DRY EYE *Disease*

Prevalence, Assessment, and Management

Dry eye disease is a chronic condition of the corneal surface marked by persistent symptoms of irritation or burning that can cause inflammatory damage to the cornea and conjunctiva if untreated. Common risk factors for this syndrome include advancing age, female sex, low humidity environments, systemic medications, and autoimmune disorders. Treatments to relieve symptoms include tear replacement, humidification, improved nutrition, and anti-inflammatory ocular agents. Home healthcare nurses can identify signs and symptoms of dry eye syndrome and initiate strategies that range from warm compresses to physician referrals for more aggressive treatment. Consistent management of this condition improves quality of life and minimizes damage to the ocular surface.

Dry eye disease (DED) is a common chronic multifactorial condition of the ocular surface characterized by failure to produce high quality or sufficient amounts of tears to moisturize the eyes (Nelson et al., 2017; Tsubota et al., 2017). Messmer (2015) indicated that DED can be categorized as "dry eye with reduced tear production (aqueous deficient) and dry eye with increased evaporation of the tear film known as the hyperevaporative type" (p. 71). Although 10% of individuals have aqueous deficient DED, more than 80% have either the hyperevaporative type related to meibomian gland dysfunction (MGD), or a combination of both.

DED can substantially affect vision and quality of life, as symptoms often interfere with daily activities, such as reading, writing, or working on video display monitors. Prevalence rates range from 5% to 50%, but can be as high as 75% among adults over age 40, with women most often affected (Stapleton et al., 2017). Among younger adults ages 18 to 45 years, only 2.7% experience DED (Farrand et al., 2017). The economic impact of DED can range from $687 per person for mild disease to $1,267 annually for severe DED. The total direct cost to the U.S. economy was projected to be $3.8 billion (Bielory & Syed, 2013; Farrand et al., 2016). These costs include over-the-counter (OTC) products, prescription drugs, and punctual plug placement.

As DED prevalence increases with age and chronic illness comorbidities, home healthcare clinicians need to be aware of the signs and symptoms in their older adult patients (Messmer, 2015). Understanding the risk factors associated with DED, medications that increase the likelihood of dry eyes, as well as normal aging effects can help clinicians identify problems associated with this chronic condition.

Patricia A. Rouen, PhD, FNP-BC, and Mary L. White, PhD, FNP-BC

Risk Factors

Several risk factors have been linked to DED (Table 1) and include personal, environmental, clinical illnesses, medications, and ocular factors (Gomes et al., 2017; Milner et al., 2017; Sullivan et al., 2017). Personal risk factors include advanced age, sex, Asian ethnicity, and contact lens use (Stapleton et al., 2017; Sullivan et al.). Environmental factors such as low-humidity environments, windy settings, air-conditioned rooms, extended periods of reading or driving or exposure to screens (e.g., computer, tablets, smart phones), and second-hand smoke exposure have been associated with DED. Clinical conditions that increase DED risk include autoimmune diseases (rheumatoid arthritis [RA], sarcoidosis, Sjögren syndrome [SS]) and chronic conditions, such as thyroid abnormalities, Bell palsy, diabetes, rosacea, hepatitis C infection, seasonal and perennial allergies, and Demodex mite allergic conjunctivitis. Persons with Parkinson disease are at high risk for DED as the normal blink reflex of 16 to 18 times per minute is reduced to 1 to 2 blinks per minute (Ekker et al., 2017). Ocular surgery or injury can also result in DED (Milner et al.).

Women are more likely to experience DED, with increased prevalence after menopause. The use of estrogen alone or with progestin can worsen symptoms (Alawlaqi & Hammadeh, 2016), and androgen treatment improves dry eye symptoms (Sriprasert et al., 2016). Low dietary intake of omega-3 fatty acids and use of continuous positive airway pressure devices are additional risk factors associated with DED (American Academy of Ophthalmology [AAO], 2013; Downie & Keller, 2015). Medications such as antihistamines, beta-blockers, decongestants, diuretics, selective serotonin reuptake inhibitors, anxiolytics, tricyclic antidepressant medications, antipsychotics, oral contraceptives, antiparkinsonian agents, and oral isotretinoin are also associated with DED (AAO; Gomes et al., 2017).

Dry eye disease can substantially affect vision and quality of life, as symptoms often interfere with daily activities, such as reading, writing, or working on video display monitors.

goodluz / shutterstock

Corneal Physiology and DED

The corneal surface of the eye provides a barrier that protects the orbital structures from ultraviolet light exposure, and infectious and harmful substances. The cornea also has an important role in vision as it refracts incoming light to the lens of the eye and onto the retina. The retinal cells convert the light into impulses that are transmitted via the optic nerve to the brain, where the impulses are translated into visual images (Delmonte & Kim, 2011). More important is the role of the corneal epithelium, a highly responsive tissue that hydrates the eye and is capable of rapid cell regeneration to heal superficial ocular traumas. In minor traumas, such as an abrasion, corneal cells regenerate within 24 hours of the injury and healing is noted within 7 to 10 days. With unmanaged severe DED, repeated trauma can result in keratoconjunctivitis where the deeper corneal layers are compromised requiring a longer recovery period that is associated with greater pain, blurred vision, and light sensitivity (Delmonte & Kim). Unmanaged DED, repeated corneal abrasions, or persistent deep tears can cause corneal scarring that compromises visual acuity (AAO, 2013).

Table 1. Risk Factors of Dry Eye Disease

Category	Risk Factor	
Personal	Sex	
	Advanced age	
	Asian ethnicity	
	Contact lenses	
	Low dietary intake of omega-3 fatty acids	
Environmental	Low humidity or windy environments	
	Air-conditioning	
	Reading for long periods	
	Driving extended periods	
	Second-hand smoke exposure	
	Prolonged exposure to display monitors (computer, tablets, etc.)	
Chronic illness	Bell palsy	Parkinson disease
	Depression	Perennial/seasonal allergies
	Diabetes	Rosacea
	Glaucoma	Thyroid disease
	Hepatitis C	
Autoimmune diseases	Rheumatoid arthritis	
	Sarcoidosis	
	Sjögren syndrome	
Medications	Anticholinergics	Estrogens
	Antipsychotics	Glaucoma medications
	Antivirals	Oral contraceptives
	Beta-blockers	Opioids
	Diuretics	Selective serotonin reuptake inhibitors
Injury	LASIX refractive surgical history	
	Ocular injury	

Note. Adapted from Gomes et al. (2017), Milner et al. (2017), and Sullivan et al. (2017).

Human tears, comprised of water, proteins, electrolytes, and lipids, function to keep the ocular surface moist, and protect the cornea from trauma and infection. The corneal epithelium has a layered structure known as the "*tear film*" that hydrates and prevents damage or infection to the cornea (Messmer, 2015; Willcox et al., 2017; Zhou & Beuerman, 2012). The three layers of the corneal epithelium consist of: (a) a topical lipid layer of oils produced from the meibomian glands in the eyelids that stabilizes and lubricates the ocular surface to prevent tear evaporation, (b) a middle aqueous layer that constitutes 90% of the tear film thickness and produces tears from the lacrimal gland to hydrate the eye, and (c) an innermost third mucin layer that provides lubricating mucus from goblet cells to stabilize the aqueous layer that provides resistance to bacterial infection (Dohlman et al., 2016; National Eye Institute, 2017; Willcox et al.). Together, these layers function to maintain moisture in the cornea and conjunctival epitheliums.

Dysfunction in any layer can lead to tear hyperosmolarity (less water in the tears) from either (a) decreased aqueous tear production, and/or (b) increased tear evaporation due to compromised production of the meibomian gland oils, or (c) reduced mucin from the goblet cells (Bron et al., 2017; Willcox et al., 2017). Milner et al. (2017) added a fourth category, exposure-related DED that is related to environmental conditions, or inability to fully close the eye leaving the cornea exposed. Tear hyperosmolarity, also known as *tear film instability*, is the primary contributing factor for DED (Nelson et al., 2017). With decreased lubrication from the lipid layer and/or reduced tear quality and quantity from the second and third layers, corneal inflammation can occur, damaging the deeper basement membrane of the corneal epithelium, resulting in visual impairment and/or persistent keratitis (Bron et al.; Willcox et al.; Zhou & Beuerman, 2012).

The cornea is one of the most innervated tissues in the body (Delmonte & Kim, 2011), and when changes in the tear film layers occur, signs and symptoms are usually noted by patients.


Kerdkanno / shutterstock

A growing body of evidence from two systematic reviews and one meta-analysis demonstrates increased intake of omega-3 fatty acids improves tear production and dry eye disease symptoms.

Presenting symptoms include eye dryness, irritation, eye fatigue, a sensation of grittiness, burning or soreness, and redness (Milner et al., 2017; Zeev et al., 2014). Patients may also report vision changes, photophobia, trouble driving at night, discomfort while watching television or reading, itching, increased blinking, or contact lens intolerance (National Eye Institute, 2017; Zeev et al.). Unmanaged DED diminishes quality of life related to vision-focused activities such as reading, driving, computer use and can adversely impact outcomes in those undergoing cataract removal or refractive procedures (Milner et al.).

Evaluation of DED

Persons with DED symptoms should be referred for a complete ophthalmologic examination. The exam should include a comprehensive medical and ophthalmological history and screening for autoimmune diseases associated with DED. Symptom scales help to quantify the severity of the condition. The three most common tools utilized are: the Ocular Surface Disease Index (OSDI; Schiffman et al., 2000), the Standardized Patient Evaluation of Eye Dryness (SPEED; Ngo et al., 2013), and the Dry Eye Questionnaire (DEQ-5; Chalmers et al., 2010).

A comprehensive ophthalmologic exam for DED includes visual acuity, a refraction evaluation to determine best-corrected visual acuity, and assessment of the orbital structures including inspection of the eyelids and lashes and palpation of the meibomian glands. If relevant, examination of the face for signs of blepharitis (suggestive of MGD) or rosacea should be included (Milner et al., 2017).

If chronic illness or autoimmune diseases are suspected, examination of the small joints to identify signs of RA and assessment of the oral mucosa to corroborate SS should be conducted (Nelson et al., 2017). SS is a chronic illness that presents with discomfort from dry eyes, mouth, and skin. Many patients who have SS also experience chronic pain from joint and eye discomfort (Grossman & Tagliavini, 2015).

Additional tests to evaluate the cornea and tear film layer are recommended. The tear film layer is assessed with in-office devices that quantify the thickness of the lipid layer. Findings from this exam also evaluate the patient's blinking patterns, as partial blinkers are prone to reduced lipid production that impacts the ocular surface. A slit-lamp biomicroscopy exam should be done to evaluate tear volume and identify superficial corneal erosions, conjunctival hyperemia, corneal surface irregularities, and MGD. Stains, such as fluorescein, illuminate abnormalities, patterns, or changes in the corneal surface consistent with DED that are visible with the slit-lamp (Downie & Keller, 2015; Milner et al., 2017). Tear function is evaluated with the tear film breakup time (TBUT) test that measures the amount of time it takes for tears in a fluorescein-stained eye to break up after blinking. After several blinks, the tear film is examined using the slit lamp and blue filter to scan for dry spots on the cornea (Dohlman et al., 2016). TBUT times under 10 seconds are abnormal, indicating tear film instability (Milner et al.). The Schirmer test measures tear production from the lacrimal gland using a sterile paper strip inserted for 5 minutes into the lower eyelid in contact with the ocular surface to measure the amount of wetting of the strip. The smaller the amount of moisture on the paper, the fewer tears produced. A value of 5 mm or less is considered abnormal (Dohlman et al.; Downie & Keller).

Other tests for DED include imaging of the tear film layer, palpation of the meibomian glands, cultures from the ocular surface, and serum antibody biomarkers for autoimmune diseases (Milner et al., 2017). In-office devices are now available to measure tear osmolarity with tear samples from

both eyes (TearLab; San Diego, CA). The sample is obtained using a test pen with a test card that gently touches the eye surface near the lower eyelid. Readings over 300 mOsm/L or a difference of 8 mOsm/L between both eyes indicates tear film instability (https://www.tearlab.com). Another screening test measures an inflammatory marker, matrix metalloproteinase 9 (MMP-9) that is consistently elevated in the tears of persons with DED. The in-office test, *InflammaDry* (Quidel Corporation; San Diego, CA) uses a sample of tears from the lower eyelid and palpebral conjunctiva to measure MMP-9. A positive result corroborates a DED diagnosis (http://www.quidel.com/immunoassays/inflammadry).

Management of DED

Although guidelines categorize DED as either an aqueous or evaporative process (Nelson et al., 2017), there is variability in symptom presentation and patients may have either evaporative or aqueous disease, a combination of both phenomena or exposure-related DED (Milner et al., 2017). Although the treatment goal is to restore tear film homeostasis (Nelson et al.), heterogeneity exists in the presentation of DED and a variety of treatments (Table 2) are used to manage this syndrome.

DED is classified as mild, moderate, or severe based on symptoms and the clinical exam findings. The management plan should be guided by

Table 2. Step Therapy Management for Dry Eye Disease

Step 1 Options	Step 2 Options	Step 3 Options	Step 4 Options
■ ***Patient education:*** regarding dry eye disease, its management and prognosis ■ ***Environmental modifications:*** humidification of home and work environments; consider a portable humidifier • avoid second-hand smoke • avoid long periods of reading, watching television, or driving • limited screen time on computers and other devices, adjust computer screens to reduce eyestrain • take short breaks to rest eyes ■ ***Dietary modifications:*** • Increased intake of foods rich in omega-3 fatty acids and vitamin A • Use of USP-verified OTC omega-3 fatty acid products • Prescriptive fish oil products: Lovaza or Vascepa ■ ***Screening for medications*** that worsen dry eye disease: anticholinergics, beta-blockers, diuretics, estrogen, oral contraceptives, opioids, antipsychotics, selective serum reuptake inhibitors, antiviral agents, isotretinoin, and drugs used to treat glaucoma and Parkinson disease ■ ***Specific treatments options*** • lid hygiene with hypoallergenic products • Warm compresses and commercially available heated eyelid masks • Ocular lubricants such as preservative-free artificial tears, gels, and ointments • Teach patients to read labels to avoid ocular products with benzalkonium chloride preservatives	■ ***Treatment Options:*** • For those with MGD, reinforce lid hygiene; tea tree oil may help with mite infestation or bacterial colonization • Moisture chamber goggles/spectacles for day and nighttime use • In-office treatments with pulsed heat or pulsed light therapy to release oils from the meibomian glands ■ ***Prescriptive Options:*** • Topical antibiotic or antibiotic/steroid ocular solutions for blepharitis • Topical corticosteroids for limited durations • If topical products did not improve the MGD, oral macrolide or tetracycline can be used • Topical secretagogues are available internationally but not approved in the US • Topical ocular immunomodulary solution: Cyclosporine 0.05% ophthalmic emulsion • Topical ocular LFA-1 antagonist solution: Lifitegrast 5% ophthalmic solution	■ ***Treatment Options:*** • Oral secretagogues: Pilocarpine, Cevimeline • Review side effects with patients: sweats, nausea, diarrhea, flushing, frequent urination • Autologous/ allogeneic serum eye drops • Therapeutic contact lens options • Soft bandage lenses • Rigid scleral lenses	■ ***Treatment options*** • Topical corticosteroids for longer duration • Amniotic membrane grafts or corneal bandage lens • Surgical punctal occlusion • Other surgical options such as salivary gland transplantation for those with Sjögren syndrome

Note. LFA-1 = lymphocyte function-associated antigen; MGD = meibomian gland dysfunction; OTC = over the counter; US = United States; USP = United States Pharmacopeia. Adapted from Jones et al. (2017) and Milner et al. (2017).

the DED severity and customized to individual patient scenarios. The Tear Film Ocular Society's Dry Eye Workshop II treatment recommendations use a step therapy approach to guide care (Jones et al., 2017). First steps emphasize patient education regarding the condition, management options and the prognosis. Patient education should cover information on environmental and dietary modifications, elimination of medications that worsen DED, and personal hygiene practices that can attenuate symptoms. Environmental strategies include the use of portable humidifiers at home or in the workplace, avoiding cigarette smoke, prolonged television viewing or reading, taking frequent breaks to rest the eyes, maintaining adequate hydration and reducing use of computer display terminals. For those using computers consistently, lowering the monitor screen to below eye level decreases the lid aperture and reduces eyestrain (AAO, 2013). Asking patients to raise their desk chairs and encouraging them to blink more often also ease dry eyes when using computers (Kwan, 2017). Lastly, *EyeLeo* (http://eyeleo.com), a free application for personal computers, reminds users to take regular breaks from their screens.

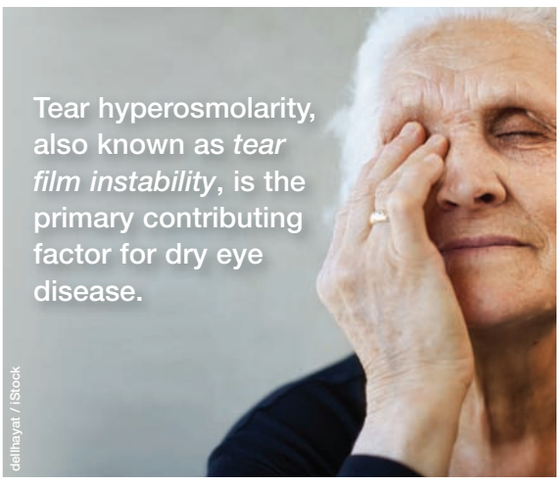
Nutritional strategies are helpful in DED management. A growing body of evidence from two systematic reviews and one meta-analysis demonstrates increased intake of omega-3 fatty acids improves tear production and DED symptoms (Kaya & Aksoy, 2016; Liu & Ji, 2014; Molina-Leyva et al., 2017). Omega-3 fatty acids (FAs) are preferred over the omega-6 or omega-9 FAs as the omega-3 FAs contain eicosatetraenoic acid (EPA) and docosahexaenoic acid (DHA) which have anti-inflammatory properties that improve tear function and DED symptoms (McCusker et al., 2016). In particular, DHA blocks oxidative reactions and prevents the release of arachidonic acid, a potent inflammatory compound (McCusker et al.). In contrast, increased intake of omega-6 FAs may incur risk for DED as when the FA is activated it releases mediators that *increase* inflammation (Funk, 2001).

Omega-3 FAs are found in many plants and vegetables including flaxseeds (the richest source); walnuts; edamame; kale; spinach; whole grains; wheat germ; black, kidney, and mung beans; squash; and broccoli (Hark et al., 2012). Omega-3 fortified eggs and milk are available in most grocery stores. Flaxseed, walnut, soy, and canola oils along with low mercury fatty fish choices such as salmon, light tuna, sardines and lake trout are rich sources of omega-3 FAs (Hark et al.). Excessive intake of mackerel, grouper, and albacore tuna is not advised given the concerns regarding heavy metal contamination (e.g., mercury) in these larger fatty fish (Bosch et al., 2016).

Two prescriptive fish oil medications are available, omega-3 acid ethyl esters that contain both EPA and DHA and icosapent ethyl capsules that only contain EPA (Weintraub, 2014). OTC fish oil supplements are also used to relieve DED symptoms. As these supplements are not regulated by the Food and Drug Administration, there is no verification that the products contain the ingredients on the label. Companies may voluntarily submit their omega-3 supplements for analysis to

Table 3. Resources for Dry Eye Disease

The National Eye Institute https://nei.nih.gov/health/dryeye/dryeye This site is part of the National Institutes of Health and includes current research on eye health and patient education resources for eye care.
Dry Eye and Meibomian Gland Dyfunction (MGD) https://dryeyeandmgd.com/ This site provides consumer education regarding MGD
American Academy of Ophthalmology https://www.aao.org/eye-health This site provides health professional and consumer information on dry eye disease and other eye conditions.
EyeLeo eyeleo.com/ This site provides a downloadable PC and MAC application that sends regular messages to take short breaks to rest your eyes.
United States Pharmacopeia (USP) Dietary Supplementation program https://www.usp.org/verification-services This USP Dietary Supplementation Verification Services are offered to manufacturers worldwide to submit their products for verification of the product's contents. Lists of verified products can be found at this site.
Dry Eye Assessment Tools *Ocular Surface Disease Index (OSDI)* Schiffman et al. (2000) http://www.dryeyezone.com/documents/osdi.pdf *Standardized Patient Evaluation of Eye Dryness (SPEED)* Ngo et al. (2013) https://dryeyeandmgd.com/wp-content/uploads/2017/04/Official-SPEED-Questionnaire.pdf *Dry Eye Questionnaire 5* Chalmer et al. (2010) To request permission to use the DEQ-5, contact copyright.com



the U.S. Pharmacopieia (USP) Dietary Supplementation program (https://www.usp.org/verification-services). If the product meets the reference standards, a *USP-verified seal* is provided and the products are published on their website (Table 3). USP verified fish oil products include supplements manufactured by the Nature Made (Pharmavite; San Fernando, CA) and Kirkland Signature brands (Costco; Issaquah, WA). Patient education should include contraindications and precautions for omega-3 FA use as these products can cause increased bleeding risk especially for those on anticoagulants. Caution is advised for patients receiving immunosuppressive therapy, as these supplements can further decrease the immune response (Hark et al., 2012). Gastrointestinal side effects include diarrhea, loose stool, and "fish" burps (Poteet, 2017).

Vitamin A plays a role in maintaining vision, especially night vision (Saffel-Shrier, 2016). A form of vitamin A, cis-retinal, is required for retinal rod cell formation; other provitamin A carotenoids have anti-inflammatory properties that attenuate cellular damage in the eye (Saffel-Shrier). Vitamin A also supports the conjunctival goblet cells to produce mucin that stabilizes the tear film (Milner et al., 2017). These data suggest increased intake of foods rich in vitamin A such as fortified cereals, apricots, cantaloupe, mangos, beets, broccoli, red peppers, mustard greens, kale, spinach, carrots, sweet potatoes and tomatoes may improve DED symptoms (Hark et al., 2012).

A review of a patient's medication list can identify pharmaceuticals that worsen DED. Medications known to aggravate DED include antihistamines, beta-blockers, decongestants, diuretics, selective serotonin reuptake inhibitors, anxiolytics, tricyclic antidepressant medications, antipsychotics, oral contraceptives, estrogen therapy, antiparkinsonian agents, and oral isotretinoin (Jones et al., 2017). Strategies to adjust the medication plan require collaboration with both primary care providers and ophthalmologists to manage both the patient's DED and their other clinical conditions.

For those with aqueous disease, first-line treatments for DED are usually OTC preservative-free artificial tears (Kwan, 2017). In addition to artificial tears, preservative-free OTC gels and ointments are available to lubricate the ocular surface and are often used at night. Patients should be cautioned to read package labels to review the ingredient list for both tears and ointments to avoid products containing the preservative benzalkonium chloride that aggravates DED. Most OTC products can be used as needed. Moshirfar et al. (2014) conducted a systematic review comparing the many brands of artificial tears, noting that most formulations provided symptom relief but some brands were superior, such as Systane Ultra (Alcon Corporation; Fort Worth, TX) and Soothe (Bausch & Lomb Corporation; Rochester, NY).

Lid hygiene is an effective strategy for MGD. Daily lid hygiene practices with warmed compresses, hypoallergic cleansing products and gentle massage to express the lipid oils is recommended (Jones et al., 2017). Commercially available heated eyelid masks are also helpful for patients with MGD. One intervention tested the effects of commercial warmed compresses on tear film stability comparing TBUT and lipid layer thickness at baseline and postintervention. Increases in the TBUT and the lipid layer thickness were observed, suggesting these products are an option to relieve DED symptoms (Bilkhu et al., 2014).

If symptoms are not improved with Step 1 treatments, Step 2 options include moisture chamber spectacles that can be used during the day and at night (Jones et al., 2017). These products were evaluated in two studies. Shen et al. (2016) noted increases in the lipid layer and tear meniscus with increased TBUT after 90 minutes of moisture chamber spectacle use, whereas Waduthantri et al. (2015) demonstrated significant relief of dry eye symptoms and improvements in corneal surface but no increase in TBUT.

Tear conservation with punctal plugs is another Step 2 option (Jones et al., 2017). The punctal plugs are inserted into the lower eyelid tear ducts to keep the natural tears from evaporating. There are two types of punctal plugs, semipermanent ones made of silicone and natural collagen plugs that are dissolvable and primarily used short term during recovery from LASIK surgery. For those with DED, the silicone plugs are preferred.

If the patient has MGD, assessment for infection with the Demodex mites should occur and if present, treatment with lid hygiene practices initiated. Tea tree oil is also a recommended treatment to eradicate the mites (Jones et al., 2017). New in-office treatments using heat or pulsed light therapies to express the meibomian gland fluids are available from trained ophthalmologists (Jones et al.). These treatments deliver precise heat treatments with direct massage to the upper and lower eyelids to remove meibomian gland blockages. A review of 30 studies using pulsed heat treatments documented that DED symptom relief lasted up to 12 months (Blackie et al., 2015). Recently, a growing body of evidence has shown in-office procedures with pulsed light (laser) treatments followed by meibomian gland massage have shown improvement in patient's DED symptoms, increased TBUT and reduced tear osmolarity (Dell et al., 2017).

Other second-line treatments include anti-inflammatory ocular preparations such as cyclosporine 0.05% ophthalmic emulsion (RESTASIS), and lifitegrast 5% ophthalmic solution (XIIDRA). These products should be prescribed by ophthalmologists and can be used for aqueous deficiency, MGD, and mucin deficiency (Jones et al., 2017). As these are immunosuppressive agents, education on proper use of the medication and monitoring for signs of eye infection should be provided.

For those with MGD, topical antibiotic ocular solutions such as azithromycin, erythromycin, or bacitracin can relieve symptoms. In severe cases, topical steroids should be used judiciously to relieve the inflammation as prolonged use suppresses the immune response and can precipitate glaucoma (Farkouh et al., 2016). If topical antibiotic solutions are ineffective, oral antibiotics are used to treat MGD (Jones et al., 2017). Two topical secretagogues are available internationally for DED but are not currently approved for use in the United States (Milner et al., 2017, p. 23).

If Step 2 treatments are ineffective, Step 3 options such as use of rigid scleral contact lenses, soft bandage lenses and therapeutic contact lens are considered. These lenses cover and protect a greater area of the corneal surface to maintain moisture and promote healing (Jones et al., 2017). Autologous serum (AS) eye drops derived from one's own plasma can be used as a Step 3 treatment (Kwan, 2017). These preparations contain anti-inflammatory factors that prevent the cornea's cascade of inflammation. A recent review evaluated five randomized controlled trials comparing AS versus artificial tears or saline, noting that there was some benefit observed with the AS solution compared with artificial tears, but no evidence of benefit after 2 weeks (Pan et al., 2017, p. 2). If Step 3 treatments are unsuccessful, Step 4 options include amniotic membrane grafts, topical corticosteroids for longer durations, or surgical punctal closure. A cryopreserved amniotic membrane biologic corneal bandage lens (Prokera®, Bio-Tissue corporation; Miami, FL) is also available for severe corneal erosion or keratitis. For patients with comorbid conditions such as rosacea, seborrheic dermatitis, RA, or SS, treatments to manage these conditions may also relieve DED (Jones et al.). Among patients with SS, salivary gland transplantation is a novel new treatment approach (Kwan).

Role of the Home Healthcare Clinician

As healthcare advocates for homebound patients, home care clinicians are in a position to identify conditions that affect quality of life. DED is one such condition that can be annoying, distracting, and painful. Home care clinicians can assess the eye symptoms with validated scales and implement strategies to reduce the effects of DED, and in collaboration with primary care providers and ophthalmologists work together to revise medication regimens to manage the patient's DED and their comorbidities.

Home care clinicians provide and reinforce patient education to their patients and family members on management of chronic conditions. When DED symptoms are identified, Step 1 treatments for relief can be initiated and implemented by the patient and/or caregivers. Nutritional interventions and resources for DED management can also be shared. If the condition continues without relief, coordination with the primary physician should occur and may lead to a referral to an

ophthalmologist. With an understanding of DED and available patient resources, patients can learn to mitigate the symptoms and discomfort associated with this chronic condition. ■

Patricia A. Rouen, PhD, FNP-BC, is a Professor, McAuley School of Nursing, University of Detroit Mercy, Detroit, Michigan.

Mary L. White, PhD, FNP-BC, is an Associate Professor, McAuley School of Nursing, University of Detroit Mercy, Detroit, Michigan.

The authors declare no conflicts of interest.

Address for correspondence: Patricia A. Rouen, PhD, FNP-BC, McAuley School of Nursing, University of Detroit Mercy, 4001 W. McNichols Road, Detroit, MI 48221 (rouenpa@udmercy.edu).

DOI:10.1097/NHH.0000000000000652

REFERENCES

AlAwlaqi, A., & Hammadeh, M. (2016). Examining the relationship between hormone therapy and dry-eye syndrome in postmenopausal women: A cross-sectional comparison study. *Menopause, 23*(5), 550-555. doi:10.1097/GME.0000000000000570

American Academy of Ophthalmology Cornea/External Disease Panel. (2013). *Preferred Practice Pattern Guidelines. Dry Eye Syndrome*. San Francisco, CA: American Academy of Ophthalmology. Retrieved from http://www.aao.org/ppp

Bielory, L., & Syed, B. A. (2013). Pharmacoeconomics of anterior ocular inflammatory disease. *Current Opinion in Allergy and Clinical Immunology, 13*(5), 537-542. doi:10.1097/ACI.0b013e328364d843

Bilkhu, P. S., Naroo, S. A., & Wolffsohn, J. S. (2014). Randomised masked clinical trial of the MGDRx eyebag for the treatment of meibomian gland dysfunction-related evaporative dry eye. *The British Journal of Ophthalmology, 98*(12), 1707-1711. doi:10.1136/bjophthalmol-2014-305220

Blackie, C. A., Carlson, A. N., & Korb, D. R. (2015). Treatment for meibomian gland dysfunction and dry eye symptoms with a single-dose vectored thermal pulsation: A review. *Current Opinion in Ophthalmology, 26*(4), 306-313. doi:10.1097/ICU.0000000000000165

Bosch, A. C., O'Neill, B., Sigge, G. O., Kerwath, S. E., & Hoffman, L. C. (2016). Heavy metals in marine fish meat and consumer health: A review. *Journal of the Science of Food and Agriculture, 96*(1), 32-48. doi:10.1002/jsfa.7360

Bron, A. J., de Paiva, C. S., Chauhan, S. K., Bonini, S., Gabison, E. E., Jain, S., …, Sullivan, D. A. (2017). TFOS DEWS II pathophysiology report. *The Ocular Surface, 15*(3), 438-510. doi:10.1016/j.jtos.2017.05.011

Chalmers, R. L., Begley, C. G., & Caffery, B. (2010). Validation of the 5-item dry eye questionnaire (DEQ-5): Discrimination across self-assessed severity and aqueous tear deficient dry eye diagnoses. *Contact Lens and Anterior Eye, 33*(2), 55-60. doi:10.1016/j.clae.2009.12.010

Dell, S. J., Gaster, R. N., Barbarino, S. C., & Cunningham, D. N. (2017). Prospective evaluation of intense pulsed light and meibomian gland expression efficacy on relieving signs and symptoms of dry eye disease due to meibomian gland dysfunction. *Clinical Ophthalmology (Auckland, NZ)*, *11*, 817-827. doi:10.2147/OPTH.S130706

DelMonte, D. W., & Kim, T. (2011). Anatomy and physiology of the cornea. *Journal of Cataract & Refractive Surgery, 37*(3), 588-598. doi:10.1016/j.jcrs.2010.12.037

Dohlman, T. H., Ciralsky, J. B., & Lai, E. C. (2016). Tear film assessments for the diagnosis of dry eye. *Current Opinion in Allergy & Clinical Immunology, 16*(5), 487-491. doi:10.1097/ACI.0000000000000307

Downie, L. E., & Keller, P. R. (2015). A pragmatic approach to dry eye diagnosis: Evidence into practice. *Optometry and Vision Science, 92*(12), 1189-1197. doi:10.1097/OPX.0000000000000721

Ekker, M. S., Janssen, S., Seppi, K., Poewe, W., de Vries, N. M., Theelen, T., …, Bloem, B. R. (2017). Ocular and visual disorders in Parkinson's disease: Common but frequently overlooked. *Parkinsonism and Related Disorders, 40*, 1-10. doi:10.1016/j.parkreldis.2017.02.014

Farkouh, A., Frigo, P., & Czejka, M. (2016). Systemic side effects of eye drops: A pharmacokinetic perspective. *Clinical Ophthalmology (Auckland, NZ), 10*, 2433-2441. doi:10.2147/OPTH.S118409

Farrand, K. F., Fridman, M., Stillman, I. Ö., & Schaumberg, D. A. (2017). Prevalence of diagnosed dry eye disease in the United States among adults aged 18 years and older. *American Journal of Ophthalmology*. Advance online publication. doi:10.1016/j.ajo.2017.06.033

Farrand, K. F., Stillman, I. Ö., Fridman, M., & Schaumberg, D. A. (2016). Impact of dry eye disease on quality of life, work productivity, daily activities, and health care resource use in a survey of 74,095 American adults. *Value in Health, 19*(3), A127. Retrieved from https://www.ispor.org/research_pdfs/52/pdffiles/PSS30.pdf

Funk, C. D. (2001). Prostaglandins and leukotrienes: Advances in eicosanoid biology. *Science, 294*(5548), 1871-1875. doi:10.1126/science.294.5548.1871

Gomes, J. A. P., Azar, D. T., Baudouin, C., Efron, N., Hirayama, M., Horwath-Winter, J., …, Wolffsohn, J. S. (2017). TFOS DEWS II iatrogenic report. *The Ocular Surface, 15*(3), 511-538. doi:10.1016/j.jtos.2017.05.004

Grossman, S., & Tagliavini, L. B. (2015). Managing Sjogren's syndrome. *Home Healthcare Now, 33*(9), 487-492. doi:10.1097/NHH.0000000000000295

Hark, L., Ashton, K., & Deen, D. (2012). *The Nurse Practitioner's Guide to Nutrition*. Hoboken, NJ: Wiley & Blackwell.

Jones, L., Downie, L. E., Korb, D., Benitez-del-Castillo, J. M., Dana, R., Deng, S. X., …, Craig, J. P. (2017). TFOS DEWS II management and therapy report. *The Ocular Surface, 15*(3), 575-628. doi:10.1016/j.jtos.2017.05.006

Kaya, A., & Aksoy, Y. (2016). Omega-3 fatty acid supplementation improves dry eye symptoms in patients with glaucoma: Results of a prospective multicenter study. *Clinical Ophthalmology (Auckland, NZ)*, *10*, 911-912. doi:10.2147/OPTH.S111473

Kwan, J. (2017, May 15). A comprehensive look at dry eye therapy. *Review of Optometry, 154*(5), 91-98. Retrieved from http://www.reviewofoptometry.com. Accessed August 26, 2017.

Liu, A., & Ji, J. (2014). Omega-3 essential fatty acids therapy for dry eye syndrome: A meta-analysis of randomized controlled studies. *Medical Science Monitor: International Medical Journal of Experimental and Clinical Research, 20*, 1583-1589. doi:10.12659/MSM.891364

McCusker, M. M., Durrani, K., Payette, M. J., & Suchecki, J. (2016). An eye on nutrition: The role of vitamins, essential fatty acids, and antioxidants in age-related macular degeneration, dry eye syndrome, and cataract. *Clinics in Dermatology, 34*(2), 276-285. doi:10.1016/j.clindermatol.2015.11.009

Messmer, E. M. (2015). The pathophysiology, diagnosis, and treatment of dry eye disease. *Deutsches Ärzteblatt International, 112*(5), 71-82. doi:10.3238/arztebl.2015.0071

Milner, M. S., Beckman, K. A., Luchs, J. I., Allen, Q. B., Awdeh, R. M., Berdahl, J., …, Yeu, E. (2017). Dysfunctional tear syndrome: Dry eye disease and associated tear film disorders—New strategies for diagnosis and treatment. *Current Opinion in Ophthalmology, 27*(Suppl. 1), 3-47. doi:10.1097/01.icu.0000512373.81749.b7

Molina-Leyva, I., Molina-Leyva, A., & Bueno-Cavanillas, A. (2017). Efficacy of nutritional supplementation with omega-3 and omega-6 fatty acids in dry eye syndrome: A systematic review of randomized clinical trials. *Acta Ophthalmologica, 95*(8), e677-e685. doi:10.1111/aos.13428

Moshirfar, M., Pierson, K., Hanamaikai, K., Santiago-Caban, L., Muthappan, V., & Passi, S. F. (2014). Artificial tears potpourri: A literature review. *Clinical ophthalmology (Auckland, NZ)*, *8*, 1419. doi:10.2147/OPTH.S65263

National Eye Institute. (2017). *Dry Eye Facts*. Washington, DC: Author. Retrieved from https://nei.nih.gov/health/dryeye/dryeye

Nelson, J. D., Craig, J. P., Akpek, E. K., Azar, D. T., Belmonte, C., Bron, A. J., …, Sullivan, D. A. (2017). TFOS DEWS II Introduction. *The Ocular Surface, 15*(3), 269-275. doi:10.1016/j.jtos.2017.05.005

Ngo, W., Situ, P., Keir, N., Korb, D., Blackie, C., & Simpson, T. (2013). Psychometric properties and validation of the Standard Patient Evaluation of Eye Dryness questionnaire. *Cornea, 32*(9), 1204-1210. doi:10.1097/ICO.0b013e318294b0c0

Pan, Q., Angelina, A., Marrone, M., Stark, W. J., & Akpek, E. K. (2017). Autologous serum eye drops for dry eye. *The Cochrane Database of Systematic Reviews*, *2*, CD009327. doi:10.1002/14651858.CD009327.pub3

Poteet, J. (2017, May 15). From alpha to omega: How fatty acids fight dry eye. *Review of Optometry, 154,* 78-83. Retrieved from http://www.reviewofoptometry.com. Accessed August 26, 2017.

Saffel-Shrier, S. (2016). Vitamin status and requirements of the older adult. In M. Bernstein & N. Munoz (Eds.), *Nutrition for the Older Adult* (2nd ed., pp. 73-86). Burlington, MA: Jones & Bartlett Learning.

Schiffman, R. M., Christianson, M. D., Jacobsen, G., Hirsch, J. D., & Reis, B. L. (2000). Reliability and validity of the ocular surface disease index. *Archives of Ophthalmology, 118*(5), 615-621. doi:10.1001/archopht.118.5.615

Shen, M., Li, J., Wang, J., Ma, H., Cai, C., Tao, A., …, Lu, F. (2009). Upper and lower tear menisci in the diagnosis of dry eye. *Investigative Ophthalmology & Visual Science, 50*(6), 2722-2726. doi:10.1167/iovs.08-2704

Sriprasert, I., Warren, D. W., Mircheff, A. K., & Stanczyk, F. Z. (2016). Dry eye in postmenopausal women: A hormonal disorder. *Menopause, 23*(3), 343-351. doi:10.1097/GME.0000000000000530

Stapleton, F., Alves, M., Bunya, V. Y., Jalbert, I., Lekhanont, K., Malet, F., …, Jones, L. (2017). TFOS DEWS II Epidemiology Report. *The Ocular Surface, 15*(3), 334-365. doi:10.1016/j.jtos.2017.05.003

Sullivan, D. A., Rocha, E. M., Aragona, P., Clayton, J. A., Ding, J., Golebiowski, B., …, Willcox, M. D. P. (2017). TFOS DEWS II sex, gender, and hormones report. *The Ocular Surface, 15*(3), 284-333. doi:10.1016/j.jtos.2017.04.001

Tsubota, K., Yokoi, N., Shimazaki, J., Watanabe, H., Dogru, M., Yamada, M., …, Liu, Z. (2017). New perspectives on dry eye definition and diagnosis: A consensus report by the Asia Dry Eye Society. *The Ocular Surface, 15*(1), 65-76. doi:10.1016/j.jtos.2016.09.003

Waduthantri, S., Tan, C. H., Fong, Y. W., & Tong, L. (2015). Specialized moisture retention eyewear for evaporative dry eye. *Current Eye Research, 40*(5), 490-495. doi:10.3109/02713683.2014.932389

Weintraub, H. S. (2014). Overview of prescription omega-3 fatty acid products for hypertriglyceridemia. *Postgraduate Medicine, 126*(7), 7-18. doi:10.3810/pgm.2014.11.2828

Willcox, M. D. P., Argüeso, P., Georgiev, G. A., Holopainen, J. M., Laurie, G. W., Millar, T. J., …, Jones, L. (2017). TFOS DEWS II tear film report. *The Ocular Surface, 15*(3), 366-403. doi:10.1016/j.jtos.2017.03.006

Zeev, M. S., Miller, D. D., & Latkany, R. (2014). Diagnosis of dry eye disease and emerging technologies. *Clinical Ophthalmology (Auckland, NZ), 8*, 581-590. doi:10.2147/OPTH.S45444

Zhou, L., & Beuerman, R. W. (2012). Tear analysis in ocular surface diseases. *Progress in Retinal and Eye Research*, *31*(6), 527-550. doi:10.1016/j.preteyeres.2012.06.002

For additional continuing nursing education activities on home healthcare topics, go to nursingcenter.com/ce.

Instructions for Taking the CE Test Online
Dry Eye Disease: Prevalence, Assessment, and Management

- Read the article. The test for this CE activity can be taken online at www.nursingcenter.com/ce/HHN. Tests can no longer be mailed or faxed.
- You will need to create a free login to your personal CE Planner account before taking online tests. Your planner will keep track of all your Lippincott Professional Development online CE activities for you.
- There is only one correct answer for each question. A passing score for this test is 14 correct answers. If you pass, you can print your certificate of earned contact hours and the answer key. If you fail, you have the option of taking the test again at no additional cost.
- For questions, contact Lippincott Professional Development: 1-800-787-8985.

Registration Deadline: March 6, 2020

Disclosure Statement:

The authors and planners have disclosed no potential conflicts of interest, financial or otherwise.

Provider Accreditation:

Lippincott Professional Development, will award 1.5 contact hours for this continuing nursing education activity.

Lippincott Professional Development is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.

This activity is also provider approved by the California Board of Registered Nursing, Provider Number CEP 11749 for 1.5 contact hours. Lippincott Professional Development is also an approved provider of continuing nursing education by the District of Columbia, Georgia, and Florida CE Broker #50-1223.

Payment:

- The registration fee for this test is $17.95.