EYEING UP EVAPORATION: GETTING AT THE HEART OF DRY EYE DISEASE


CECELIA KOETTING, OD, FAAO, DIPABO, AND MILE BRUJIC, OD, FAAO

Dry eye disease (DED) is a common disorder affecting millions of Americans[1] and is a common reason people seek eye care.[2] DED is a known contributor to fluctuating vision, and in addition to causing pain and discomfort, severe DED requiring surgery has been shown to cause a decrease in patients' quality of life comparable with that in dialysis, severe angina, and disabling hip fractures.[3] At the heart of DED is the overall health of the ocular surface. Cecelia Koetting, OD, FAAO, DipABO, and Mile Brujic, OD, FAAO, participated in a recent webinar to discuss the underlying mechanisms and pathology of the disease.

TEAR FILM AND OCULAR HOMEOSTASIS

The ocular tear film is composed of three integrated layers: lipids that prevent tears from evaporating between blinks, aqueous that comprises the bulk of the tear film, and mucin that helps spread the tears across the ocular surface (Figure 1).[4] A healthy tear film lubricates the eyes for comfort, protects them from injury and infection, washes away foreign particles and debris from the turnover of epithelial cells, and maintains the smooth refractive surface necessary for clear vision.[5]

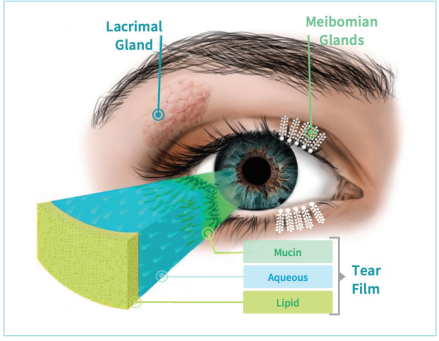

Figure 1. Tear film composition.[4]

Ocular surface homeostasis depends on a balance of tear production and evaporation, and when this is disrupted, DED is usually present.[6] Deficiencies of one or a combination of lipids, aqueous, or mucin,[6] as well as certain lifestyle conditions,[7] can cause evaporation to exceed supply. This leads to desiccation stress and a cycle of tissue damage and inflammation (Figure 2).[8]

DRY EYE DISEASE ETIOLOGY

Dry eye disease can be classified into evaporative or aqueous deficient etiologies. Evaporative etiology comprises at least half of all cases, while around 14% stem from a pure aqueous deficiency; interestingly, at least 36% of cases involve both etiologies.[6] "Nearly 90% of DED cases have an evaporative etiology (Figure 3).[9] In my experience, a large percentage of my patients show a combination," said Dr. Brujic.

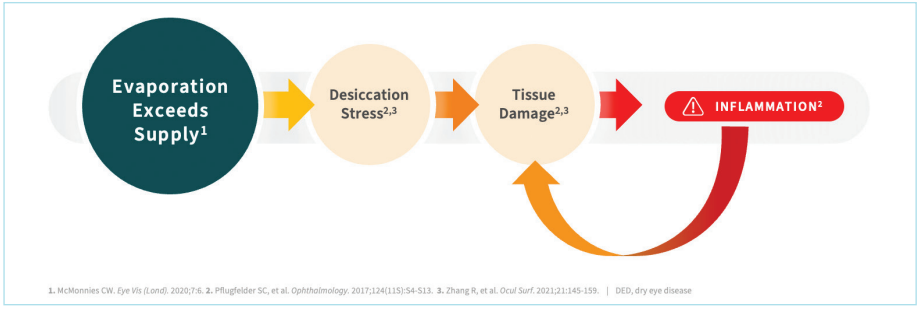

Figure 2. Excessive tear evaporation leads to inflammation, damage, and disease progression in DED.

Multiple factors contribute to excessive tear evaporation and tear film instability, including lifestyle factors, lipid deficiency, and low aqueous volume (Figure 4). "We have to think about anatomy and blink rates and how our patients spend their day—screen time, their job, environment, and sleep patterns," said Dr. Koetting.

THE ROLE OF MEIBOMIAN GLAND DYSFUNCTION IN DED

Meibomian gland dysfunction (MGD) is a major contributor to DED.[6] The main consequence of MGD is excessive evaporation, and when meibomian gland function is disrupted, tear film composition—and consequently ocular

Figure 3. In up to 90% of cases, DED has an evaporative etiology.[6]

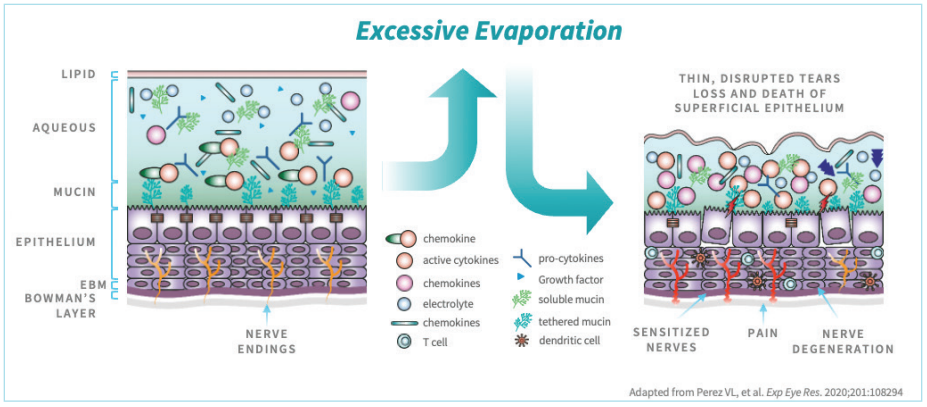

Figure 4. Multiple factors contribute to excessive tear evaporation in DED.[12]

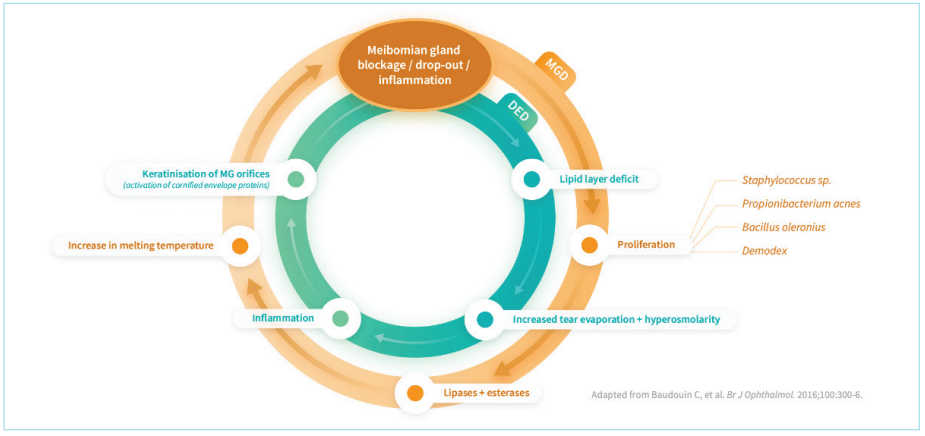

Figure 6. MGD-induced evaporation leads to ocular surface damage and inflammation.[11]

surface health—is negatively impacted due to the decline in quality and quantity of meibum secreted.[10]

"Tear evaporation tends to be at a higher rate in individuals with MGD so they are losing tears off the ocular surface more quickly (Figure 5). For ocular homeostasis to be present, the oil layer must be the appropriate thickness and consistency. If it is not, openings occur within the tear film and allow more evaporation to occur, which can lead to desiccation of the epithelial cells. The lipid layer thickness is directly associated with meibomian gland loss. The fewer functioning meibomian glands there are, the thinner the lipid layer is," said Dr. Brujic.

MGD-induced evaporation also leads to inflammation and damage, potentially triggering a cascade effect of desiccation stress and ocular surface inflammation that can damage the cornea (Figure 6).[11] "Evaporation leads to more stress on epithelial cells, and that desiccation stress leads to tissue damage and creates a feedback cycle. We see dead and devitalized cells that can get worse depending on what's going on. This leads to MMP-9s and other inflammatory markers swimming around and perpetuating the cycle. It is no longer a healthy functioning tear film," said Dr. Koetting.

DISRUPTING THE CYCLE OF OCULAR SURFACE DAMAGE AND INFLAMMATION

DED is a multi-factorial disease perpetuated by lifestyle factors, lipid deficiency, and low aqueous volume that adversely impacts millions. DED etiology can be divided into aqueous deficient and evaporative; however, over one-third of cases are mixed, with most cases having an evaporative etiology. MGD is present in most cases and is the primary reason for lipid layer insufficiency, and, consequently, reducing its function of protecting the ocular surface. Ultimately, this leads to rapid tear film disruption, tear film instability, and faster tear evaporation, kicking off a perpetual cycle of ocular surface desiccation and inflammation. "We need to look at the whole picture to determine what is at the heart of patients' DED. Then, we can begin the steps that will lead them down a path to ocular surface homeostasis," concluded Dr. Brujic. ■

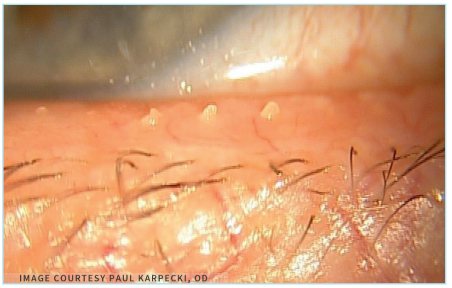

Figure 5. Plugged meibomian glands lead to lipid deficiency.

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