Inflammation in Dry Eye Disease
Blair Lonsberry, OD, FAAO

Dry eye is an inflammatory disorder. Management should start with improved identification of affected patients in everyday practice and treating underlying causes of inflammation.

Dry eye disease (DED) affects an estimated 20 million individuals in the US. In the coming decades, prevalence will likely escalate as certain risk factors—including older age, diabetes, and dependence upon lit-screen technology—also increase and the number of patients with multiple risk factors mounts.

General practice optometrists see patients with DED every day. Yet too many cases remain undiagnosed because eyecare providers (ECPs) often fail to ask about and look for DED. There has been an explosion of research, tools, and resources in recent years aimed at making management of DED more reliable and effective, from new diagnostics and therapeutics to a proliferation of management guidelines and protocols. However, these advances have yet to translate into consistent screening and detection by the majority of our colleagues.

Optometrists are in a perfect position to be leaders in the field and to make a difference for individuals with DED, particularly for patients in early stages of disease when treatment is most effective. At least 80% of patients I see in Southern Alaska have signs and/or symptoms of DED, a function of the dry Northern climate, continuous exposure to indoor heat, and a patient population with high rates of contact lens wear and prolonged daily computer use. Places with higher elevations and colder, dryer climates—such as Alaska and Colorado—have particularly high rates of DED. But even in less extreme climates, if you look for DED among your patients, you will find it and be poised to make a powerful impact on patient’s long-term ocular surface health.

Cycle of Inflammation

In December 2014, the first Dry Eye Summit—of which I was a part—met in Dallas, Texas, in an effort to address the substantial gap between the current DED knowledge base and everyday optometric practice. What emerged was a collection of basic, easily implemented recommendations, several of which are discussed here. According to the summit report, DED is “an inflammatory disorder of the tears and ocular surface that impacts the eye’s ability to refract correctly and that, if left untreated, can have a serious impact on functional vision, eye discomfort, and patient quality of life.”

Aqueous-deficient DED originates from inadequate aqueous production (such as in patients with autoimmune disease involving the lacrimal gland) and represents a minority of DED cases. Evaporative DED, the more common type, is due to lipid layer insufficiency or altered quality, which allows evaporation of the aqueous tear layer. Evaporative DED is predominantly related to meibomian gland dysfunction (MGD), in which altered meibum production changes the overall composition of the tear film, allowing aqueous to evaporate and elevating tear osmolarity and pH. Hyperosmolarity triggers a cascade of inflammatory events on the ocular surface, including activation of signaling molecules, cytokines, matrix metalloproteinases (MMPs), and inflammatory cells. Increased amounts of interleukin (IL)-1α and mature IL-1β, the proinflammatory forms of IL-1, have been found in the tear fluid of DED patients. A vicious cycle of inflammation, cellular damage, and glandular compromise is then set...
into motion.

Various factors that promote evaporation stoke the inflammation cycle, such as low ambient humidity, wind exposure, or reduced blink rate or incomplete lid closure associated with computer/smartphone viewing.4 Androgens are thought to support meibomian glandular function; low levels of circulating androgens in women contribute to a higher rate of DED among women compared to men. Declining androgen levels following menopause also contribute to DED in postmenopausal women.7 In men and women, aging induces structural changes to meibomian glands and eyelids (eg, hyperkeratinization of meibomian gland ductal epithelium, aging gland atrophy, and chronic blepharitis), which increase meibum viscosity, ductal plugging, and tear film instability.7 Other risk factors for tear film disequilibrium include eyelid or lacrimal gland damage, contact lens wear, refractive surgery, and certain topical or systemic medications that promote drying, such as antihistamines and antidepressants.

Testing

The Dry Eye Summit suggested that all patients suspicious for dry eye undergo (1) eyelid examination, including of the meibomian glands, (2) corneal staining, and (3) an assessment of tear film stability, such as tear break-up time (TBUT) or osmolarity testing.3

Osmolarity

Tear film hyperosmolarity is clearly linked with ocular surface inflammation and is a core mechanism associated with DED.8 As a sensitive, quantitative test, tear osmolarity is useful for establishing the diagnosis of DED, quantifying severity, and measuring treatment response.9,10

In my practice and clinical experience, patients with dry eye complaints undergo osmolarity testing first—before drops, air puff, or other evaluation or manipulation of the eye—to carefully ascertain the patient’s baseline state. Osmolarity testing is noninvasive, so it causes little to no interference with subsequent tests. Keep in mind that the device for testing osmolarity is sensitive to environmental conditions including ambient humidity, which can vary from one room to the next even within a single office suite. For highest accuracy, the osmolarity unit and cartridges should be maintained in a consistent location and not moved; patients should be brought to it for testing.

Occasionally I hear the objection that nearly all patients have abnormally elevated tear osmolarity, implying the test is not useful. I would suggest that the opposite is true: osmolarity testing is supremely useful in demonstrating the

KEY ISSUES IN OCULAR SURFACE DISEASE — ISSUE 2

STATEMENT OF NEED

Although sometimes used as a synonym for dry eye disease, the term “ocular surface disease” refers to a cluster of anterior eye disorders that includes dry eye (evaporative or due to tear insufficiency), bacterial and viral infections, blepharitis, meibomian gland dysfunction, allergic conjunctivitis, ocular surface problems associated with glaucoma treatment, and the ocular manifestation of systemic inflammatory diseases and endocrine disorders (eg, Sjögren’s syndrome, arthritis, and thyroid disease). While prevalence data vary considerably based on the population studied and disease definition, all of these conditions are common.1,3 In addition, they share pathogenic mechanisms, have overlapping clinical signs and symptoms, and are often comorbid.4 For example, allergic conjunctivitis, blepharitis and Sjögren’s syndrome—like dry eye disease—are inflammatory conditions that affect the ocular surface and share a number of symptoms, including discomfort, itching, dryness, and irritation.5,4

Diagnosis and treatment of ocular surface disease are clearly important, but they are rendered difficult by a number of factors: the frequency of comorbid conditions with similar signs and symptoms; incomplete understanding of the underlying pathogenesis; frequently poor correlation between signs and symptoms; occasional systemic disease as an underlying factor; and the absence of simple, clear diagnostic tests. Even after diagnosis, adherence to best practice in patient management is complicated by the number of agents available and competing claims for them in the marketplace. Each installment of Key Issues in Ocular Surface Disease will look at two important topics in the management of ocular surface disease in order to support optometric’s clinical reasoning and decision-making abilities and navigating the growing body of sometimes contradictory evidence on ocular surface disease. The benefits are substantial: accurate diagnosis and effective treatment of ocular surface disease will contribute greatly to patient comfort and satisfaction, help patients enjoy comfortable contact lens wear, and significantly enhance outcomes in cataract and corneal refractive surgery.

References


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high rates of DED present in the population, underscoring the imperative to test and treat.

**MMP-9**

Matrix metalloproteinase 9 (MMP-9) is a gelatinase, a potent inflammatory mediator capable of cleaving epithelial membrane tight junctions and inducing corneal ulceration. Elevated levels of MMP-9 are present in patients with DED and other conditions associated with ocular tissue damage and remodeling.

InflammaDry® (RPS, Sarasota, FL) is a rapid, disposable, qualitative test for the presence of elevated MMP-9 in tears. It is currently the only commercially available, point-of-service assay for an ocular inflammatory mediator. In our clinic, we use InflammaDry as a second test for DED, performed after the collection process is slightly more invasive and may influence subsequent tests.

A positive InflammaDry test indicates a tear MMP-9 concentration of at least 40 ng/mL, a sign of significant ocular surface inflammation or remodeling. Many ECPs interpret a positive InflammaDry result, like corneal staining, as indicating advanced DED, and there is some evidence to suggest that is true. However, other studies show a lack of correlation between DED severity and MMP-9 status. A negative InflammaDry result indicates MMP-9 within normal range (< 40 ng/mL) and must also be carefully interpreted. Since MMP-9 is but one of many potentially harmful mediators of inflammation, inflammation may still be present despite a negative MMP-9 test.

Although it is not necessary to establish the diagnosis, meibography—such as LipiView® (TearScience, Morrisville, NC)—adds a highly valuable visual dimension to diagnosis and patient counseling. Showing patients images of atrophied meibomian glands, which are lost glands that will not recover, makes a powerful impression and can improve compliance with therapy (Figures 1 and 2).

**Treating DED**

For most patients, DED develops over years to decades and cannot be corrected in a few weeks. ECPs should treat the underlying causes of DED rather than only aiming for short-term palliative relief. According to recommendations made by the Dry Eye Summit, minimum recommendations for treatment for all patients include (1) eyelid heat and hygiene, (2) omega-3-fatty acid supplementation, and (3) artificial tears for palliative relief.

**Lid Hygiene**

Lid hygiene is directed at the crux of MGD: meibum inspissation. Normal healthy meibum is a thin, easily-expressed liquid like olive oil; by contrast, unhealthy meibum is thick like vegetable shortening and has a higher melting point. Basic lid hygiene consists of warm compress application followed by lid massage. Heat liquefies meibum, while gentle massage liberates it from the ducts.

Reusable lid-warming masks can be heated in the microwave and placed on the eye to facilitate meibum expression. A reasonable goal is to achieve warmth between 40º and 45º C for 5 to 10 minutes once or twice daily. In one ex-vivo study, a warm wet cloth (the standard recommendation) performed poorly, only achieving the targeted temperature for 3 minutes before quickly losing heat. By contrast, five commercially available masks (MGDRx EyeBag®, EyeDoctor®, Bruder®, Tranquileyes® goggles, and Thera-Pearl®) retained heat above 40º C for at least 5 minutes. My clinic generally recommends the Bruder® mask (Bruder Healthcare, Alpharetta GA) which contains heat-retaining Medibeads® and stays above 50º C for a longer time compared with other masks. Patients should always use caution to avoid burning the skin with any microwave-heated mask.

Lid hygiene might also include anti-Demodex and/or antibacterial foams (preferable, in my view, to the more abrasives scrubs) to treat blepharitis.
**Oral Omega-3-Fatty Acids**

Oral supplementation with omega-3-fatty acids is another therapy aimed at an underlying cause of evaporative dry eye and MGD with chronic ocular surface inflammation—an imbalance of omega-3 (not enough) and omega-6-fatty acids (too much) in the typical Western diet. Research suggests that omega-3-fatty acid deficiency impairs meibomian gland function and that supplementation with oral omega-3-fatty acids can improve meibomian gland function and reduce DED symptoms.15,17 Higher consumption of fruits, vegetables, and fish, as well as greater exposure to sunshine (for vitamin D), are also advisable. A vitamin D supplement may be necessary in winter or if vitamin D levels are inadequate.

**Other Therapies**

Artificial tears are palliative remedies in widespread use among DED sufferers for immediate relief of stinging, dryness, and other symptoms. Without a prescription, patients often make the mistake of choosing over-the-counter eyedrops that ultimately do not serve them, such as those that contain a decongestant and are not meant for long-term use. To avoid this, prescribe a particular artificial tear or topical ocular lubricating agent.18

All patients who use cell phones, tablets, or computers should be taught the value of taking frequent screen breaks, whether they have DED yet or not. Concentration on near tasks decreases the rate of spontaneous, ocular surface-refreshing blinking and causes incomplete blinking, both of which dry out the eye, destabilize the tear film, and induce inflammation. Patients who report blinking deliberately to focus their vision or whose ocular complaints are worst by end of day almost always have DED.

Instructing patients to follow the 20-20-20 rule—every 20 minutes, take a 20 second break by looking 20 feet away—helps reestablish a more natural blinking pattern and can improve symptoms of DED over time. I call this “visual hygiene.”

In my view, it is best to implement the above measures directed at long-term control for at least several weeks and give them a chance to work.2,19 At that point, if more aggressive treatment is needed, the ECP can prescribe one of the available antiinflammatory medications, including cyclosporine A, lifitegrast, corticosteroids, or doxycycline. For more severe cases, patients might benefit from placement of punctal plugs; an amniotic membrane ring such as Prokera® (BioTissue, Doral, FL) or AmbioDisk™ (IOP Ophthalmics, Costa Mesa, CA); drops made from autologous serum; or—for postmenopausal women—drops compounded with testosterone and progesterone.17,18,20 In the pipeline, an elevated concentration cyclosporine A drop formatted with nanomicellar technology (Seciera™ or OTX-101, Sun Pharmaceuticals) has recently entered phase 3 development.

**Conclusion**

Optometrists should ask their patients about symptoms and behaviors that might be related to DED, including computer use; examine their lids and ocular surface; test the composition, volume, and stability of their tear film; suggest visual hygiene, lid hygiene, and fish oil supplementation; and prescribe an artificial tear as part of an early intervention. Looking for the patient with low-grade disease and starting with small interventions helps build success and increase confidence in incorporating DED care into one’s practice.

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Communicating with Dry Eye Patients

Whitney Hauser, OD

Skillful communication—the cornerstone of a good doctor–patient relationship—is vital for effective dry eye disease management.

Dry eye is an alarmingly prevalent condition, affecting 5% to 34% of adults globally, including at least 20 million Americans. Prevalence increases with age, digital technology use, and other risk factors such as comorbid diabetes. Thus, eyecare providers should expect that a substantial proportion of patients they are seeing in everyday practice are affected and that that proportion will be increasing over time.

Dry eye disease (DED) has been defined as “an inflammatory disease of tears and ocular surface that impacts the eye’s ability to refract correctly and that, if left untreated, can have a serious impact on functional vision, eye discomfort, and patient quality of life.” As with any progressive chronic disease, identifying the condition in its early stages and offering effective counseling and treatment provides long-term benefits to patients, typically beyond what they may be able to appreciate at the time of the initial discussion.

My academic practice is unusual in that it is comprised mainly of patients with dry eye—about 80%—nearly all of whom have already seen multiple practitioners; every eyecare provider, however, regardless of the composition of their practice, should be vigilant for signs and symptoms of dry eye, familiar with key questions for uncovering it, and ready to have at least a brief, straightforward discussion about dry eye, its implications, and management. In this article, I offer practical strategies for optimizing communication with dry eye patients for successful management and long-term patient retention and satisfaction.

Who Is at Risk?

DED describes a wide range of disorders with diverse pathogenic mechanisms that share a specific endpoint—deficient ocular surface moisture. We typically think in terms of two broad categories of DED: insufficient production of aqueous (aqueous insufficiency or tear-deficient dry eye); or increased evaporation of aqueous related to some imbalance (evaporative dry eye). While all adult patients are at risk for DED—and risk increases with age—higher rates exist among females, contact lens wearers, those with lacrimal gland or eyelid damage, and patients who have undergone refractive or cataract surgery.

Patients with ocular comorbidities, particularly those that directly affect the ocular surface (eg, allergic conjunctivitis) or that require chronic topical therapies (eg, glaucoma), and patients with systemic autoimmune disease (eg, Sjogren’s syndrome, rheumatoid arthritis, thyroid disease, diabetes) are at increased risk for DED. Systemic medications that may exacerbate ocular dryness include antihistamines, decongestants, antihypertensives, antidepressants, and beta blockers. Environmental and lifestyle risk factors that may contribute to dry eye include low environmental humidity, air pollution, cigarette smoking, alcohol use, and caffeine consumption.

Taken together, one can see that most patients have multiple risk factors for DED. For example, woman over 50 already have two risk factors; add antidepressant use (about 20% among this population) and computer/digital technology use (nearly 100% of professional women and men) and risk substantially mounts.

The ubiquitous nature of digital technology—computers, tablets, smartphones, and now wearable technology—is likely having a profound effect on ocular surface health within the general and DED populations because of its inhibitory effect on spontaneous blinking.

Studies have found that we blink less frequently and less completely when our attention and gaze are focused on near tasks like reading, and that the extended interblink interval (time between blinks) is even more marked when engaged with a digital screen than hard copy text. When visually concentrated, ocular surface exposure increases beyond the tear film’s capacity to refresh it.

Communication Tip 1: Talk about Blinking

Many patients who use computers—nearly everyone in this day and age—have an incomplete blink or lid closure that is likely having a profound effect on ocular surface health within the general and DED populations because of its inhibitory effect on spontaneous blinking.

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closure during spontaneous blinking. Performing “blink exercises” at regular intervals throughout the day will eventually restore the natural full-closure blink, increase tear film meibum, reduce evaporative water loss, and improve dry eye symptoms (Figure 1).

**Suspect and Detect**

Patients with DED may present with any number of ocular symptoms related to dryness, including irritation, discomfort, burning, grittiness, foreign body sensation, blur, redness, or annoying epiphora. They may have difficulty tolerating (or simply grown less enthusiastic about) contact lens wear. They may express that their symptoms worsen as the day goes on.4,9

Many patients with objective signs of DED have little in the way of symptoms or are not cognizant of or concerned by mild symptoms. The discordance between symptoms and signs of DED presents a challenge to eyecare providers; we should be vigilant for subclinical presentations rather than waiting for patients to volunteer their complaints. For example, the patient who hesitates mid-line during Snellen visual acuity testing—only to proceed after being instructed to blink and refresh their tears—may have unrecognized DED.

Eyecare providers who proactively ask their patients about symptoms of DED are far more likely to detect it compared with those who wait for patients to volunteer their complaints. The wide array of screening questionnaires and a lack of consensus around the most appropriate or “right” one—compounded by uncertainty around best practices for diagnosis and management—can lead to a kind of “paralysis of analysis.” While it’s good to have a plan, overthinking the methodology at the expense of action does not serve patients’ or your practice’s best interest.

The best screening tool is always the one that fits with the logistics of your practice, that is—the one that you actually use. In our academic practice, we use two validated surveys: the Ocular Surface Disease Index (OSDI) and the Standard Patient Evaluation of Eye Dryness (SPEED). A private practice might select a single survey, the one that is most useful and efficient for patients and for the practice. Alternatively, providers or technicians might simply ask patients verbally about symptoms during visits (see Communication Tip 2).

**Communication Tip 2: Ask**

Experts assembled at the 2014 Dry Eye Summit suggested, that in the absence of a formal screen, these four questions are among the most likely to uncover symptoms of dry eye:3

- Do your eyes ever feel dry or uncomfortable?
- Are you bothered by changes in your vision throughout the day?
- Are you ever bothered by red eyes?
- Do you ever use or feel the need to use drops?

**Managing DED**

Successful DED management is more likely when patients have an established diagnosis, have a working understanding of their disease, and are motivated to participate actively in their own therapeutic success. Good communication lays the groundwork for a solid long-term therapeutic alliance.

My practice is to explain a little bit about the structure and function of the tear film, focusing on which layers are deficient in the patient’s individual case. It is also enormously helpful to help patients make sense of objective findings and images, such as meibomian gland obstruction and atrophy visible with meibography.

**Communication Tip 3: Tell, but Also Show**

Taking pictures and showing them to patients levels the playing field, so to speak, because it allows the clinician to say objectively, “Here’s what I see.” Seeing the changes with her own eyes, a patient may be more tuned in and open to the rest of the conversation, including what to expect and options for treatment.

**Communication Tip 4: Form a Long-Term Alliance**

Aim to be clear and straightforward but also sensitive when communicating about DED with patients. As a lifelong degenerative disease, DED can be a difficult diagnosis to receive; and some will not see the benefit of intervening at first. My practice is to present their imaging results; explain what they can expect if

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**FIGURE 1** Performing blink exercises every 10 to 12 minutes for 30 days can restore the full blink. Pause, close eyes for two seconds, then open. Pause, close eyes for two seconds, squeeze eyes tight for two seconds, then open and relax.
they don’t treat; and suggest a course of what action that they can start right away. I’ll often say, “Here’s what I would do if I were you, and I would start today.”

No matter how well you make the case, however, some asymptomatic or mildly symptomatic patients will not be compelled to start treatment. This is not a failure or a reason to push harder; rather it is an opportunity to demonstrate respect for their autonomy and position yourself as a trusted resource and long-term ally who will be there for them should they reconsider. If you leave the door open, the chronicity of the disease will likely draw them back to you.

**Communication Tip 5: Maintain an Attitude of Service**

Patients with painful chronic diseases may have recurring difficulties and complaints; dry eye is no exception. In fact, there is evidence to suggest that some patients with persistent DED symptoms have central sensitization, ie, a form of neuropathic pain.10

Patients who call frequently or express dissatisfaction at office visits should not be dismissed by staff members as a nuisance; rather, they should be respected as individuals with “complaints” that need to be addressed. Maintaining an attitude of service by listening, building rapport, and working toward a common mission of being symptom-free is essential for providers and every member of the staff who is in contact with patients. Referral may be necessary for patients who are not experiencing relief with treatment.

**Communication Tip 6: Manage Expectations**

Be honest with patients about what you can and cannot do for them; in other words, be careful not to undersell or oversell the available therapies. There are an increasing number of available therapies—from artificial tears to topical antiinflammatory agents to advanced treatments such as autologous serum and amniotic membrane treatment—and new treatments in development; but there is currently no cure for DED. That said, identifying and diagnosing DED and its underlying cause, communicating clearly, and being truthful about limitations often helps patients accept their situation and move forward with confidence.

**Conclusion**

DED is already common and increasing in prevalence within general optometric practice. As with any chronic disease, successful DED management hinges on direct, compassionate communication within a solid, trusting doctor-patient relationship. Working to improve identification of DED and refining our methods for and discussion around diagnosis and management with patients are integral to serving patients well in the 21st century.

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1. Which of the following screening questions is NOT among those recommended by Dry Eye Summit experts to uncover subtle symptoms of DED in optometry patients?
   A. Do your eyes ever feel dry or uncomfortable?
   B. Are you bothered by changes in your vision throughout the day?
   C. Do you prefer to wear glasses to contact lenses?
   D. Do you ever use or feel the need to use drops?

2. The most common cause of DED is:
   A. Excess androgen production
   B. Autoimmune disease
   C. Meibomian gland dysfunction
   D. None of the above

3. The 20-20-20 rule is:
   A. A standard for visual acuity improvement following DED therapy
   B. A guideline for relaxing near stress and rebooting the natural blink reflex
   C. A guideline for rebalancing dietary intake of essential fatty acids
   D. A tear cytokine profile used to determine appropriate antiinflammatory therapy

4. Working on a computer exacerbates DED by:
   A. Increasing full lid closure
   B. Increasing interblink interval
   C. Increasing blink rate
   D. Both A and B

5. Which of the following is NOT important to long-term DED management success?
   A. Persuading patient to start therapy at diagnosis
   B. Managing expectations, including limitations of therapy
   C. Taking patient complaints seriously
   D. Having a common goal with patients

6. Which of the following medications increases DED risk?
   A. Antihypertensives
   B. Antidepressants
   C. Antihistamines
   D. All of the above

7. Which of the following statements is true?
   A. Computer use does not significantly affect the tear film
   B. A cold dry climate and the winter season are risk factors for DED
   C. Patients who wear contact lenses are increased risk for DED
   D. Both B and C are true

8. According to the 2014 Dry Eye Summit:
   A. Optometrists should identify DED in their patients
   B. Optometrists should not attempt to manage DED
   C. Osmolarity and meibography should be performed on all patients
   D. All of the above

9. According to the 2014 Dry Eye Summit, which of the following is NOT a basic testing recommendation for DED diagnosis?
   A. Lid examination
   B. Tear film stability or osmolarity
   C. MMP-9
   D. Corneal staining

10. Which of the following is NOT associated with increased risk for DED?
    A. Advanced age
    B. Female sex
    C. Tropical climate
    D. Computer use