Contact Lens Update
CLINICAL INSIGHTS BASED IN CURRENT RESEARCH

Summary: Pathophysiology report

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Bron et al.: TFOS DEWS II pathophysiology report. Ocul Surf 2017;15(3): 438-510.

The report on pathophysiology lists two main sub-types of dry eye: aqueous tear-deficient and evaporative, initially established in the 1995 NEI/Industry workshop on dry eye disease[1] and further described in the 2007 dry eye report from the Tear Film and Ocular Surface Society (TFOS).[2]

Aqueous tear-deficient dry eye

Aqueous deficient dry eye (ADDE) describes eyes that have a normal rate of tear evaporation, but where insufficient tears are secreted (for example due to lacrimal gland damage), resulting in hyperosmolarity of the tears. Typical causes of ADDE include lacrimal gland obstruction, the use of systemic drugs such as anti-histamines or Betablockers, or immune cell infiltration of the lacrimal glands as found in patients with auto-immune disorders such as Sjögren syndrome. Patients with ADDE often report dry eye symptoms that include blurred vision, grittiness and ocular discomfort, along with clinical signs such as tear film instability and conjunctival staining.

Evaporative dry eye

Evaporative dry eye (EDE) describes eyes with normal lacrimal function but excessive evaporation of the tear film, leading to tear hyperosmolarity. Meibomian gland dysfunction is considered the major cause of EDE. Hybrid forms of ADDE and EDE exist, as found in Sjögren's syndrome, which can include both lacrimal deficiency as well as meibomian gland disease.

According to the pathophysiology sub-committee, the use of the terms EDE and ADDE should be retained to describe the initiating basis of dry eye disease. Depending on its progression, any form of dry eye disease may take on additional evaporative features, and should be described and referred to accordingly.

Mechanisms of dry eye

Dry eye disease is initiated by desiccating stress and a chain of inflammatory events that lead to hyperosmolar tissue damage and ultimately a vicious circle (Figure 1) of disease.

The core mechanism of dry eye disease is tear hyperosmolarity, which:

- is caused by evaporation from the ocular surface, in both ADDE and EDE. Irrespective of dry eye disease type, additional evaporation may occur due to environmental (e.g. wind, humidity) and/or personal factors (blink rate, aperture size);
- causes ocular surface damage, both directly and by initiating inflammation;
- sets up a series of signaling events within epithelial cells on the ocular surface. Due to these events, various inflammatory mediators and proteases are released, which may lead to a loss of both epithelial cells and goblet cells, and decreased surface wettability, which is associated with a quicker tear film break-up and increased hyperosmolarity.

The vicious circle of dry eye disease: multiple entry points

Although tear hyperosmolarity is a frequent starting point for dry eye disease, any dry eye etiology may have other (and at times multiple) entry points into the vicious circle (Figure 1), with tear hyperosmolarity and inflammatory events being the common denominators after it has been entered.

Tear film instability is an example of a different entry point into the vicious circle in patients with EDE, where tear film instability may be caused by conditions such as ocular allergy or because of topical preservative use or contact lens wear; in these cases, tear film instability is considered to be the initiator for tear hyperosmolarity to occur.

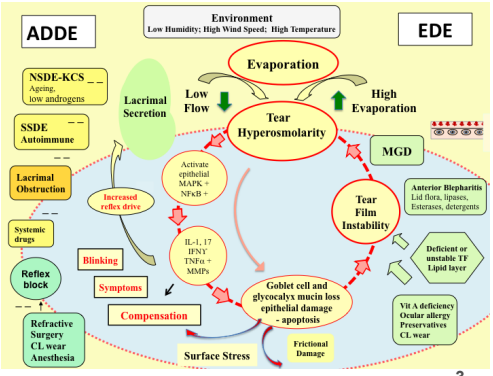

Figure 1: The vicious circle of dry eye disease[3]

REFERENCES

1. Lemp MA. Report of the National Eye Institute/ industry workshop on clinical trials in dry eyes. *CLAO J* 1995;21:221-2.
2. Report of the international dry eye workshop (DEWS). *Ocul Surf* 2007;5:65-204.
3. Bron et al.: TFOS DEWS II pathophysiology report. *Ocul Surf* 2017;15(3): 438-510.