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From evidence to practice.
A summary of the TFOS DEWS III Diagnostic Methodology Report

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Dry eye disease (DED) affects one in three UK adults[1] making it one of the most prevalent ocular conditions seen in High Street practice. However, diagnosis remains challenging due to the well-documented lack of consistency between clinical signs and symptoms, as well as the use of differing diagnostic criteria. Symptoms of DED should not be underestimated as they can have a significant negative impact on a patients' quality of life – affecting daily activities and mental health even in the absence of sight-threatening disease[2].

The Tear Film and Ocular Surface Society (TFOS) produces internationally recognised consensus reports on DED, synthesising current evidence compiled by multi-disciplinary panels of experts from around the world. The first TFOS report of the International Dry Eye Workshop (DEWS) was published in 2007; TFOS DEWS II followed in 2017 and the latest TFOS DEWS III was published in June 2025[3]. These latest reports provide a long-awaited update of TFOS DEWS II.

This CPD article summarises the key updates in the TFOS DEWS III Diagnostic Methodology Report, and how the diagnostic framework can be applied in clinical practice to improve consistency and diagnostic accuracy. This latest report focuses on targeting the underlying causes of DED, rather than just providing symptomatic relief. This is particularly relevant for dispensing opticians (DO), contact lens opticians (CLO), and optometrists (OO), who increasingly find themselves on the frontline of eyecare services.

In this article, the TFOS DEWS III Diagnostic Methodology Report will simply be referred to as DEWS III; different reports from TFOS DEWS III will be fully specified.

DED DEFINITION

*According to DEWS III, "Dry eye is a multifactorial, **symptomatic disease** characterised by a loss of homeostasis of the tear film **and/or ocular surface**, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities are **etiological factors**"*[4].

Table 1 provides a detailed comparison of the changes from the TFOS DEWS II[5] definition compared to DEWS III[4] with key points as follows:

- Reinforces that DED is strictly a symptomatic disease; if there are clinical signs without symptoms, this would indicate another ocular surface disease
- The inclusion of "and/or ocular surface" acknowledges the understanding of the interplay between the tear film and ocular surface as central to dry eye pathology
- Aetiological factors can drive symptoms and disease
- Emphasises that dry eye is a 'disease' rather than a syndrome, as there is more understanding of the causal processes

DRY EYE QUESTIONNAIRES

DED is a symptomatic condition; therefore, in the absence of symptoms, another ocular surface disease is indicated. To establish a standardised metric for symptom assessment, a single, validated dry eye symptom questionnaire is required. Ideally, such a questionnaire should have:

- A low response burden (easy and time efficient for patients to complete)
- Be simple to score
- Contribute to the assessment of severity and monitoring of treatment efficacy

	Constantly	Mostly	Often	Sometimes	Never
Have you experienced any of the following *during a typical day within the last month?*					
1. Eyes that are sensitive to light?	4	3	2	1	0
2. Vision blurring between blinks (with your refractive correction if needed)?	4	3	2	1	0
	Symptoms and visual disturbance subscale ⇨				
Have problems with your eyes limited you in performing any of the following *during a typical day within the last month?*					
3. Driving or being driven at night?	4	3	2	1	0
4. Watching TV, or a similar task?	4	3	2	1	0
	Visual function / tasks subscale ⇨				
Have your eyes felt uncomfortable in any of the following situations *during a typical day within the last month?*					
5. Windy conditions?	4	3	2	1	0
6. Places or areas with low humidity?	4	3	2	1	0
	Environmental subscale ⇨				

FIGURE 1. OSDI-6 questions, with the summed score ≥4 being the diagnostic cut-off[4]

A questionnaire must reliably measure what it is intended to measure, distinguishing between individuals with and without dry eye symptoms. It must be reliable and consistent, producing the same results when repeated. Furthermore, it should possess sufficient responsiveness to detect meaningful changes over time, thereby reflecting variations in symptom severity resulting from treatment effects or disease progression.

The recommendation to adopt a single questionnaire represents one of the updates introduced in DEWS III[4]. Previously, TFOS DEWS II had recommended two questionnaires for assessing dry eye symptoms: the 12-item Ocular Surface Disease Index (OSDI); and the 5-item Dry Eye Questionnaire (DEQ-5). However, the two questionnaires were not directly comparable, leading to inconsistencies in how dry eye symptoms were evaluated in both research and clinical practice.

In 2018, the OSDI-6 (**Figure 1**) was introduced as a simplified, shorter version of the OSDI-12, with an easy-to-use scoring structure[6]. The diagnostic cut-off for a positive result is ≥4, with severity indexed as: 0-3 normal, 4-8 mild to moderate and >8 severe. These categories support clinical decision-making when used alongside diagnostic tests to evaluate treatment efficacy and disease progression.

CRITERIA	TFOS DEWS II (2017)[5]	TFOS DEWS III (2025)[4]
Definition	*"Dry eye is a multifactorial disease of the ocular surface characterised by loss of homeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles"*	*"Dry eye is a multifactorial, symptomatic disease, characterised by loss of homeostasis of the tear film and/or ocular surface, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities are etiological factors"*
Symptom emphasis	The presence of ocular symptoms is required for diagnosis but the link between signs/symptoms was less explicit	DED is explicitly described as always symptomatic; signs alone are not sufficient for a diagnosis
And/or ocular surface	The loss of homeostasis described relative to the tear film	The loss of homeostasis now encompasses the tear film and/or the ocular surface, recognising the key role of both
Aetiological factors	Instability, hyperosmolarity, inflammation, damage, neurosensory abnormalities play aetiological roles	Instability, hyperosmolarity, inflammation, damage, neurosensory are aetiological factors and the 'and/or ocular surface' recognise both the tear film and ocular surface changes may drive symptoms and disease
Disease vs. syndrome	Defined DED as a disease rather than a syndrome after progress in understanding causality	Reinforces why it is a disease, as there is more understanding of the causal processes impacting diagnosis and management

TABLE 1. Definition comparison: TFOS DEWS II vs. TFOS DEWS III

Although no validated dry eye questionnaires currently exist for children, they have been shown to be reliable in paediatric populations[7]. The simplified shorter format of the OSDI-6 should, therefore, be beneficial. A positive result from the OSDI-6 identifies those patients who require further diagnostic testing to confirm if DED is present.

During a CPD workshop session at the 2025 ABDO Clinical Conference, delegates were asked to complete the OSDI-6 questionnaire as an initial task. Many were surprised by how quick and easy this process was, highlighting its utility in clinical practice. By early identification of individuals with dry eye symptoms, further diagnostic testing could be initiated before significant disease progression occurs. This could improve patient outcomes through earlier intervention and tailored management.

DISCORDANCE BETWEEN SIGNS AND SYMPTOMS

A systematic review looked at the association between signs and symptoms of DED analysing 175 individual sign-symptom associations[8]. Of these, 148 reported correlation co-efficients ranging from -0.4 to 0.4, indicating a generally weak and inconsistent relationship.

This suggests that only around 15 per cent of the variance in patient-reported symptoms can be explained by commonly-measured clinical signs, such as tear break-up time, Schirmer test, ocular surface staining, or tear osmolarity. In simple terms, the majority of what patients feel, and report, cannot be reliably predicted from these objective diagnostic markers alone. Several factors contribute to this discordance, and understanding these can help minimise diagnostic uncertainty in practice.

MEASUREMENTS

Firstly, there is subjectivity in the assessment of both signs and symptoms; patients self-report symptoms and practitioners subjectively grade/score objective tests. The order in which tests are performed can also influence results. DEWS III recommends tests should be conducted from the least to most invasive (**Table 2**). However, it is not necessary to complete every test listed. To improve consistency and diagnostic accuracy, discussion between practitioners within a

ORDER	DIAGNOSIS	SUB-TYPE DRIVER	CUT-OFF POINT
1	OSDI-6 Questionnaire		Score ≥4 triggers further DED assessment
2		Blink/lid exam rate/completeness /lid seal	Normal spontaneous blinking 10-15 blinks per minute, (blink rate can be affected by fatigue, attention/concentration, environmental conditions and neurological or ophthalmic conditions)
3		Tear meniscus height preferably using infrared illumination Tear volume, indicator for aqueous deficient dry eye disease (ADDE)	≤2mm is an indicator for ADDE. Measured centrally in primary gaze directly below pupil midline, a single measurement using infrared or visible white light. OCT also provides a cross-sectional view of the tear prism. Measurement can be distorted by LIPCOF, conjunctivochalsis or disruption from lid shape
4	Non-invasive tear break-up time	Tear film stability	Measured time interval between a blink and the first appearance of discontinuity in the tear film, cut-off point < 10 seconds
5		Anatomical lid/globe misalignment, e.g. pterygia, pinguecula, lid-parallel conjunctival folds (LIPCOF) and conjunctivochalasis. Raised lesions disrupt the smooth ocular surface affecting tear flow and positioning/function of the glands	
6		Inflammation – redness Inflammation is both a cause and consequence of DED	
7		Lipid – interferometry assesses the lipid layer thickness non-invasively	
8	Osmolarity	Increased osmolarity is a core feature of DED	Osmolarity ≥308 mOsm/L in either eye or interocular difference > 8 mOsm/L, differences between eyes may have a slightly improved diagnostic utility than absolute values, cut-offs were established using TearLab only
9		Lid margin – eyelashes, lid margin, diagnostic expression MGD. Anterior blepharitis is often accompanied by debris around the lashes	
10	Fluorescein tear break-up time		<5 seconds indicates instability Fluorescein increases tear volume and reduces stability, giving shorter break-up times
11	Ocular surface staining	Ocular surface damage – corneal, conjunctival and lid wiper staining	>5 corneal punctate spots (fluorescein) or >9 conjunctival punctate spots (lissamine green) or lid margin staining ≥2mm length and ≥25 per cent sagittal width
12		Lid margin – meibography	
13		Neural dysfunction – corneal nerves/sensation Hypoesthesia seen in diabetes, herpes simplex/zoster, ocular surgery leading to epithelial damage without symptoms Hyperesthesia, neuropathic ocular pain, symptoms of burning stabbing pains outweighing signs. Systemic analgesics, antidepressants and ocular surface therapy	

TABLE 2. TFOS DEWS III sequence of tests, sub-type drivers and diagnostic cut off points[4]

practice is essential so that the same tests are being conducted in the same order and using the same grading scales.

Some practitioners may instil fluorescein early in an examination, even though it is 10th in the recommended test sequence, after all non-invasive tests (**Table 2**). Wolffsohn *et al*[9] found that although practitioners may lack confidence when grading to one decimal place, discrepancies between practitioners tended to be smaller when finer grading scales were used. A later study concluded that 0.5 grading increments were optimal, but recommended that the scale should be visible during assessments[10].

MULTIFACTORIAL AND DYNAMIC NATURE OF DED

Patients may present with more than one underlying cause of DED, with conditions such as depression, chronic pain and allergies – and factors like age, sex and medication influencing symptoms. Research using heterogeneous patient groups could also dilute the correlations between signs and symptoms.

The dynamic nature of this disease means that over time it evolves, often in response to both internal and external factors so symptoms and signs can even fluctuate throughout the day.

The TFOS Lifestyle Reports[12] explore influencing factors in detail (**Table 3**), providing valuable insights that support a more holistic understanding of DED and facilitate identification and management of underlying causes. Recording the time tests are conducted, and if ocular

lubricants were recently instilled, provides additional insight when identifying underlying causes[5].

The updated DED definition emphasises aetiological factors reflecting a broader understanding of DED pathophysiology, notably incorporating neurosensory abnormalities. This revision acknowledges both neuropathic and neurotrophic mechanisms as key contributors alongside tear film instability, hyperosmolarity and ocular surface inflammation. This highlights that neurosensory dysfunction often requires distinct clinical consideration and appropriate multidisciplinary interventions.

With neuropathic pain (pain without stain), patients present with severe symptoms, often a burning sensation, but minimal signs caused by a dysfunction in the somatosensory nervous system involving the corneal nerves (peripheral corneal nerves or central pathways)[13]. Importantly, conventional treatments such as ocular lubricants often fail to provide the expected symptomatic relief as they do not address the underlying neural mechanisms. Conditions include post-surgical nerve injury, chronic pain syndromes such as fibromyalgia, and psychological conditions[4].

With neurotrophic keratopathy (stain without pain), patients present with significant clinical findings but few or no symptoms. This is caused by altered corneal sensitivity from abnormalities in the ophthalmic division of the trigeminal nerve. This dysfunction can also impact the blink reflex leading to ocular surface staining and damage[13,14]. Causes include diabetes, herpetic keratitis, refractive surgery and ocular surface neoplasia or trauma[4].

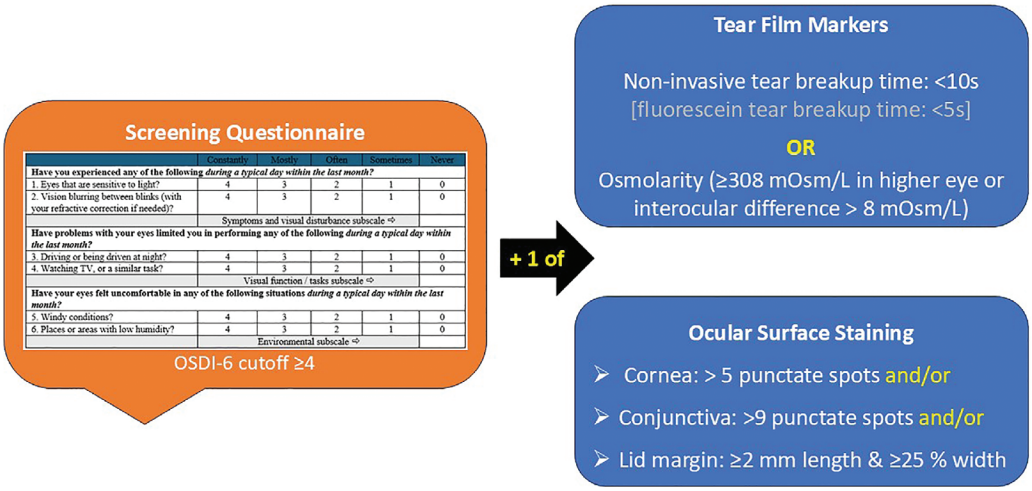


FIGURE 2. TFOS DEWS III diagnostic algorithm[4] ©TFOS DEWS III

Environmental conditions such as pollution and humidity
Digital device use is ubiquitous with abnormal blinking and increased digital eye strain
Contact lens wear especially if wearer behaviours and hygiene are poor
Cosmetic products and procedures introduce allergens, carcinogens and irritants
Depression, anxiety, sleep disorder and chronic pain
Nutrition, deficiencies in omega-3 fatty acids, vitamin A, B12, C and D

TABLE 3. Lifestyle factors affecting ocular health[11]

DIAGNOSTIC ALGORITHM

DEWS III introduces an updated algorithm (**Figure 2**), designed to streamline and personalise the process for both the patient and the practitioner[4]. It should be remembered that currently there is no gold standard for the diagnosis of DED. The DEWS III authors advocate DED diagnosis should not rely on a single test. Instead, they recommend an approach that combines reproducible markers – capturing both patient-reported symptoms and objective clinical signs.

STEP ONE

SYMPTOMS: Using the OSDI-6 questionnaire to standardise measurement of patient reported symptoms, ≥4 a positive marker for dry eye symptoms indicating the need for further testing.

STEP TWO

TEAR FILM MARKERS: Non-invasive tear break-up time (NIBUT) <10s or osmolarity ≥308mOsm/L in the higher eye, or an interocular difference >8mOsm/L. Either of these metrics, if positive, supports a DED diagnosis alongside a positive symptom result using the OSDI-6 questionnaire.

Dry eye diagnosis = positive OSDI-6 score + *one* positive clinical sign from tear film markers

OCULAR SURFACE MARKERS: If NIBUT is not available, or to further support the diagnosis, ocular surface markers can be assessed using fluorescein (corneal staining) and lissamine green (conjunctival staining). For a positive result >5 corneal punctate spots or >9 conjunctival punctate spots, or lid margin staining ≥2mm in length and ≥25 per cent width. These cut-off points allow diagnostic consistency across clinical settings.

TEAR FILM MARKERS

TEAR BREAK-UP TIME

The most commonly-used tear film stability test in practice is fluorescein tear break-up time (FBUT). However, instillation of fluorescein itself disturbs the tear film, temporarily increasing tear volume leading to a shorter break-up time. NIBUT can be measured using a keratometer, although this only provides information relating to the central 3-3.5mm of tear film covering the cornea[15].

Ideally, an objective measurement using a Placido disc, which not only detects the first break in the tear film but also maps the disruption across the corneal surface, is preferred. As the cost of such instrumentation continues to decrease, it is likely that these technologies will be found more routinely in practice. Where FBUT is used, it should follow all other non-invasive tests. The cut-off point for FBUT is <5 seconds.

OSMOLARITY

Tear osmolarity refers to the dissolved electrolytes within the tears. In simple terms, it is a measure of how salty the tears are, expressed in milliosmoles per litre (mOsm/L)[16].

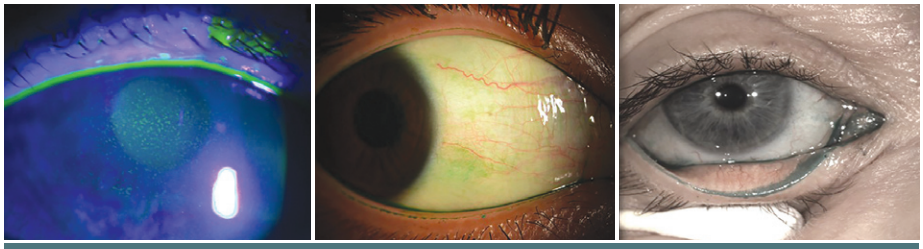


FIGURE 3. L-R: fluorescein corneal staining; lissamine green conjunctival staining; lissamine green staining of lower lid wiper epitheliopathy

Tear hyperosmolarity refers to an increased concentration of solutes, which can arise from either increased evaporation or reduced decreased lacrimal secretions or a combination of both. Evaporation between blinking causes the tear film to thin leading to increased osmolarity[17]. The imbalance in osmolarity readings between eyes can also be a more sensitive indicator for DED than simply taking the highest value from each eye[18].

It is important to note that osmolarity testing is not widely adopted in clinical practice, and the diagnostic cut-off points were established using only the TearLab system.

OCULAR SURFACE STAINING

CORNEAL STAINING

Instillation of fluorescein via fluorescein strips moistened with sterile saline or fluorescein solution is used to assess corneal staining. The optimum time after instillation for corneal assessment is one to four minutes[19].

The DEWS III diagnostic algorithm recommends counting punctate spots for higher consistency[4]. However, the significance of staining location is not comprehensively addressed. For example, contact lens edge staining beneath the eyelids may not necessarily represent a positive indicator for DED, whereas characteristic 'smile' staining should be considered clinically relevant[20].

CONJUNCTIVAL AND LID MARGIN STAINING

Instillation of lissamine green is preferred for conjunctival staining and lid margin assessment. A lissamine green strip is also moistened with sterile saline, which is allowed to soak in for at least five seconds before applying the drop to the eye[21]. This allows the dye to fully dissolve into the liquid, ensuring maximum concentration is applied to the eye.

The optimal time for conjunctival assessment has been shown to be immediately after application[20] – although DEWS III refers to a consensus of between one and five minutes. Again, results are assessed by counting punctate spots.

Lid wiper staining refers to the marginal conjunctiva of both the upper and lower eyelids, which makes direct contact with the ocular surface during blinking[22]. The upper lid wiper plays a key role in distributing the tear film across the eye, whereas the function of the lower lid wiper in tear spreading remains less well understood. This area is known to be highly sensitive, and damage in that area is known as lid wiper epitheliopathy (LWE).

LWE is defined as a form of epithelial damage affecting part of the eyelid's marginal conjunctiva linked to inadequate lubrication and increased friction between the eyelid and the ocular surface[23] (**Figure 3**). To assess this staining involves a subjective estimation of the length and the sagittal depth of the staining. This means assessing if the staining is of 2mm or greater in length over at least 25 per cent of the sagittal width[4].

SUB-CLASSIFICATION BASED ON AETIOLOGICAL DRIVERS

This is one of the more significant changes from TFOS DEWS II to DEWS III: a shift from sub-classifying DED purely as aqueous deficient dry eye (ADDE), evaporative dry eye (EDE), or mixed towards an aetiological driver-based factor. This reflects a more nuanced understanding of DED as multifactorial.

The traditional ADDE/EDE framework had limitations because many patients present with overlapping or unclear features, and a significant number of patients exhibit no clear signs of reduced tear volume deficiency or MGD.

The current approach focuses on identifying specific aetiological drivers linking to the TFOS DEWS III Management and Therapy Report for a more tailored treatment protocol[24].

1. TEAR FILM DEFICIENCIES

Although evidence remains limited, fluorescein tear break-up patterns have been used to identify specific tear film layer disruptions; an area break was linked to a severe ADDE, a spot, dimple or line break were associated with poor wettability, and a random break linked to increased evaporation[25].

- **LIPID LAYER:** thickness can increase in hypersecretory MGD and decrease in obstructive MGD, but a direct link between lipid layer thickness and tear evaporation rate remains unproven[26]. Nonetheless, the lipid layer plays a critical role in maintaining tear film stability and regulating evaporation
- **AQUEOUS LAYER:** represents the bulk of the tear volume and is assessed by measurement of the tear meniscus height (TMH). TMH of ≤0.2mm is a differentiating metric for ADDE[27]. TMH should be measured directly below the pupil as eyelid morphology can influence measurement[28]. Measurement can also be affected by lid anatomy like lid parallel conjunctival folds and conjunctivochalasis. Optical coherence tomography (OCT) allows for a cross-sectional view of the TMH so any disruptions can be easily identified
- **MUCIN/GLYCOCALYX:** goblet cells are essential for maintaining the mucin layer and glycocalyx; when there is a reduction in goblet cells, the wettability of the ocular surface is affected[27]

2. EYELID ANOMALIES

- **BLINKING:** essential for tear film distribution, debris removal, maintaining ocular surface health and optical clarity. Normal spontaneous blink rate is 10-15 blinks per minute, although this can vary according to factors such as age, attention, fatigue, environmental conditions, neurological disease and medication use[29,30]. Altered blinking patterns are both a feature and a biomarker of DED. Increased blink rate can occur as a response to ocular irritation, whereas reduced blink rate is observed in both DED and non-DED during prolonged screen use or intense visual attention. Incomplete blinking is often seen in evaporative DED. Blinking is controlled by

neuromuscular components, the orbicularis oculi, levator palpebrae superioris and Muller's muscle[31]. Sensory feedback from the cornea and conjunctiva modulates the blink reflex. Blinking can be spontaneous, reflex or voluntary. Blinking exercises to modify poor blinking can improve DED symptoms and show some positive changes in clinical signs[32] although, in the author's opinion, require consistent clear patient messaging as compliance is difficult to maintain

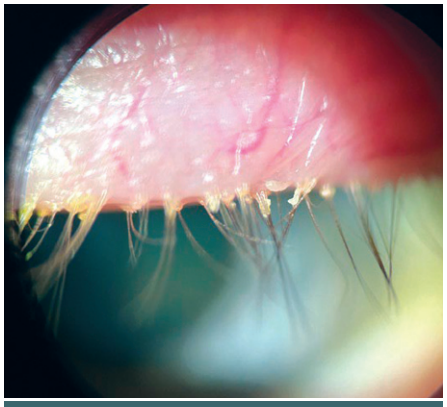


FIGURE 4. Cylindrical dandruff around the eyelash bases in anterior blepharitis

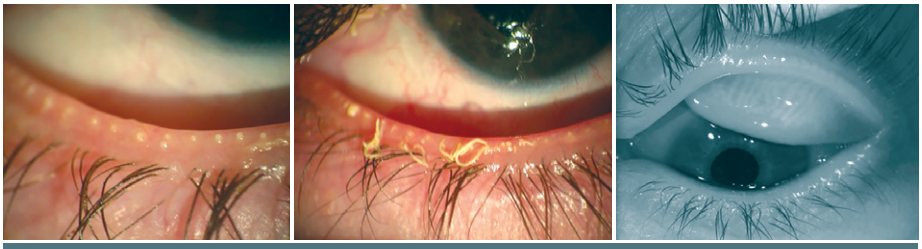


FIGURE 5. L-R: blocked meibomian gland; thick toothpaste like meibum; upper lid meibography with meibomian gland truncation

- **LID MARGIN DISEASE:** anterior blepharitis (inflammation of the lid margin) can present with debris or dandruff around the lashes. Periocular bacteria build biofilms, which can thrive on the eyelid's moist, nutrient rich surface. **Figure 4** shows cylindrical dandruff around the base of the lashes in anterior blepharitis. Demodex blepharitis should also be considered[33]
- **POSTERIOR BLEPHARITIS OR MGD (Figure 5):** a chronic abnormality of the meibomian glands classified as either terminal duct obstruction and/or changes in the glandular secretions[34]. Severity is, again, subjectively classified according to patient symptoms and signs. There are more meibomian glands in the upper eyelid compared to the lower eyelid, and the glands in the upper eyelid are longer. Advanced assessment can be achieved by non-invasive infrared photography or meibography[35]

3. OCULAR SURFACE ABNORMALITIES

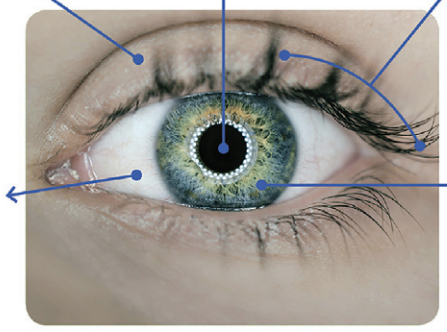
- **ANATOMICAL MISALIGNMENT:** leading to a disruption of the smooth ocular surface such as pterygia, pinguecula, lid parallel conjunctival folds or conjunctivochalasis. Most of these signs would be easily visible during gross observation
- **NEURAL DYSFUNCTION:** as detailed in discordance between signs and symptoms is now recognised as an aetiological factor for DED

Eyelid-related disorders	Key differential features
Anterior blepharitis (bacterial, fungal, parasitic, viral, seborrhoic)	Crusts, collarettes, or debris on lashes, hyperaemic swollen lid margins, lid notching, marginal keratitis
Meibomian gland dysfunction	Altered meibomian gland secretion, meibomian gland plugging and pouting, lid margin telangiectasia, lid notching
Chalazion/hordeolum	Raised lesions on the eyelids
Eyelid malposition (ectropion, entropion)	Part or whole eyelid everted from the globe (ectropion) or inverted (entropion), tearing, mucus discharge.
Trichiasis and distichiasis	Eyelashes directed towards the globe (trichiasis), extra row of eyelashes more posterior (distichiasis), epithellopathy where lashes touch eye
Floppy eyelid syndrome	Severe eyelid laxity, spontaneous eversion of upper lid, papillary conjunctivitis, lash ptosis, history of sleep apnea
Ocular rosacea	Telangiectasia, erythema lid margin, meibomian glad dysfunction, history of rosacea (can present without skin involvement)
Blepharospasm	Episodic uncontrolled blinking or closure of the eyelids
Bell's palsy and/or lagophthalmos	Inability to close eye due to VII nerve palsy, punctate epithelial defects in lower cornea.
Canaliculitis	Swollen and inflamed nasal lid, chronic punctal discharge

Non-ocular surface disorders	Key differential features
Uncorrected refractive error	Blurry vision, headache, squinting, eye strain
Digital eye strain	Eye strain, ache around the eyes, blurred vision, headache, shoulder and neck pain, associated with prolonged screen use
Intermittent angle closure	Shallow anterior chamber, high intraocular pressure, severe conjunctival hyperaemia, corneal edema, fixed mid-dilated pupil
Posterior scleritis	Severe pain, proptosis, vision loss, choroidal folds, exudative retinal detachment, papilledema
Anterior uveitis	Anterior chamber cells and flare, keratic precipitates, pain and severe photophobia
Tolosa-Hunt syndrome	Severe unilateral retrobulbar or periorbital pain, restricted eye movements
Thyroid eye disease	Lid retraction, proptosis, lid swelling, double vision, restricted eye movements, history of thyroid disease
Carotid cavernous fistula	Pulsatile exophthalmos, orbital bruit, conjunctival chemosis and engorged vessels, restricted eye movements
Dacryocystitis	Purulent punctal discharge, painful erythematous swelling around lacrimal sac
Nasolacrimal duct obstruction	Continuous tearing and watery eyes, reflux with lacrimal syringing
Sinusitis	Nasal drainage, hyposmia or anosmia, facial pain or pressure, nasal obstruction

Other mixed and miscellaneous ocular surface disorders	Key differential features
Limbal stem cell deficiency	Stippled staining pattern, loss of clarity epithelium, conjunctivalization of the cornea, superficial corneal vascularization
Episcleritis and anterior scleritis	Diffuse or sectoral hyperaemia ocular surface, nodules, subacute presentation, boring pain (scleritis)
Mucus fishing syndrome	Repetitive eye-rubbing and self-extraction of mucus, epithelial damage of the nasal and temporal conjunctiva (lissamine green staining)
Ocular neuropathic pain	Increased perception of pain to non-painful stimuli (e.g. aircon or wind), history of refractive laser or ocular surgery, diagnosis of exclusion when symptoms outweigh signs
Foreign body	Foreign body on cornea, conjunctiva, or under upper eyelid, acute onset
Contact lens intolerance and contact lens -induced conjunctivitis	3 and 9 o'clock staining, conjunctival hyperaemia, improvement after discontinuing lens, (giant) papillae
Toxic conjunctivitis and keratopathy	Inferior papillary or follicular reaction, inferior conjunctival and corneal staining, use of (preserved) eye drops or other triggering systemic medication
Thermal burns, UV-radiation, traumatic and chemical injuries	Severe pain, history of trauma, welding or chemical injury, interpalpebral cornea epithelial defect
Superior limbic keratoconjunctivitis	Papillary reaction upper palpebral conjunctiva, sectorial conjunctival hyperaemia, keratinization and ciliary injection, punctate staining or erosion of superior cornea

Conjunctiva-related disorders	Key differential features
Allergic eye disease	Itch as main symptom, papillary conjunctivitis, atopic history, lid swelling and chemosis
Infectious conjunctivitis (viral, bacterial)	Acute onset, conjunctival hyperaemia, discharge, papillae or follicles, recent upper respiratory infection, lymphadenopathy
Cicatricial conjunctivitis (Stevens-Johnson syndrome, ocular cicatricial pemphigoid, trachoma)	Inflammation and scarring of conjunctiva, subconjunctival fibrosis, fornix shortening, symblepharon
Ligneous conjunctivitis	Fibrinous pseudomembranes on palpebral conjunctiva
Conjunctivochalasis	Loose, redundant, non-oedematous folds in the conjunctiva (often asymptomatic). Lid-parallel conjunctival folds (LIPCOF) may be a milder version
Pinguecula, pterygium	Grey-white to yellowish lesion of the bulbar interpalpebral conjunctiva (pinguecula), wing-shaped fibrovascular tissue migrating over cornea (pterygium) (often asymptomatic)
Conjunctival concretions	Discrete yellow-white deposits on palepebral conjunctiva (can be symptomatic when they erode through conjunctiva)



Differential diagnoses of dry eye disease (DED) grouped by anatomy, and key differential features compared to DED.

Cornea-related disorders	Key differential features
Neurotrophic keratopathy	Decreased corneal sensitivity, corneal epithelial defects and stromal ulceration, history of herpes keratitis or LASIK
Corneal dystrophies and degenerations (e.g. Salzman and epithelial basement membrane dystrophy)	Corneal depositions, opacities and/or nodules, varying corneal erosion or edema
Bullous keratopathy	Corneal edema, epithelial bullae, history of intraocular surgery or Fuchs endothelial corneal dystrophy.
Infectious keratitis	Acute onset, infiltrate or ulcer in cornea, severe pain, conjunctival hyperaemia, photophobia
Thygeson superficial punctate keratitis	Discrete grey-white focal corneal lesions, often central and elevated, lack of conjunctival hyperaemia
Recurrent corneal erosion	Recurrent attacks of acute ocular pain, often during the night or while awakening, corneal erosion, history of corneal dystrophy or degeneration
Mooren ulcer and other peripheral ulcerative keratitis	Painful thinning and ulceration of cornea, often starting in peripheral cornea (Mooren ulcer). History of autoimmune disease, ocular/systemic infection or CL-wear (other peripheral ulcerative keratitis).

FIGURE 6. Key differential diagnoses of DED grouped by anatomy[4]

- **PRIMARY INFLAMMATION:** a key factor in the pathophysiology of DED, influencing disease severity, progression risk and treatment decisions. It can be both a cause and consequence of DED, arising from ocular surface damage or systemic autoimmune conditions like Sjögren's disease where immune cell infiltration damages the lacrimal glands[36]. While inflammation is associated with DED, it is not sensitive to early stages of the disease[37]

4. SYSTEMIC DISEASES

- **AUTOIMMUNE DISEASES:** conditions like rheumatoid arthritis, lupus, Stevens-Johnsons syndrome and Sjögren's disease are closely associated with DED.[4] A multi-disciplinary team approach is important, as addressing the underlying systemic disease is key to improving ocular symptoms
- **HORMONE IMBALANCE:** hormonal fluctuations, particularly in women, can influence tear production and quality. The impact of hormonal imbalance and its management in DED is discussed in detail in the TFOS DEWS III Digest[38]
- **METABOLIC DISEASES:** diabetes affects ocular tissues from hyperglycaemia and glycation-related damage impairing lacrimal gland and corneal nerve function, leading to reduced tear production, increased osmolarity and altered sensitivity[39]. Good glycaemic control can help mitigate these effects

DIFFERENTIAL DIAGNOSIS

DED shares symptoms and signs with a range of ocular conditions, making accurate diagnosis essential to avoid mismanagement. Conditions like allergic conjunctivitis, blepharitis, corneal dystrophies and neurotrophic keratitis, and embedded corneal foreign bodies, require specific management or immediate referral depending upon professional qualification and scope of practice[40]. DEWS III has several useful infographics, which can be downloaded and used in practice, one of which is for differential diagnoses of DED grouped by anatomy (**Figure 6**). This topic is too big to cover within this article.

PATIENT COMMUNICATION

A final new theme for DEWS III is actively involving patients in discussions regarding their symptoms, listening to concerns and educating on lifestyle modifications that would help to manage their DED, explaining the chronic nature of this condition, as well as setting realistic expectations. Using written information that a patient can take away alongside visual aids, demonstrating treatments like how to instil ocular lubricants correctly or a lid hygiene routine for anterior blepharitis and MGD, is recommended. This involvement and shared decision making, from diagnosis to management, hopefully will result in better compliance and improved outcomes.

Involving the entire practice team supports consistent messaging and more informed patient discussions. Explaining the professional roles of each practice member, alongside the diagnostic algorithm, aetiological drivers and the importance of a differential diagnosis, sets the foundation to not only educate patients on DED but also inform patients about the knowledge and skills that DOs, CLOs and OOs provide.

CONCLUSION

The DEWS III provides practitioners with a comprehensive and practical diagnostic framework for use in clinical practice. It includes a validated, easy-to-use dry eye questionnaire and a clear step-by-step diagnostic algorithm to improve consistency, diagnostic accuracy and patient outcomes.

The updated definition emphasises that dry eye disease is a symptomatic disease and moves towards sub-classifications based on aetiological drivers. In addition, the downloadable Infographics serve as valuable tools that support diagnostic decision-making and differential diagnosis, and facilitate patient education.

Finally, this evidenced-based resource promotes a standardised diagnostic approach that can be applied across both clinical and research contexts, enabling a more accurate understanding of the true prevalence and impact of DED.

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All figures from Wolffsohn JS *et al.* TFOS DEWS III Diagnostic Methodology. *American Journal of Ophthalmology* 2025;279:387-450.

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References can be found when completing this CPD module. For a PDF of this article with references, email abdocpd@abdo.org.uk

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LEARNING OUTCOMES FOR THIS CPD ARTICLE

DOMAIN: Communication

2.1,2.2: *Communicate effectively with patients the dynamic, chronic and multifactorial nature of dry eye disease, explaining the need for ongoing management, potential lifestyle modifications and realistic expectations.*

DOMAIN: Clinical practice

5.3: *Analyse the latest evidence from the TFOS DEWS III diagnostic methodology report to accurately interpret the updated diagnostic criteria for dry eye disease and apply this to the care you provide.*

6.1,6.2: *Recognise and work within the limits of your scope of practice for the diagnosis and management of dry eye disease including when to refer to ensure safe and effective patient care.*

DOMAIN: Specialty CPD – contact lens optician

Critically evaluate the latest evidence from the TFOS DEWS III diagnostic methodology report for the diagnosis and management of patients with dry eye disease and apply this to inform the care you provide.

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