Dry Eye Syndrome

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*A/E = androgen/estrogen ratio

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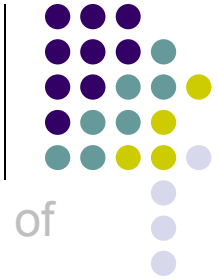

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--clearing debris from the corneal surface; and
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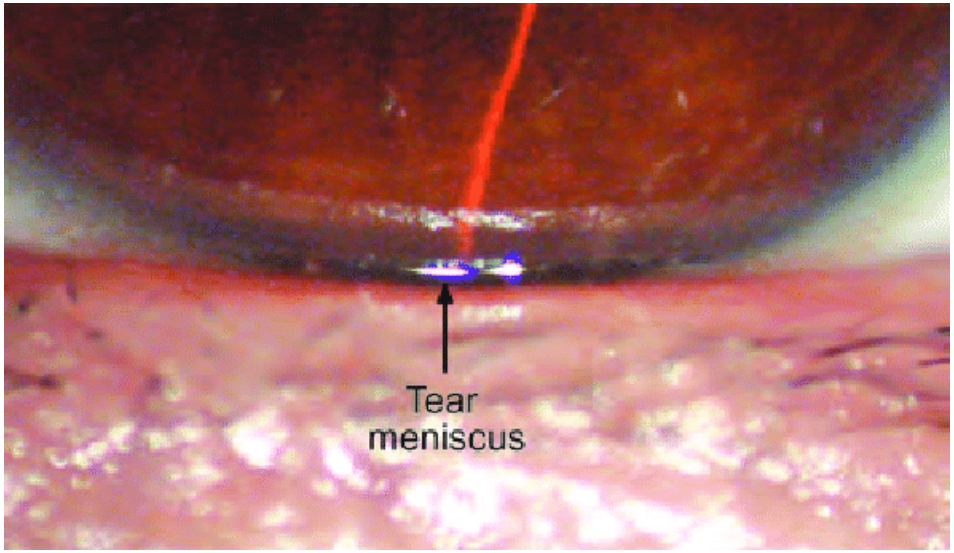

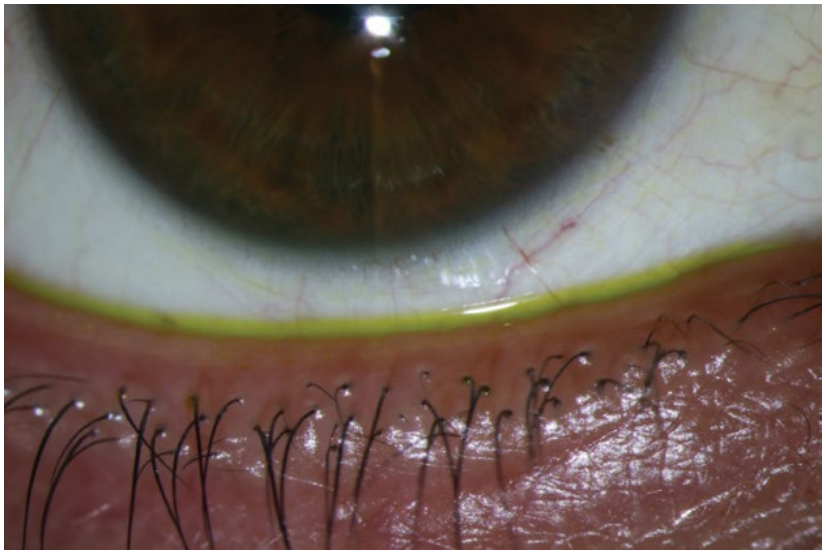

Tear lake (aka tear strip; tear meniscus)

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Dry Eye Syndrome

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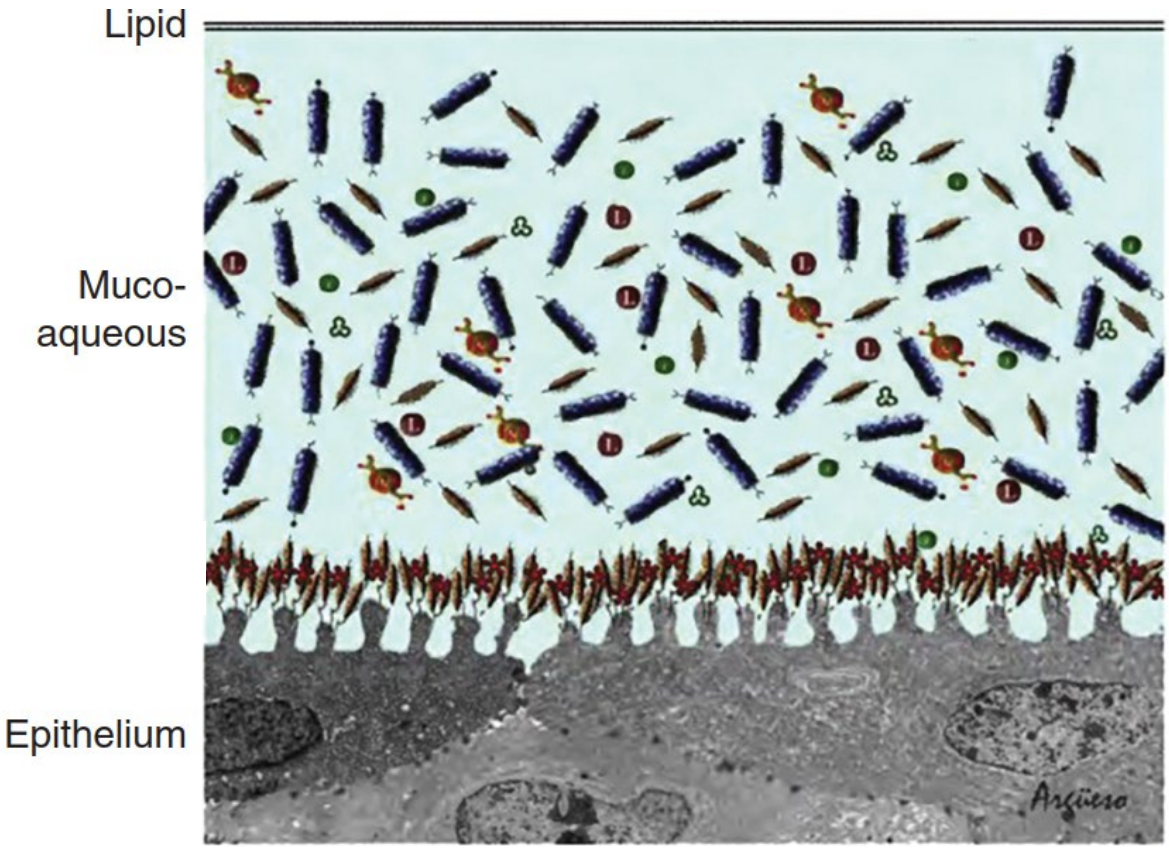

Two-phase model of the tear film. Schematic drawing of the structure of the tear film showing the outer lipid layer and the mucoaqueous layer.

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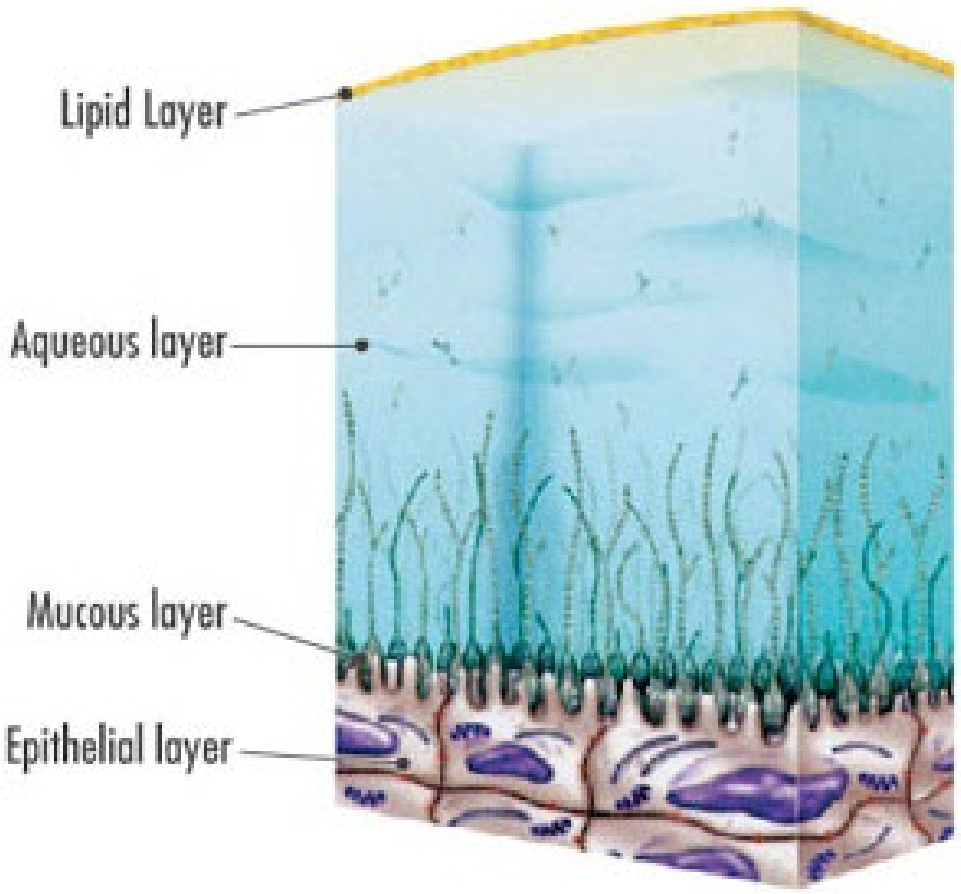
The older *tripartite model* of the tear film posited that the three components formed distinct mucus (inner), aqueous (middle) and lipid (outer) layers, but the consensus now is this model is incorrect, and it has largely been supplanted by the two-phase model.

Dry Eye Syndrome



The old/obsolete *tripartite model* of the tear film

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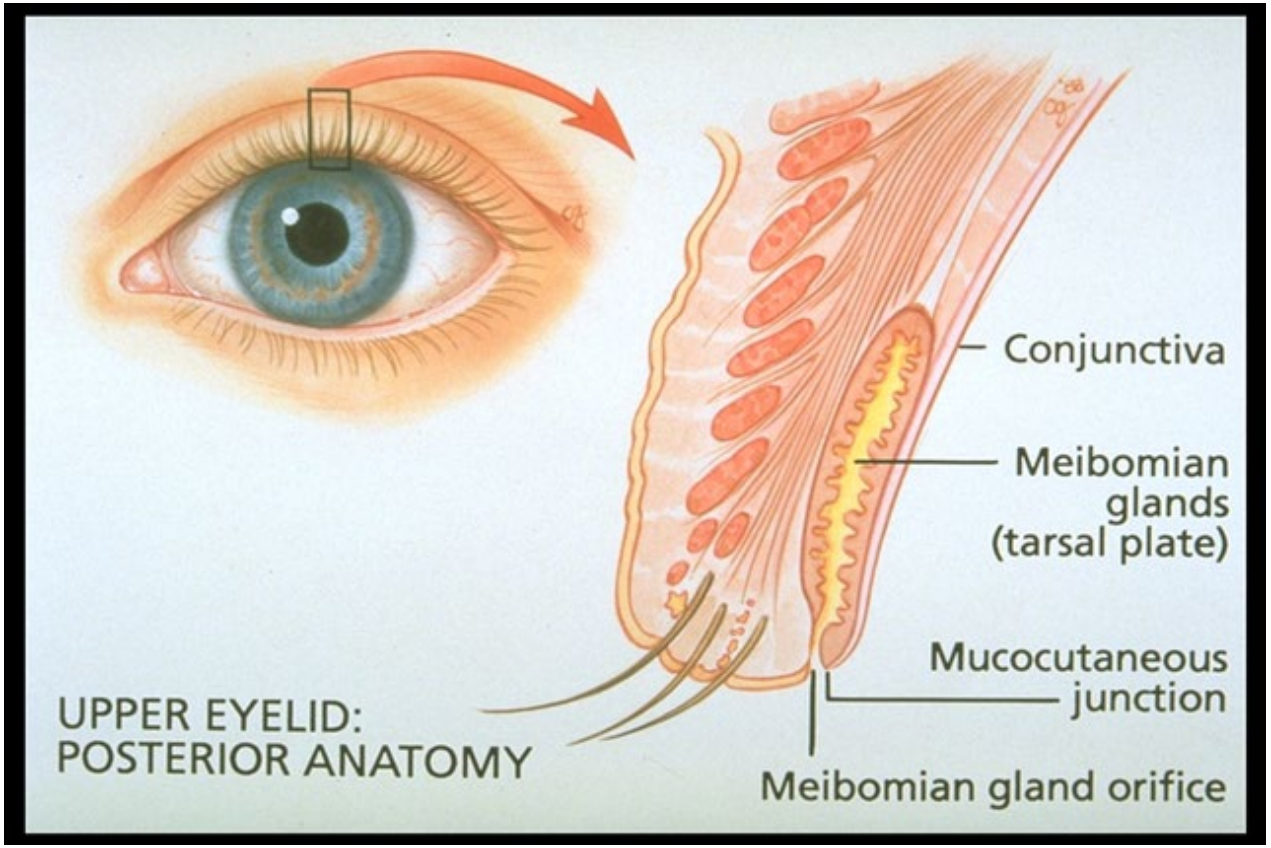
The specific lipid is **meibum**, a product of the *meibomian glands*. These glands are embedded within the tarsal plates of both the upper and lower lids. There are ~2x as many glands in the upper lids. They are innervated by the parasympathetic system.

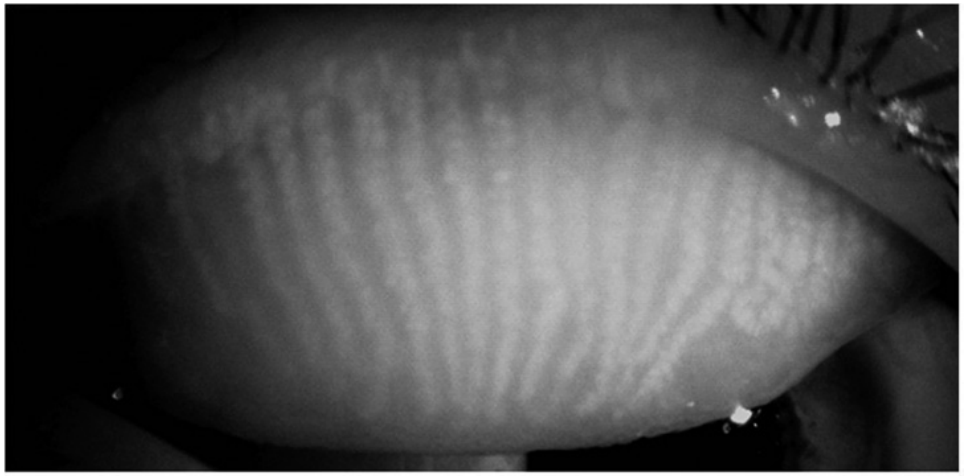
Dry Eye Syndrome



Meibomian glands

Dry Eye Syndrome

Upper lid



Lower lid

Meibomian glands

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The lipid layer makes several key contributions to the stability and effectiveness of the tear film. It inhibits tear film evaporation and reduce its surface tension, both of which serve to maintain the tear film on the eye. (Without a lipid layer, surface tension would pull the tear film down off the surface.) The lipid layer also facilitates visual acuity by producing a glassy-smooth surface at the air-tear film interface.

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Aqueous is produced by the *lacrimal glands*, which includes the *main lacrimal gland* (found in the superotemporal orbit) and the accessory lacrimal glands of *Krauss* and *Wolfring* (found scattered throughout the forniceal and palpebral conj). All of the lacrimal glands are innervated by parasympathetics as well.

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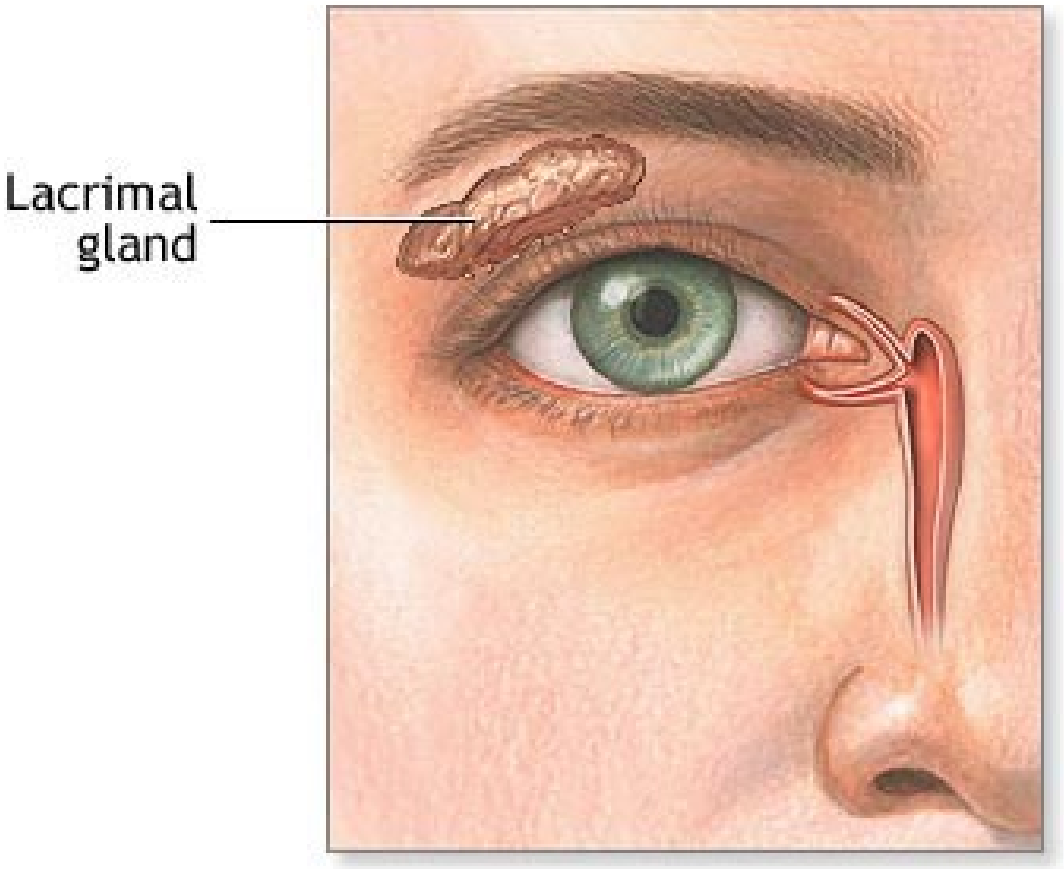
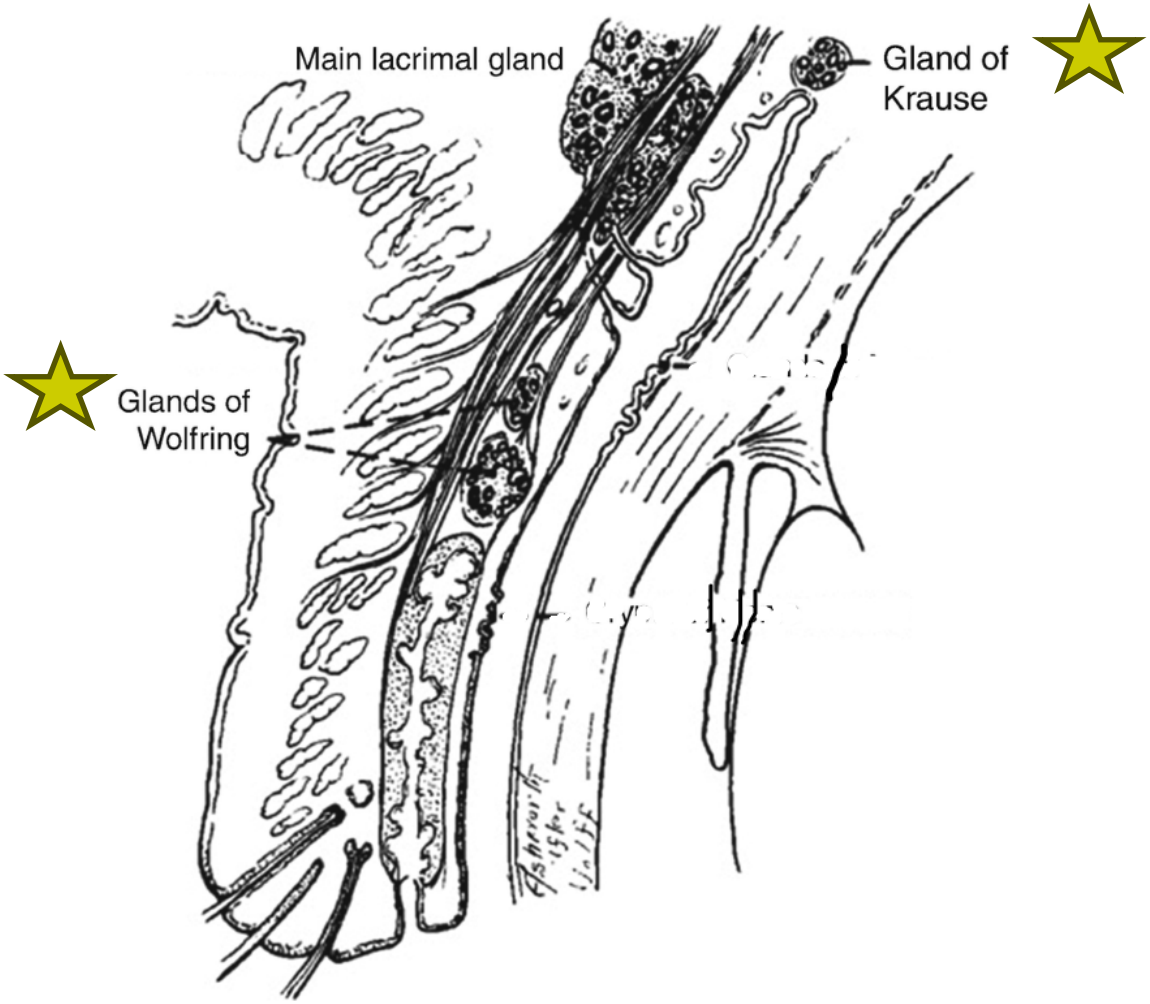
Dry Eye Syndrome



The main lacrimal gland

Dry Eye Syndrome



The lacrimal glands of Krause and Wolfring

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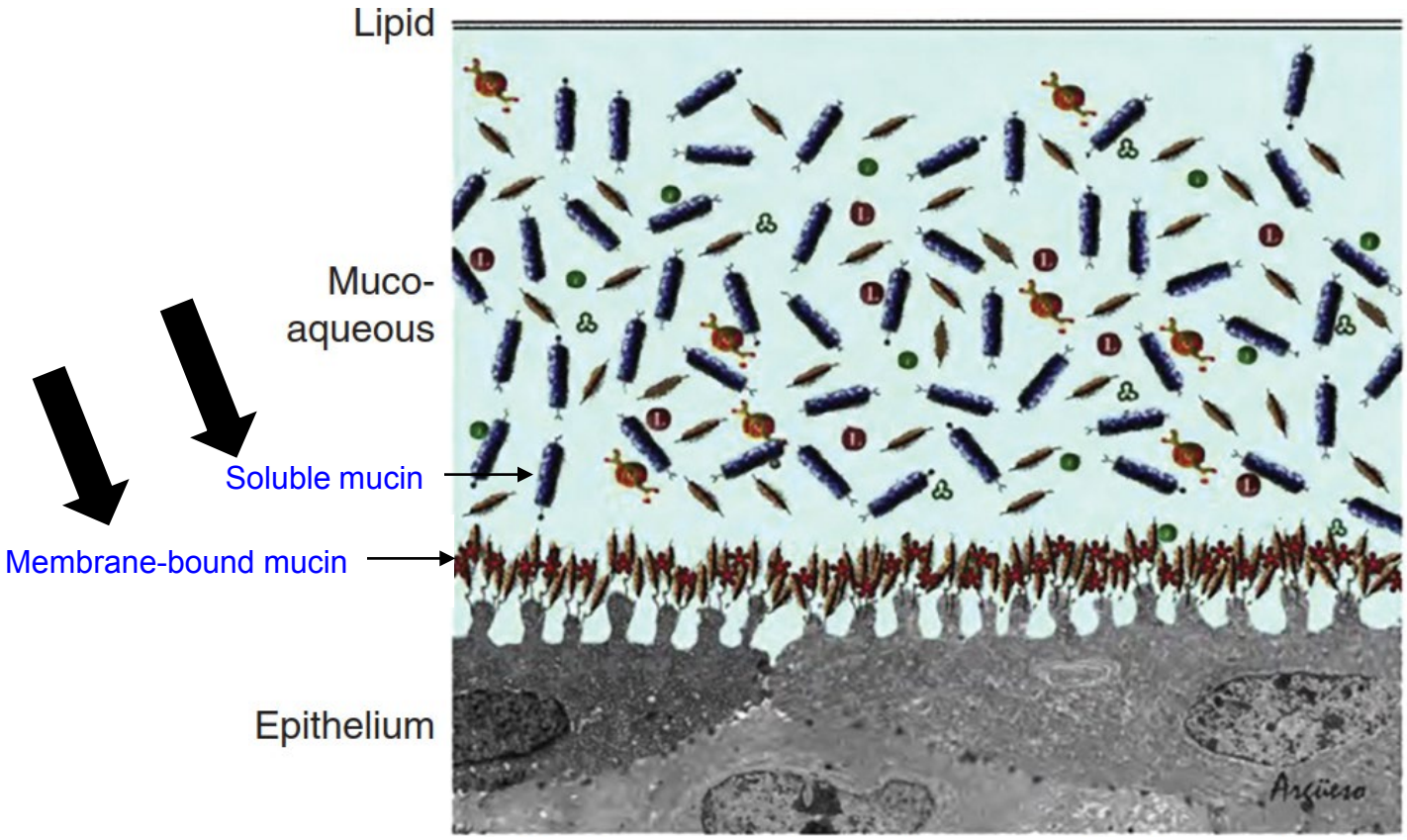
Dry Eye Syndrome



We saw this depiction of the *two-phase model of the tear film* earlier in the set. But are now ready to note the presence and location of mucin. Note that there are 'membrane-bound' mucins in the glycocalyx of the corneal epithelium.

Dry Eye Syndrome

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The primary role of electrolytes in the tear film is to regulate **tear-film osmolarity**. Recall that osmolarity refers to the concentration of solutes in a fluid—literally, the number of solute-particles in a given amount of fluid. Normal tear osmolarity is 296 ± 10 milliosmoles per liter (mOsm/L).

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But why is tear-film osmolarity important? For one reason*: The osmotic-pressure gradient it can exert on the underlying ocular-surface epi cells*. The membranes of these cells are freely permeable to water but not solutes (ie, they are *semi-permeable*). Recall the rule regarding semi-permeable membranes: **Solvent follows solute**.

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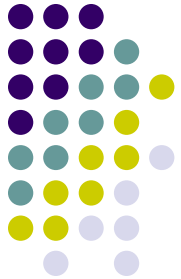
Dry Eye Syndrome



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Dry Eye Syndrome

Recall that a reflex arc has three components: A *sensory limb* consisting of sensory receptors and afferent nerves, a *motor limb* consisting of efferent nerves and the effector end-organ, and a *CNS integration center* that connects the afferent and efferent limbs.

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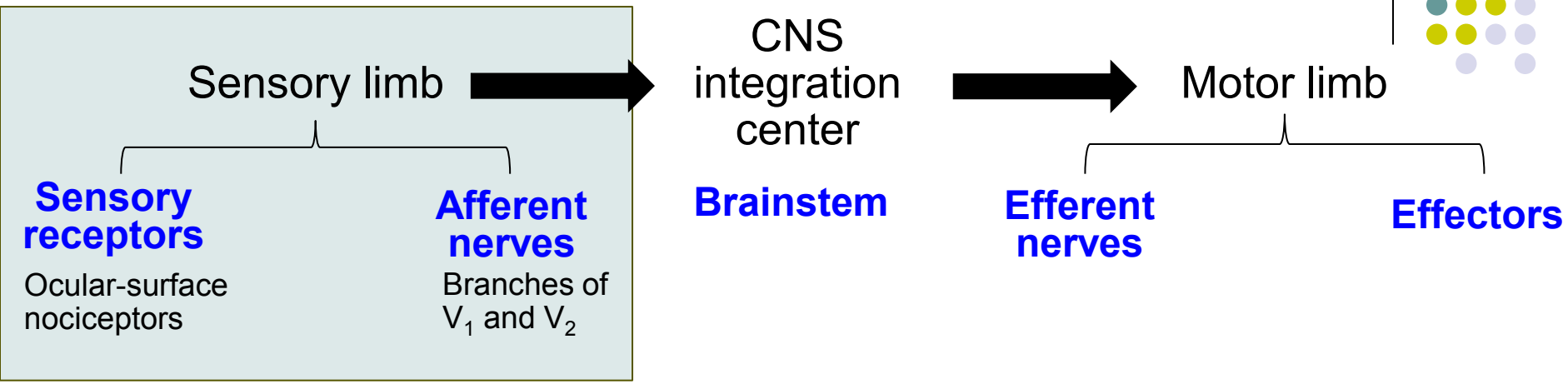
Dry Eye Syndrome

The LFU



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Dry Eye Syndrome

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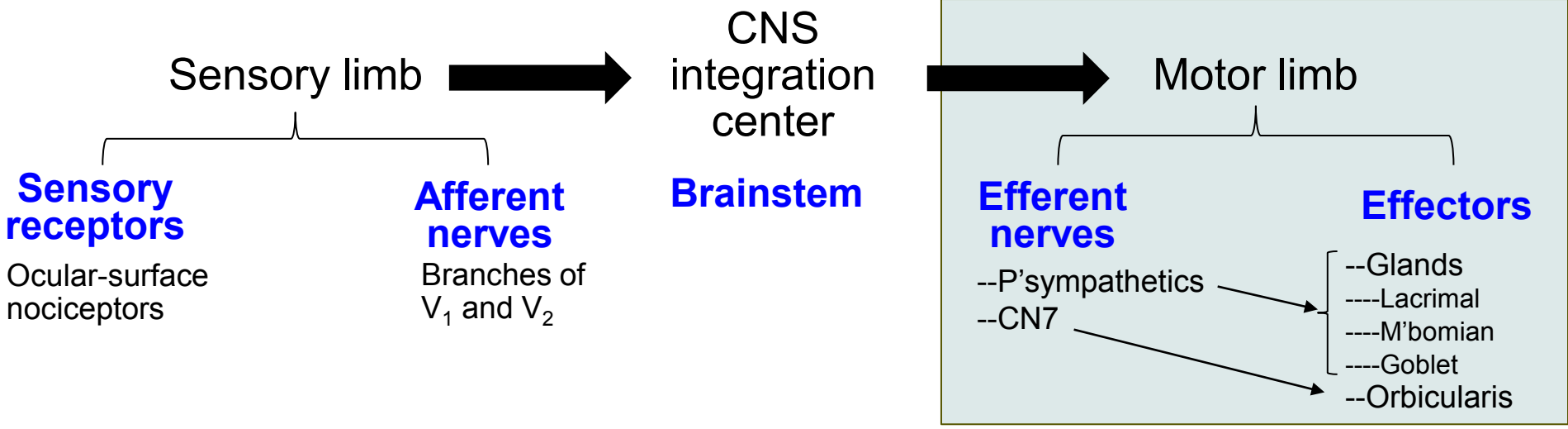

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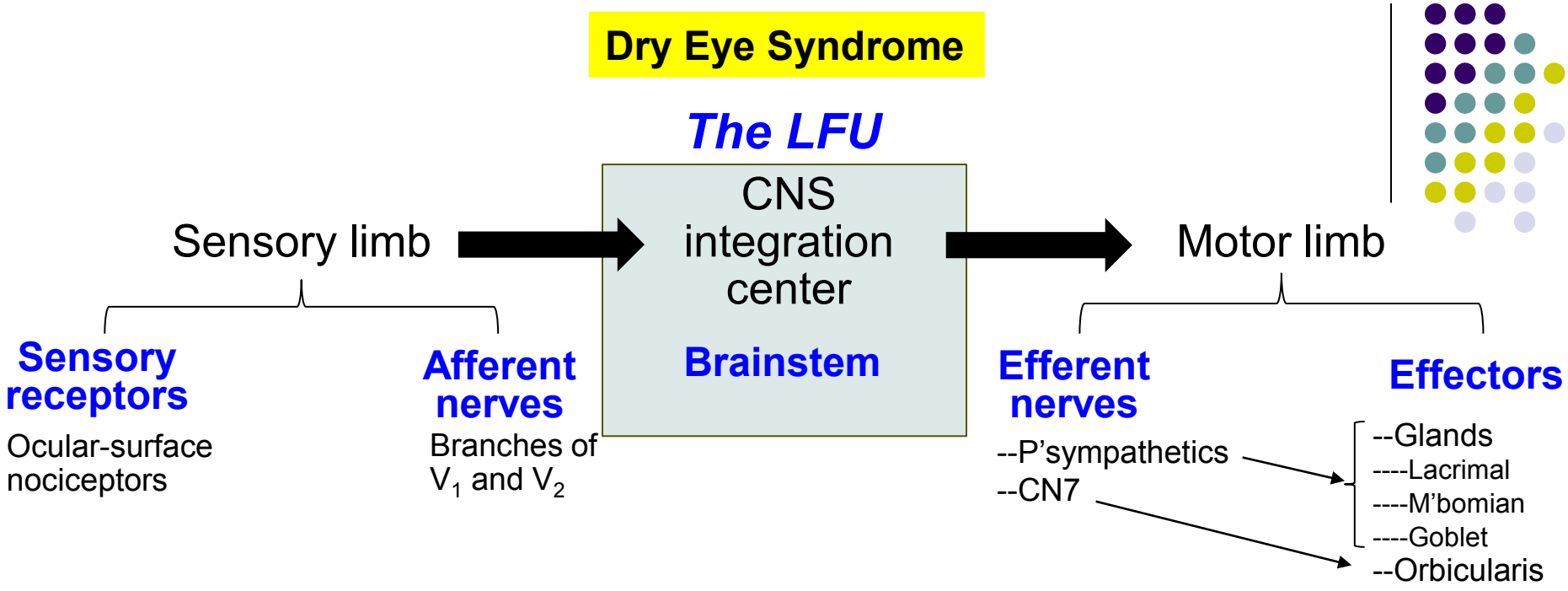
Dry Eye Syndrome



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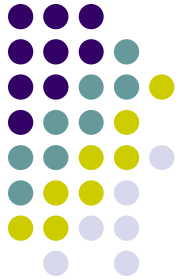
Dry Eye Syndrome



We are ready (finally!) to tackle the pathophysiology of DES…

Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**

We are ready (finally!) to tackle the pathophysiology of DES…
Which commences with tear hyperosmolarity

Tear hyperosmolarity

Dry Eye Syndrome

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There are two basic ways in which the status of the aqueous component of the tear film could lead to tear hyperosmolarity:

Tear
hyperosmolarity

Dry Eye Syndrome

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or…

Tear hyperosmolarity

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Tear hyperosmolarity

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This state is known as…

Aqueous Tear Deficiency

→ Tear hyperosmolarity

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Evaporative Dry Eye

Tear hyperosmolarity

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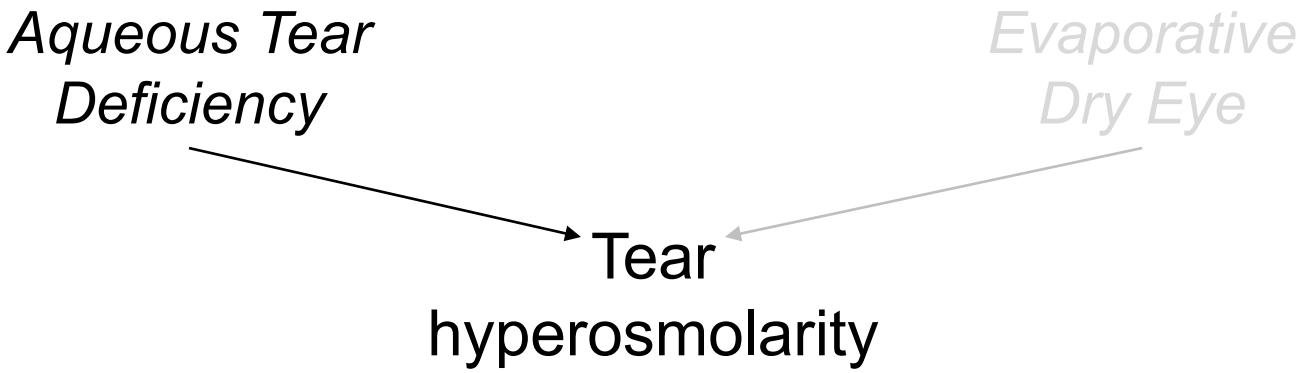
While it's a bit of an oversimplification, we can associate the components of the tear film with the pathologic states underlying DES:



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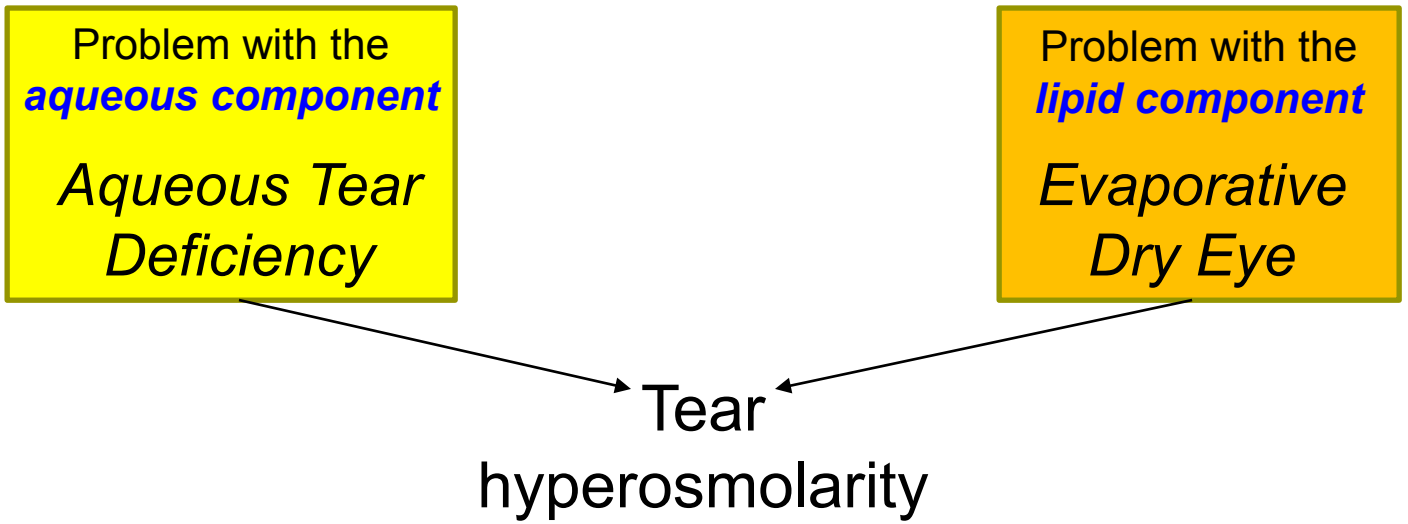
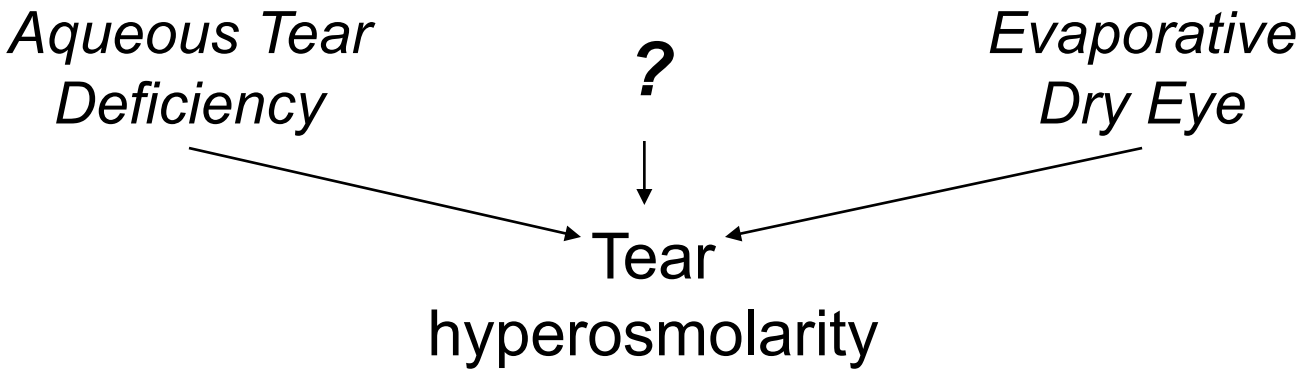


Dry Eye Syndrome

The pathophysiology for DES damage starts with damage/dysfunction of the tear film in the form of

Head's up: Shortly we're gonna add a *third* mechanism leading to tear hyperosmolarity

There ~~are~~ three two basic ways in which the status of the aqueous component of the tear film could lead to tear hyperosmolarity:



Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**

Let's drill down on both, starting with ATD.

Problem with the ***aqueous component***

Aqueous Tear Deficiency

Problem with the ***lipid component***

Evaporative Dry Eye

Tear hyperosmolarity

Dry Eye Syndrome

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But first—

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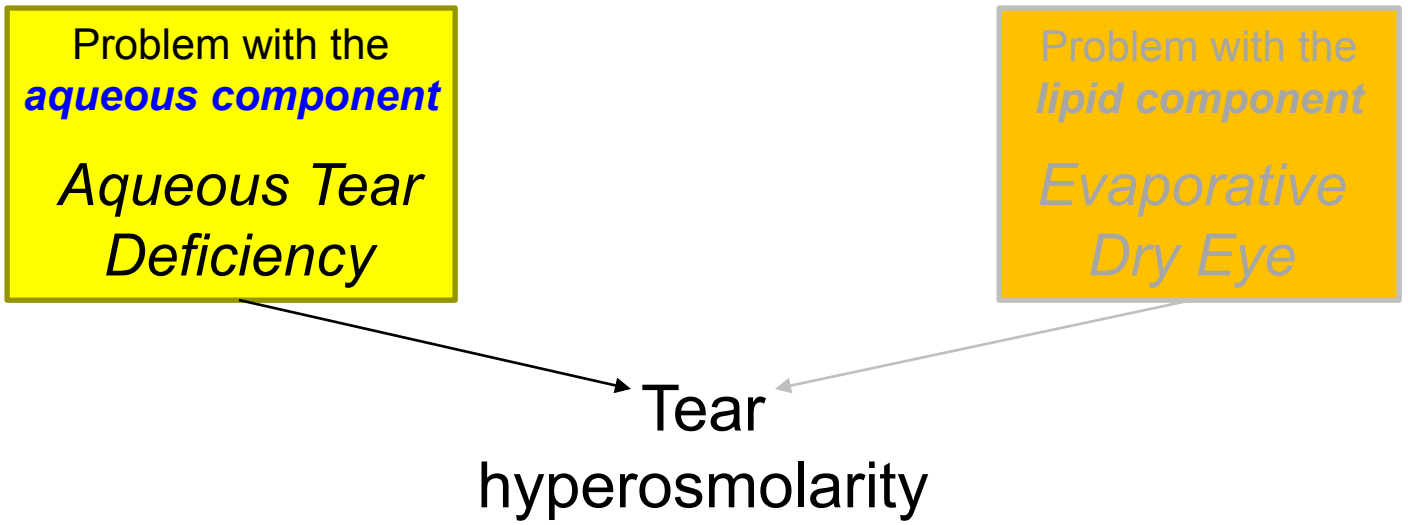

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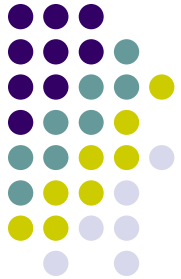
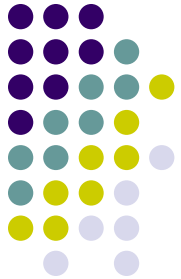


But first—there are three classic tests of aqueous tear production:

Test name			
Basal secretion test			
Schirmer I			
Schirmer II			

Dry Eye Syndrome



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What each assesses:

Test name	Assesses…		
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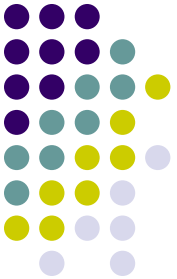
Dry Eye Syndrome



But first—there are three classic tests of aqueous tear production:

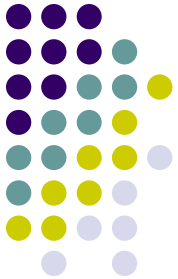
What each assesses: How each is performed:

Test name	Assesses…	Protocol	
Basal secretion test	Basal secretion (duh)	Instill anesthetic, blot, place strip, measure saturation at 5 min	
Schirmer I	Basal *and* reflex secretion	Same, but **without** instilling anesthetic	
Schirmer II	Reflex secretion only	Instill anesthetic, blot, place strip, irritate nasal mucosa w/ a cotton-tip	

Dry Eye Syndrome

Aqueous tear production testing

Dry Eye Syndrome

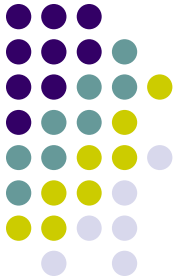


But first—there are three classic tests of aqueous tear production:

What each assesses: How each is performed: How each is interpreted:

Test name	Assesses…	Protocol	Interpretation
Basal secretion test	Basal secretion (duh)	Instill anesthetic, blot, place strip, measure saturation at 5 min	Less than 3 mm wetting after 5 min = ATD
Schirmer I	Basal *and* reflex secretion	Same, but **without** instilling anesthetic	Less than 5 mm wetting after 5 min = ATD
Schirmer II	Reflex secretion only	Instill anesthetic, blot, place strip, irritate nasal mucosa w/ a cotton-tip	Less than 15 mm wetting after 2 min = reflex secretion defect

Dry Eye Syndrome



Sjögren's **Non-Sjögren's**

ATD is subdivided into two categories: *Sjögren's* and *non-Sjögren's*

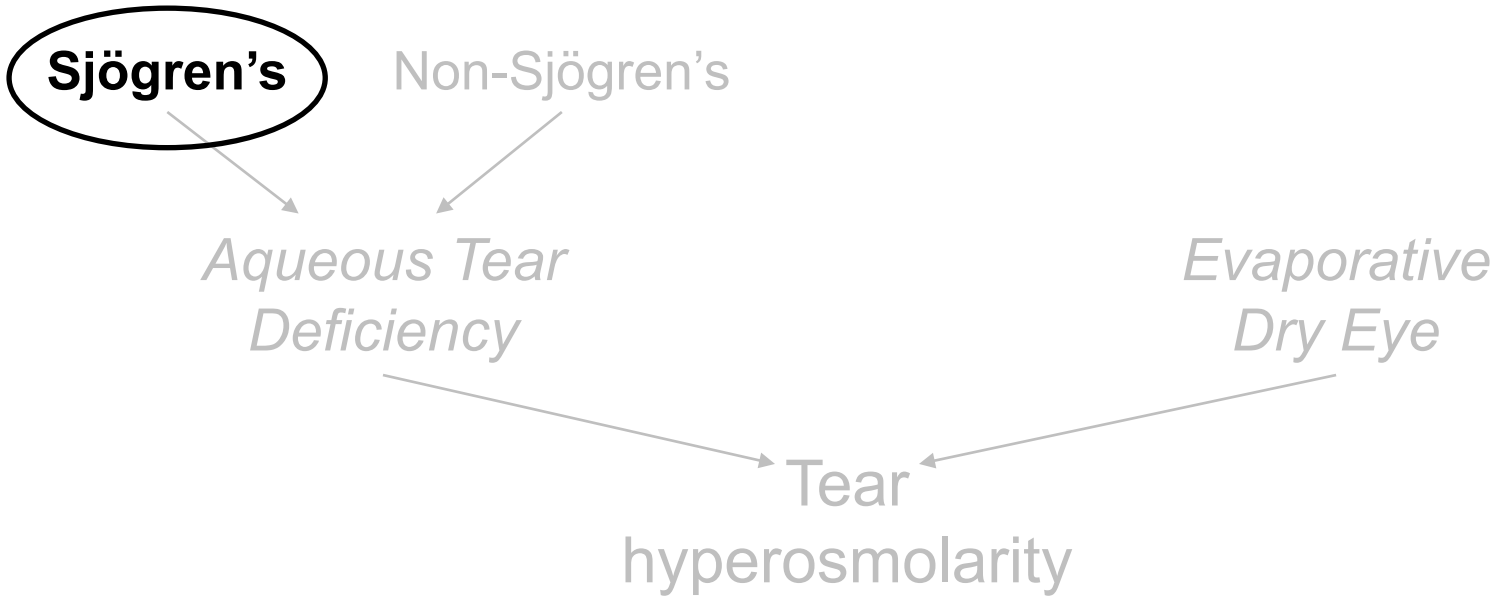
Aqueous Tear Deficiency

Evaporative Dry Eye

Tear hyperosmolarity

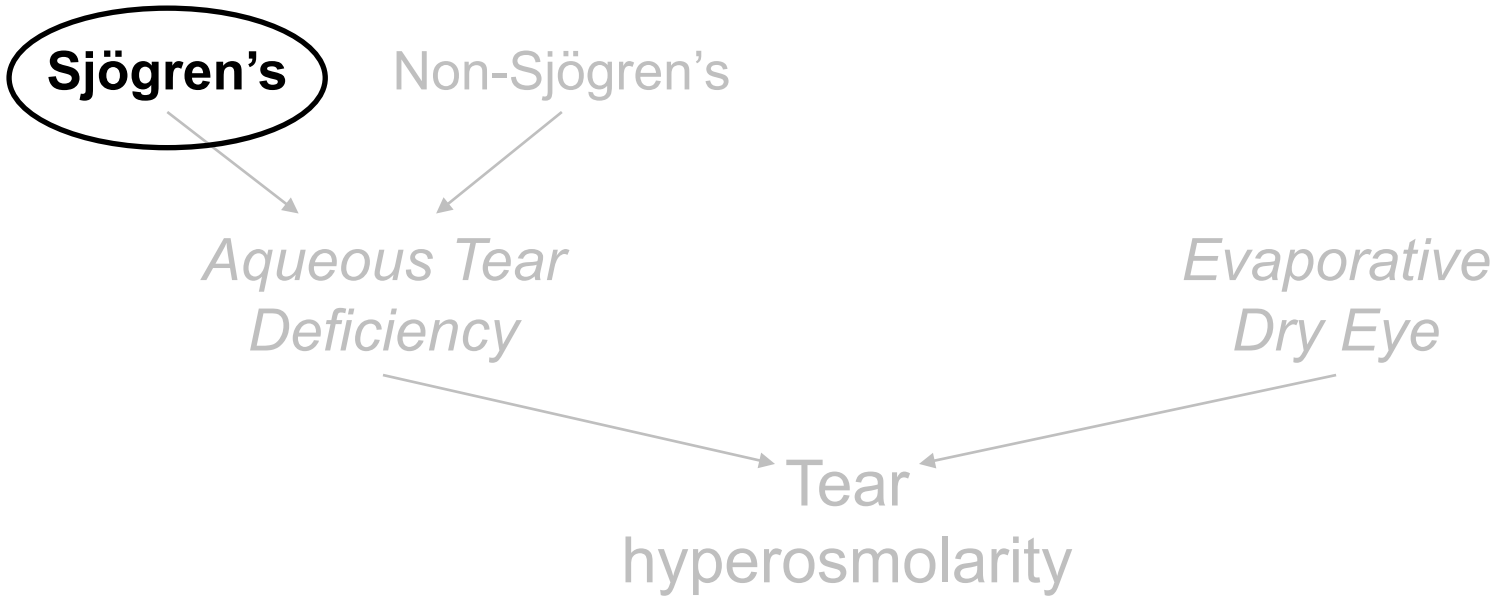
Dry Eye Syndrome

Sjögren's syndrome (SS) is a chronic autoimmune disorder characterized by lymphocytic infiltration of exocrine glands. The vast majority of pts are female. It is divided into *primary* and *secondary* forms, the key distinction being that secondary SS is associated with a systemic connective-tissue disease (eg, RA, SLE, scleroderma).



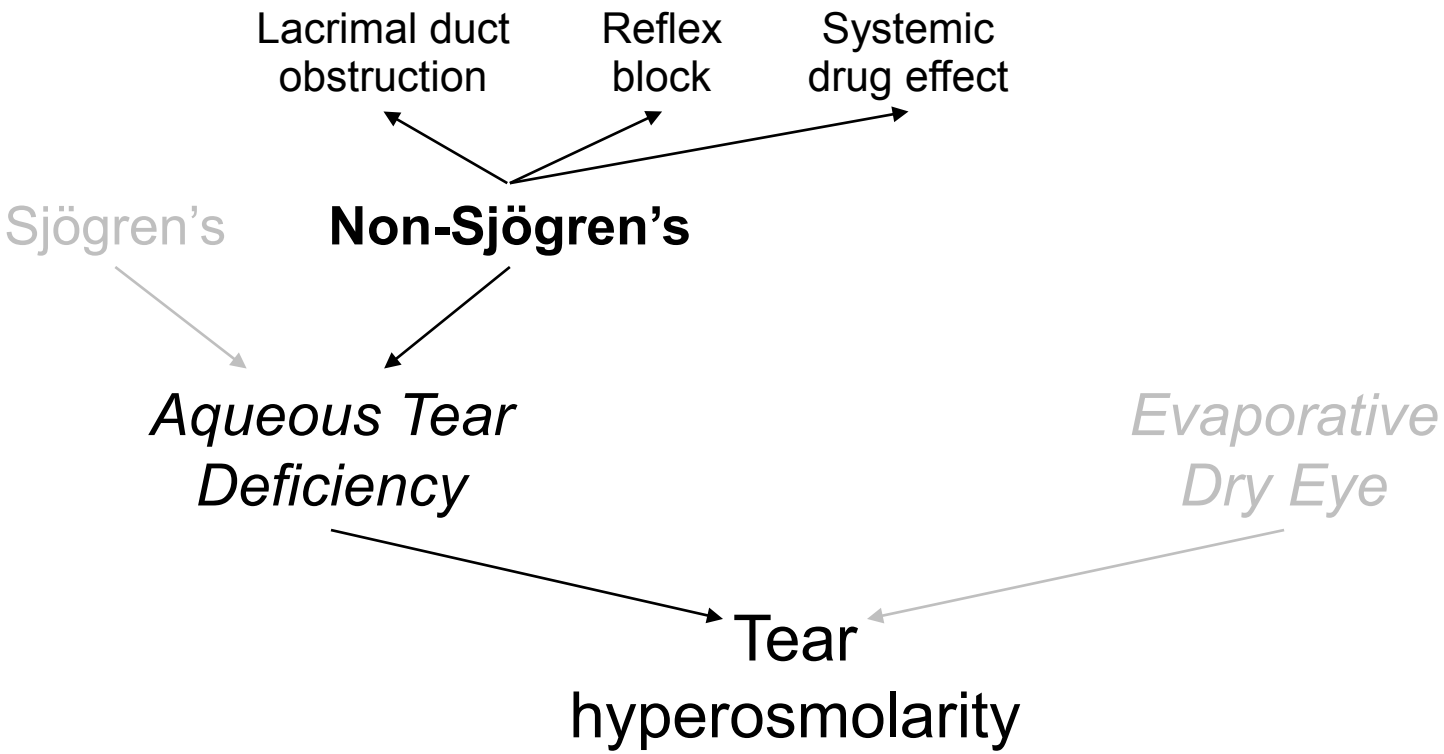
Dry Eye Syndrome

Sjögren's syndrome (SS) is a chronic autoimmune disorder characterized by lymphocytic infiltration of exocrine glands. The vast majority of pts are female. It is divided into *primary* and *secondary* forms, the key distinction being that secondary SS is associated with a systemic connective-tissue disease (eg, RA, SLE, scleroderma). In both forms, aqueous hyposecretion (and thus ATD) results from autoimmune-mediated lymphocytic infiltration of the lacrimal glands.



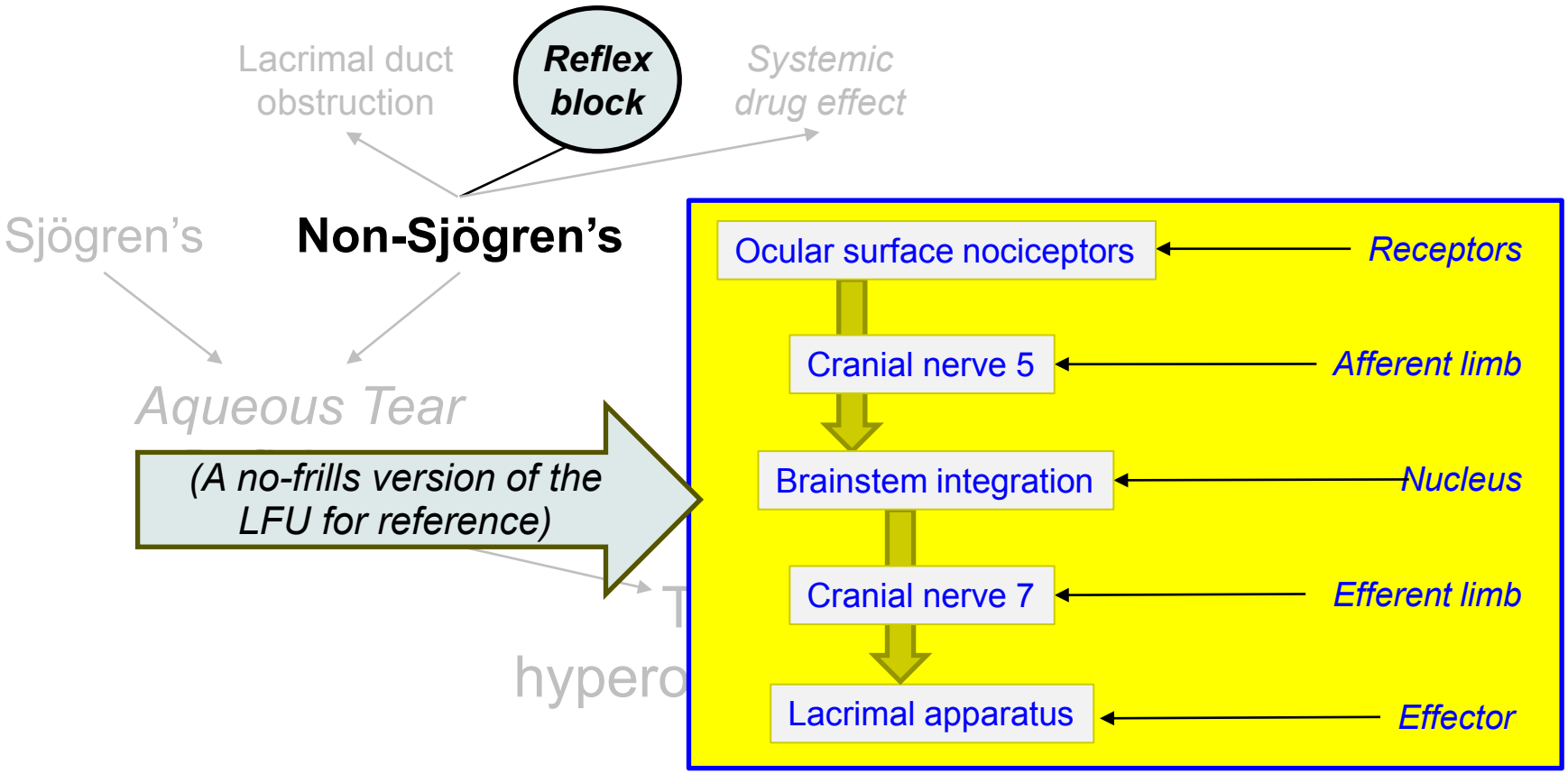
Dry Eye Syndrome

In **non**-Sjögren's ATD, other causes of lacrimal gland hyposecretion are at work:



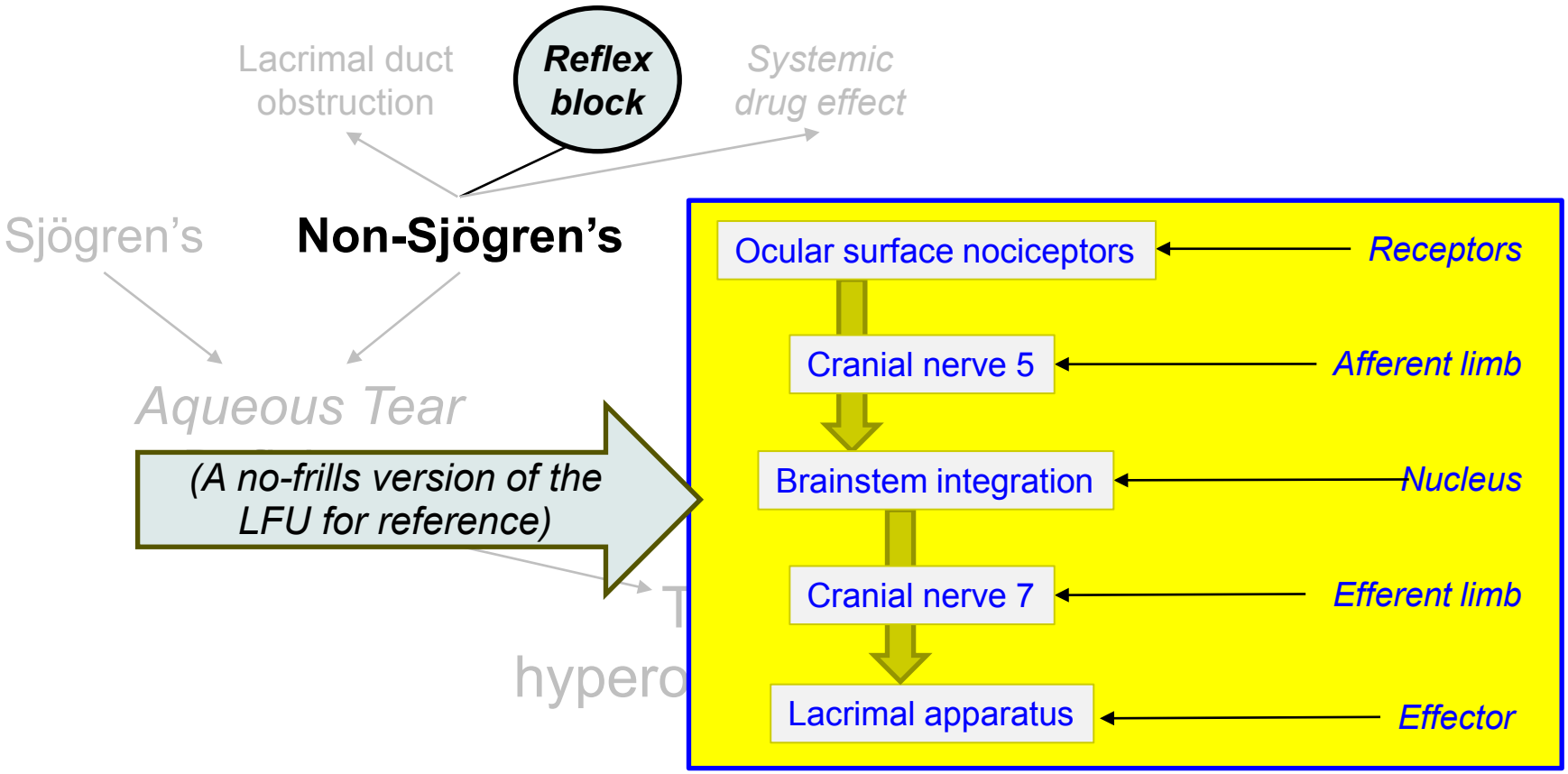
Dry Eye Syndrome

Reflex block refers to anything that disrupts the normal functioning of the LFU ‘reflex circuit.’



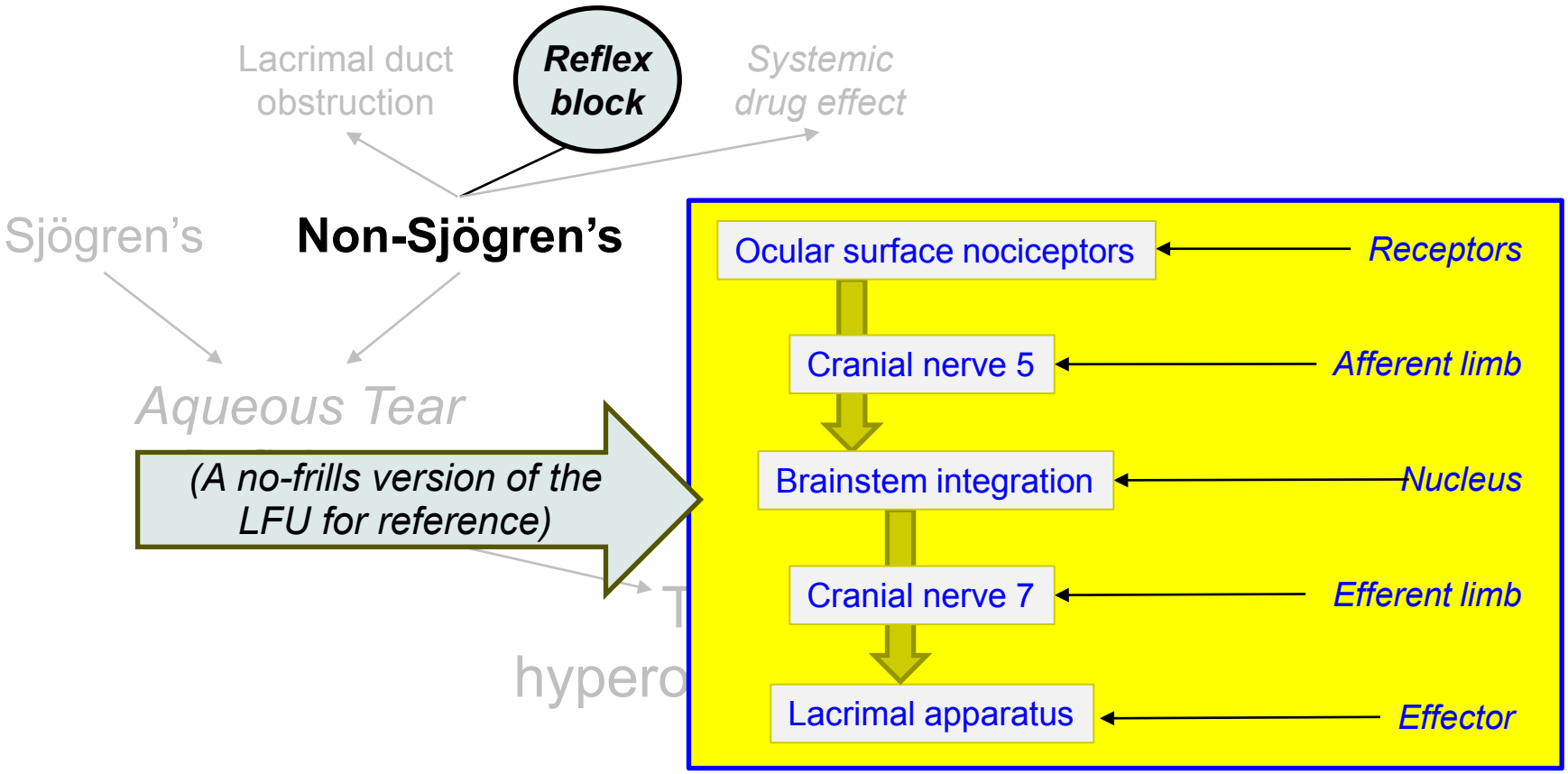
Dry Eye Syndrome

Reflex block refers to anything that disrupts the normal functioning of the LFU 'reflex circuit.' *Afferent* limb block is often due to corneal hypoesthesia from corneal surgery, post-herpetic neuropathy, and contact-lens wear.



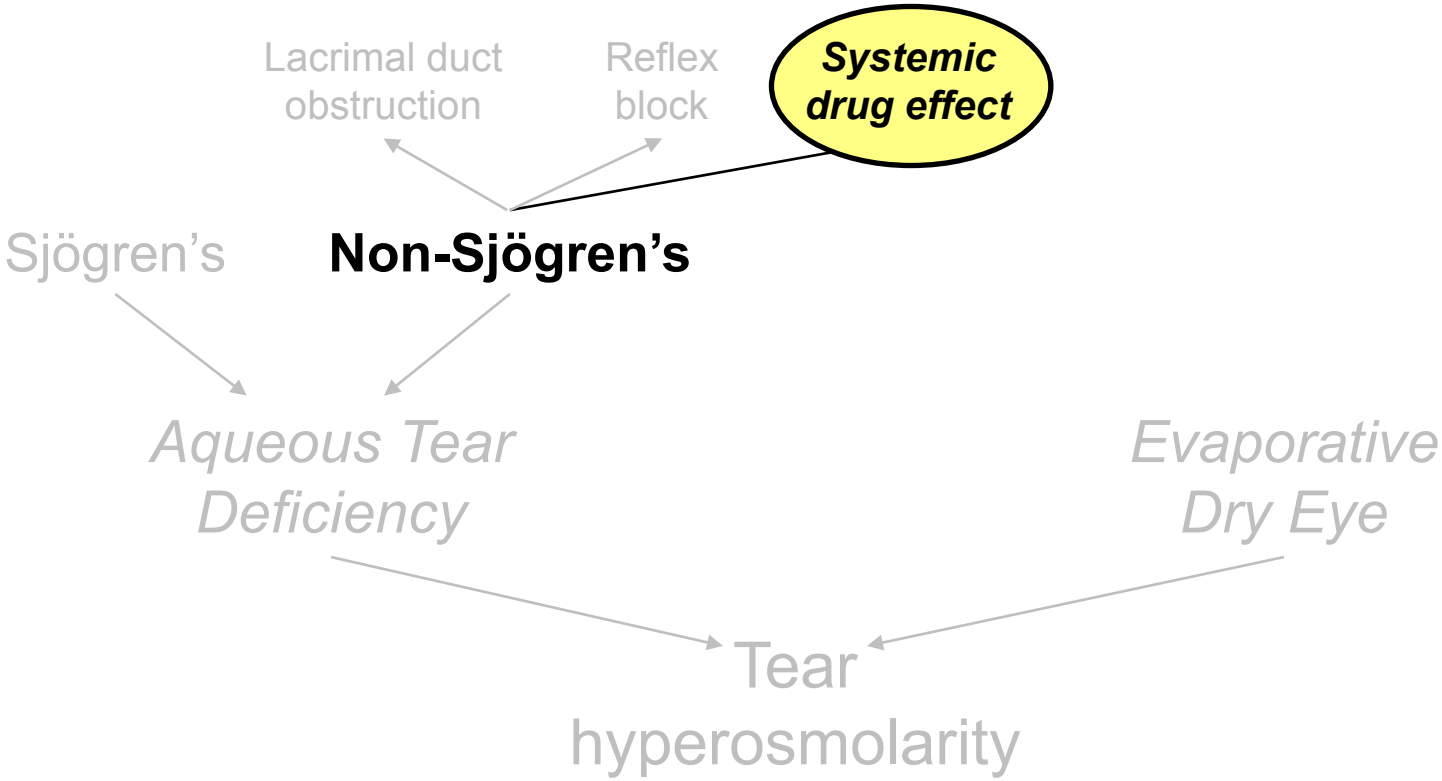
Dry Eye Syndrome

Reflex block refers to anything that disrupts the normal functioning of the LFU 'reflex circuit.' *Afferent* limb block is often due to corneal hypoesthesia from corneal surgery, post-herpetic neuropathy, and contact-lens wear. *Efferent* limb block is usually due to compromised CN7 function, eg, Bell's palsy.



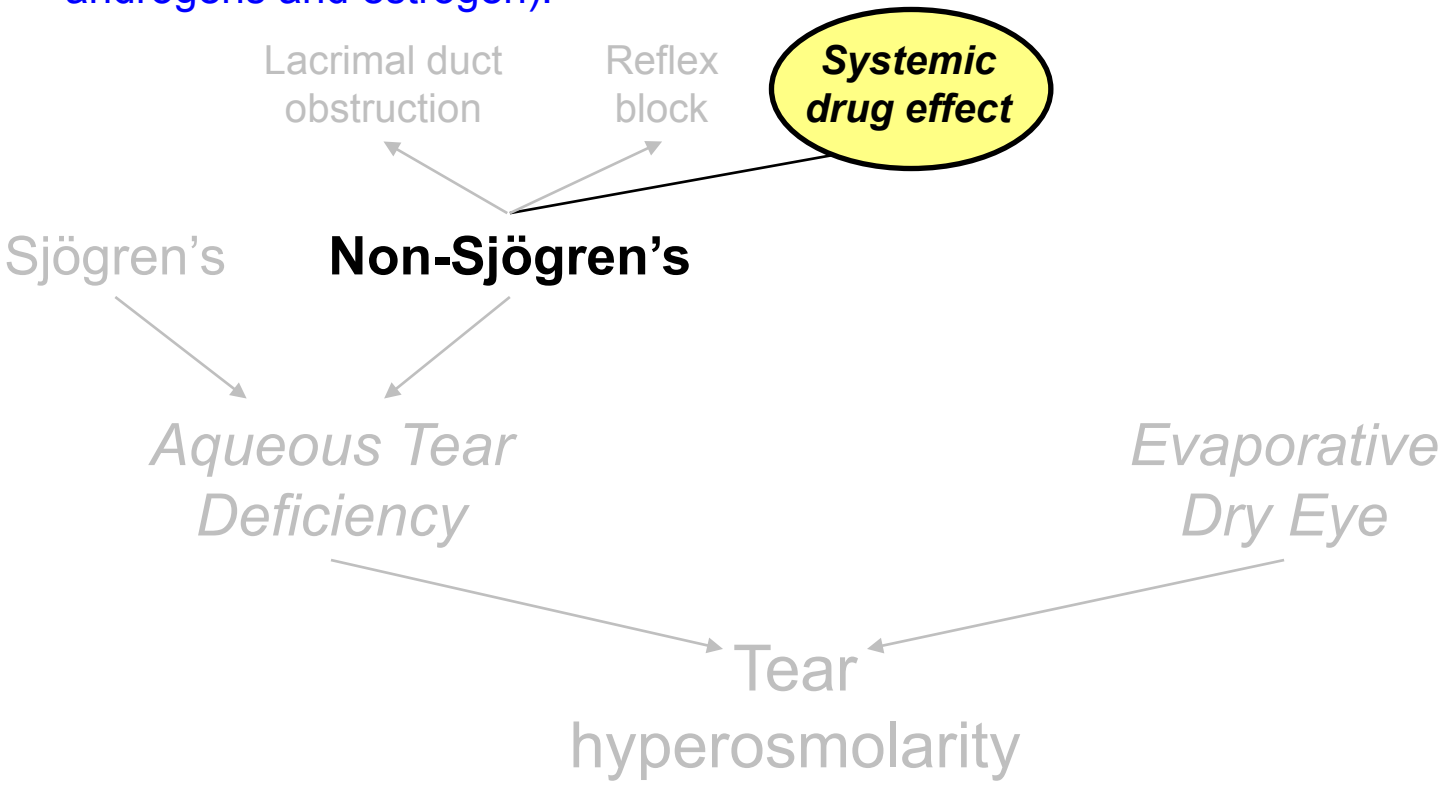
Dry Eye Syndrome

A substantial number of systemic drugs are implicated in inducing DES.
(Eg, 22 of the 100 best-selling drugs in the US list ‘dry eye’ as a side effect!)

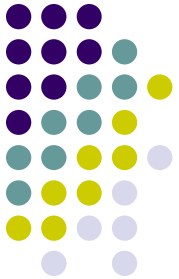


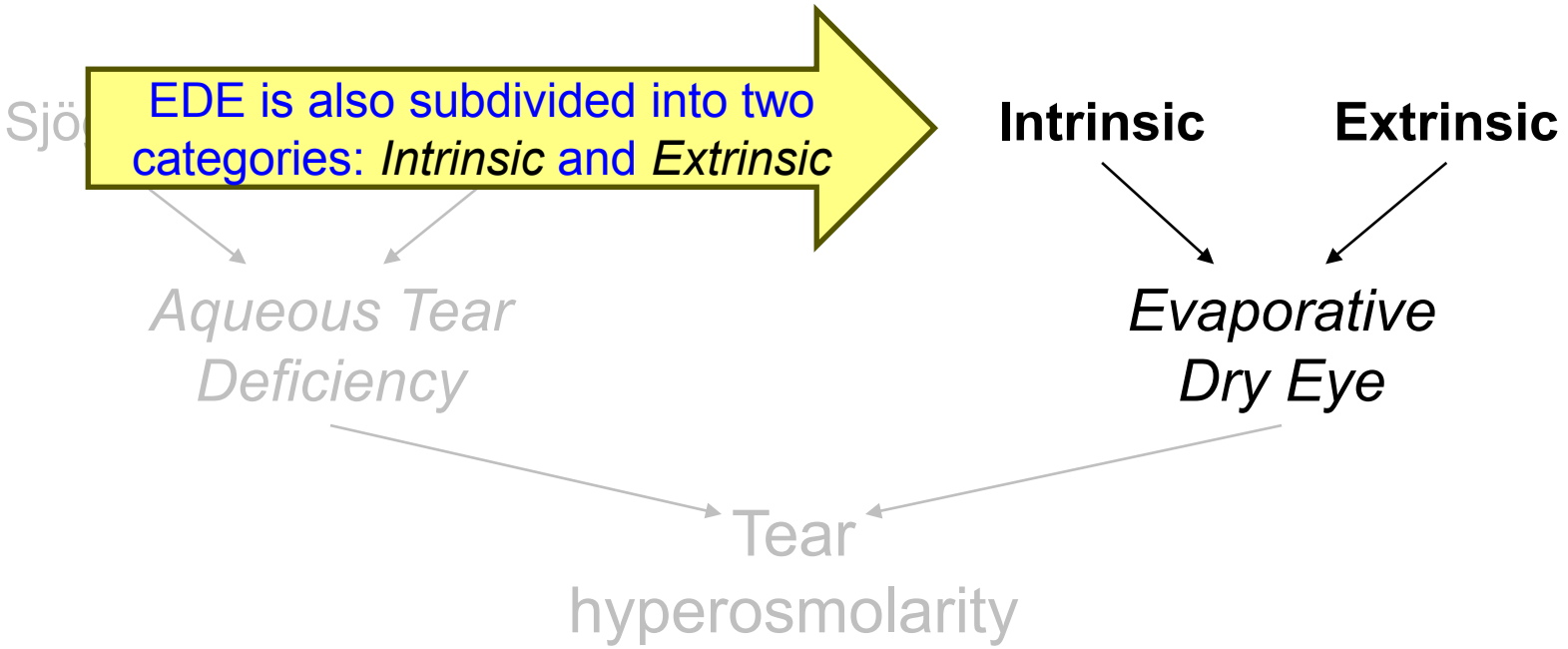
Dry Eye Syndrome

A substantial number of systemic drugs are implicated in inducing DES. (Eg, 22 of the 100 best-selling drugs in the US list 'dry eye' as a side effect!) These include anti-histamines, anti-cholinergics (eg, antidepressants), anti-hypertensives, Parkinson's meds, and OCPs (because of their effect on androgens and estrogen).



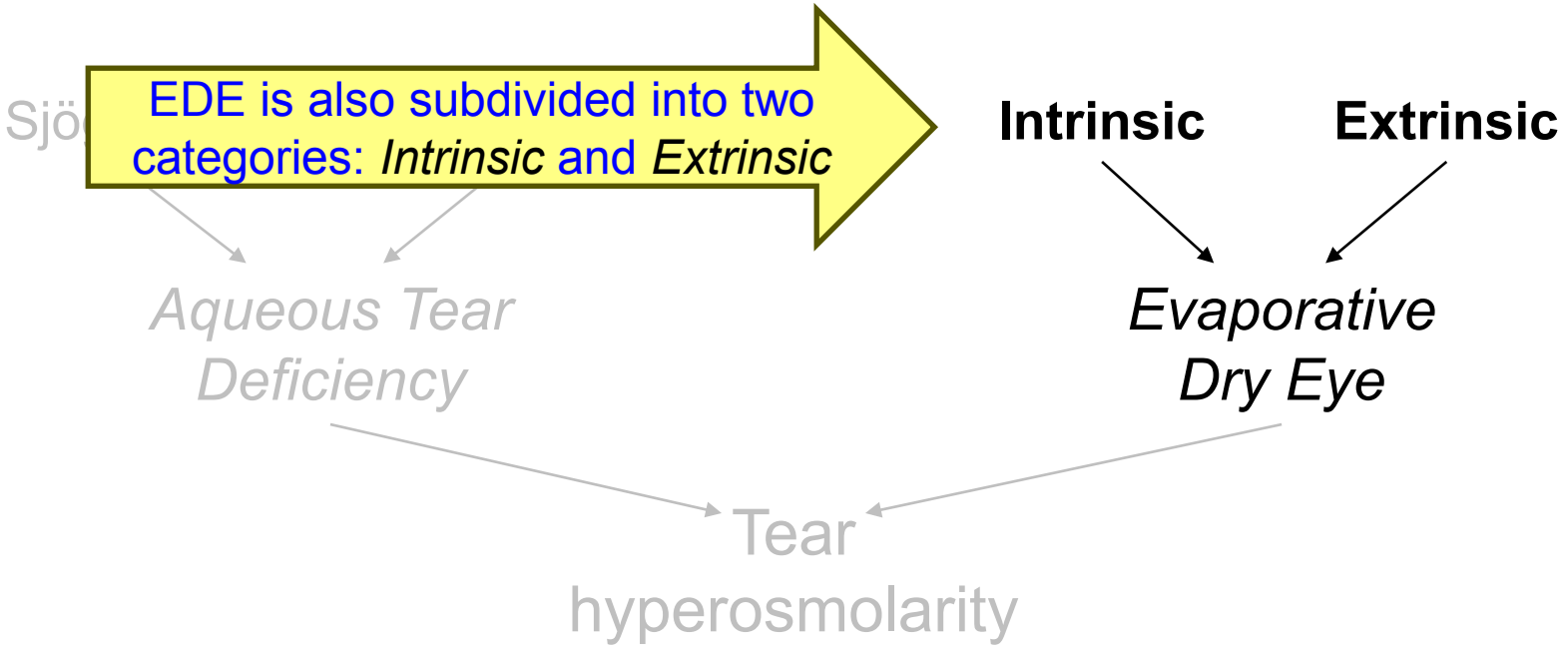
Dry Eye Syndrome





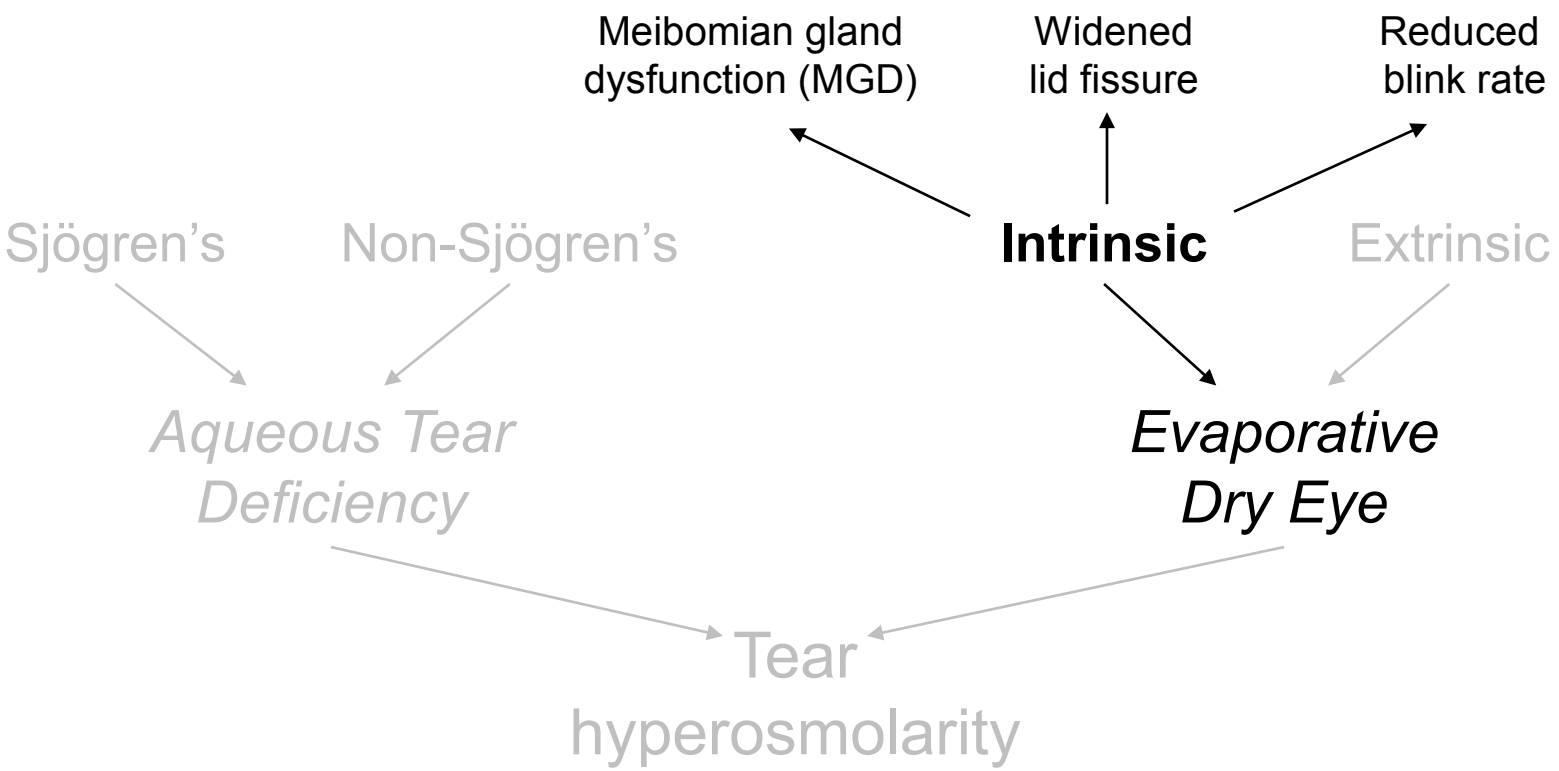
Dry Eye Syndrome

In this context, *intrinsic EDE* refers to any cause related to the eyelids, whereas *extrinsic EDE* refers to any non-eyelid factor that promotes evaporation.



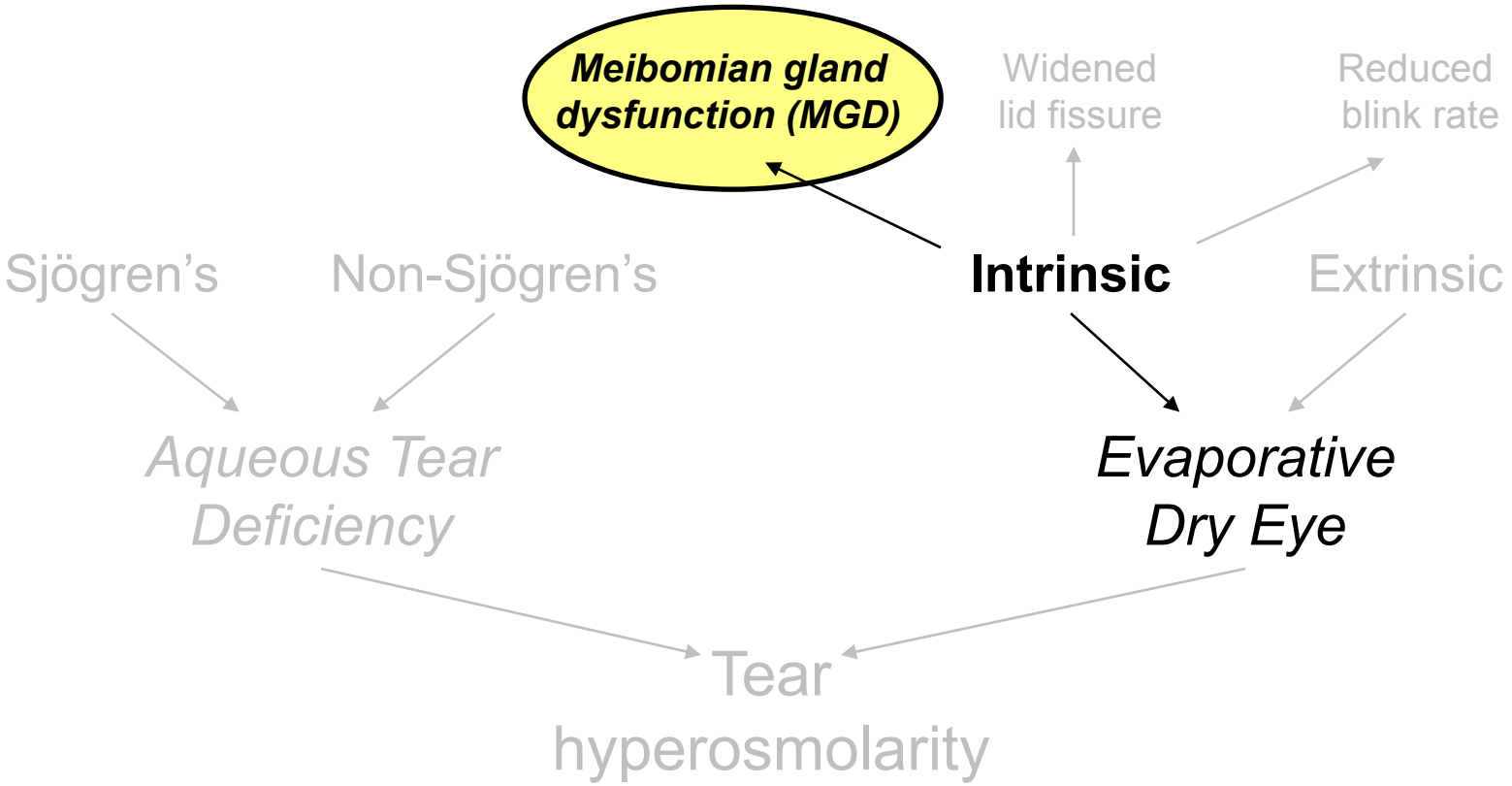
Dry Eye Syndrome

There are three common causes of intrinsic EDE:



Dry Eye Syndrome

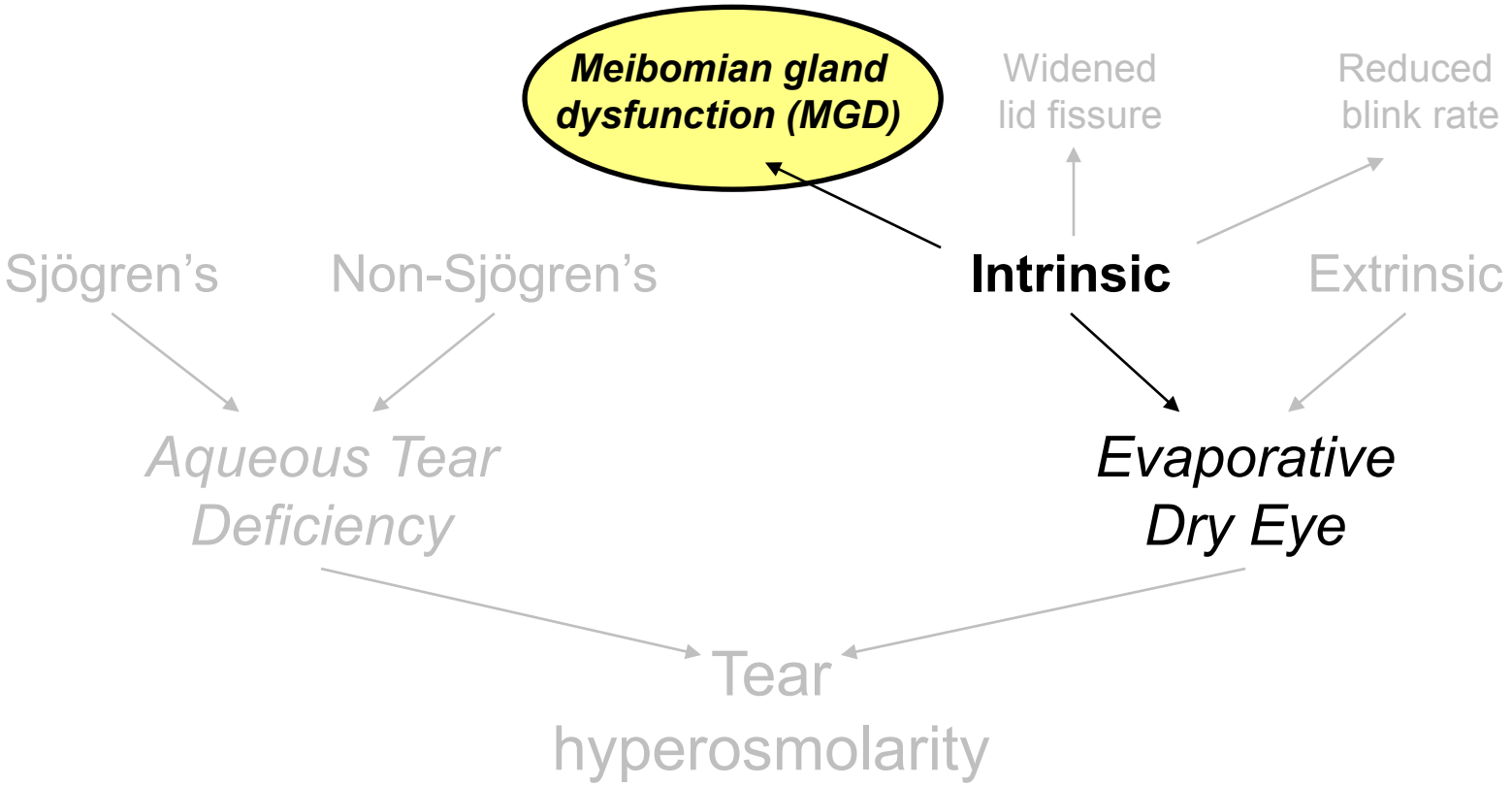
MGD refers to a broad group of disorders characterized by dysfunction of the meibomian glands. Individual of Asian heritage are at especially high risk.



Dry Eye Syndrome

MGD refers to a broad group of disorders characterized by dysfunction of the meibomian glands. Individual of Asian heritage are at especially high risk.

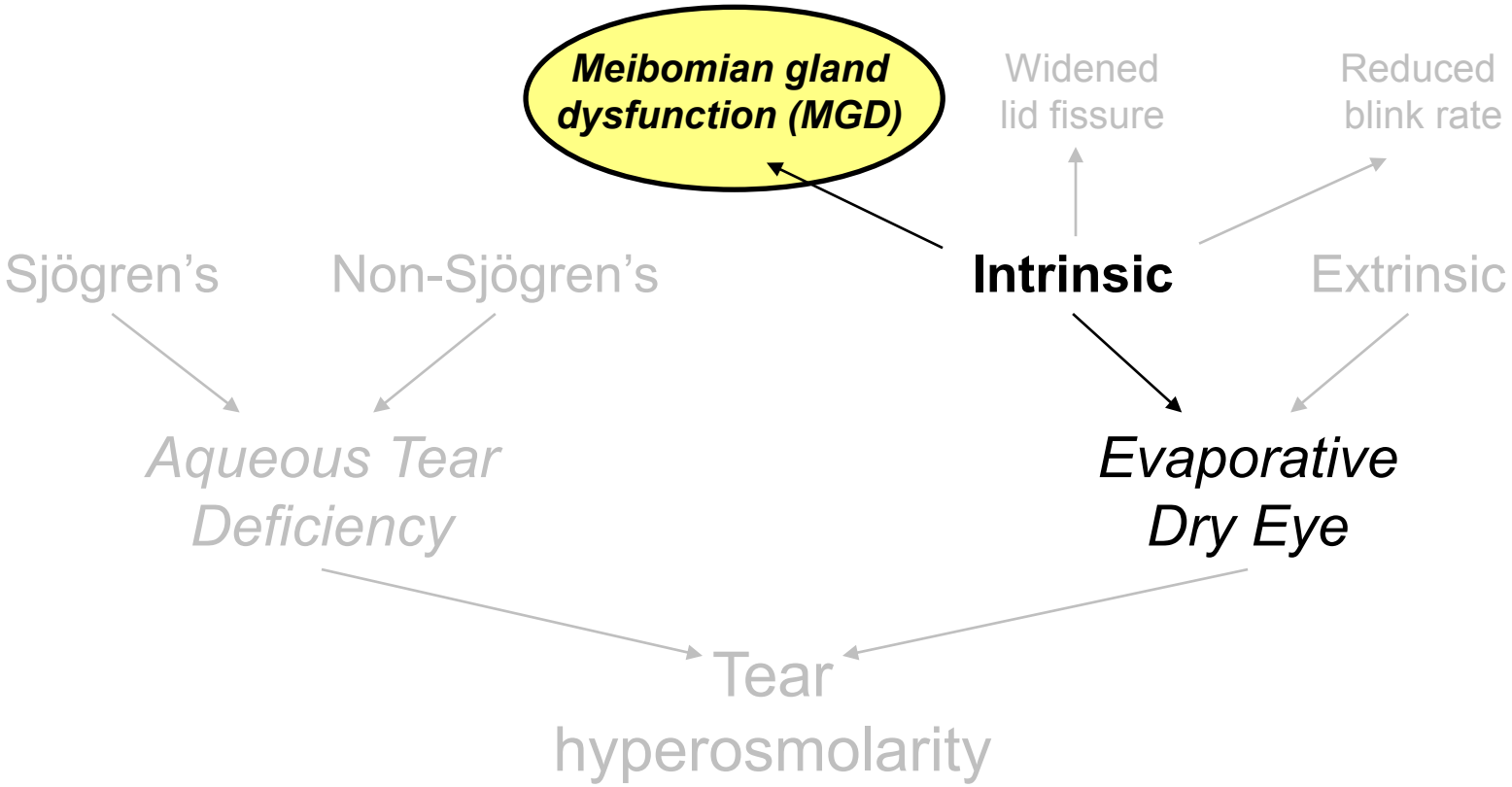
In many cases, the dysfunction is due to obstruction of gland output leading to an inadequate volume of meibum in the tear-film; in others, the meibum's chemical composition has been altered, rendering it ineffective. (Chemical alteration and obstruction commonly co-exist in the same pt.)



Dry Eye Syndrome

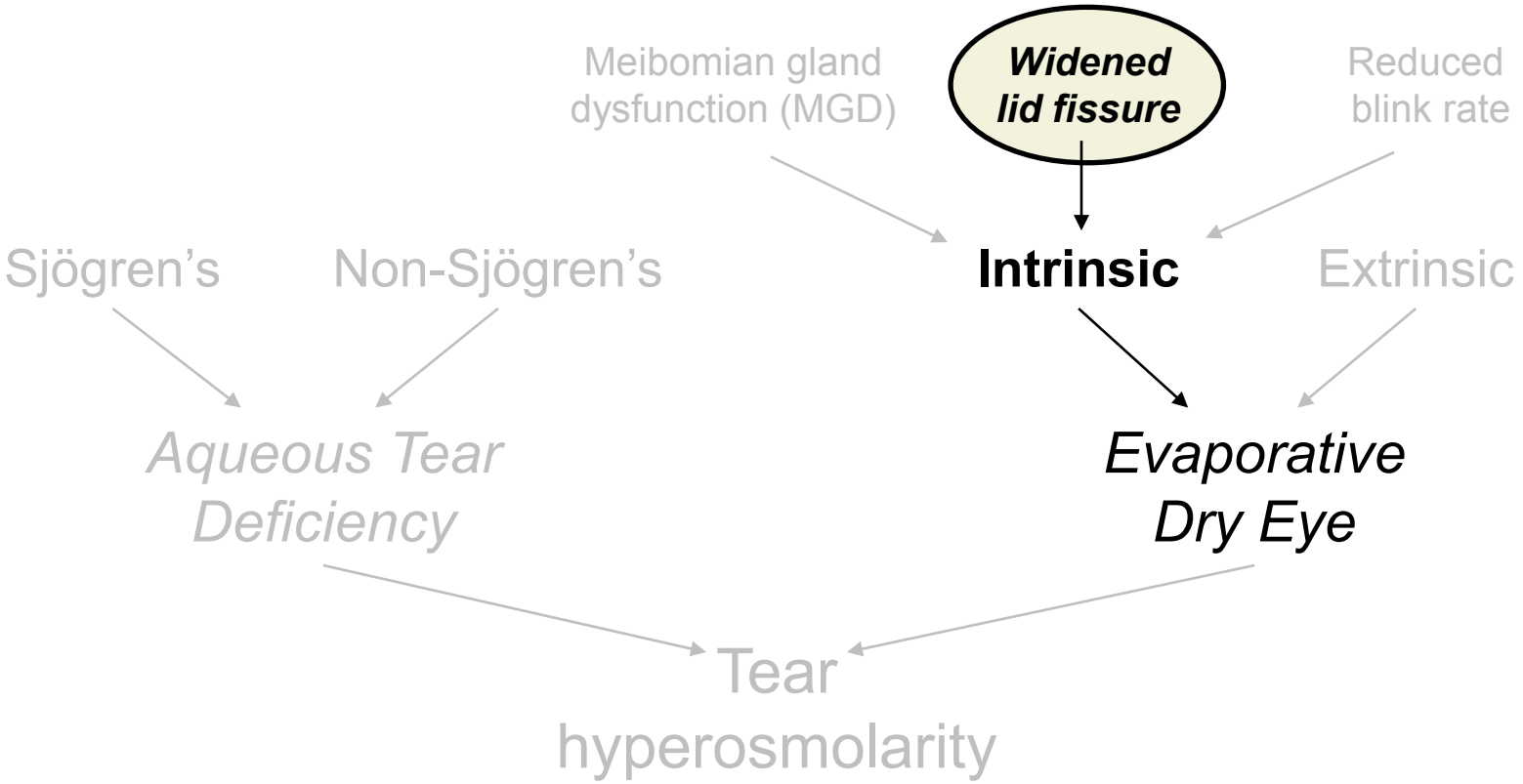
MGD refers to a broad group of disorders characterized by dysfunction of the meibomian glands. Individual of Asian heritage are at especially high risk.
In many cases, the dysfunction is due to obstruction of gland output leading to an inadequate volume of meibum in the tear-film; in others, the meibum's chem al alteration and obstruction commonly co-exist in the same pt.)

We will have more to say about MGD later in the set



Dry Eye Syndrome

A **widened lid fissure** can be 2ndry to forward displacement of the globe (ie, proptosis/exophthalmos); increased innervation to the lid retractors such as occurs in thyroid eye disease; and/or to congenital craniofacial malformations resulting in shallow orbits (eg, Crouzon syndrome).



Dry Eye Syndrome

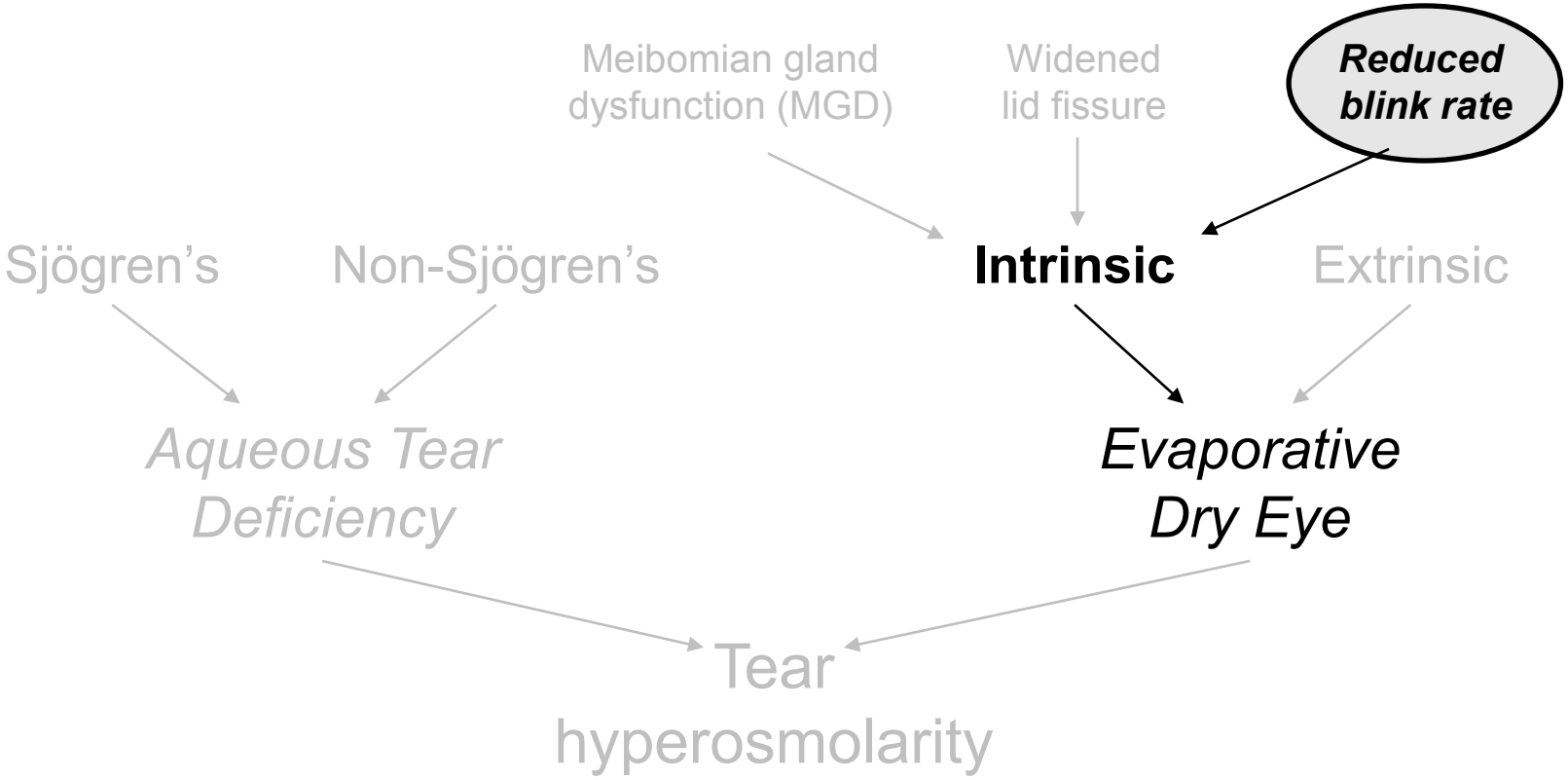
TED: Lid retraction + exophthalmos->surface exposure->EDE

Dry Eye Syndrome

Crouzon syndrome: Shallow orbits->surface exposure->EDE

Dry Eye Syndrome

Causes of **reduced blink rate** include normal phenomena such as seen during sustained participation in a visually intensive task (eg, reading; computer work) as well as pathological causes such as Parkinson's dz.



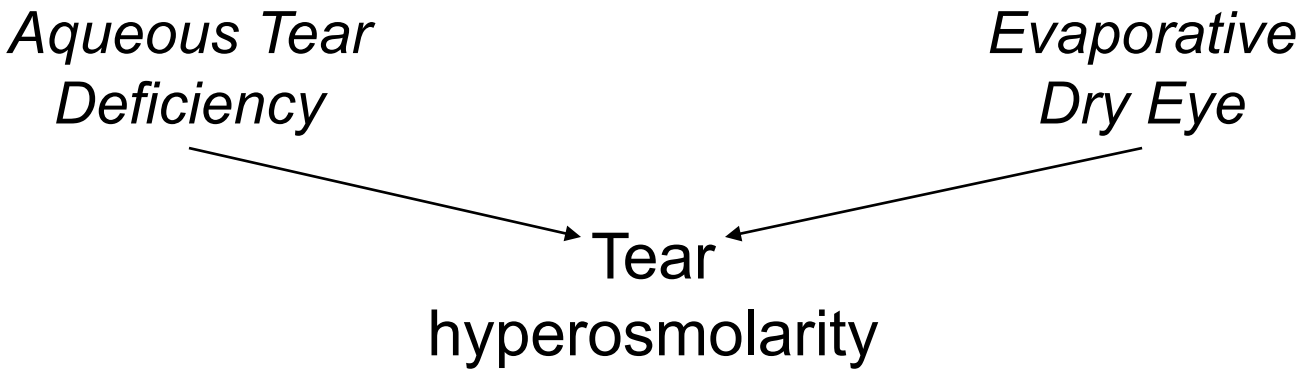
Dry Eye Syndrome

The pathophysiology for DES damage starts with disarrangement of the tear film in the form of

Head's up: Shortly we're gonna add a *third* mechanism leading to tear hyperosmolarity

There ~~are~~ three two basic ways in which the status of the aqueous component of the tear film could lead to tear hyperosmolarity:

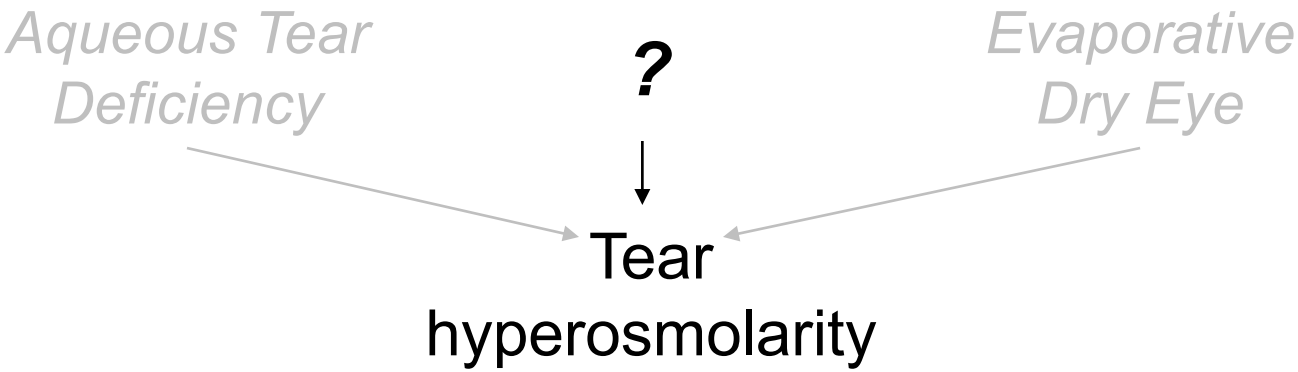
Recall that earlier in the set we alluded to a *third* means by which tear-film status could produce hyperosmolarity and dry eye. The time to address this has arrived!



Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**

The ***other*** *fundamental way could the status of the tear film lead to tear hyperosmolarity:*

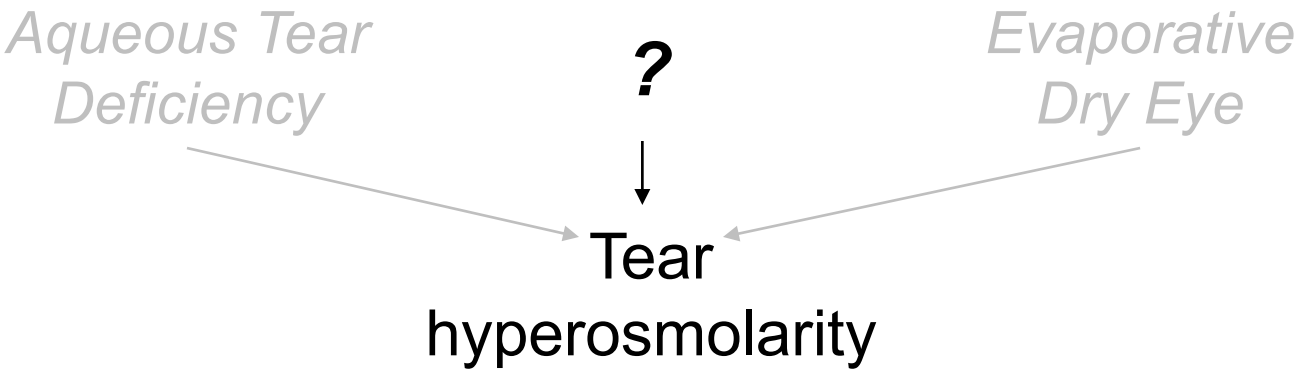


Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**

The ***other*** *fundamental way could the status of the tear film lead to tear hyperosmolarity:*

The tear film can break up too quickly, exposing the ocular surface.



Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**

The ***other*** *fundamental way could the status of the tear film lead to tear hyperosmolarity:*

The tear film can break up too quickly, exposing the ocular surface.
This state is known as one of…

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

↓

Tear hyperosmolarity

Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**

Recalling our answers to **this** issue previously:

While it's a bit of an oversimplification, we can associate the components of the tear film with the pathologic states underlying DES:

Problem with the ***aqueous component***

Aqueous Tear Deficiency

Tear Film Instability

Problem with the ***lipid component***

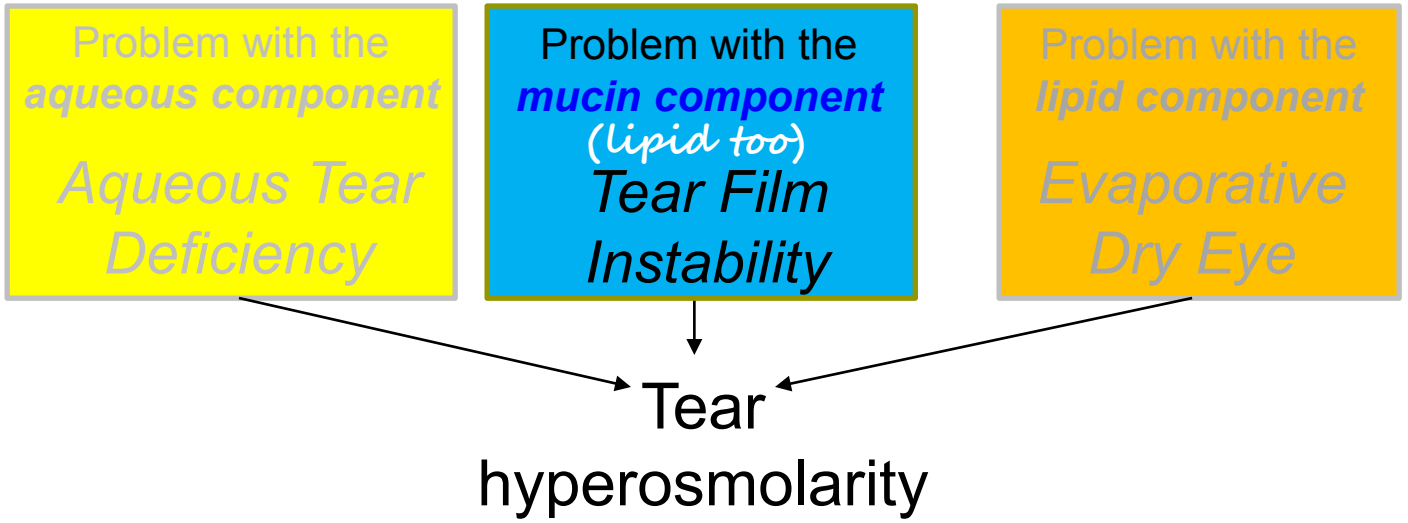
Evaporative Dry Eye

Tear hyperosmolarity

Dry Eye Syndrome

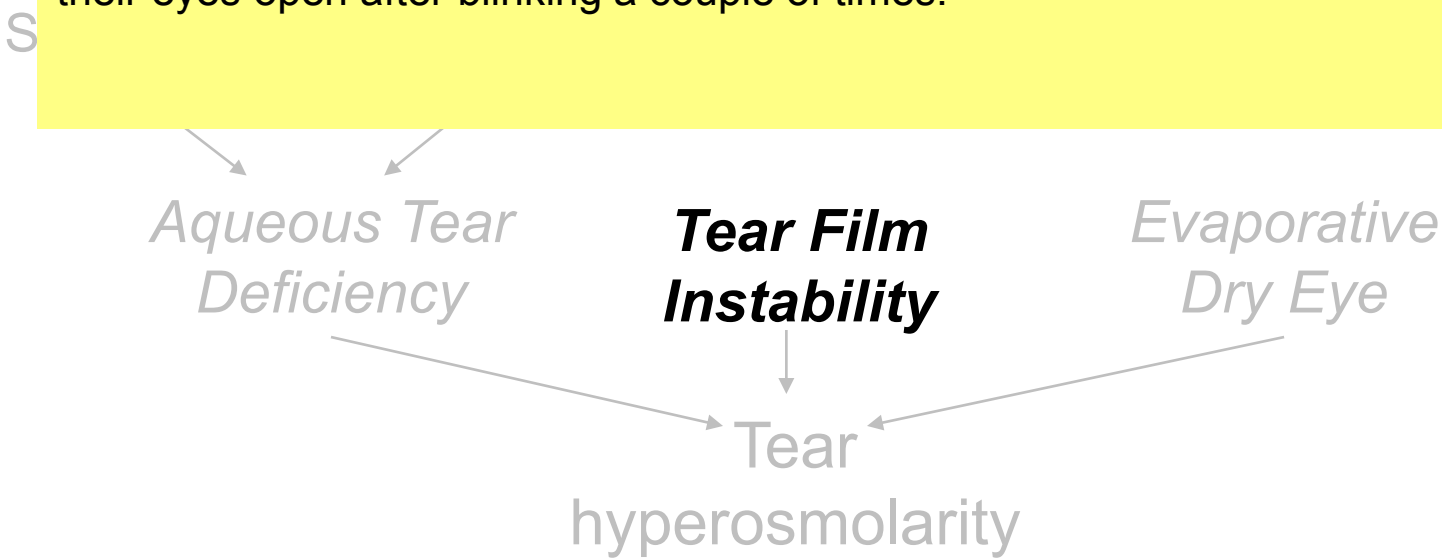
The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity.**

Recalling our answers to **this** issue previously:
With respect to tear-film instability, problems with the mucin (and lipid) components lead to tear-film instability.



Dry Eye Syndrome

Tear-film instability is quantified via the **tear-film break-up time** (TBUT or TFBUT) assessment. A little fluorescein is instilled, and the pt is asked to hold their eyes open after blinking a couple of times.



Dry Eye Syndrome

Tear-film instability is quantified via the **tear-film break-up time** (TBUT or TFBUT) assessment. A little fluorescein is instilled, and the pt is asked to hold their eyes open after blinking a couple of times. The tear film is observed with the cobalt-blue filter in place, and the length of time that passes until a dry spot appears is noted. A TBUT of less than ~10s is considered abnormal.

S

Aqueous Tear Deficiency

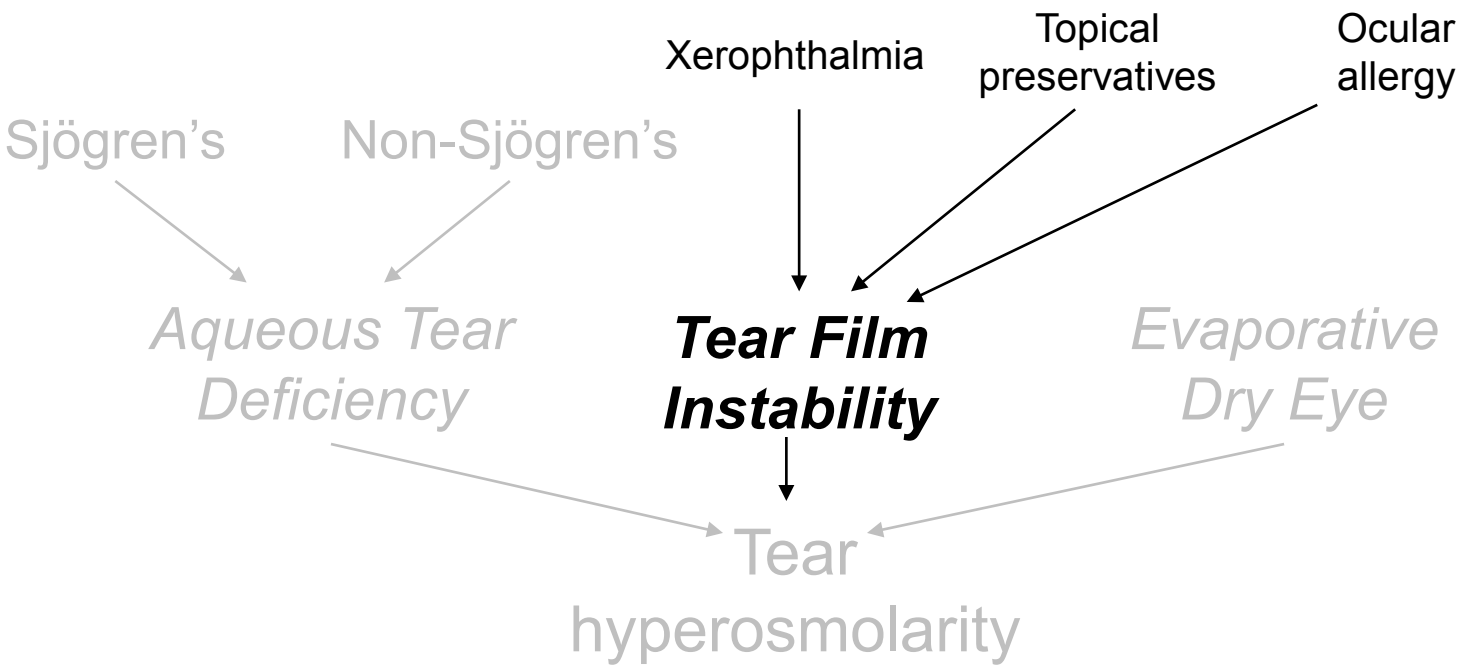
Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity

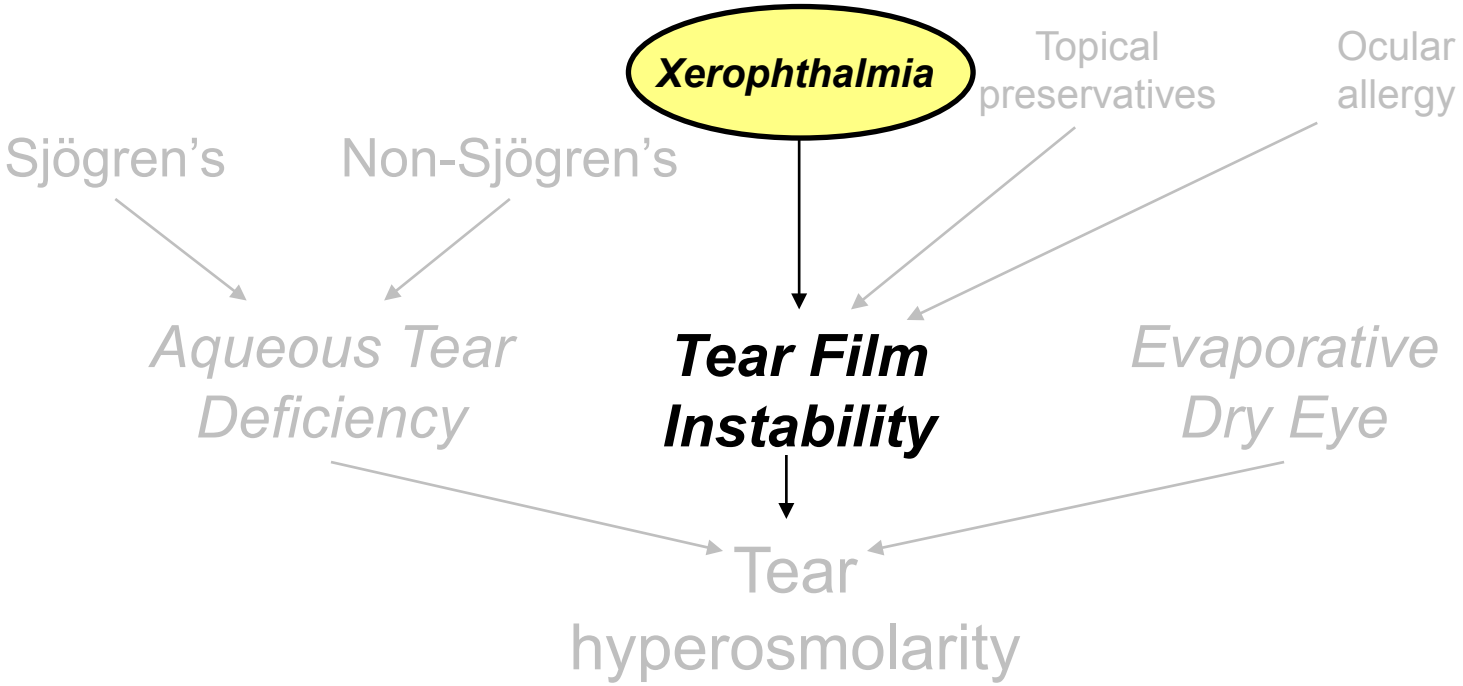
Dry Eye Syndrome

Three categories of conditions leading to TFI have been identified:



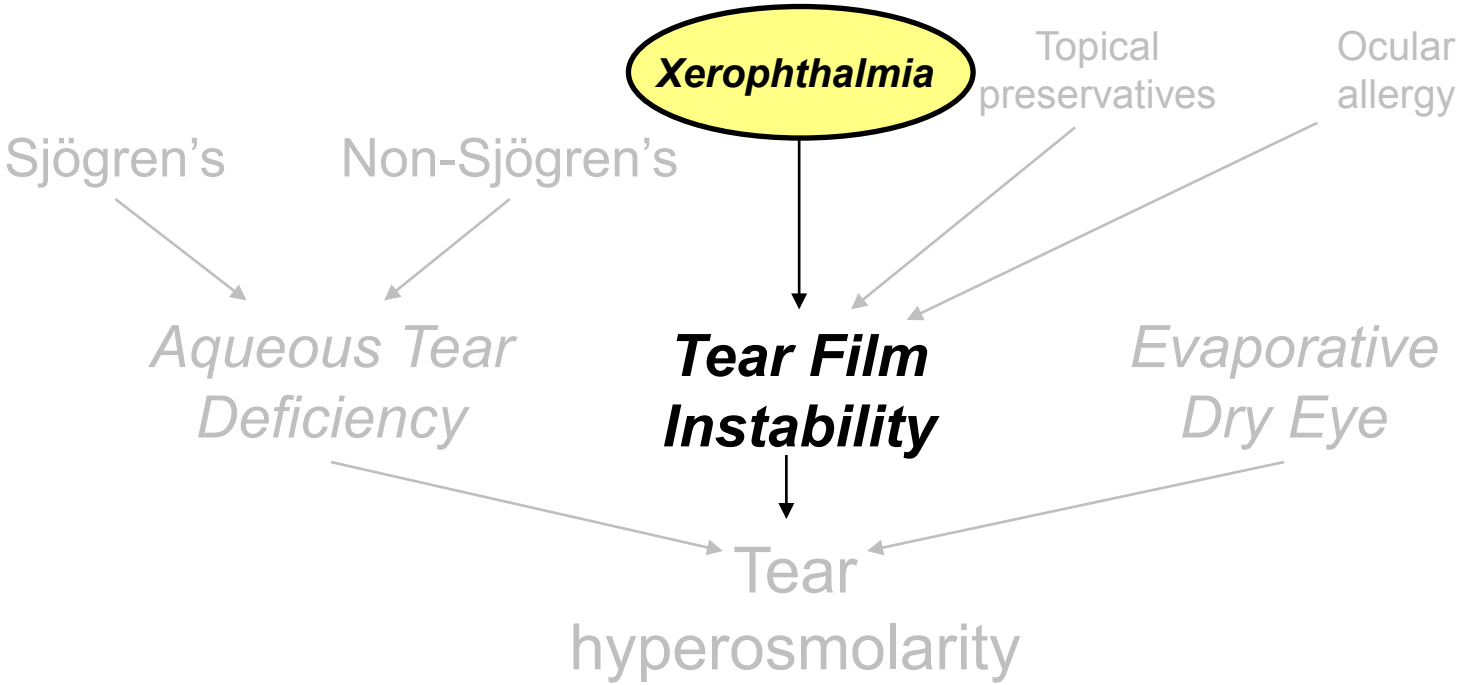
Dry Eye Syndrome

The leading cause of xerophthalmia worldwide is **hypovitaminosis A**, a potentially fatal condition.

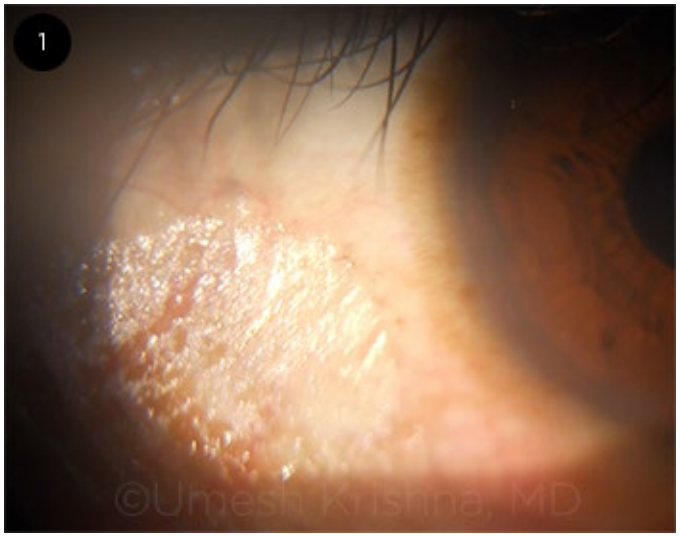


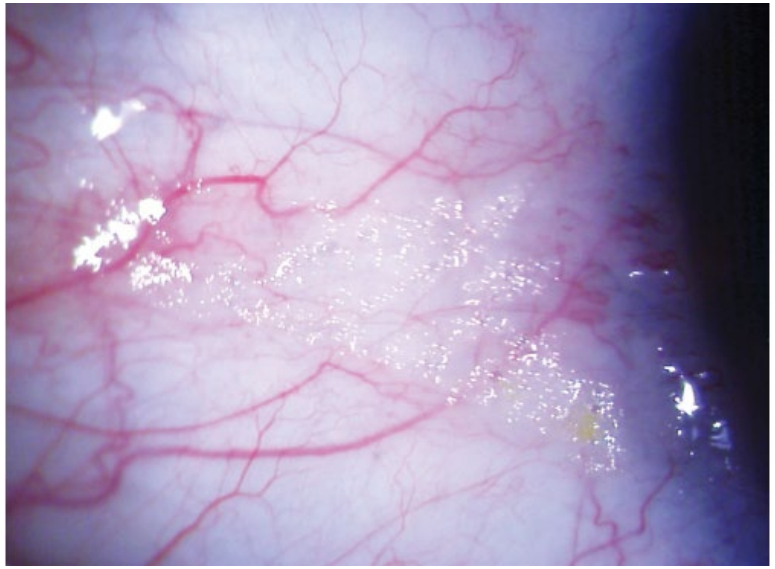
Dry Eye Syndrome

The leading cause of xerophthalmia worldwide is **hypovitaminosis A**, a potentially fatal condition.
Hypovitaminosis A xerosis of the ocular surface produces **Bitôt spots**—foamy, white/gray area on the interpalpebral conjunctiva.



Dry Eye Syndrome

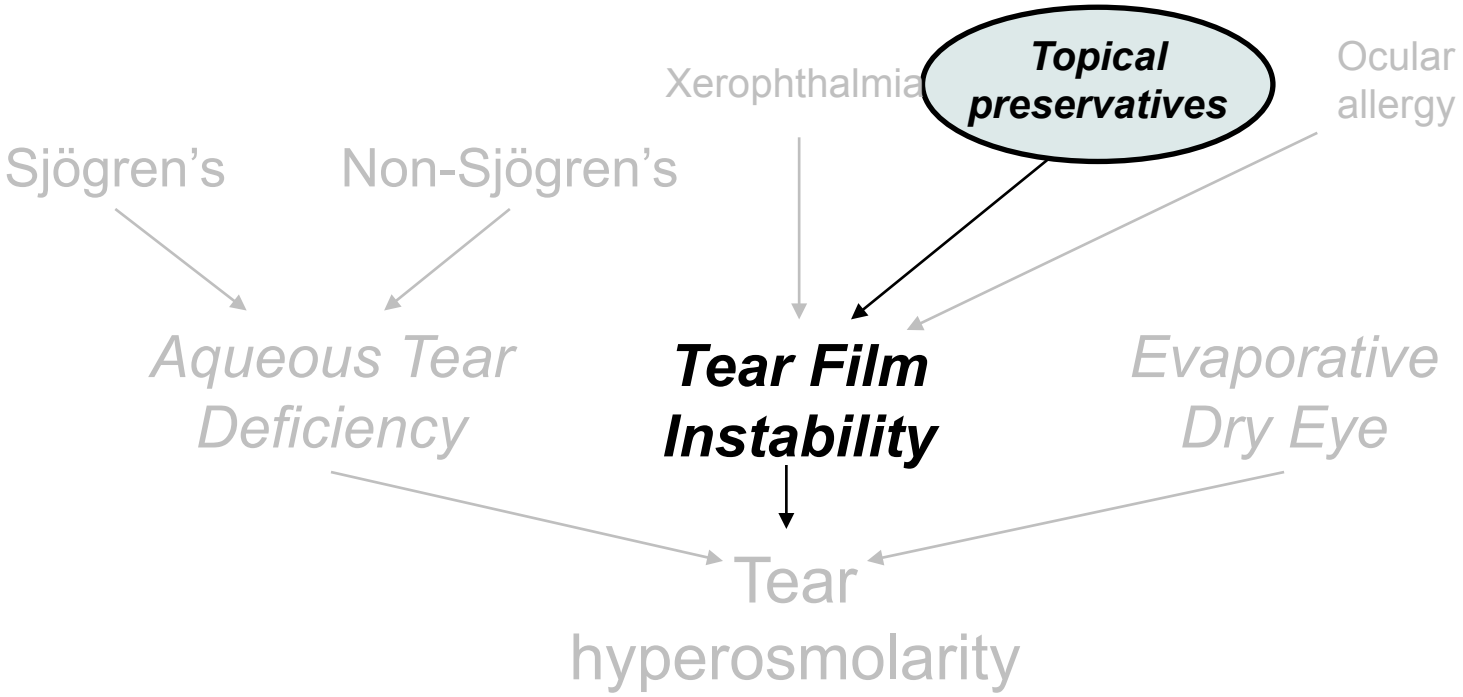




Bitôt spots: Conj finding temporal to the cornea, with typical dry/foamy appearance

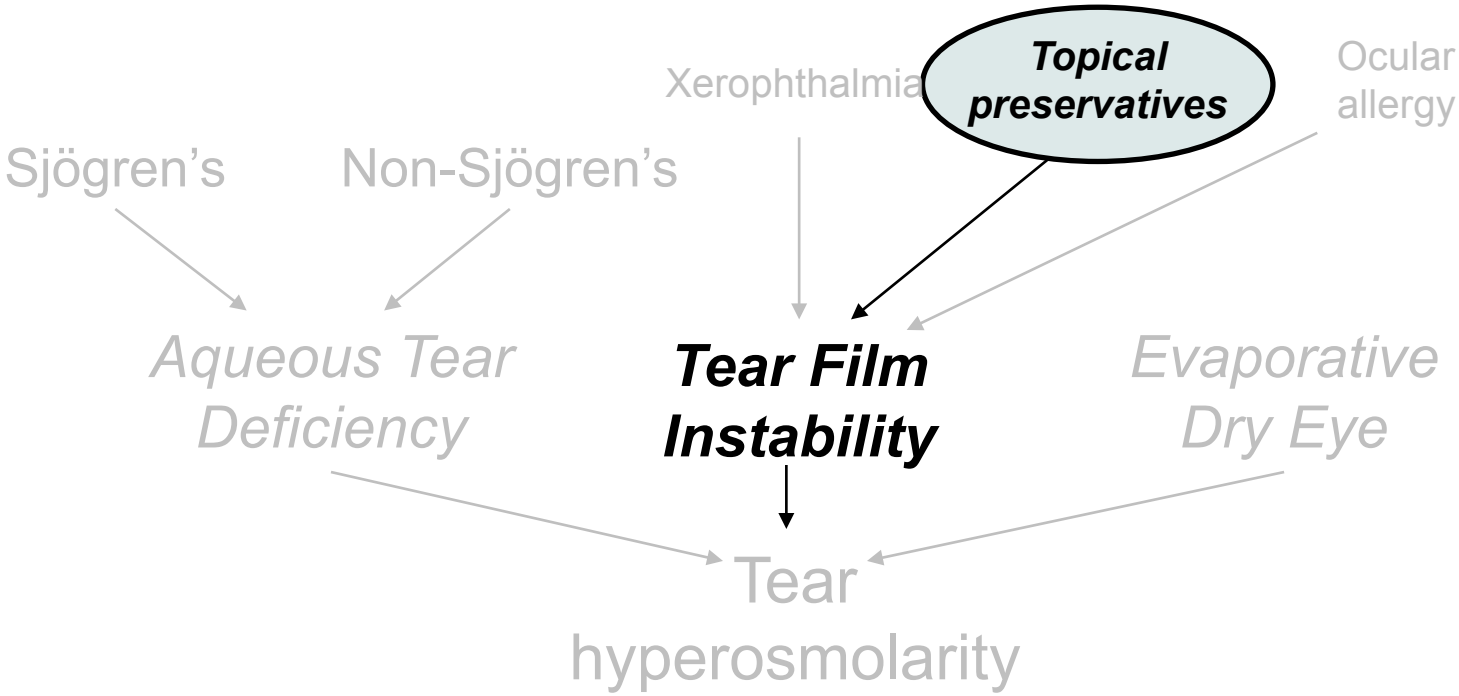
Dry Eye Syndrome

Preservatives in ophthalmic preparations lead to TFI by provoking an inflammatory response in the conj epithelium, which in turn promotes goblet cell apoptosis.



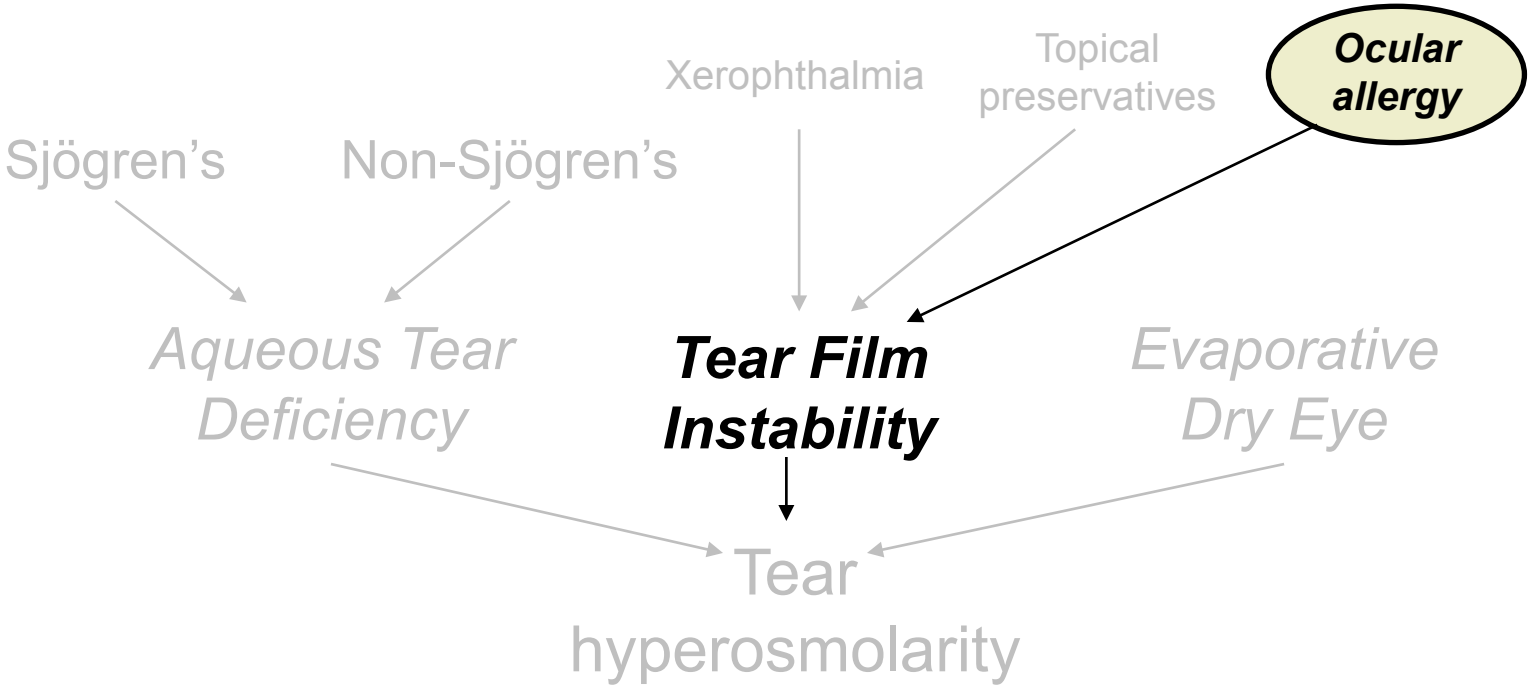
Dry Eye Syndrome

Preservatives in ophthalmic preparations lead to TFI by provoking an inflammatory response in the conj epithelium, which in turn promotes goblet cell apoptosis. The preservative **benzalkonium chloride** (aka BAK or BAC) is especially notorious for doing this.



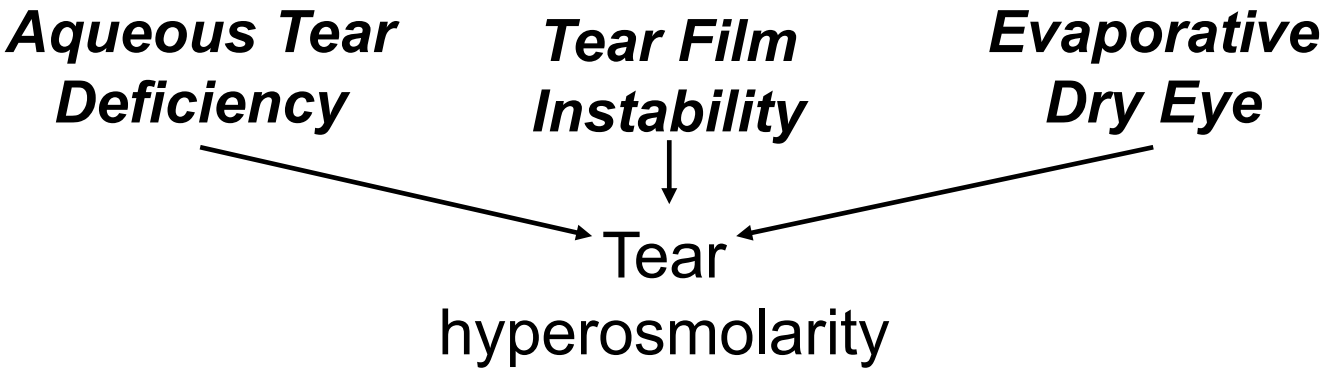
Dry Eye Syndrome

Allergic antigens produce TFI by initiating an IgE-mediated inflammatory cascade, leading to goblet-cell loss.

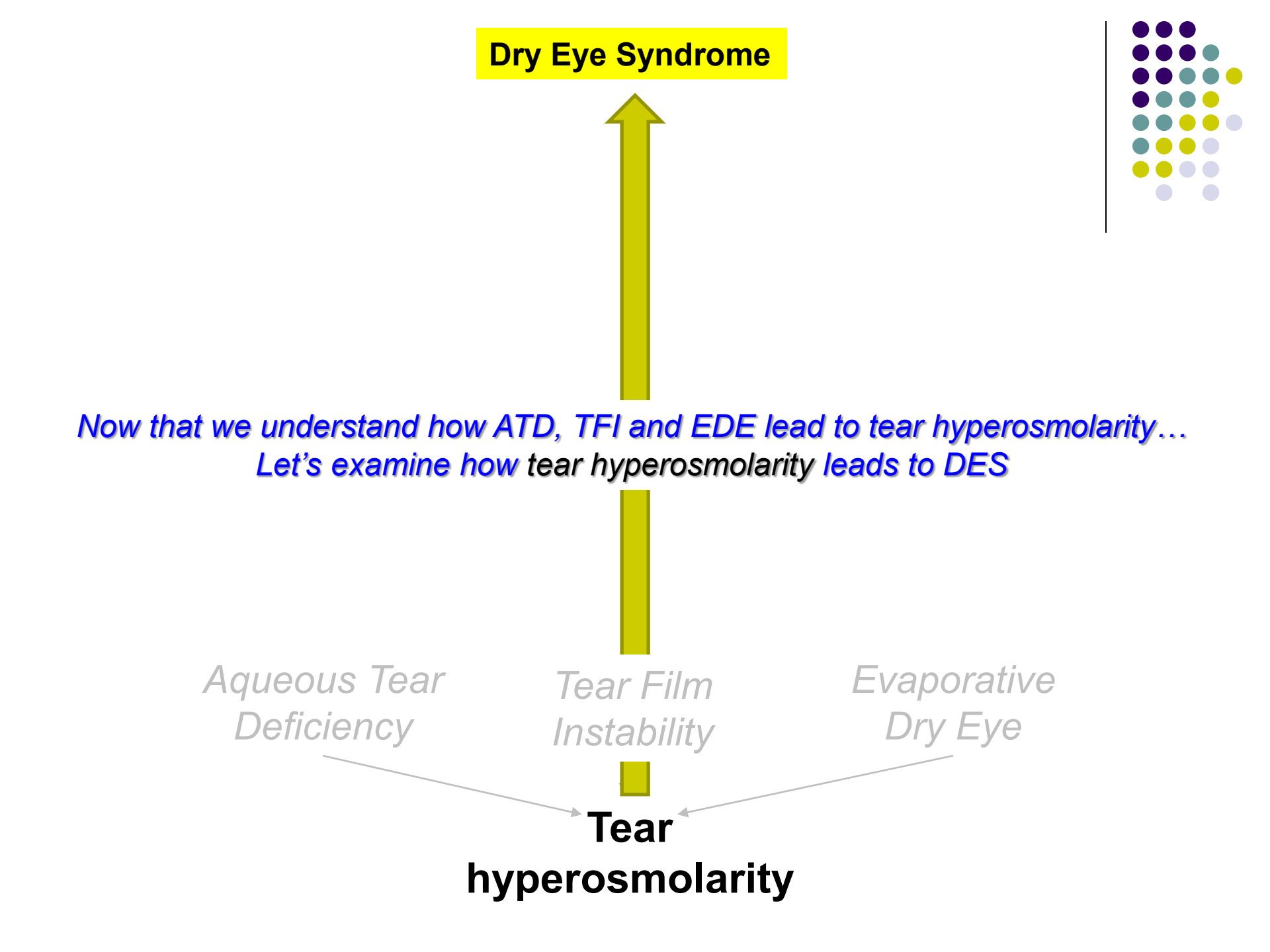


Dry Eye Syndrome

Now that we understand how ATD, TFI and EDE lead to tear hyperosmolarity…



Dry Eye Syndrome



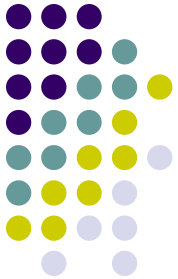
Now that we understand how ATD, TFI and EDE lead to tear hyperosmolarity…
Let's examine how tear hyperosmolarity leads to DES

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity



Dry Eye Syndrome

Surface epithelium damage

Hyperosmolar stress

Aqueous Tear Deficiency

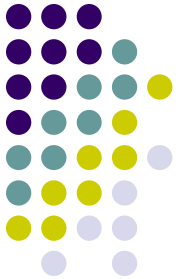
Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity

First:

Tear-film hyperosmolarity initiates the DES cascade by stressing surface epithelium, significantly damaging it.



Dry Eye Syndrome

Then:

In turn, damaged epi cells release cytokines that promote and/or facilitate inflammation. These cytokines include TNF, MMP-9, and IL-1.

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

Tear-film hyperosmolarity initiates the DES cascade by stressing surface epithelium, significantly damaging it.

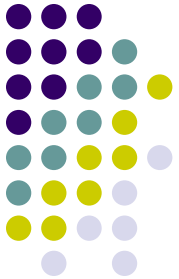
Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity

Dry Eye Syndrome



Then:

In turn, damaged epi cells release cytokines that promote and/or facilitate inflammation. These cytokines include TNF, MMP-9, and IL-1. TNF and IL-1 promote epi-cell apoptosis, whereas MMP-9 cleaves epi cells from their BM.

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

Tear-film hyperosmolarity initiates the DES cascade by stressing surface epithelium, significantly damaging it.

Aqueous Tear Deficiency

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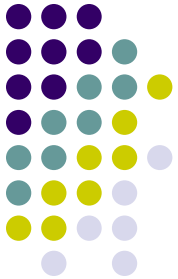
Tear hyperosmolarity

Dry Eye Syndrome



Inflammatory cytokine release

DES feedback loop!

Surface epithelium damage

In turn, damaged epi cells release cytokines that promote and/or facilitate inflammation. These

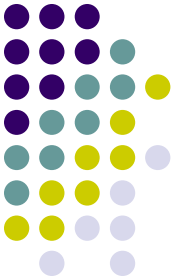
Note that surface epi damage induces cytokine release…*And cytokine release induces surface epi damage*. Thus, a vicious cycle/circle develops in which *epi damage leads directly to further epi damage*.

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity



Dry Eye Syndrome

Note that

Cytokines also impede function of the afferent arm of the LFU reflex arc…

Neural reflex arc disruption

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

In turn, damaged epi cells release cytokines that promote and/or facilitate inflammation. These cytokines include TNF, MMP-9, and IL-1. TNF and IL-1 promote epi-cell apoptosis, whereas MMP-9 cleaves epi cells from their BM.

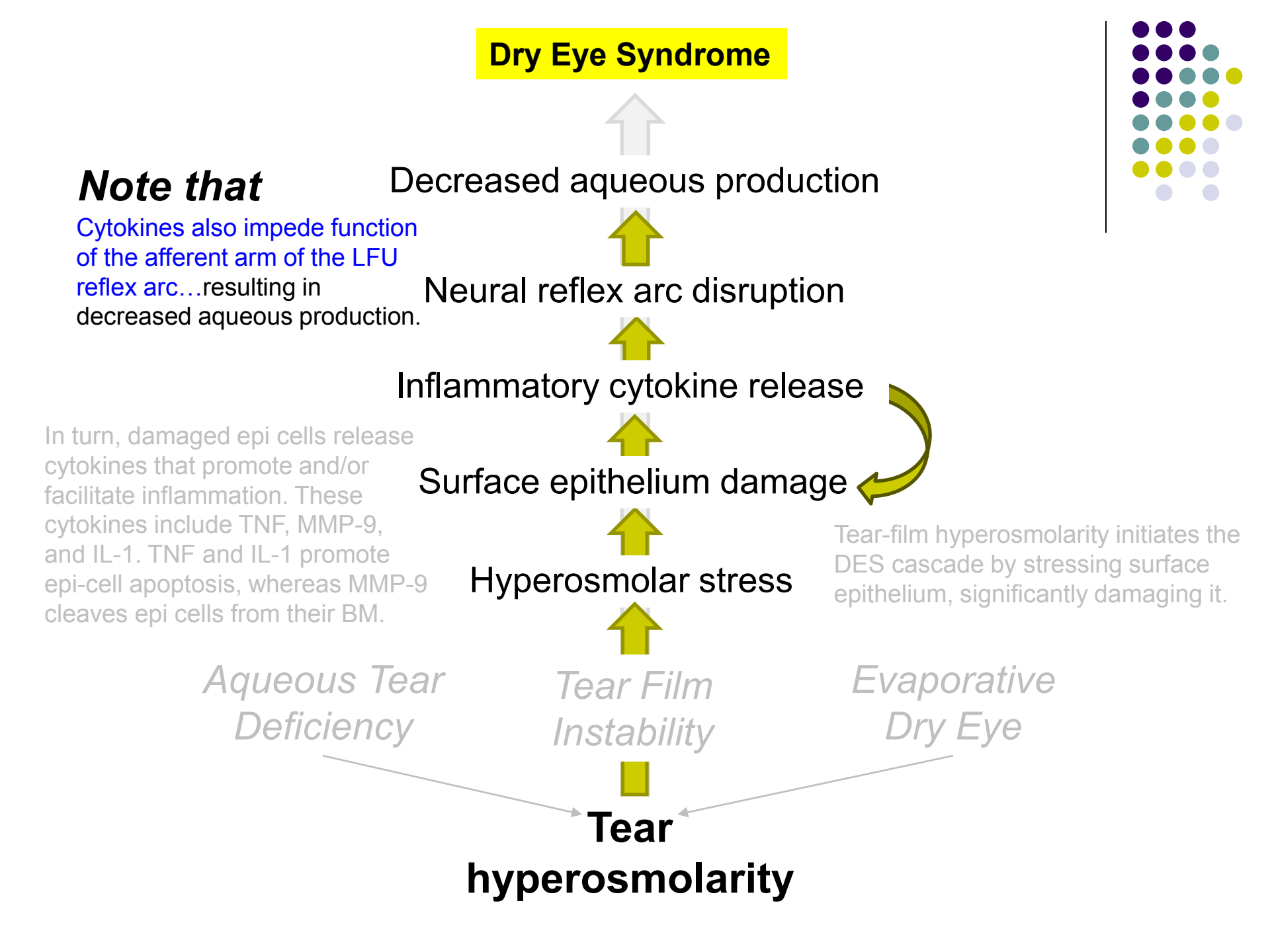
Tear-film hyperosmolarity initiates the DES cascade by stressing surface epithelium, significantly damaging it.

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity



Note that

Cytokines also impede function of the afferent arm of the LFU reflex arc…resulting in decreased aqueous production.

In turn, damaged epi cells release cytokines that promote and/or facilitate inflammation. These cytokines include TNF, MMP-9, and IL-1. TNF and IL-1 promote epi-cell apoptosis, whereas MMP-9 cleaves epi cells from their BM.

Tear-film hyperosmolarity initiates the DES cascade by stressing surface epithelium, significantly damaging it.

Dry Eye Syndrome

Decreased aqueous production

Neural reflex arc disruption

So neural reflex arc disruption decreases aqueous production…

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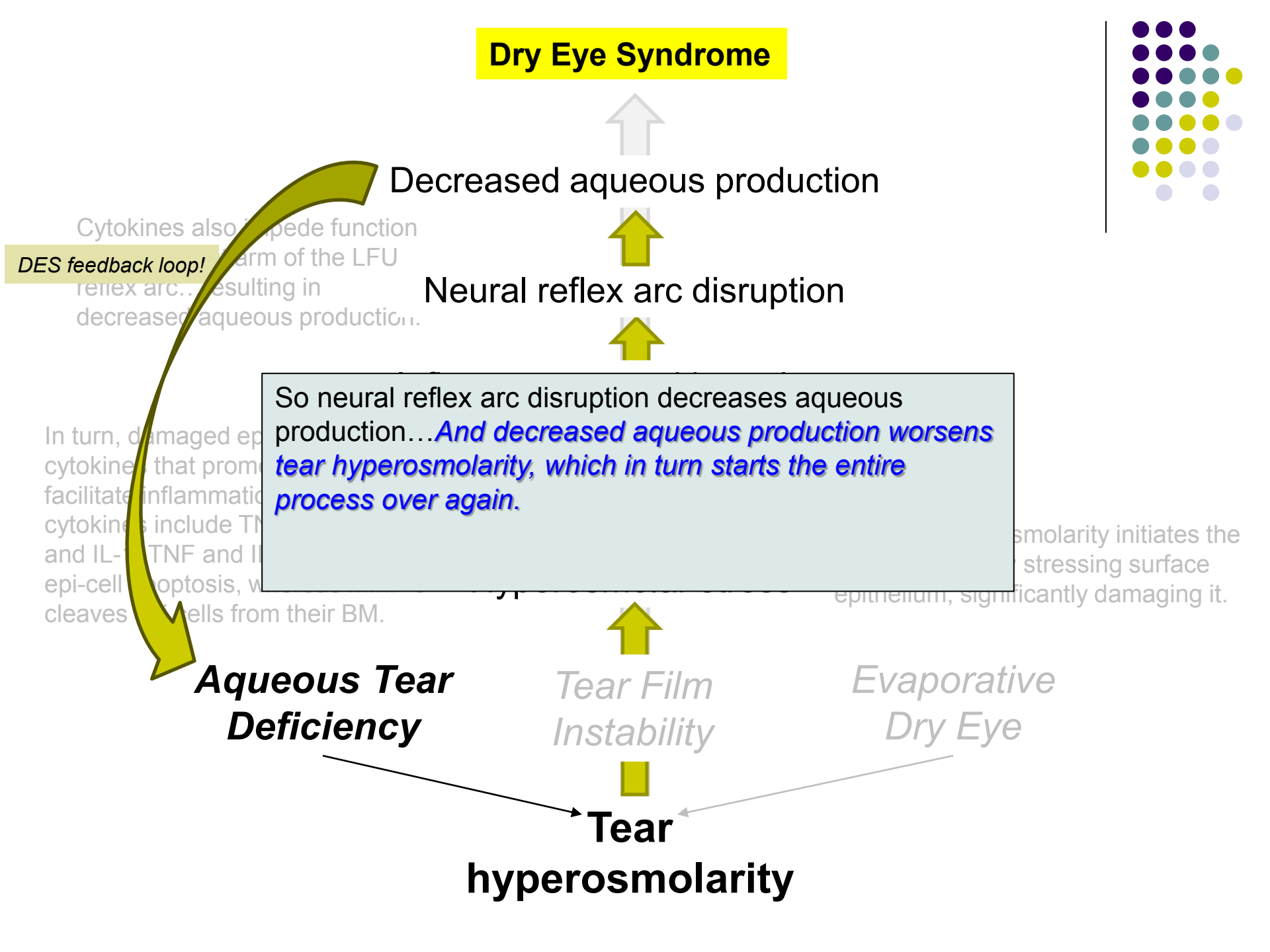
…smolarity initiates the …stressing surface epithelium, significantly damaging it.

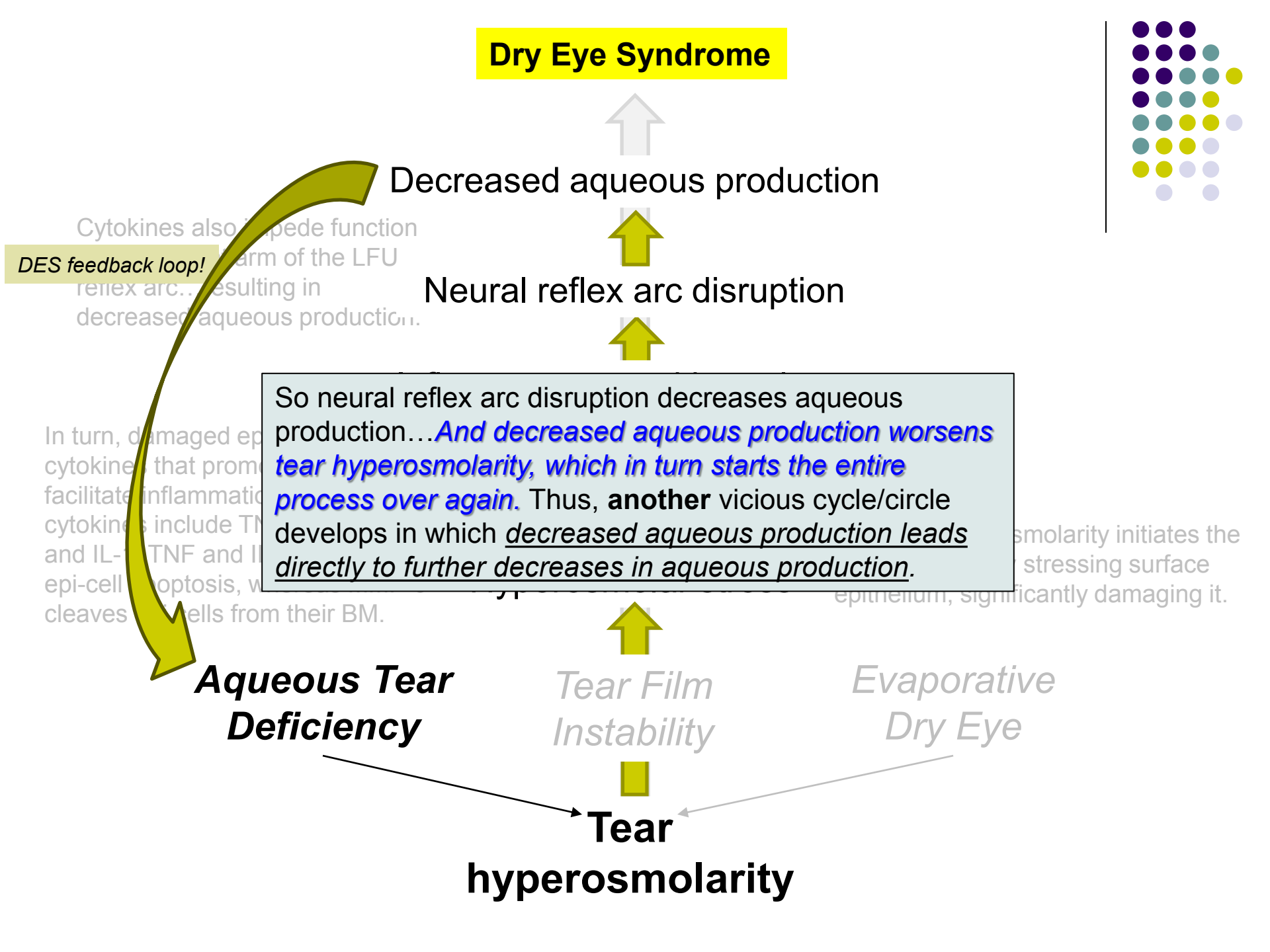
Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity


Dry Eye Syndrome
Decreased aqueous production
Neural reflex arc disruption
So neural reflex arc disruption decreases aqueous production…And decreased aqueous production worsens tear hyperosmolarity, which in turn starts the entire process over again.
Aqueous Tear Deficiency
Tear Film Instability
Evaporative Dry Eye
Tear hyperosmolarity


Dry Eye Syndrome
Decreased aqueous production
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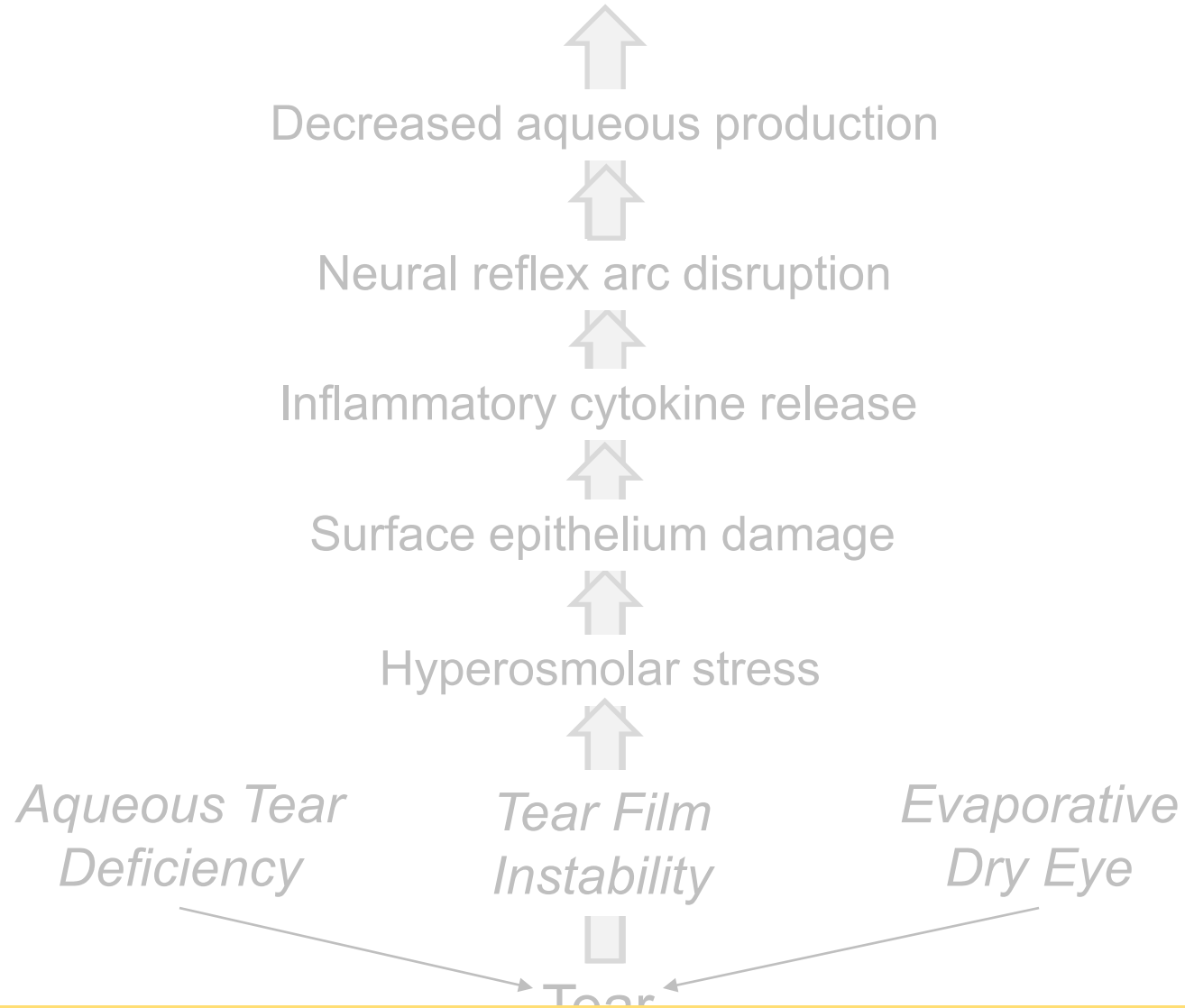
So neural reflex arc disruption decreases aqueous production…And decreased aqueous production worsens tear hyperosmolarity, which in turn starts the entire process over again. Thus, another vicious cycle/circle develops in which decreased aqueous production leads directly to further decreases in aqueous production.
Aqueous Tear Deficiency
Tear Film Instability
Evaporative Dry Eye
Tear hyperosmolarity

Dry Eye Syndrome



With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

Dry Eye Syndrome

Decreased aqueous production

Neural reflex arc disruption

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

1) Increase tear volume

Tear volume

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

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3) Prevent cytokine release and/or mitigate their effects

Inflammatory cytokine release

Surface epithelium damage

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Inflammatory cytokine release

The most straightforward means of increasing aqueous volume is supplementing the tear lake with **artificial tears**. (The *Cornea* book says tear substitutes are “the mainstay of treatment for aqueous tear deficiency.”)

1) Increase tear volume

2) Decrease tear evaporation

Tear volume

Tear Film Instability

Tear evaporation

Dry Eye

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3) Prevent cytokine release and/or mitigate their effects

Inflammatory cytokine release

The most straightforward means of increasing aqueous volume is supplementing the tear lake with **artificial tears**. (The *Cornea* book says tear substitutes are "the mainstay of treatment for aqueous tear deficiency.") In more severe cases **punctal occlusion** may be indicated.

1) Increase tear volume

Tear volume

2) Decrease tear evaporation

Tear evaporation

Tear Film Instability

Evaporative Dry Eye

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

Dry Eye Syndrome

Decreased aqueous production

Neural reflex arc disruption

3) Prevent cytokine release and/or mitigate their effects

Inflammatory cytokine release

The mainstay tx of EDE is **lid hygiene**. (The *Cornea* book says lid hygiene is "an essential part [of tx] at all stages of the disease.")

1) Increase tear volume

2) Decrease tear evaporation

Tear volume

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear evaporation

Tear

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Inflammatory cytokine release

The mainstay tx of EDE is **lid hygiene**. (The *Cornea* book says lid hygiene is "an essential part [of tx] at all stages of the disease.")
The two fundamental steps involved in lid hygiene are:
1) Application of heat to the eyelids to soften the abnormal meibum; and
2) Compression/massage of the lid margin to express the abnormal meibum.

1) Increase tear volume

2) Decrease tear evaporation

Tear volume

Aqueous Tear Deficiency

Tear Film Instability

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Tear evaporation

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With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

Dry Eye Syndrome

Decreased aqueous production

Quick sidebar on ‘abnormal meibum’: What makes it abnormal is its chemical composition, which has been altered (and not for the better). Because of this altered composition, its melting point is higher than normal. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil resting on the lid margin.

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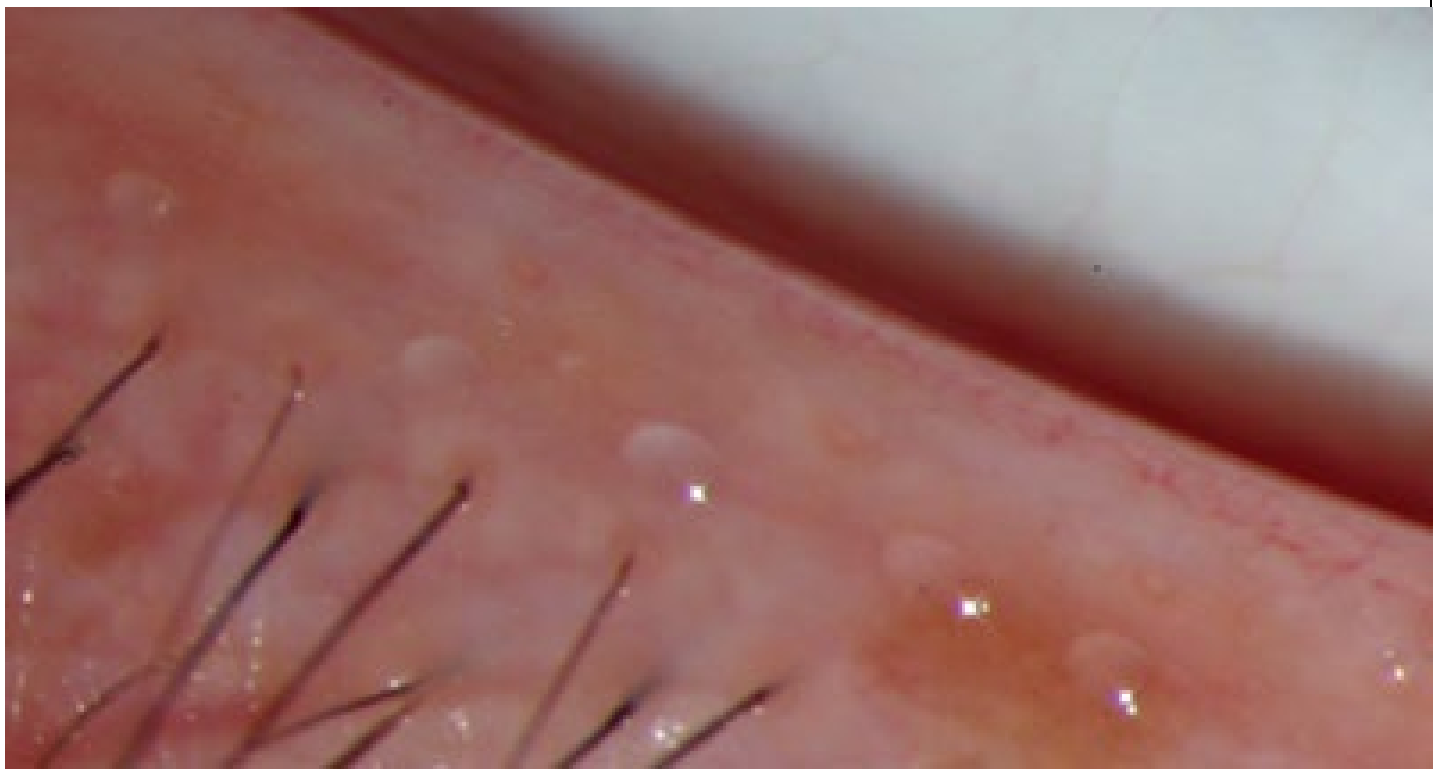

Normal meibum

Dry Eye Syndrome

Decreased aqueous production

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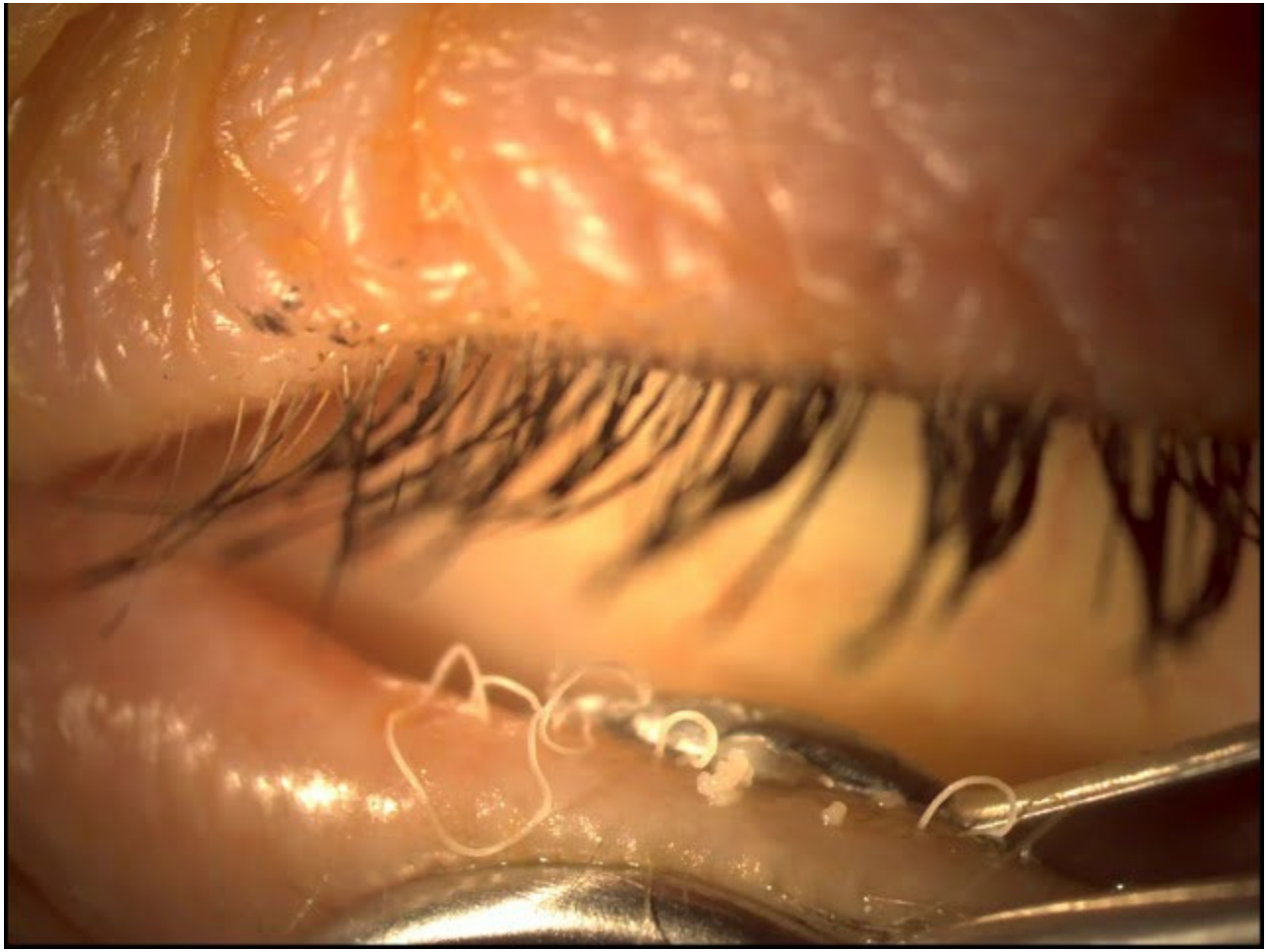

Ewwww

Dry Eye Syndrome

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So to reiterate, the logic underpinning lid hygiene is:
--**Step 1**: Liquify the semisolid abnormal meibum clogging the glands
--**Step 2**: Express the now-liquefied abnormal meibum from the glands

1) Application of heat to the eyelids to soften the **abnormal meibum**, and
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Tear Volume

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear evaporation

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

Dry Eye Syndrome

Decreased aqueous production

But using heat and massage to get the abnormal meibum flowing is not sufficient. Why? Because once the lids cool back down to body temp, the subsequent (abnormal) meibum will again be semisolid—that is, unless steps are taken to normalize its chemical composition.

Hyperosmolar stress

1) Increase tear volume

2) Decrease tear evaporation

Tear volume

Tear Film Instability

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3) F
miti

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--Topical abx to reduce bacterial load (bacterial lipases play an important role in altering meibum's chemical composition)

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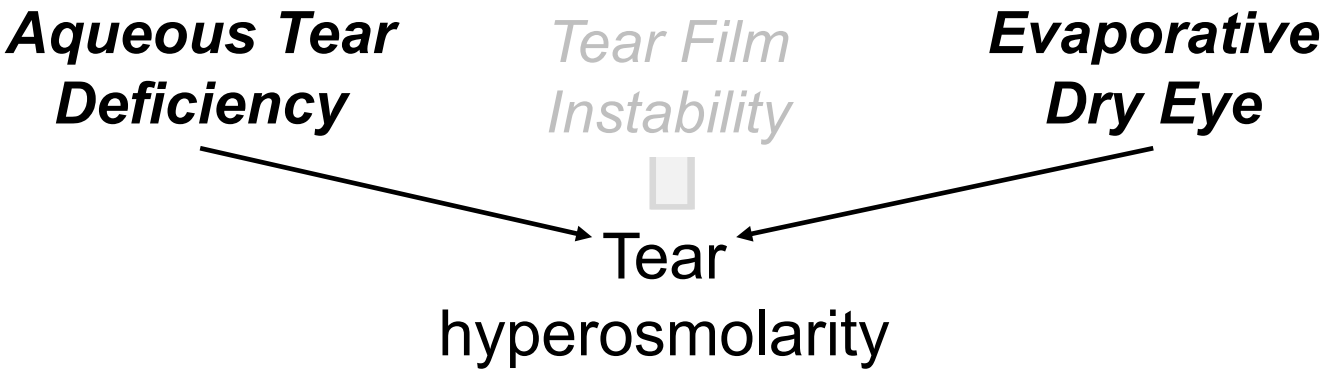
To this point we've discussed treatment strategies for ATD and EDE as distinct entities (which they are). However, ATD and EDE frequently coexist in DES pts, and this has important implications for management. Most interventions (ATs, anti-inflammatory meds) are useful in both conditions. However, there is one relatively common ATD intervention that must be used with caution in pts who also have MGD: Punctal occlusion.



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Aqueous Tear Deficiency → *Tear Film Instability* ← ***Evaporative Dry Eye***

Tear hyperosmolarity

Dry Eye Syndrome

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Aqueous Tear Deficiency + *Tear Film Instability* + ***Evaporative Dry Eye***

Tear hyperosmolarity

Dry Eye Syndrome

Finally: The *Cornea* book discusses several conditions that mimic DES in their presentation:

--Conjunctivochalasis
--Superior limbic keratoconjunctivitis (SLK)
--Floppy eyelid syndrome
--Nighttime lagophthalmos
--Parkinson's
--Mucous-membrane pemphigoid/OCP

Dry Eye Syndrome

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--P

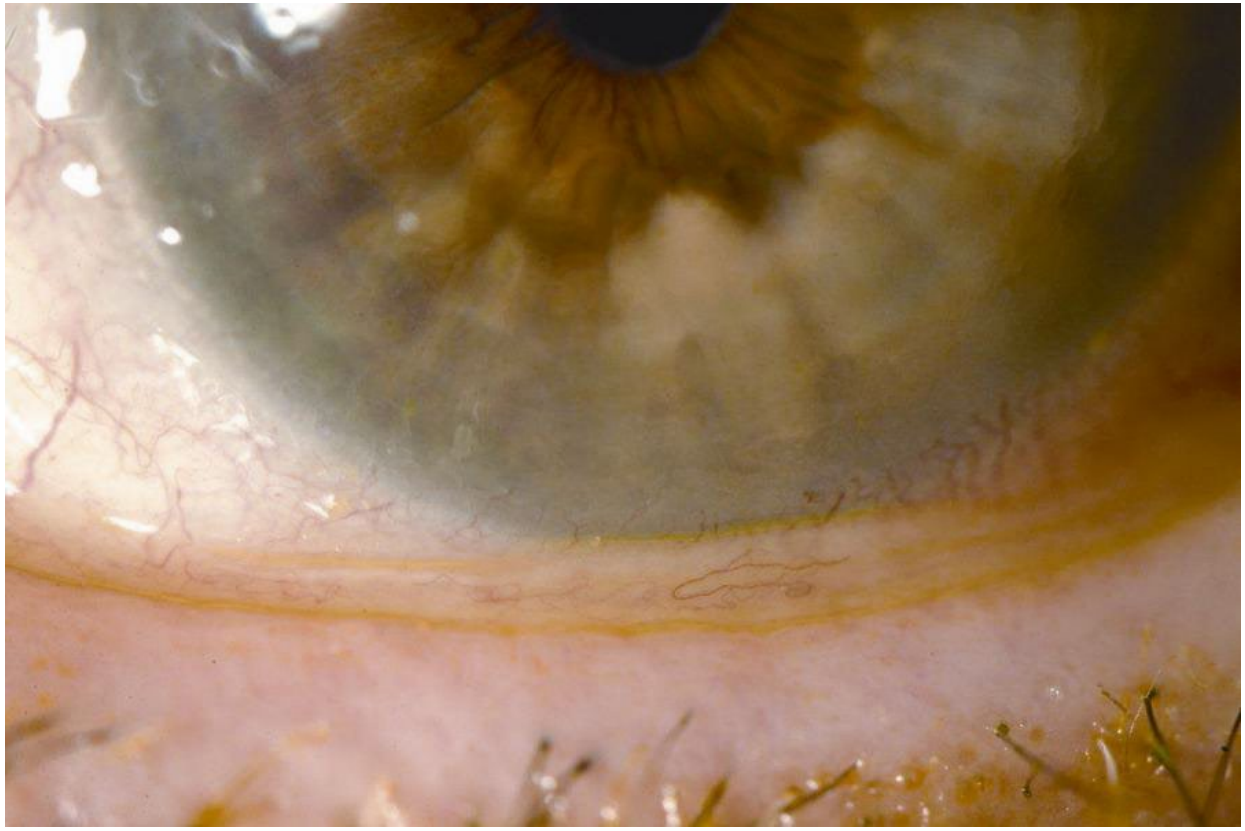
--N

--P

--M

Conj'chalasis refers to loose, redundant, nonedematous conj. It usually manifests as a 'fold' of conj draping on the lower-lid margin.

Dry Eye Syndrome



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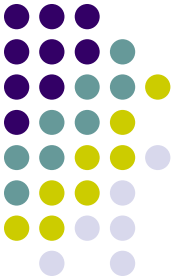
Dry Eye Syndrome



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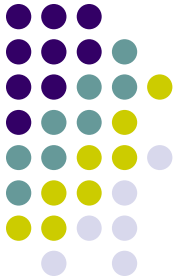
Conj'chalasis is managed in a stepwise manner. It's reasonable to start with ATs, antihistamines, steroids etc—although one of the characteristics of conj'chalasis is that it doesn't respond well to DES-tx maneuvers. Often, surgical intervention (in the form of excision or thermal cicatrization) is required for resolution.

Dry Eye Syndrome



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SLK is a chronic/recurrent inflammatory condition of the superior limbal cornea and adjacent conj. It is rare, and the vast majority of sufferers are women. SLK pts share many of the same symptoms as DES pts (FBS, red eyes, and tearing). However, the *signs* in the two differ enough to allow them to be distinguished from one another.

Dry Eye Syndrome

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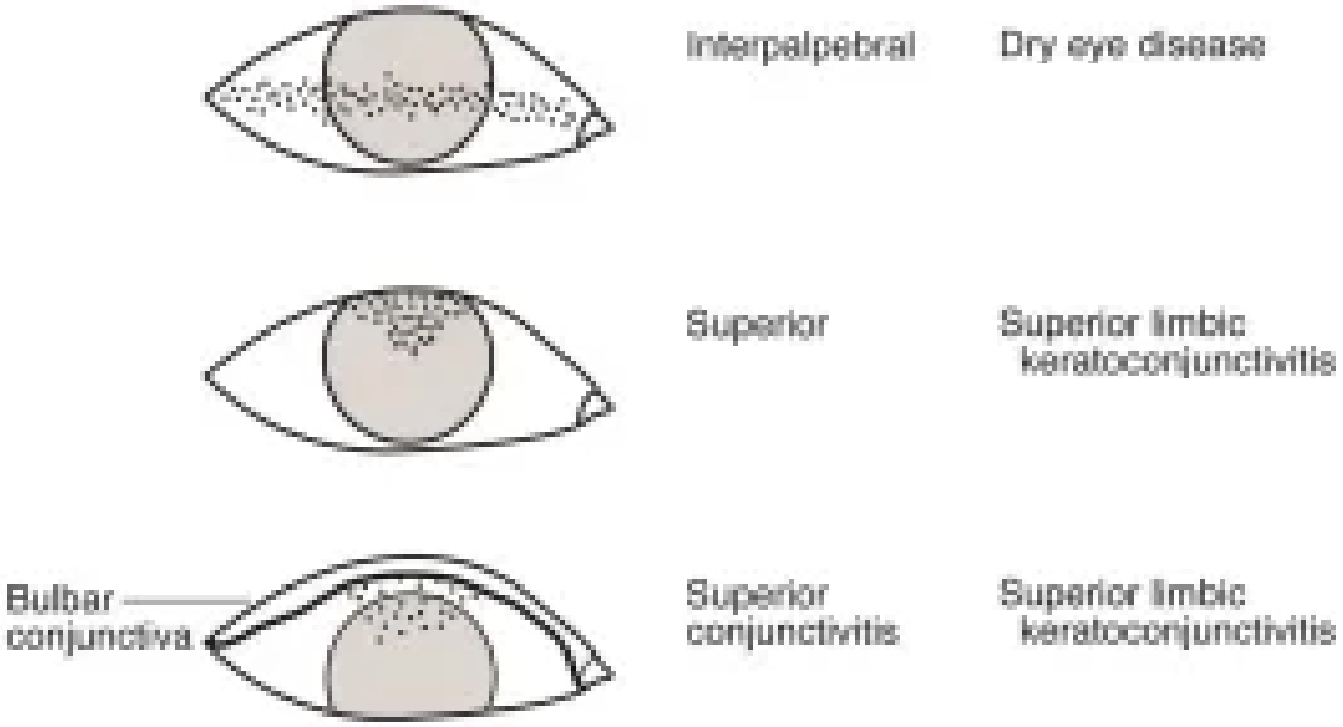
Dry Eye Syndrome

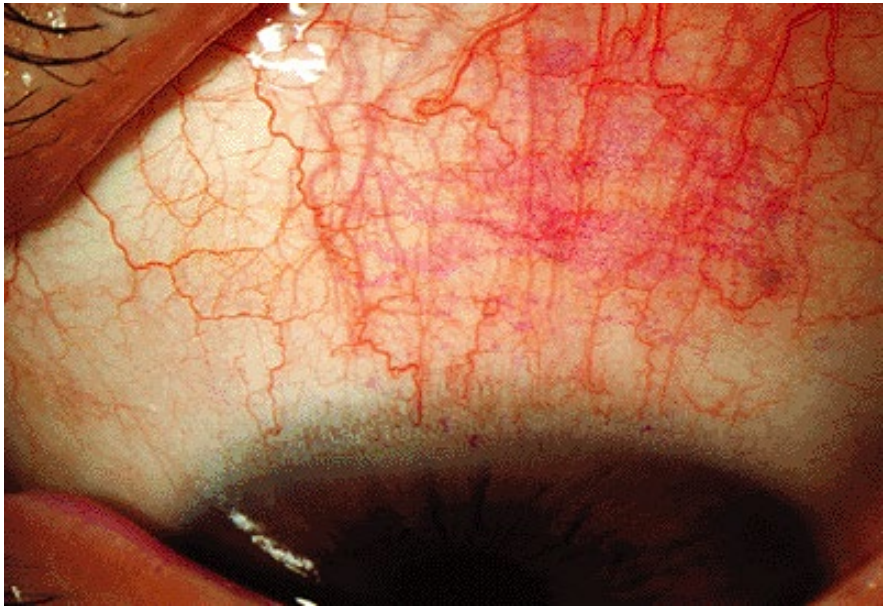


K and conj staining in SLK vs DES

Dry Eye Syndrome

SLK: Superior conj injection

Dry Eye Syndrome



Superior rose bengal staining

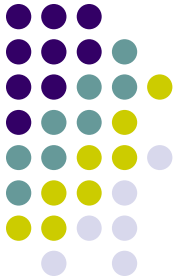
Superior lissamine green staining

SLK: Superior conj staining

Dry Eye Syndrome

SLK: Superior corneal filaments

Dry Eye Syndrome



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Dry Eye Syndrome

SLK: Superior tarsal conj papillary rxn

Dry Eye Syndrome

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The pathogenesis of the superior conj/cornea damage in SLK stems from excessive contact and rubbing between the upper lid and the superior conj/cornea. SLK pts have overly tight superior lids, usually as a result of orbital congestion stemming from thyroid eye dz—a classic (and highly testable) association with SLK.

Dry Eye Syndrome

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--**Floppy eyelid syndrome**

Floppy eyelid syndrome (FES) is a condition characterized by upper-lid laxity along with chronic inflammation of the ocular surface. FES pts complain of FBS and mucous discharge that are worse in the morning. The main risk factor for FES is obesity.

Dry Eye Syndrome

FES. Wow.

Dry Eye Syndrome

If you can’t tell, that’s an upper lid so lax it can be pinched like this

Dry Eye Syndrome

FES. Note the fine papillary rxn (another common finding) on the easily-everted lid

Dry Eye Syndrome

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The presumed pathogenic process in FES is that during sleep, their laxity allows the upper lids to evert in response to face-rubbing against a pillow while sleeping in the prone position. Lid eversion results in contact between the eye and the bedding which traumatizes the ocular epithelia.

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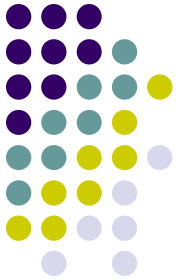
The presumed pathogenic process in FES is that during sleep, their laxity allows the upper lids to evert in response to face-rubbing against a pillow while sleeping in the prone position. Lid eversion results in contact between the eye and the bedding which traumatizes the ocular epithelia.

Initial management is conservative—ointment qHS, and preventing eversion by shielding the eye or taping them shut during sleep. If FES fails to respond to this, surgical tightening of the lax upper lid is in order. FES is strongly associated with obstructive sleep apnea, and <u>all FES pts should be evaluated for OSA.</u>



That's it! Go through this slide-set a couple of times (at least) until you feel like you have a handle on it. When you're ready, do slide-set *K48*, which covers this material in a Q&A format (and more detail).