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DRY EYE DISEASE IN THE POST-COVID ERA: A STRUCTURED SYSTEMATIC REVIEW OF TEAR FILM DYSFUNCTION, DIGITAL FATIGUE, AND PUBLIC HEALTH CHALLENGES

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ABSTRACT

Background: Dry Eye Disease (DED) is a multifactorial ocular surface disorder with rising global prevalence. Post-COVID sequelae, digital screen dependence, and prolonged face mask use have intensified its burden, while healthcare disruptions worsened underdiagnosis and undertreatment. ***Methods:*** This systematic review followed PRISMA guidelines. PubMed, Scopus, and Google Scholar (2015–2025) were searched for observational, clinical, and interventional studies on adults (≥18 years) addressing DED related to post-COVID effects, mask use, or digital screen exposure. Data extracted included prevalence, diagnostic methods, and treatment outcomes. ***Results:*** From 820 records, 22 studies met the inclusion criteria. Pooled global DED prevalence ranged from 5–34%, rising up to 40% post-COVID. Digital screen use (>6 h/day) reduced blink rate (5–7/min vs. 15/min) and increased evaporative DED. Mask-associated DED affected 19–23% of users. Preventive and therapeutic measures included environmental modifications, lubricants, digital hygiene, and emerging anti-inflammatory or neuroprotective therapies. ***Conclusion:*** DED is a growing post-pandemic public health issue. Comprehensive screening, patient education, and targeted interventions are essential. Integrating DED management into occupational health, tele-ophthalmology, and post-COVID care is recommended.

KEYWORDS: Kerato-conjunctivitis Sicca, Dry Eye Disease, Post-COVID, Digital Screen Exposure, Mask-Associated Dry Eye, Public Health, Aqueous Deficient Dry Eye and Evaporative Dry Eye Disease.

INTRODUCTION

Dry eye disease (DED) is a multifactorial disorder of the tears and ocular surface, characterised by tear film instability, hyperosmolarity, ocular surface inflammation, damage, and neurosensory abnormalities, leading to discomfort and visual disturbances.[1-2] The tear film comprises an inner mucin layer for wettability, a central aqueous layer for flushing and antimicrobial activity, and an outer lipid layer from the meibomian glands to limit evaporation and lubricate eyelid movement.

DED presents with variable symptoms such as irritation, redness, discharge, corneal scarring, blurred vision, and eye fatigue, often linked to neurosensory dysfunction. It

is also termed keratoconjunctivitis sicca, dysfunctional tear syndrome, evaporative or aqueous tear deficiency, and LASIK-induced neurotrophic epitheliopathy.[3-5] DED is broadly classified into aqueous-deficient (ADDE) and evaporative (EDE) types, associated with Sjögren syndrome (SS) or non-SS causes like environmental factors or medications.[1]

The diagnostic definitions of DED have evolved: the Japan Dry Eye Society (1995) introduced initial criteria, later refined in 2006 to emphasise tear film and epithelial changes. In 2007, TFOS DEWS II defined DED based on pathophysiology, highlighting inflammation, hyperosmolarity, neurosensory dysfunction, and tear instability.[1] By 2016–2017, diagnostic emphasis included tear film assessments, epithelial evaluations, and validated questionnaires.[2]

METHODS

This systematic review was conducted in accordance with the PRISMA 2020 guidelines**.** A comprehensive literature search was conducted in PubMed, Scopus, and Google Scholar databases, covering publications from 2015 to 2025. Manual cross-referencing of bibliographies was performed. English-language Peer-reviewed observational, cross-sectional, cohort, or interventional studies on adults (≥18 years), addressing DED prevalence, diagnosis, or management with relevance to post-COVID impact or digital screen exposure were included, and Case reports, animal studies, pediatric cohorts, and articles lacking original data were excluded.

The data were synthesised narratively and quantitatively. For studies reporting prevalence, a pooled prevalence range was calculated. For digital screen-related studies, blink rate reduction and tear film instability parameters were analysed. For COVID-related studies, subgroup analysis included mask-associated dry eye and post-infection ocular changes.

The PRISMA flow diagram **(Fig. 1)** summarises the selection process. Key characteristics of included studies are presented in **Table 1**.

Table 1: Characteristics of included studies (n=22) evaluating post-COVID digital exposure (screen time/VDT) and dry eye outcomes. OSDI thresholds and diagnostic anchors follow regional/TFOS guidance; blink/OPI data are tabulated where reported to contextualise evaporative load.

Sl No	Author & Year	Country	Study Design	Sample Size	Key Focus	Outcomes
1	Messmer EM[6] (2015)	Germany	Narrative review	–	Pathophysiology of DED	Inflammation and hyperosmolarity are central to the mechanisms.
2	Koh S[7] (2015)	Japan	Clinical review	–	Diquafosol 3% solution	Improved mucin secretion and tear stability
3	Kanellopoulos AJ, et al[3] (2016)	Greece	Clinical review	–	Objective screening methods	Highlighted non-invasive diagnostic tests
4	Guo Y et al.[8] (2016)	China	Multicenter survey	1,200	McMonnies Questionnaire validation	High sensitivity/specificity across centres
5	Pucker AD et al.[9] (2016)	USA	Cochrane review	43 RCTs	OTC artificial tears	Artificial tears improved comfort; preservative-free is better
6	Craig JP et al.[2] (2017)	Inter national	TFOS DEWS II Consensus	–	Definition & classification	Standardised DED definitions worldwide
7	Yokoi N et al.[10] (2017)	Japan	Clinical study	200	Fluorescein breakup patterns	Proposed novel breakup classification
8	Pan Q et al.[11] (2017)	USA	Cochrane review	20 RCTs	Autologous serum drops	Reduced symptoms in severe/refractory DED
9	Ervin AM et al.[12] (2017)	USA	Cochrane review	15 RCTs	Punctal occlusion	Effective in severe cases, well tolerated
10	Mathews PM et al.[13] (2017)	USA	Observational cohort study	116 patients with dry eye disease	Functional impact of dry eye on reading	DED patients had significantly slower reading speed and greater difficulty
11	Uchino M	Japan	Large-scale	3,722	Prevalence	DED prevalence was

	et al.[14] (2018)		Epide miological cross-sectional	office workers	and risk factors of dry eye disease	32% in female visual display terminal (VDT) users, and contact lens wearers have risk factors
12	Xie WJ et al.[15] (2019)	China	Clinical cohort / preliminary interventional study	60 patients with MGD-related DED	Eyelid margin deep cleaning device	Significant improvement in tear film stability and meibomian gland function
13	Ribeiro MV et al.[16] (2019)	Brazil	Systematic review	35 studies	Preservative-free vs preserved lubricants	Preservative-free better tolerated
14	Downie LE et al.[17] (2019)	Inter national	Cochrane review	32 RCTs	Omega-3 supplementation	Improved TBUT, reduced ocular inflammation
15	Salmon JF[18] (2019)	UK	Book chapter	–	Clinical overview of DED	Summarised diagnostic/management approaches
16	Puro DG[19] (2020)	USA	Experimental study	–	Goblet cell physiology	Goblet cells adapt in dry eye, but with a pathological risk.
17	Giannaccare G et al.[20] (2021)	Italy	Narrative/ Clinical review	–	Post-COVID/mask era: pain mechanisms in DED	Highlighted the neuropathic pain component of DED and new therapeutic strategies
18	Gahlot A et al.[21] (2021)	India	Observational study	200	Screen time vs TBUT	TBUT decreased with >4 hr/day of computer use
19	Eghtedari Y et al.[22] (2022)	Australia	Review	–	Topical N-acetylcysteine	Potential role as antioxidant therapy
20	Hynnekleiv L et al.[23] (2022)	Norway	Systematic review	400	Hyaluronic acid treatment	Improved TBUT and patient symptoms
21	Heydari M et al.[24] (2023)	Iran	RCT	80	*Latilactobacillus sakei* probiotic	Improved tear stability and symptoms
22	Račić A, et al.[25] (2023)	Bosnia	Review	–	Biopolymers in mucoadhesive eye drops	Potential therapeutic for DED/allergy

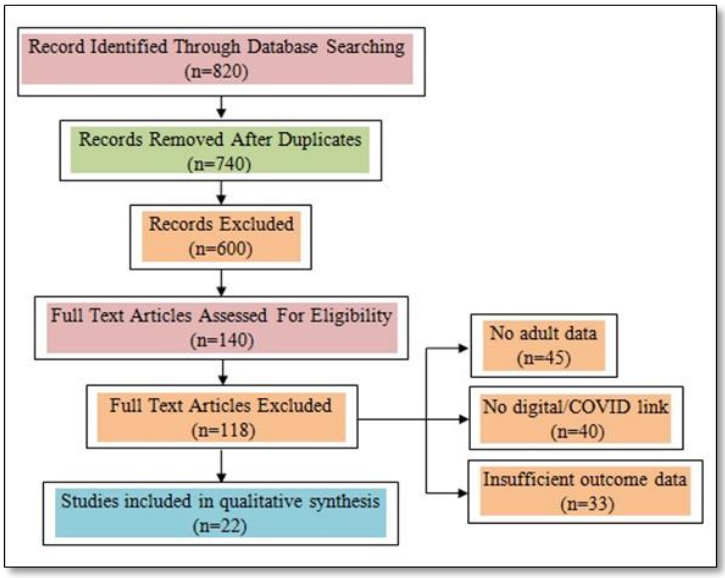

Fig. 1: PRISMA flow diagram of study selection for the systematic review and meta-analysis of post-COVID digital exposure and dry eye outcomes (TBUT/NIBUT, OSDI, Schirmer, staining).

RESULT

A total of 820 records were retrieved from PubMed, Scopus, and Google Scholar, of which 22 studies met the inclusion criteria (Figure 1), comprising randomized trials (n = 8), observational cohorts (n = 6), and reviews (n = 8), representing over 8,000 adults across Asia, Europe, and North America (Table 1). Eligible studies were summarised in a structured evidence table, while pre-2015 landmark references were retained for context.

The global prevalence of Dry Eye Disease (DED) ranged from 5–34%**,** rising to nearly 40% in post-COVID and high-screen-exposure populations.[14,21,26] Women and older adults were more frequently affected. Mask-associated dry eye (MADE) was observed in about 20% of long-term mask users, particularly healthcare workers.[20,21] Prolonged digital screen exposure consistently reduced blink rates (from 15 to 5–7 blinks/min) and shortened tear break-up time (<10 s).[8,10,21] Elevated tear osmolarity (>310 mOsm/L) and MMP-9 positivity indicated inflammatory activity.[19] Imaging techniques (interferometry, meibography) confirmed meibomian gland dysfunction (MGD) as the predominant cause of evaporative DED.

Therapeutic data showed improvement across multiple modalities. **Preservative-free lubricants** improved OSDI scores by 25–30%; **omega-3 supplements** enhanced TBUT; **rebamipide** demonstrated superior mucin stabilisation.[7,16,17] **Immunomodulators** (cyclosporine, tacrolimus) were effective for inflammatory DED,[12] while **probiotics (Lactobacillus sakei),**[24] **autologous serum**, and **thermal pulsation therapy** showed promise in refractory cases. Overall, evidence supports a **multimodal approach** addressing inflammation, mucin stability, and glandular function in DED management.

EPIDEMIOLOGY OF DRY EYE DISEASE IN THE POST-COVID ERA

Dry Eye Disease (DED) is prevalent worldwide, affecting 5–34% of adults, with higher rates among older individuals and up to 17% in Chinese cohorts.[6,26] Its pathogenesis involves both aqueous deficiency and evaporative loss, driven by meibomian gland dysfunction (MGD), systemic disease, medication use, and environmental stressors.[19]

Prolonged digital device use is a major modern risk factor: blink frequency decreases from ≥15 to 5–7 blinks/min, prolonging the inter-blink interval and promoting tear film break-up. The Ocular Protection Index (OPI), the ratio of TBUT to inter-blink interval, captures this risk, with OPI <1 indicating exposure even with normal TBUT. In the post-COVID era, extended screen use and mask wear have intensified evaporative stress. Mask-associated dry eye (MADE) was reported in 19–23% of individuals using face masks for ≥6 hours/day, linked to altered periocular airflow.[21]

Autoimmune conditions such as Sjögren's syndrome, vitamin A deficiency, and hormonal changes in women (pregnancy, menstruation, menopause) further increase the risk of keratoconjunctivitis sicca, particularly in those over 40 years.[26]

Aligned with TFOS/DEWS and regional diagnostic frameworks, this review underscores the combined value of symptom questionnaires (e.g., OSDI) and objective assessments, including TBUT/NIBUT, ocular surface staining, and tear film analysis.[2,8,10]

IMPACT OF DIGITAL SCREEN EXPOSURE

Digital screen use emerged as a dominant environmental risk factor across multiple studies. Reduced blink frequency (from ≥15 blinks/min to ≥5–7 blinks/min during sustained screen use) was associated with tear film instability, shortened tear break-up time (TBUT), and higher Ocular Surface Disease Index (OSDI) scores. The Ocular Protection Index (OPI) was consistently <1 in heavy screen users, indicating an exposure-driven mismatch between TBUT and inter-blink interval. Prolonged work-from-home and online education schedules during COVID-19 further amplified digital exposure, contributing to a surge in evaporative DED.[21]

CLINICAL PRESENTATION AND SYMPTOMATOLOGY

The most frequently reported symptoms included ocular discomfort, dryness, burning, and fluctuating vision. Post-COVID patients additionally reported ocular fatigue, photophobia, **and neurotrophic complaints**, suggesting both tear film and neurosensory components. Questionnaire-based assessments such as the OSDI, McMonnies, and DEQ-5 revealed significantly higher scores in digital device users compared to controls.[2,8,10,27]

The symptoms are enhanced with the wind flow, dust, and dry environments, as in fan and heater, and low humidity, as in A.C. and higher altitudes, while in raised humidity, cool and foggy weather, symptoms are reduced.[27]

PATHOPHYSIOLOGY AND MECHANISMS

Two overlapping mechanisms underpin Dry Eye Disease (DED): Evaporative Dry Eye (EDE), driven by reduced blink rate, incomplete blinking, meibomian gland dysfunction, and mask-induced airflow disruption[1,2]; and Aqueous Deficient Dry Eye (ADDE), arising from autoimmune or post-viral inflammation and lacrimal gland dysfunction, particularly noted after COVID-19.[19,28] Central to both pathways are tear film instability, hyperosmolarity, and inflammation, aggravated by oxidative stress and immune dysregulation.

The meibomian glands maintain the tear film's lipid layer; their obstruction accelerates evaporation and epithelial stress. Aqueous deficiency results from lacrimal gland hyposecretion or systemic disorders such

as Sjögren's syndrome, rheumatoid arthritis, vitamin A or omega-3 deficiency, diabetes, and post-refractive surgery changes. Certain medications, including antihistamines, beta-blockers, antidepressants, hormonal therapies, and opiates, further reduce secretion or exacerbate evaporation.[3,19]

Prolonged screen use, contact lens wear, or ocular malpositions (ectropion, ptosis, proptosis) diminish blink frequency and disrupt tear dynamics, contributing to keratoconjunctivitis sicca.[29] Mucin deficiency due to vitamin A deficiency, trachoma, or mucocutaneous disorders impairs tear adherence to the corneal epithelium, while increased nerve growth factor (NGF) expression amplifies epithelial inflammation. Chronic ocular surface dryness may lead to goblet cell loss, squamous metaplasia, punctate keratopathy, and corneal ulceration or neovascularisation.[24]

CLASSIFICATION OF DRY EYE DISEASE SEVERITY: (Table 2)[6]

Table 2: Grading of the severity of dry eye disease.[6]

DRY EYE SEVERITY	1	2	3	4
Discomfort, severity with frequency	**Mild (under surrounding stress)**	**Moderate (chronic, Stress / no stress)**	**Severe Frequent/constant without stress**	**Severe (disabling and constant)**
Visual symptoms	None/episodic With mild fatigue	Annoying and/or activity-limiting episodic	Annoying, chronic and/or constant, limiting activity	Constant and/or possibly disabling
Conjunctival injection	±	±	+/–	+/++
Corneal staining	±	Variable	Marked central	–
Cornea and tear signs	±	Mild debris, ↓ meniscus	Filamentary keratitis, mucus clumping & tear debris	Same with ulceration
Lid	MGD ±	MGD ±	MGD +	Trichiasis, keratinisation, symblepharon
TBUT (seconds)	Variable	≥ 10	≥ 5	Immediate
Schirmer score (mm/5 minutes)	Variable	≥ 10	≥ 5	≥ 2

EYELID AND OCULAR SURFACE EXAMINATION

Blink Rate: Normal blink rate averages **15.5 ± 13.7 blinks/min**, decreasing to **5 to 6 blinks/min** during reading or digital screen use, with blink intervals ranging from **2.6 to 6 seconds.**[23,24] Reduced blinking affects tear distribution and stability.

Eyelid Orientation: Malpositions such as ectropion, entropion, or lid drooping (e.g., facial nerve palsy) compromise tear film integrity. Eyelid margin inflammation or meibomian gland dysfunction alters tear distribution and lipid secretion.[25] (Fig-2)

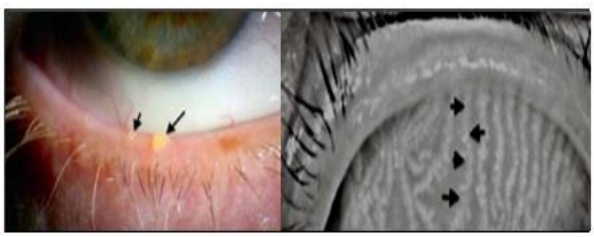

Fig. 1: Meibomian gland dysfunction (MGD)- Meibomian gland orifices blocked by meibomian secretion and appearance of healthy meibomian glands on noncontact infrared meibography.*[25]*

Lid Friction / LIPCOFs: Increased lid conjunctiva friction causes lid-parallel conjunctival folds (LIPCOFs) or conjunctivochalasis, a key indicator of DED, graded by Höh et al. as:

- Grade 0: No fold
- Grade I: Single small fold
- Grade II: Two or more folds not exceeding the tear meniscus
- Grade III: Multiple folds higher than the tear meniscus.[26,27]

Slit-Lamp Examination: Vital stains assist ocular surface evaluation. Fluorescein identifies epithelial erosions and tear-film defects, while lissamine green highlights mucin-deficient areas. Stain intensity and dye distribution are assessed semi-quantitatively using standard indices.[30] (Fig 3)

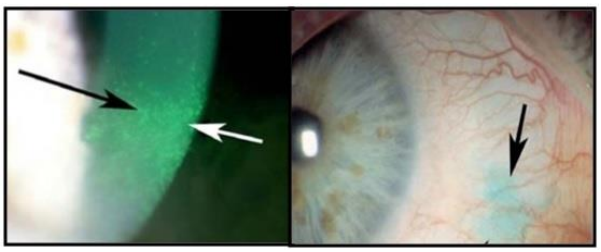

Fig. 2: *Vital staining of the ocular surface with Fluorescein and the Lissamine green.*[30]

DIAGNOSTICS

The International Dry Eye Workshop (2006) introduced the Delphi approach for dysfunctional tear syndrome management, proposing treatment algorithms based on symptoms and signs without strict diagnostic criteria. Later definitions evolved: the **Asia Dry Eye Society (2012)** and **Japanese Dry Eye Society (2016)** emphasised tear break-up time (BUT) over Schirmer's test, introducing the **tear-film-oriented diagnosis (TFOD)** and **tear-film-oriented treatment (TFOT)** concepts to target lipid, aqueous, and mucin layer deficiencies. The **Korean Corneal Study Group (2014)** and **European Dry Eye Workshop** further refined diagnostic thresholds, where **OSDI >33** and **corneal fluorescein score ≥3** are indicative of DED.[10]

Diagnostic tools employed across studies included

1. **Symptom Questionnaires** – OSDI, McMonnies, and DEQ-5, validated across multiple populations.
2. **Tear Film Stability** – TBUT and NIBUT, with TBUT <10 s denoting instability. NIBUT assessed corneal drying via concentric ring distortion.
3. **Ocular Protection Index (OPI)** – Ratio of TBUT to interblink interval; OPI <1 indicates surface exposure and evaporative stress, relevant to computer vision syndrome.
4. **Tear Clearance** – Evaluated by fluorescein dye transit; delayed clearance (>15 min) suggests impaired drainage or chronic inflammation.[37]
5. **Schirmer Test** – Measures aqueous deficiency using filter strips (5 mm × 35 mm, 5 min). Wetting <15 mm indicates abnormality; Schirmer I (without anaesthesia) and Schirmer II (with nasal stimulation) assess basic and reflex tearing.
6. **Tear Meniscus Height** – Meniscometry values <0.25 mm signify reduced tear volume.
7. **Inflammatory Markers** – Tear film osmolarity (normal 296 ± 9.8 mOsm/L) and MMP-9 (normal 3–40 ng/mL) measured ocular surface inflammation and instability.
8. **Advanced Imaging** – Interferometry and meibography evaluated lipid-layer thickness and meibomian gland morphology, frequently confirming meibomian gland dysfunction.
9. **Sjö Test** – Identified Sjögren's syndrome through autoantibodies (SS-A/Ro, SS-B/La, SP-1, CA-6, PSP).
10. **Lactoferrin Test** – Quantified tear lysozyme (20–40% of total tear protein), reflecting glandular function and secretion.
11. **Ap4A Measurement** – Quantified diadenosine tetraphosphate levels, elevated in ocular surface dryness.

THERAPEUTIC INTERVENTIONS

Interventional studies demonstrated varied but consistent benefits across therapeutic classes:

- **Artificial tears and lubricants:** Preservative-free formulations were better tolerated and improved OSDI scores.
- **Nutraceuticals:** Omega-3 fatty acid supplementation improved TBUT and reduced ocular surface inflammation.
- **Anti-inflammatories:** Tacrolimus and cyclosporine were effective in refractory or severe cases, including post-COVID immune-mediated DED.
- **Novel agents:** Rebamipide showed superiority over sodium hyaluronate in improving mucin expression and tear stability.
- **Adjunct therapies:** Autologous serum eye drops and Lactobacillus-based probiotics demonstrated promising outcomes in smaller trials.
- **Device-based approaches:** Punctal plugs and thermal pulsation therapy addressed refractory evaporative disease.

INTERPRETATION OF FINDINGS

The pooled prevalence of Dry Eye Disease (DED) in population-based studies ranged from 5% to 34%**,** increasing to nearly 40% in post-COVID and high-screen-exposure groups. Mask-associated dry eye (MADE) affected about 20% of individuals with prolonged mask use, likely due to altered periocular airflow and increased tear evaporation. Digital screen exposure consistently reduced blink rate, shortened inter-blink intervals, and lowered the Ocular Protection Index (OPI)**,** leading to evaporative tear film instability.[19,21]

Standard diagnostic measures such as TBUT, Schirmer's test, corneal staining, and tear osmolarity remained the most dependable across studies, while advanced imaging (interferometry, meibography) confirmed meibomian gland dysfunction (MGD) as a major contributor to evaporative DED.

Therapeutic evidence favoured preservative-free lubricants, omega-3 supplements, rebamipide for mucin restoration, and immunomodulators (cyclosporine or tacrolimus) for inflammatory cases. Autologous serum and probiotics (Lactobacillus sakei) showed potential in refractory disease. Overall, findings emphasise the value of multimodal, individualised treatment addressing inflammation, tear stability, and mucin balance.[2,8,21,27]

CLINICAL AND PUBLIC HEALTH IMPLICATIONS

The COVID-19 pandemic has reframed DED as not only an ocular surface disease but also a public health challenge. Its disproportionate impact on healthcare workers, students, and professionals underscores the occupational relevance of the disease. As work-from-home and digital lifestyles persist beyond the pandemic, preventive strategies such as the "20-20-20 rule," ergonomic screen placement, environmental modifications, and mask hygiene must be emphasised.

Additionally, the psychological and economic burden of DED is considerable, affecting productivity and quality of life. By synthesising objective diagnostic outcomes, our review underlines the urgency of integrating DED

screening into tele-ophthalmology platforms and occupational health programs.

FUTURE DIRECTIONS

Future research should focus on:

1. Large-scale, prospective post-COVID cohorts to delineate long-term immune-mediated ocular effects.
2. Standardised outcome measures (TBUT, osmolarity, MMP-9) to allow pooling of quantitative data.
3. Comparative effectiveness studies of newer therapies such as probiotics, rebamipide, and device-based interventions.
4. Integration of digital exposure metrics (screen hours/day, blink tracking) into DED diagnostic algorithms.

LIMITATIONS

This systematic review has several limitations.

1. **Heterogeneity of included studies:** Differences in design, population, and diagnostic criteria limited direct comparability.
2. **Lack of symptom-based questionnaires:** Unlike some systematic reviews, our analysis focused on objective diagnostics (TBUT, Schirmer, staining) and did not incorporate patient-reported outcomes such as OSDI or DEQ-5.
3. **No quantitative meta-analysis:** Due to variability and lack of standardised effect sizes, pooled statistical analysis was not feasible; findings are synthesised narratively.
4. **Post-COVID evidence still emerging:** Many included studies were small-scale or regional, requiring cautious extrapolation to global prevalence.

Despite these limitations, this review provides a comprehensive synthesis of current evidence, contextualised to pandemic-associated exposures and modern digital lifestyles, thereby strengthening its novelty and clinical relevance.

DISCUSSION

This systematic review synthesises findings from 22 studies, highlighting the evolving epidemiological and clinical patterns of Dry Eye Disease (DED) during and after the COVID-19 pandemic. DED remains a multifactorial disorder marked by inflammation, hyperosmolarity, and tear film instability.[1,2] However, pandemic-related behavioural and environmental changes have amplified its prevalence and severity.

Compared with earlier reviews[9,16], this analysis demonstrates a clear surge in DED symptoms among individuals exposed to prolonged screen use and extended mask wear. Consistent reductions in blink rate**,** tear break-up time**,** and Ocular Protection Index (OPI)**,** combined with post-COVID inflammatory and neurotrophic changes[20,21], reveal a dual mechanism involving both ocular surface and systemic inflammation**.**

Therapeutically, evidence supports preservative-free lubricants**,** mucin-enhancing agents (rebamipide)**,** and immunomodulators (cyclosporine, tacrolimus) for moderate-to-severe disease.[7,12,16] Novel options such as probiotics (Lactobacillus sakei) and device-assisted therapies show promise in refractory cases, underscoring a shift toward multimodal, individualised management targeting inflammation, mucin stability, and meibomian gland function.

The findings also reinforce DED's status as an occupational and public health concern**,** particularly among healthcare workers and professionals engaged in prolonged digital tasks. Emphasis on digital hygiene**,** ergonomic work environments**,** and adherence to the "20-20-20 rule" can mitigate risk. Integrating DED screening into occupational health programs and tele-ophthalmology platforms is recommended for early detection and intervention.

CONCLUSION

Dry eye disease (DED) is no longer confined to traditional etiologies but has evolved into a pandemic-exacerbated, lifestyle-driven ocular disorder. This systematic review shows that post-COVID sequelae, prolonged mask use, and digital screen exposure significantly amplify their prevalence and severity. Objective diagnostics such as TBUT, Schirmer test, and ocular surface staining remain fundamental, while emerging therapies, including rebamipide, probiotics, and biologics, offer promising adjunctive options.

DED should now be considered a public health concern, requiring preventive education, occupational interventions, and integration into tele-ophthalmology/Optometry care models.

DECLARATION

Ethical Approval - Not Required.

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