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Introduction

Dry eye disease (DED) afflicts hundreds of millions of people throughout the world, is one of the most frequent causes of patient visits to eye care practitioners, and has no cure. Moderate to severe DED is associated with significant pain, limitations in performing daily activities, reduced vitality, poor general health, and often depression.

To increase our understanding of DED, the Tear Film & Ocular Surface Society (TFOS), a non-profit organization, launched the TFOS Dry Eye Workshop II (TFOS DEWS II). This Workshop involved the efforts of 150 clinical and basic science research experts from around the world and required more than 2 years to complete.

The TFOS DEWS II report was published in the July 2017 issue, and the Executive Summary in the October 2017 issue, of The Ocular Surface and downloadable versions are available on the TFOS website: www.TearFilm.org.

Some highlights of the conclusions and recommendations of the TFOS DEWS II are presented in this summary.

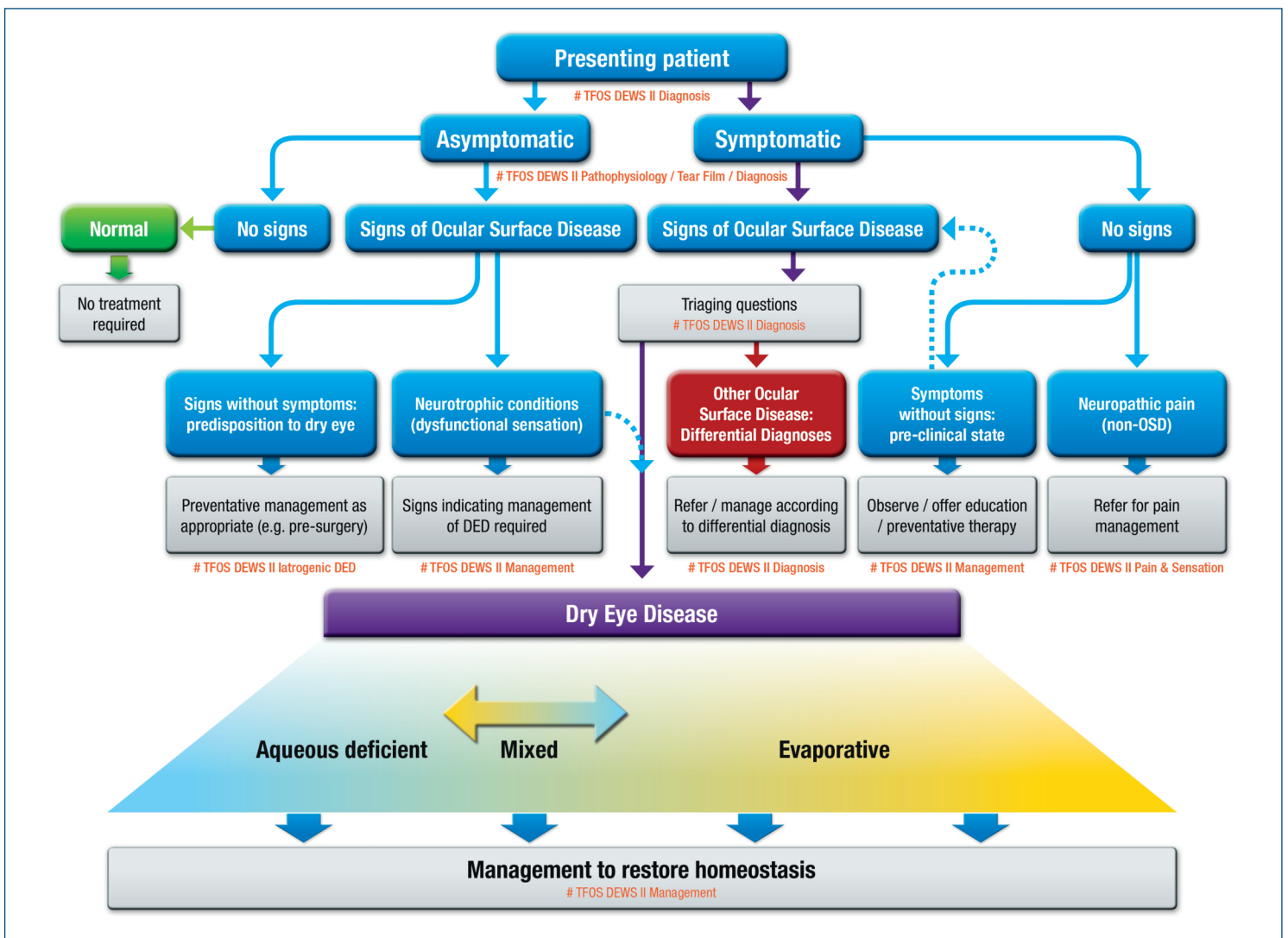


Figure 1. Classification of DED

What is DED?

TFOS DEWS II defined DED as follows:

“Dry eye is a multifactorial disease of the ocular surface characterized by a loss of homeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles.”

DED is classified etiologically into two predominant and non-mutually exclusive categories: aqueous deficient (ADDE) and evaporative (EDE) (Figure 1). Most DED is evaporative in nature.

The unifying characteristic of DED is the loss of tear film homeostasis. Such imbalance may be detected as an abnormality in any one or more of a myriad of tear film and ocular surface features.

Why does DED occur?

The core mechanism of DED is evaporation-induced tear hyperosmolarity, which is the hallmark of the disease (Figure 2). It damages the ocular surface both directly and by initiating inflammation, which can lead to a self-perpetuating, vicious cycle of DED. The major cause of EDE is meibomian gland dysfunction (MGD).

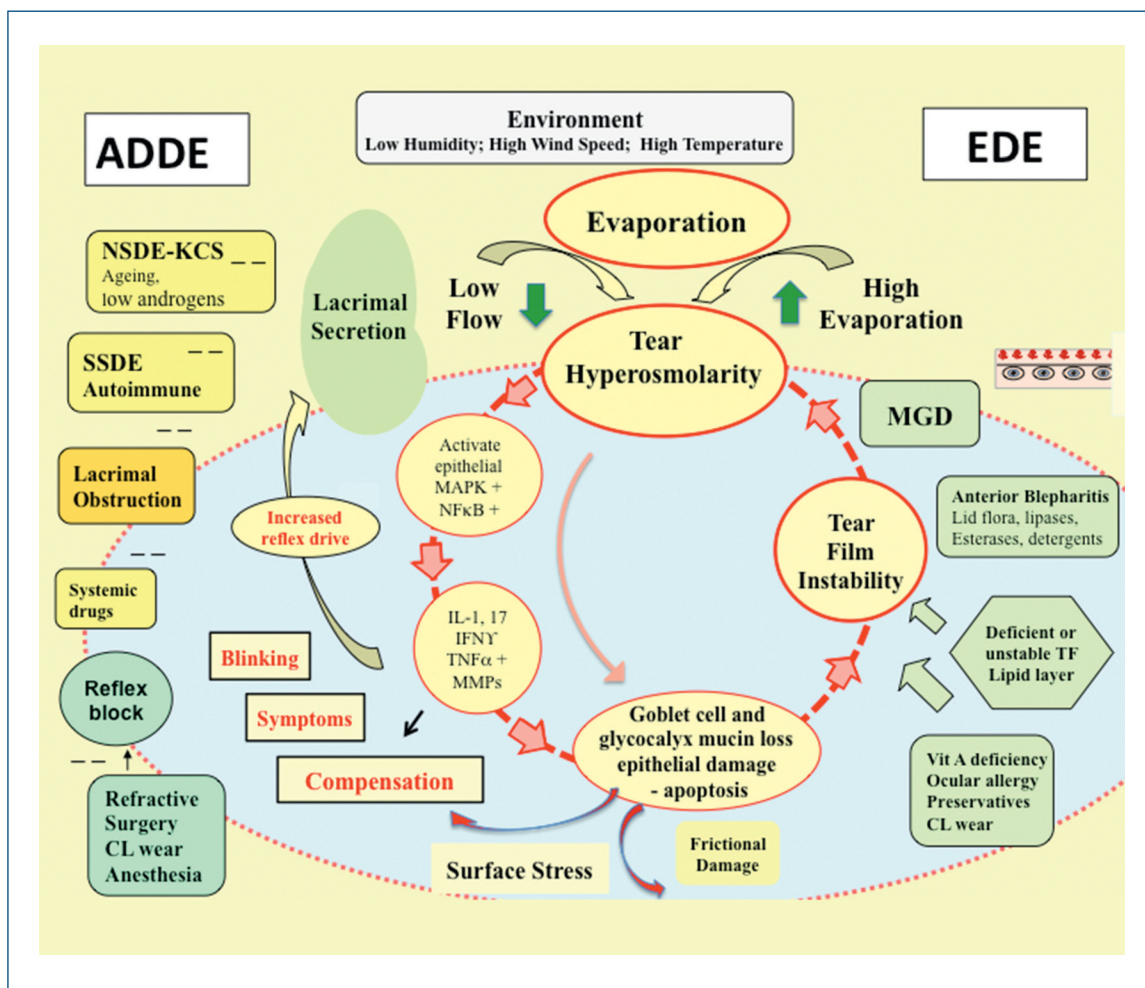


Figure 2. Pathophysiology of DED

Consistent risk factors for DED include age, sex, race, MGD, connective tissue disease, Sjögren syndrome, androgen deficiency, computer use, contact lens wear, estrogen replacement therapy, hematopoietic stem cell transplantation, certain environmental conditions (such as pollution, low humidity, and sick building syndrome) and medication use (for example, antihistamines, antidepressants, anxiolytics, and isotretinoin). Iatrogenic DED is also common and can be induced by a number of clinical interventions, including many topical and systemic drugs, and ophthalmic surgical (e.g. refractive, cataract, glaucoma, vitreoretinal) and non-surgical (e.g. botulinum toxin application, cosmetic) procedures.

How is DED diagnosed?

TFOS DEWS II recommends a sequence of tests for diagnosis of DED and assessment of its severity (Figure 3). This diagnostic process first utilizes triaging questions to exclude conditions that mimic DED. A DED diagnosis then requires a positive score on one of two specific symptom questionnaires, followed by at least one positive clinical sign indicating reduced non-invasive break-up time, elevated or a large interocular disparity in osmolarity, or ocular surface staining.

How is DED treated?

TFOS DEWS II also recommends a series of staged management and treatment options with proven efficacy, that aim to restore tear film homeostasis. These are listed in the Table below.

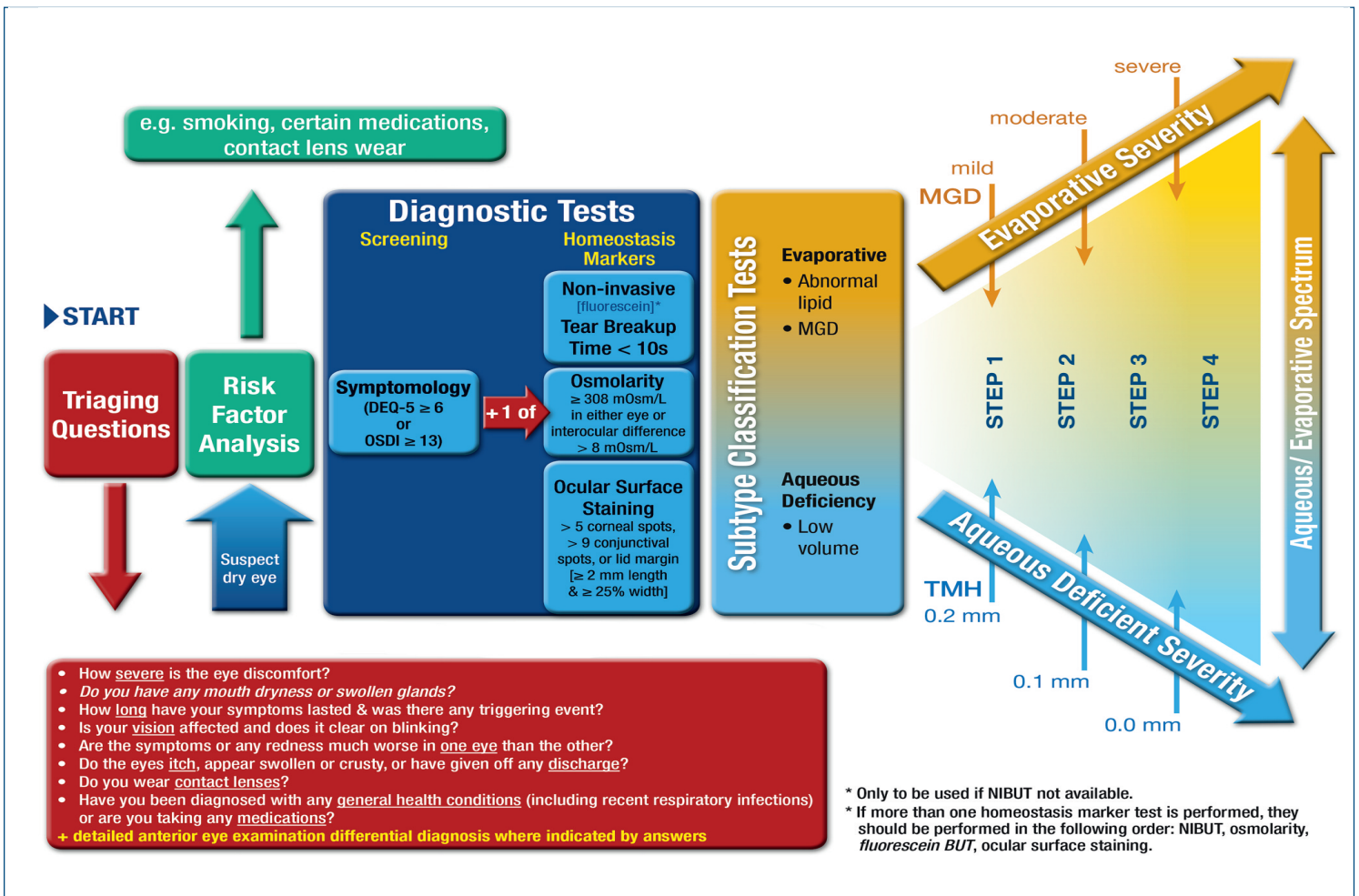


Figure 3. Diagnostic approach for DED

STEP	TFOS DEWS II recommendations for the staged management and treatment of DED
1	<ul style="list-style-type: none"> • Education regarding the condition, its management, treatment and prognosis • Modification of local environment • Education regarding potential dietary modifications (including oral essential fatty acid supplementation) • Identification and potential modification/elimination of offending systemic and topical medications • Ocular lubricants of various types (if MGD is present, then consider lipid-containing supplements) • Lid hygiene and warm compresses of various types
2	<p>If above options are inadequate consider:</p> <ul style="list-style-type: none"> • Non-preserved ocular lubricants to minimize preservative-induced toxicity • Tea tree oil treatment for Demodex (if present) • Tear conservation <ul style="list-style-type: none"> o Punctal occlusion o Moisture chamber spectacles/goggles • Overnight treatments (such as ointment or moisture chamber devices) • In-office, physical heating and expression of the meibomian glands (including device-assisted therapies) • In-office intense pulsed light therapy for MGD • Prescription drugs to manage DED <ul style="list-style-type: none"> o Topical antibiotic or antibiotic/steroid combination applied to the lid margins for anterior blepharitis o Topical corticosteroid (limited-duration) o Topical secretagogues o Topical non-glucocorticoid immunomodulatory drugs o Topical lymphocyte function-associated antigen-1 antagonist drugs o Oral macrolide or tetracycline antibiotics
3	<p>If above options are inadequate consider:</p> <ul style="list-style-type: none"> • Oral secretagogues • Autologous/allogeneic serum eye drops • Therapeutic contact lens options <ul style="list-style-type: none"> o Soft bandage lenses o Rigid scleral lenses
4	<p>If above options are inadequate consider:</p> <ul style="list-style-type: none"> • Topical corticosteroid for longer duration • Amniotic membrane grafts • Surgical punctal occlusion • Other surgical approaches (eg tarsorrhaphy, salivary gland transplantation)

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