

Updates in Ocular Surface Disease Treatments for 2026

1 hour

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Description

This course reviews latest advancements in diagnosis and management of ocular surface diseases (OSD) based on the Tear Film and Ocular Surface Society Dry Eye Workshop III. This will include research updates, emerging diagnostics, in-office procedures, prescription therapies, over-the-counter options, and pipeline innovations. An emphasis is placed on identifying root cause first and individualize treatments based on this cause.

Objectives

- Describe the updated definition and pathophysiology of dry eye disease from TFOS DEWS III and learn about the newest research related to OSD treatments.
- Evaluate new and emerging diagnostics tools, in-office treatments, pharmacologic therapies for OSD, and new drugs in the pipeline for future FDA approval.
- Integrate evidence-based OTC and cosmetic considerations into patient care.

Outline

I. Definition and Disease Overview

A. Updated Definition (TFOS DEWS III, 2025)

- Dry eye as a multifactorial, symptomatic disease
 - Loss of homeostasis of tear film and/or ocular surface
- Core etiologic factors:
 - Tear instability
 - Hyperosmolarity
 - Inflammation
 - Neurosensory abnormalities

B. Evolution from DEWS II

- Inclusion of ocular surface (not just tear film)
- Reinforcement of symptom requirement for diagnosis

C. Epidemiology and Burden

- ~16 million diagnosed DED patients in the U.S.
- 7–9 million moderate-to-severe cases
- ~80% experience symptom flares

D. Economic and Societal Impact

- ~\$2.59 billion annual U.S. cost
- ~\$55 billion societal burden
- Functional impact:
 - Reduced productivity
 - Impaired vision (reading, driving)
 - Contact lens intolerance

II. Research and Clinical Framework Updates

A. TFOS DEWS III Management Algorithms

- Algorithm 1: Tear film deficiencies
- Algorithm 2: Eyelid abnormalities
- Algorithm 3: Ocular surface abnormalities

B. Clinical Paradigm Shift

- Move toward:
 - Root-cause diagnosis
 - Multimodal treatment
 - Restoration of ocular surface homeostasis

C. Updated Screening Guidelines (Hydroxychloroquine/Plaquenil)

- Primary tests:
 - SD-OCT, Wide-field FAF
- Secondary confirmation:
 - HVF (10-2, 24-2C), mfERG
- Discontinued tests:
 - Amsler grid, Color vision , Time-domain OCT

III. Diagnostics in 2026

A. Symptom-Based Questionnaires

- OSDI-6 (new standard)
 - Streamlined (6 questions)
 - Improved clinical usability
 - Better longitudinal tracking
- SPEED
 - Quick 8 questions
 - Based on 28 as highest, higher is more symptomatic
 - Cut off is 7/28

B. Tear Film and Biomarker Testing

1. Tear Osmolarity

- Central mechanism in DED pathophysiology
- Clinical implications:
 - Corneal toxicity
 - Inflammation
 - Refractive variability

2. Clinical Relevance

- Essential for:
 - Pre-surgical screening
 - Monitoring disease severity

IV. In-Office Procedures

A. Dynamic Muscle Stimulation (DMSt)

- Targets:
 - Lid laxity
 - Blink dysfunction
- Outcomes:
 - ↑ TBUT (86%)
 - ↑ blink quality
 - ↓ lid laxity

B. Thermo-Mechanical Action (TMA)

- Mechanism:
 - Controlled heat delivery
 - Meibum liquefaction

- Benefits:
 - Improved gland function
 - Increased TBUT
 - Periocular skin tightening

C. Clinical Integration

- Treatment protocols:
 - 3 sessions (2–3 weeks apart)
 - Maintenance every 3–6 months

IV. Prescription Medications

A. Demodex Blepharitis (DB)

1. Pathophysiology

- Infestation by *Demodex folliculorum*
- Mechanisms:
 - Mechanical damage - claws and teeth
 - Bacterial transmission – concomitant staph bacteria overgrowth
 - Inflammatory cascade - reaction to chiton and Demodex digestive enzymes are pro-inflammatory on the lid margins

2. Clinical Signs

- Collarettes (pathognomonic) - at the base of the lashes
- Lid inflammation - erythema and thickening of lid margins
- MGD association - thickened meibum and clogged glands

B. Lotilaner 0.25%

- Mechanism:
 - Targets mite GABA channels → paralysis and death
- Clinical outcomes:
 - ~50% collarette resolution
 - Improved MG function - MGYLS – Meibomian glands yielding liquid secretions
 - Symptom improvement - improvement in fluctuating vision, less stinging, burning and itching of the eyelid margins

C. Cyclosporine 0.1% in Perfluorobutylpentane

- Mechanism:
 - Immunomodulation + enhanced drug delivery due to the water-free vehicle
- Advantages:
 - Faster onset (2–4 weeks) of decrease in signs
 - Improved bioavailability - due to vehicle which reduces evaporation of the tear film
- Additional benefit:
 - Lipid layer stabilization from the vehicle

D. Perfluorohexyloctane (PFHO)

- Mechanism:
 - Mimics natural meibum provides a protective layer to decrease evaporation, similar to meibum
 - Reduces evaporation (~80%)
- Clinical benefits:
 - Improved tear stability
 - Reduced friction with the eye lids

E. Acotremone 0.003% ophthalmic solution

- Neuromodulation Mechanism:

- o TRPM8 receptor activation via the trigeminal nerve
- o Stimulates basal tear production from the lacrimal functional unit – lacrimal gland, goblet cells, meibomian glands

- Outcomes:

- o Increased tear volume by 10mm on Schirmer's strip from baseline
- o Improved lipid concentration - in the tears stimulated by drop

VI. Over-the-Counter Therapies

A. Artificial Tear Ingredients

- Hyaluronic acid:

- o Hydration and epithelial healing
- o Binds water to the surface of the cornea and conjunctiva

- Trehalose:

- o Osmo protection
- o Naturally occurring anti-desiccation substance

- Castor oil:

- o Lipid layer support
- o Reduces evaporation of the tears

B. Adjunctive Therapies

- Hyperhydration supplements

- Warm compress masks:

- o 104°F for 8–12 minutes

VII. Cosmetics and Ocular Surface Health

A. Under-Eye Products – used to reduce swelling and appearance of fine lines and wrinkles

- Silicate-based tightening agents
- Hydrating and antioxidant ingredients

B. Ocular Safety Considerations

- Formulations Avoid:
 - Prostaglandins
 - Harsh preservatives

VIII. Pipeline and Emerging Therapies

A. AZR-MD-001 (Keratolytic Therapy)

- Mechanism:
 - Targets keratinization in MGD
 - Dissolves capped and clogged meibomian glands
- Outcomes:
 - Improved gland function
 - Symptom resolution

B. MR-146 Gene Therapy

- Target:
 - Moderate – Severe Neurotrophic keratitis
- Mechanism:
 - Adeno-Associated Virus (AAV) gene therapy - mediated tear film restoration through increased aqueous production
 - Targets lacrimal glands

C. Vezocolmitide (ST-100)

- Mechanism:
 - Extra Cellular Matrix repair and collagen restoration
 - Benefits:
 - Phase 3 FDA trial failed to show improved tear production
 - However corneal damage was reduced and vision improved within days
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IX. Clinical Integration and Takeaways

A. Key Clinical Shifts

- Treat root cause, not just symptoms
- Integrate:
 - Tear film
 - Eyelids
 - Ocular surface

B. Multimodal Treatment Approach

- Combine:
 - Diagnostics
 - In-office therapies
 - Pharmacologic treatments

C. Future Direction

- Personalized medicine
- AI-driven diagnostics
- Disease-modifying therapies