-being (Tsou, 2022). Moreover, the rapid development of digital technologies has led to a significant increase in the time spent in front of computer and smartphone screens, both in professional and private life (Wolffsohn et al., 2023). Prolonged exposure to screens has been linked to symptoms known as digital eye strain (DES) or computer vision syndrome, including eye strain, blurred vision, headaches and dry eye syndrome (Pucker et al., 2024).

The COVID-19 pandemic has also increased the frequency and need for digital device use. Devices have come to be viewed not only as work tools but also as a way to educate and interact with family and friends (Aldukhayel et al., 2022). Growing evidence suggests that prolonged exposure of the eyes to digital screens may significantly increase the risk of developing and exacerbating dry eye symptoms (Kim et al., 2017).

An important mechanism contributing to this process is the reduced spontaneous blinking rate when using screens, often combined with incomplete blinking (Portello et al., 2013). This causes disruption of the tear film distribution on the ocular surface, increased tear evaporation and ultimately contributes to tear film instability (Tsubota & Nakamori, 1995; Mehra & Galor, 2020). Furthermore, prolonged exposure to digital devices can increase the ocular surface's exposure to external factors and promote dysfunction of the meibomian glands, responsible for lipid production. Insufficient quality or quantity of these glands in the tear film leads to accelerated evaporation from the ocular surface, contributing to the symptoms of dry eye syndrome (Fjærvoll i in., 2022; Narang i in., 2023).

The increasing number of people dependent on digital devices across all age groups highlights the importance of understanding the relationship between digital screen exposure and dry eye disease so that this information can be used in clinical practice. The aim of this review is to summarize the current knowledge on the pathophysiological mechanisms linking digital screen exposure to dry eye disease, assess the available clinical evidence, and discuss practical preventive strategies that may reduce the severity of symptoms in individuals affected by this condition (Dresp-Langley & Hutt, 2022).

2. Methodology

This literature review examined publications regarding the association between exposure to digital devices and dry eye syndrome (DED). The literature search was conducted using PubMed, Scopus, and Google Scholar. The search terms used included "dry eye disease," "digital screen exposure," "visual display terminal," "digital eye strain," "computer vision syndrome," "meibomian gland dysfunction," and "tear film instability." The primary focus was on English-language publications published between 2000 and 2025. Additionally, selected previous studies considered relevant to understanding the pathophysiological mechanisms and diagnosis of DED were also reviewed. The analysis primarily included clinical trials, epidemiological studies, review articles. The publications analyzed addressed the mechanisms linking long-term use of digital devices to ocular surface disorders, as well as epidemiological data, clinical symptoms, and available methods for preventing and treating screen-related dry eye. Papers not directly related to the ocular surface or not providing significant data on digital exposure were omitted.

3. Results

3.1 Pathophysiology of Dry Eye Disease

To understand the nature of dry eye syndrome, it is essential to examine the pathophysiological mechanisms leading to this condition. To maintain tear osmolarity within appropriately narrow limits, tear production must be continuously regulated (Sullivan et al., 2012). This occurs reflexively in the body through the tear functional unit (LFU). This unit consists of the ocular surface, the secretory appendages, and the nerve fibers that connect them (Stern et al., 2004). The afferent portion of the feedback loop regulating tear production innervates the ocular surface epithelia, including the cornea, conjunctiva, and eyelid margins. The efferent portion includes parasympathetic fibers innervating the lacrimal glands, meibomian glands, and conjunctival goblet cells. The nasolacrimal duct is also part of the reflex system (Paulsen, 2003). Sensory signals from corneal receptors, including TRPM8, polymodal nociceptors, and mechanoreceptors, are transmitted via the trigeminal nerve to the brainstem, where stimuli related to ocular surface dryness are integrated. In response, efferent signals are sent via parasympathetic fibers, primarily within the facial nerve, to the lacrimal gland, triggering tear production and the blink reflex. Disruption of this pathway at any level can disrupt tear film stability and lead to the pathological changes observed in DED. Chronic stimulation of these receptors can lower their excitability threshold and enhance the transmission of pain stimuli within the brainstem. This results in a disproportionately severe pain response with minimally effective stimuli (Bron et al., 2017).

Dry eye disease can be divided into two subtypes: aqueous deficient dry eye (ADDE) and evaporative dry eye (EDE). In clinical practice, these subtypes may coexist in approximately 40% of cases (Donthineni et al., 2019). ADDE syndrome is associated with lacrimal gland dysfunction leading to decreased secretion, whereas EDE is most often associated with generalized meibomian gland dysfunction (MGD). In this form, terminal duct obstruction and/or qualitative/quantitative changes in glandular secretion may occur (Nelson et

al., 2011). As a result, increased tear evaporation may occur, leading to a shortened tear film breakup time (TBUT). This is a key parameter used in clinical practice. It measures the time from blinking to complete tear distribution on the ocular surface and is one of the primary indicators of tear film stability. TBUT is assessed after fluorescein injection into the conjunctival sac during a slit-lamp examination. In healthy individuals, it typically exceeds 10 seconds, while values below this threshold indicate tear film instability and are commonly observed in patients with DED. Reduced TBUT levels correlate with increased ocular surface light exposure and contribute to the development of hyperosmotic stress (El Barche et al., 2024). Interestingly, EDE due to MGD is probably one of the most common complaints in ophthalmological practice, accounting for approximately 42.5–57.2% of cases (Yadav et al., 2022). The predominance of evaporative mechanisms in many populations highlights the importance of lipid layer integrity for maintaining tear film stability (Narang et al., 2023).

The tear film serves as a barrier between the ocular epithelial surface and the external environment. Its estimated thickness in the precorneal layer is approximately 3 μm (Pflugfelder & Stern, 2020). It consists of a surface lipid layer, a central aqueous phase, and a mucin-rich interface with the epithelial glycocalyx. Each layer serves distinct biological functions. The lipid layer limits evaporation and, by lowering the surface tension of tears, enables uniform coverage of the ocular surface with a film and stabilizes its integrity. The aqueous layer provides hydration, nutrients, and antimicrobial protection (Lemp et al., 1970). The mucin layer, composed of both secreted mucins (e.g., MUC5AC) and membrane-bound mucins (MUC1, MUC4, MUC16), is essential for maintaining surface wettability and ensuring even tear distribution. Impaired expression leads to reduced tear film adhesion and increased susceptibility to local drying (Georgiev et al., 2019).

Tear hyperosmolarity is another important mechanism responsible for dry eye syndrome. In healthy individuals, tear film osmolarity is estimated to be between 296 and 302 mOsm/l, whereas in patients with DED, higher values of approximately 316 and 360 mOsm/l are observed. Hyperosmolarity leads to a decrease in cell volume and an increase in solute concentration. Through complex mechanisms, these processes can activate intracellular signaling pathways, particularly mitogen-activated protein kinase (MAPK) and nuclear factor kappa B (NF-κB). This can then lead to the production of proinflammatory cytokines such as interleukin-1β, interleukin-6, and TNF-alpha. These mediators can contribute to epithelial cell apoptosis, tight junction damage, and, consequently, loss of the tear film's barrier function (Baudouin et al., 2013).

Inflammation is both a consequence and a key driver of DED progression. Hyperosmotic stress and epithelial damage activate the innate immune response and subsequently recruit adaptive immune cells, including helper lymphocytes. These cells release interferon gamma and interleukin 17, which contribute to goblet cell loss and reduced mucin production (Nguyen et al., 2011). Matrix metalloproteinase 9 (MMP-9), a recognized biomarker of ocular surface inflammation, is often elevated in patients with DED. MMP-9 degrades tight junction proteins and extracellular matrix components, leading to increased epithelial permeability and further destabilization of the tear film (Chotikavanich i in., 2009; Luo i in., 2004).

A key feature of DED pathogenesis is the mutual exacerbation of the mechanisms described, worsening the patient's clinical condition. Tear film instability promotes hyperosmolarity, which causes inflammation and epithelial damage. Inflammation, in turn, further disrupts tear film components, impairs glandular function, and disrupts neural regulation. Over time, this cycle leads to chronic ocular surface dysfunction and persistent symptoms. External factors, including environmental conditions and behavioral patterns, can exacerbate this cycle and accelerate disease progression (Bron et al., 2017).Understanding these interrelated mechanisms is important for assessing the influence of environmental factors, including exposure to digital screens, on the development and progression of DED.

3.2Impact of Digital Screen Exposure on the Ocular Surface

With the widespread use of digital devices, the number of people using them daily has increased significantly (Ha et al., 2025). A growing body of research indicates that digital screen use may affect tear film homeostasis through behavioral and environmental mechanisms, contributing to the development or exacerbation of DED symptoms (M. Uchino et al., 2013; Wang et al., 2021).

One of the main mechanisms underlying ocular surface disorders associated with screen use is a reduction in spontaneous blinking. Under physiological conditions, the average blink rate at rest is approximately 17 per minute (Bentivoglio et al., 1997), but when using digital screens this rate can drop significantly. One study showed a decrease in blink rate from 18.4 blinks/minute before working at a computer to 3.6 blinks/minute while using the device (Hunter et al., 1991).

In addition to the reduced frequency, incomplete blinking is observed, which is caused by the upper eyelid not completely covering the cornea. Some studies suggest that incomplete blinking may play a greater role in the pathogenesis of dry eye than blink frequency alone, because tear film stability can be maintained with an insufficient number of blinks if the blinks are complete (Hirota et al., 2013). Incomplete blinking can cause the tear film at the base of the cornea to thin, leading to its breakdown. These processes also contribute to increased tear evaporation (McMonnies, 2007).

The use of digital screens is also associated with increased exposure of the eye surface to external factors. Users tend to maintain wider palpebral fissures and prolonged fixation, especially when screens are placed at eye level. This results in greater exposure of the ocular surface to ambient air, accelerating tear evaporation (Rosenfield, 2011). Environmental factors commonly associated with screen use, such as low humidity, air conditioning, the presence of fans, and airborne dust and toner particles, can further exacerbate tear loss through evaporation and destabilize the tear film (Blehm et al., 2005).

Another key mechanism is meibomian gland dysfunction. During blinking, approximately 1/30 of the meibomian glands' contents are released from their ducts, forming a lipid coating approximately 42 nm thick that limits evaporation of the tear film's aqueous layer (Bron et al., 2004; Butovich et al., 2008). However, blinking alone is not sufficient to achieve a regular distribution of the appropriate volume of sebaceous secretion. It has also been suggested that thinning of the lipid layer in the upper segment of the cornea may play a significant role, causing increased surface tension and the release of glandular secretions through the network of ducts in the lower eyelid (King-Smith et al., 2004). Reduced and incomplete blinking when using screens leads to insufficient stimulation of the glands, resulting in decreased lipid secretion and stagnation of meibomian secretions. Studies have shown that individuals who regularly use digital devices are more likely to exhibit features of MGD. These include abnormalities of the eyelid margins, decreased quality of meibomian secretions, and increased meibomian gland atrophy on meibography (Mehra & Galor, 2020). Results from other studies show that individuals with MGD have a statistically significant higher incidence of conjunctival symptoms and lower basal tear secretion (Fenga et al., 2008).

Screen-related disorders don't just affect the lipid layer of the tear film. The importance of the mucin layer, and in particular MUC5AC secreted by goblet cells in the conjunctiva, should be emphasized. Its hydrophilic nature allows it to moisten the ocular surface, thus contributing to its cleansing of contaminants (Gipson, 2007; Gipson et al., 2004; Inatomi et al., 1996). A study was conducted which showed that people who used VDT for more than 5 hours had statistically lower MUC5AC expression levels compared to the group using VDT for a shorter period (Y. Uchino et al., 2014).

Prolonged exposure to screens can also contribute to inflammation of the ocular surface. Elevated levels of numerous proinflammatory cytokines, including IL-2, IL-4, IL-5, IL-6, IL-10, IFN-γ, TNF-α, IL-1β, and the chemokine IL-8, are observed in the tears of patients with dry eye disease. Their levels correlate with disease severity. The increased expression of corresponding genes in the conjunctiva suggests that the increased levels of these mediators are primarily due to their excessive production and not solely to the tear-thickening effect of evaporation (Massingale et al., 2009). Other studies have shown that levels of the same cytokines are significantly higher in people who use computers for more than 3 hours a day compared to those with shorter exposure times (Gupta et al., 2025).

Neurosensory mechanisms are also involved in ocular surface dysfunction associated with screen use. Prolonged visual attention and reduced blinking rate can disrupt normal feedback loops within the tear function unit. Over time, these changes can affect corneal sensitivity, leading to a discrepancy between the severity of symptoms reported by patients and the degree of objective clinical changes observed during ophthalmological examination (Galor et al., 2018).

These mechanisms reinforce each other, creating a self-perpetuating cycle of ocular surface destabilization. Reduced blinking and increased evaporation promote tear film instability and hyperosmolarity, which in turn induce inflammatory reactions and epithelial damage. Current research highlights that exposure to digital screens acts as a significant factor in modifying this cycle, amplifying underlying vulnerabilities to ocular surface damage.The mechanisms linking prolonged digital screen use to ocular surface disorders and the development of dry eye syndrome are illustrated in Figure 1.

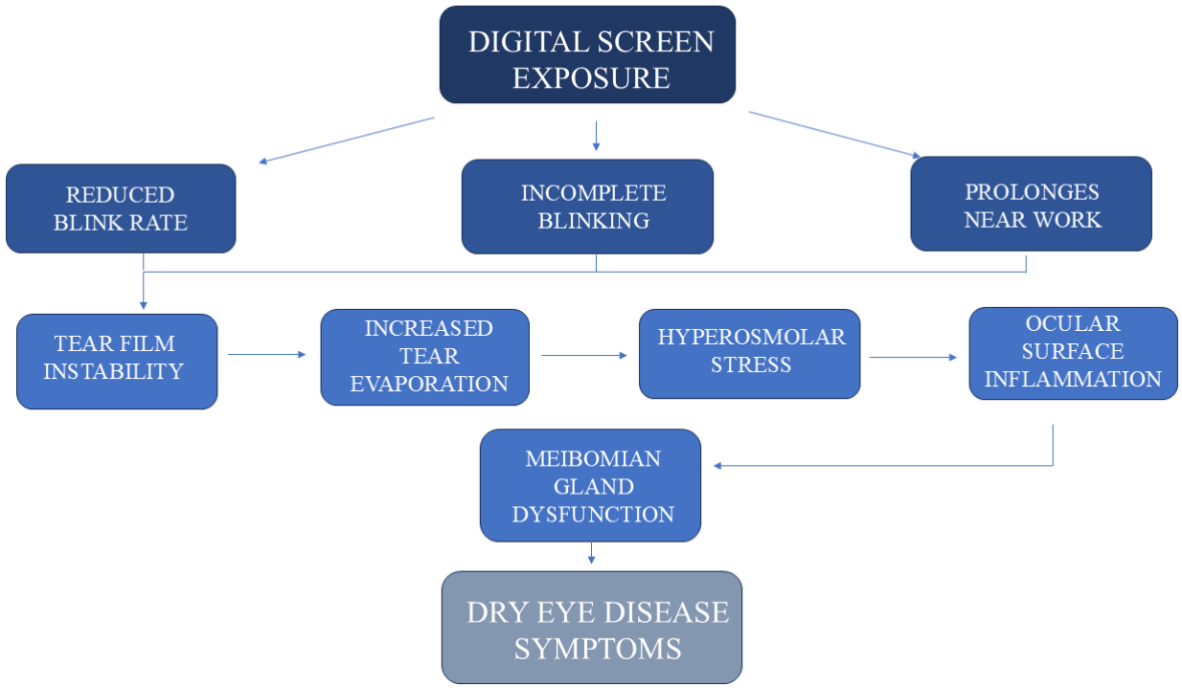

Fig. 1. *Proposed mechanisms linking prolonged digital screen exposure with tear film instability and dry eye disease.*

3.3 Epidemiological and Clinical Evidence of Screen-Related Dry Eye Disease

Studies indicate that the prevalence of dry eye symptoms varies significantly depending on the population studied, the diagnostic criteria used, and the duration of exposure to screens (Aljarousha et al., 2024; Shanti i in., 2020).

One of the best-studied occupational groups is office workers. One study found that the prevalence of definite or probable DED among this group can range from 26% to 70%. Screen exposure time has also emerged as a significant factor. Even 1-2 hours of daily VDU exposure has been shown to be associated with the development of DED (Fjaervoll et al., 2022). In another study, participants using display screens for approximately six hours per day underwent a comprehensive ophthalmological assessment, including questionnaires regarding dry eye symptoms and their impact on quality of life. Clinical tests, such as fluorescein and lissamine green staining of the cornea and conjunctiva, tear film break-up time (TBUT), and the Schirmer test, were also performed. The high prevalence of symptoms, particularly dryness, eye fatigue, and difficulty keeping the eyes open for 10 seconds, led the authors to conclude that symptoms consistent with DED may be present in up to approximately 60% of VDT users (Kawashima et al., 2015).

Studies also highlight the significant impact of DED on job performance. One study estimated annual productivity losses among Japanese touchscreen users with DED at approximately $6,160 per employee in total productivity and $1,178 in wage losses (M. Uchino et al., 2014).

Similar findings have been observed in younger populations. In a cross-sectional study of undergraduate business and medical students in Saudi Arabia, 55.5% suffered from mild, moderate, or severe dry eyes associated with prolonged computer use. Business students were also found to be approximately 1.6 times more likely to develop computer vision syndrome (CVS) than medical students (Al Tawil et al., 2020).

Children and adolescents constitute another group particularly at risk for developing DED symptoms associated with digital exposure. Studies conducted in the pediatric population have shown that children who use smartphones for extended periods had a shorter tear film breakup time and a higher incidence of dry eye symptoms compared to children with less exposure to digital devices. The prevalence of smartphone use was 65.1% among older children and 50.9% in the younger group, while the prevalence of DED reached 9.1% and 4%, respectively. A significant observation was the improvement in both subjective symptoms and clinical parameters after 4 weeks of smartphone restriction in children with DED, which may suggest the partially reversible nature of ocular surface changes associated with long-term digital exposure (Moon et al., 2016).

The impact of increased exposure to digital devices has also become particularly evident during the COVID-19 pandemic. This is confirmed by the results of a French survey conducted during the first lockdown. The survey, active between days 8 and 13 of quarantine and involving 11,391 respondents, showed that 64% of participants reported increasing their screen time during the period of social isolation (Rolland et al., 2020). Furthermore, a study conducted among children aged 13 ± 2.45 years found that during the pandemic, the average time spent using digital devices increased to 3.9 ± 1.9 hours per day compared to the pre-COVID-19 period. The increase was particularly pronounced in intensive screen use. 36.9% of the children surveyed used digital devices for more than 5 hours per day during the pandemic, compared to only 1.8% before the pandemic (Mohan et al., 2021).

3.4 Prevention and Management Strategies

The increasing incidence of DED highlights the importance of effective strategies aimed at reducing symptoms in patients. An example and most common strategy used to prevent dry eye syndrome caused by excessive digital device use is the 20-20-20 rule. This involves taking regular 20-second breaks every 20 minutes of screen time and focusing your eyes on an object approximately 6 meters (20 feet) away. This reduces visual strain and promotes more frequent blinking. This strategy has also been shown to reduce subjective symptoms such as dry eyes and eye strain while promoting normal blinking patterns (Talens-Estarelles et al., 2023).

Conscious blinking training is also an important element of prevention. Excessive and consistent use of digital screens is associated with reduced blinking frequency, increased incomplete blinking, tear film instability and, ultimately, visual impairment due to evaporation. A key strategy in this case is to consciously increase blinking frequency and ensure a complete blink. Proper blinking allows for even distribution of the tear film across the eye's surface, limiting excessive evaporation and reducing corneal friction. It also promotes the distribution of lipids produced by the meibomian glands, which stabilizes the tear film's lipid layer. Regular blinking also supports the function of the orbicularis oculi muscle, which is involved in the proper outflow and distribution of tears, which may contribute to reducing the severity of DED symptoms (Arita et al., 2025).

Workstation modifications can play a significant role in reducing dry eye symptoms. Placing a digital screen approximately 15-20 degrees below eye level reduces the width of the palpebral fissure, limiting exposure to the ocular surface, which translates into reduced tear evaporation (JASCHINSKI* et al., 1999). Maintaining the proper eye distance from the screen is also crucial. Around 50–70 cm is considered optimal. Research indicates that properly adjusting the monitor's brightness and contrast, and avoiding excessive exposure to light from both the screen and overhead lighting or windows, can also be beneficial. Using blinds, curtains, lower-intensity light bulbs, and anti-reflective filters to reduce reflections from the monitor surface can also be helpful. Properly arranging reference materials is also important, as it reduces the need for frequent head movements and varying eye exposure to varying light levels (Kahal et al., 2025).

The evaporation of tears, and therefore the intensification of DED symptoms, is also promoted by low air humidity, the influence of air conditioning, and unfavorable conditions in the work room. To avoid this, it's recommended to use humidifiers, avoid direct airflow towards the eyes, and improve ambient air quality, especially in environmental conditions. Adhering to these strategies helps maintain proper tear film stability (Chlasta-Twardzik et al., 2021; Hirayama et al., 2013).

If symptoms persist despite behavioral modifications, artificial tear supplementation is recommended, which is the cornerstone of treatment. These restore tear film stability and reduce corneal friction, leading to relief of symptoms such as dryness, discomfort, and fatigue. In recent years, numerous studies have been conducted to determine the effectiveness and compare various artificial tear preparations. It turns out that even over-the-counter products can be safely used (Pucker i in., 2016), but it's crucial that they be preservative-free.

Individuals with meibomian gland dysfunction are at higher risk of developing DED symptoms than the healthy population. Therefore, treating meibomian gland dysfunction is crucial, especially among individuals who frequently use digital screens. Eyelid temperature and the melting point of lipid secretions are fundamental to the proper release of meibomian gland secretions. In healthy individuals, the eyelid temperature is 33-37°C. The melting point ranges from 19.5°C to 33.8°C in healthy individuals, while in patients with MGD it is higher, ranging from 32.2°C to 35.3°C (Terada et al., 2004). To prevent meibomian gland dysfunction, warm compress therapy is recommended, which improves the production of secretions and the quality of the lipid layer. Instead of compresses, it is also suggested to use special eyelid warming devices, the effectiveness of which has been confirmed by research (Arita et al., 2015). Proper eyelid hygiene is also important. Regular eyelid cleansing strategies can significantly reduce glandular obstruction, improving tear film stability and thus reducing the

severity of DED symptoms. Furthermore, regular removal of crusts around the eyelid margins reduces the risk of bacterial infection, which can contribute to the development of meibomian gland failure.

In situations where eyelid margin cleansing alone is not sufficient, in-office treatment using intense pulsed light (IPL) therapy may be considered (Craig et al., 2015). This method involves delivering light pulses of a carefully selected wavelength to the tissues of the eyelid region, which leads to a selective thermal effect. This may result in improved fluidity of meibomian gland secretions, reduced obstruction of the tear ducts, and stabilization of the lipid layer of the tear film. Reports also indicate that IPL may have anti-inflammatory effects and reduce the severity of symptoms in patients with evaporative DED associated with MGD (Craig et al., 2015).

In some individuals with severe symptoms or who have not benefited from primary treatment, pharmacological treatment may be necessary. Topical cyclosporine therapy has been shown to reduce ocular surface inflammation, improve tear production as measured by the Schirmer test (in 15% of patients compared to 5% in controls), and reduce the severity of subjective symptoms in patients with DED. The anti-inflammatory effect of cyclosporine is understood to mean a reduction in the number of cells that activate lymphocytes, which ultimately leads to the interruption of the ongoing immune response in the body (Kunert et al., 2000; Sullivan et al., 2012). Cyclosporine, also having antiapoptotic functions, is responsible for the protection of conjunctival epithelial cells by blocking intrinsic and extrinsic apoptotic pathways, but at the same time it stimulates the apoptosis of T lymphocytes by increasing the activity of Fas/FasL and caspases (Gao et al., 2013). Additionally, it turns out that cyclosporine has the ability to increase the density of goblet cells, as well as the production of the immunoregulatory factor – TGF-β2 in the bulbar conjunctiva (Pflugfelder et al., 2008).

Physicians play a crucial role in DED prevention, as they are responsible for educating patients about the impact of digital exposure on the ocular surface. Many patients are still unaware of the impact digital screens have on the ocular surface. Strategies such as regular breaks, conscious blinking, and environmental modifications form the basis of preventative recommendations and play a crucial role in alleviating patient discomfort.The most commonly recommended preventive and therapeutic strategies for screen-related dry eye are summarized in Figure 2.

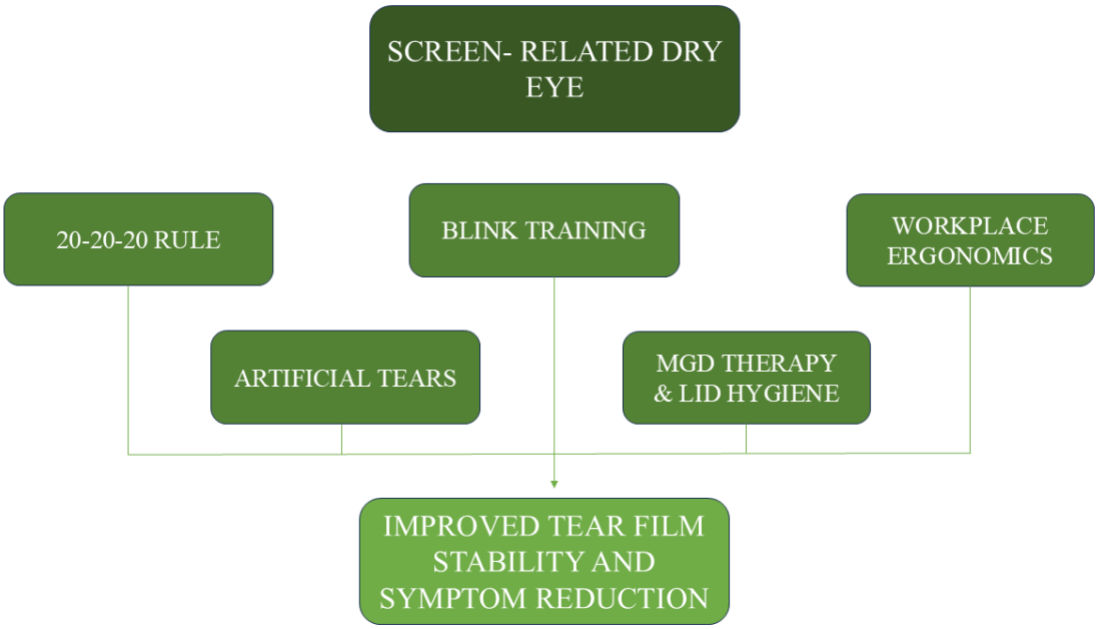

Fig. 2. *Overview of preventive and therapeutic strategies for screen-related dry eye disease*

Achieving optimal therapeutic outcomes requires an approach that combines behavioral interventions, environmental modification, and targeted therapy. This combination allows for better adaptation to individual risk factors and patient needs, ultimately achieving the best possible clinical outcomes.

3.5 Future Directions

Despite significant progress in understanding the relationship between digital screen exposure and dry eye disease, current research remains plagued by numerous limitations. Addressing these issues will be crucial to improving the diagnosis, prevention, and treatment of ocular surface disorders associated with digital device use.

One key limitation of current research is the predominance of cross-sectional studies. Although such studies consistently demonstrate an association between long-term screen exposure and DED symptoms, they do not allow for a clear cause-and-effect relationship, but rather merely indicate the co-occurrence of the phenomena analyzed at a specific point in time. As emphasized in the studies, there is a need for well-designed longitudinal and prospective studies to better understand the temporal relationship between environmental risk factors and the development of DED (Stapleton et al., 2017).

Another significant challenge is the lack of standardization in both the diagnosis of DED and the assessment of digital screen exposure. Comparison between studies is significantly hampered by variability in diagnostic criteria resulting from differences in symptom questionnaires and objective clinical tests. This problem is particularly evident in parameters such as tear film breakup time (TBUT) and tear osmolarity. An additional limitation is that most available studies rely on subjective assessments of screen time, which may impact the accuracy and reproducibility of the obtained results. Therefore, future research should focus on developing more standardized and objective methods for assessing digital exposure and ocular surface parameters (Wolffsohn et al., 2017).

The long-term effectiveness of preventive strategies is another area requiring further research. Although interventions such as regular breaks, blink awareness training, and ergonomic adjustments are widely recommended, evidence supporting their effectiveness remains limited. Existing studies emphasize the importance of developing evidence-based behavioral recommendations for individuals exposed to prolonged digital exposure.

Furthermore, future research should examine the interaction between digital screen use and other risk factors, such as low humidity, environmental pollution, indoor conditions, and hormonal influences. The multifactorial nature of DED suggests that these variables may act synergistically, influencing both symptom severity and disease progression (Galor et al., 2023).

Furthermore, further research may help optimize the management of screen-related dry eye. The TFOS DEWS II Management and Therapy Report emphasized the importance of individualizing treatment strategies, particularly in patients with evaporative dry eye and meibomian gland dysfunction. A better understanding of the specific mechanisms associated with prolonged digital exposure may help optimize treatment recommendations in the future (Jones et al., 2017).

In summary, future research should focus on long-term research projects, standardized methodologies, objective exposure assessment, and the development of targeted preventive and therapeutic strategies. A better understanding of these relationships may be crucial for effective prevention, diagnosis, and treatment of dry eye disease in the face of the increasing digitalization of everyday life.

4. Discussion

Modern lifestyles are associated with increasing exposure to digital devices, the impact of which on the ocular surface is gaining significant clinical significance. Although dry eye syndrome remains a multifactorial disease, the presented data suggest that prolonged use of digital devices may exacerbate the underlying pathophysiological mechanisms of DED, particularly in individuals predisposed to impaired tear film homeostasis (Bron et al., 2017; Craig et al., 2017; Wolffsohn et al., 2023).

The behavioral changes observed during screen use are consistent with the mechanisms responsible for the development of evaporative DED. Reduced blink frequency, an increase in the number of partial blinks, and prolonged visual attention lead to tear film destabilization and increased evaporative stress. These disturbances simultaneously affect several components of the tear function, encompassing both the integrity of the lipid layer and the neurosensory regulation of the ocular surface. In clinical practice, this may partially explain why symptoms characteristic of the evaporative form and coexisting meibomian gland dysfunction predominate in individuals who use digital devices extensively (Bron et al., 2017; Nichols et al., 2011; Wolffsohn et al., 2023).

The changing epidemiological profile of dry eye disease is receiving increasing attention. DED has traditionally been associated primarily with advanced age, hormonal changes, and autoimmune diseases. However, a growing body of evidence now indicates the frequent occurrence of ocular surface symptoms in

younger population groups, including students, children, and adolescents (Mohan et al., 2021; Moon et al., 2014; Zarban et al., 2024). This may suggest that contemporary lifestyle factors, particularly chronic exposure to digital devices, play an increasingly important role in shaping the epidemiology of DED.

Digital exposure is particularly important in the pediatric population. Unlike traditional risk factors for dry eye syndrome, digital device use now begins in early childhood and often extends to many hours a day. The potential impact of long-term exposure on the developing ocular surface remains poorly understood, but the observed increase in the incidence of dry eye symptoms in children may have significant future health consequences (Mohan et al., 2021; Moon et al., 2014). This phenomenon is gaining importance in the context of the ongoing digitalization of education and the growing dependence of everyday functioning on mobile devices.

The discrepancy between patient-reported symptom severity and clinical findings remains problematic. In ophthalmological practice, patients who use screens extensively often report significant discomfort despite relatively minor deviations in standard diagnostic tests. This may indicate the involvement of neurosensory mechanisms and the limitations of currently used methods for assessing DED (Belmonte et al., 2017; Bron et al., 2017). It is also indicated that the assessment of patients with suspected screen-related dry eye should include not only the classic tear film parameters, but also the analysis of visual behavior, work environment and the subjective impact of symptoms on daily functioning.

Symptoms associated with screen use aren't always solely due to ocular surface disorders. Digital eye strain also includes accommodative, muscular, and ergonomic components related to prolonged close-up work and visual system overload (Rosenfield, 2011). The line between digital eye strain and DED is often blurry, and symptoms of both conditions can coexist and exacerbate each other. In practice, this can make it difficult to clearly differentiate the two conditions and require a more thorough patient assessment.

Of significant clinical importance is the fact that digital exposure represents a potentially modifiable risk factor. Unlike many classic determinants of DED, such as age or systemic disease, screen-related behavior can be partially managed through patient education, improved work ergonomics, and appropriate preventative strategies. Even relatively minor behavioral changes can limit tear film destabilization and reduce symptom severity in individuals with chronic digital exposure (Rosenfield, 2011; Wolffsohn et al., 2023).

Available data suggest that screen-related dry eye should now be considered not only an individual problem but also a growing public health challenge. The prevalence of digital devices, increasingly earlier exposure to screens, and the predicted further increase in screen time mean that the importance of technology-related ocular surface disorders will likely continue to grow. Considering the impact of digital behaviors in daily ophthalmological practice can improve both early diagnosis of DED and the effectiveness of preventive and therapeutic management.

5. Conclusions

Daily use of digital devices has become an integral part of modern lifestyle and is increasingly associated with ocular surface symptoms (Mehra & Galor, 2020; Rosenfield, 2011). The increasing incidence of dry eye symptoms, also among young people, highlights the importance of lifestyle factors and the digital environment in modern ophthalmology (Mehra & Galor, 2020; M. Uchino et al., 2008). Importantly, however, digital exposure remains a potentially modifiable factor, enabling the implementation of preventive measures that can limit the severity of symptoms and improve patient well-being. In clinical practice, considering digital behavior, visual work conditions, and environmental factors when assessing patients with symptoms of dry eye and digital eye strain is becoming increasingly important. As everyday life becomes increasingly digital, the problem of ocular surface disorders associated with screen use will continue to grow. Therefore, ocular surface disorders associated with screen use will likely pose an increasing clinical and health challenge.

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