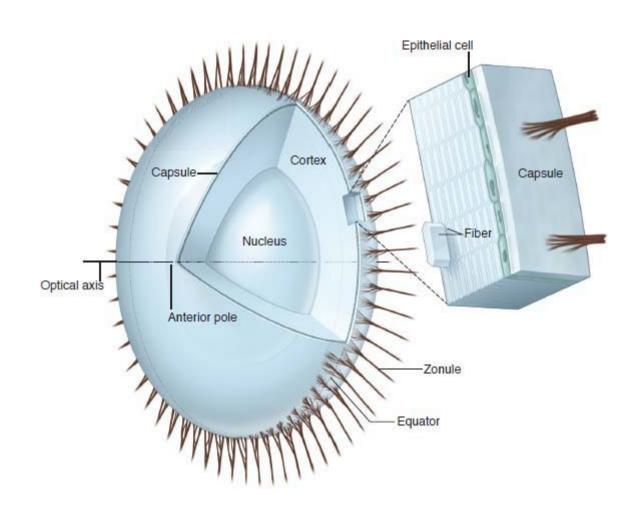
- Anatomy of the mature* lens
 - Capsule
 - Epithelium



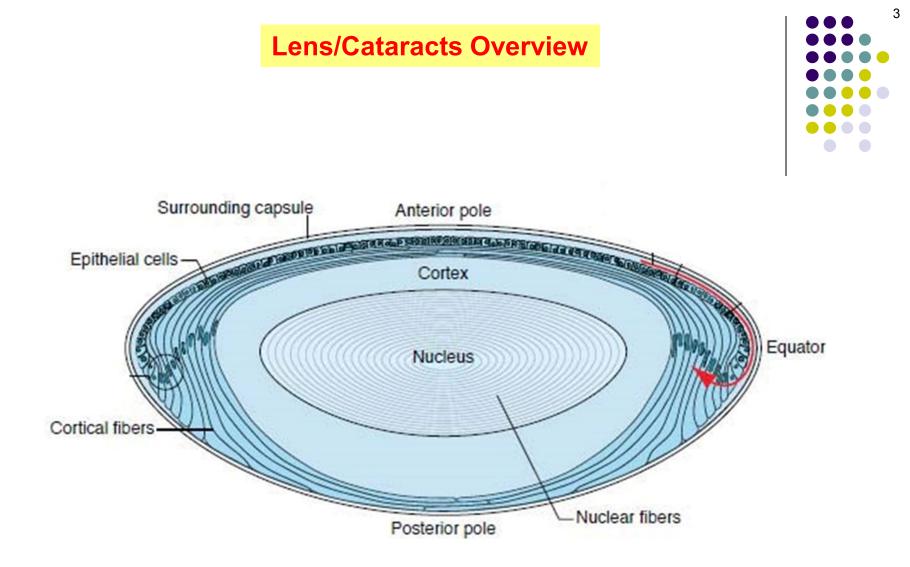
Nucleus
The human lens has five basic components

Cortex
Zonules

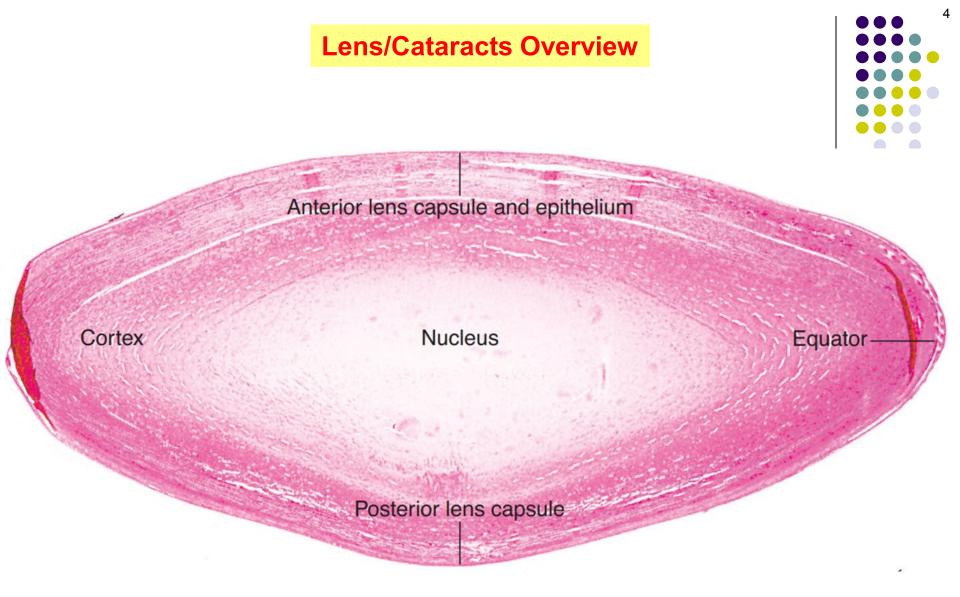
*'Mature' meaning 'postnatal;' **not** referring here to a 'mature' cataract



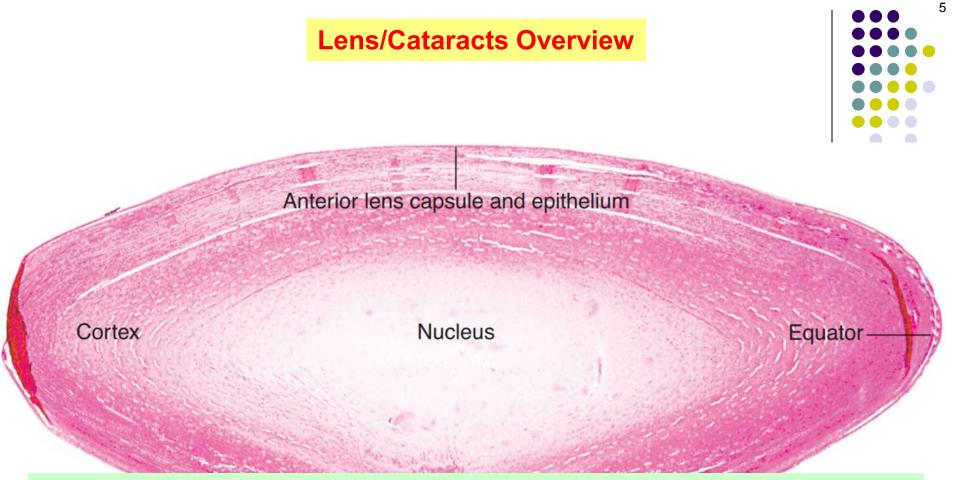
Basic components of the mature lens



Basic components of the mature lens: Another depiction



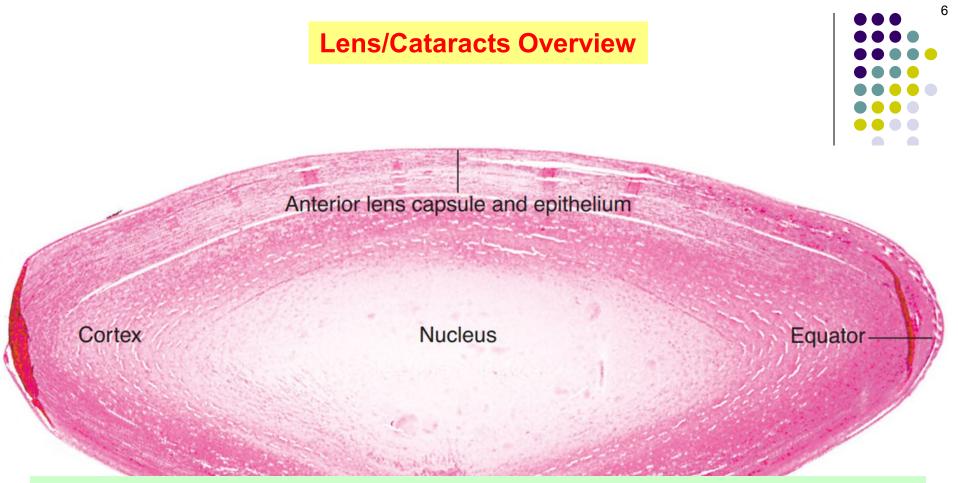
Basic components of the mature lens: Photomicrograph



Note on a nomenclature-related source of confusion if you've ever sat in on cataract surgery:

--During the case, the surgeon likely made reference at some point to the cataract's *epinucleus*, and you may be wondering why no such layer is depicted above.

--More puzzling, the surgeon may have used the term *cortex* in referring to a very thin, sticky layer adherent to the capsule—a tissue *nothing* like the cortex depicted here. *What's going on?*



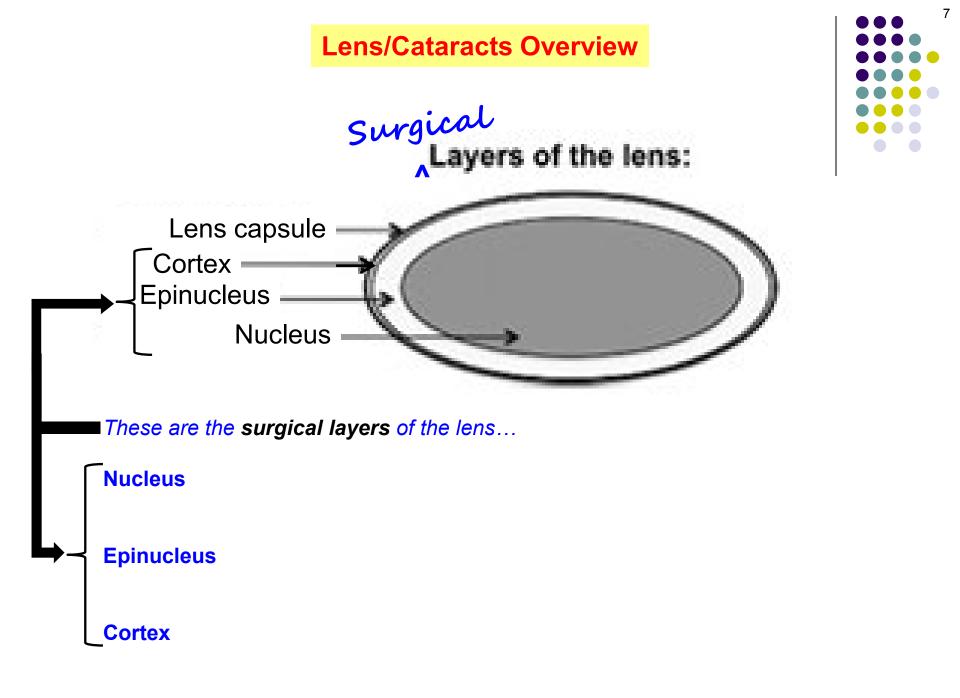
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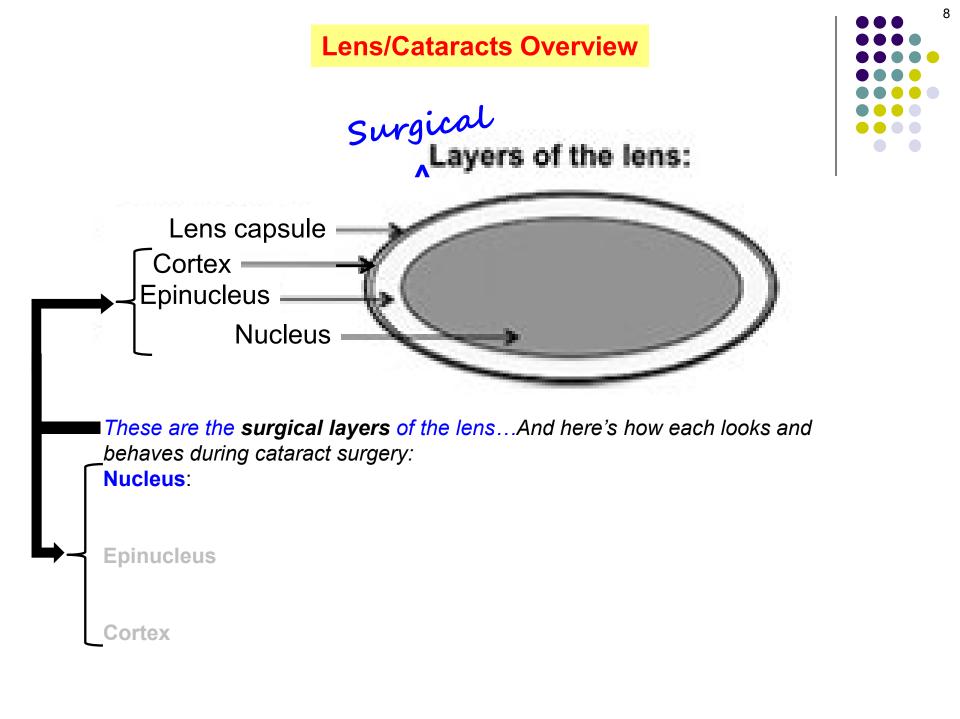
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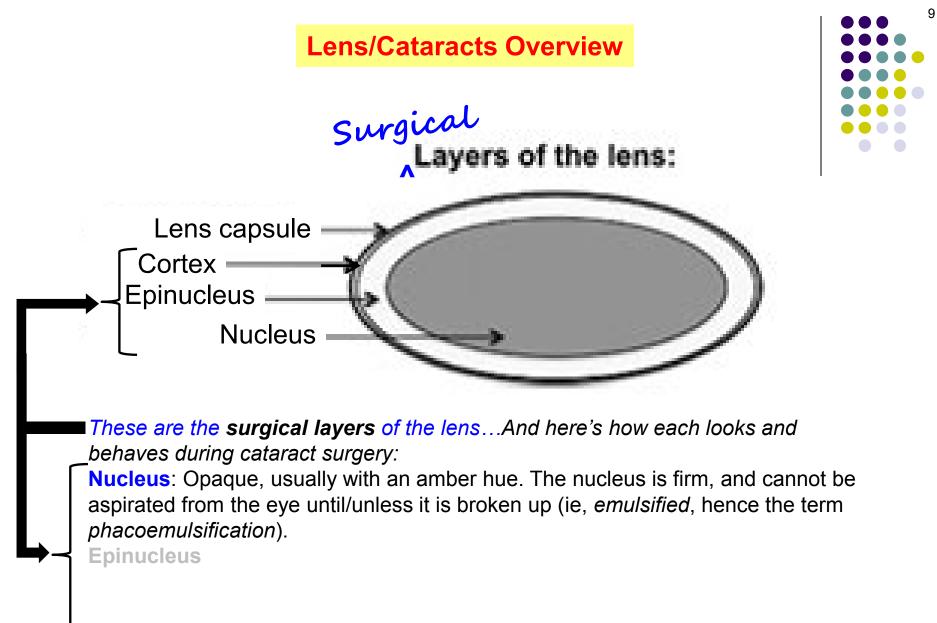
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What's going on?

What's going on is that the surgeon was referring to the *surgical* layers of the lens, not its *anatomic* layers. During cataract surgery, portions of the lens 'behave' very differently from one another, and it is with respect to these behaviors that surgeons conceptualize the makeup of the lens. (Cont)







Cortex

10 Lens/Cataracts Overview Surgical s of the lens: Lens capsule Cortex -Epinucleus Nucleus

These are the surgical layers of the lens... And here's how each looks and behaves during cataract surgery:

Nucleus: Opaque, usually with an amber hue. The nucleus is firm, and cannot be aspirated from the eye until/unless it is broken up (ie, *emulsified*, hence the term *phacoemulsification*).

Epinucleus: Clear to cloudy. The epinucleus is soft, and can be aspirated without emulsification (although emulsifying energy is often employed during epinucleus removal in order to make the process faster/more efficient).

Cortex

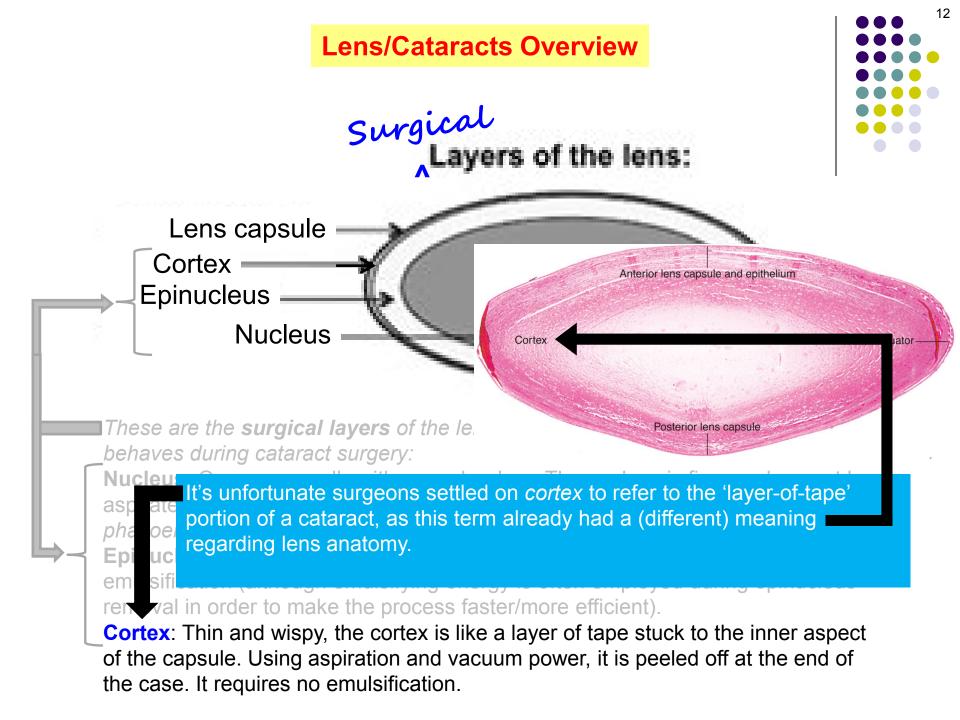
11 Lens/Cataracts Overview Surgical Layers of the lens: Lens capsule Cortex -Epinucleus Nucleus

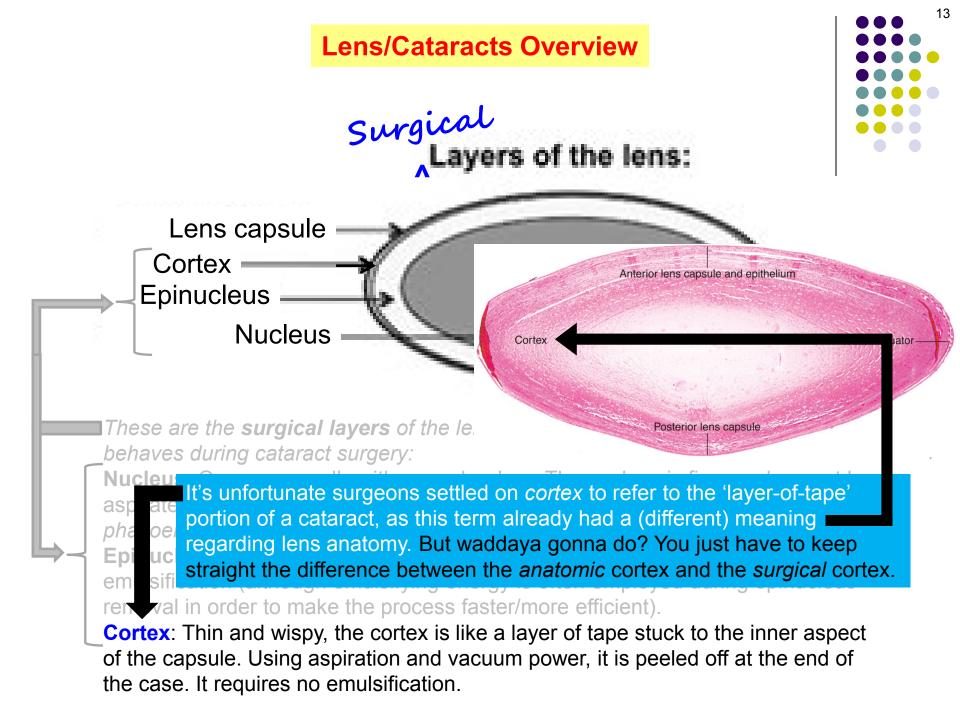
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Epinucleus: Clear to cloudy. The epinucleus is soft, and can be aspirated without emulsification (although emulsifying energy is often employed during epinucleus removal in order to make the process faster/more efficient).

Cortex: Thin and wispy, the cortex is like a layer of tape stuck to the inner aspect of the capsule. Using aspiration and vacuum power, it is peeled off at the end of the case. It requires no emulsification.





- Anatomy of the mature lens
 - Capsule
 - Type IV collagen
 - Epithelium

The capsule is comprised mainly of type IV collagen. (And that's all we'll have to say about that.)

- Nucleus
- Cortex
- Zonules



- Anatomy of the mature lens
 - Capsule
 - Type IV collagen
 - Epithelium

Nucleus

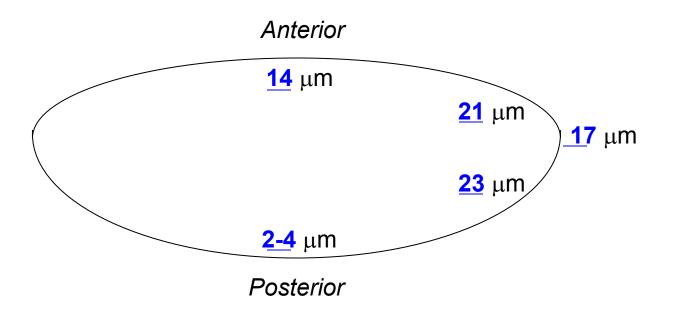
A weird-but-important point about the capsule: Despite the fact that it is on the outside of the lens, the capsule is the basement membrane of the underlying lens epithelium. (We will see how this counterintuitive histologic relationship comes to be when we look at lens embryology later.)

• Cortex

• Zonules

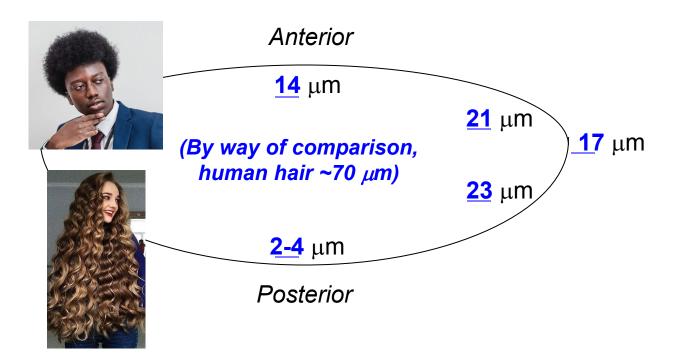


Lens capsule thickness has important clinical and surgical implications.





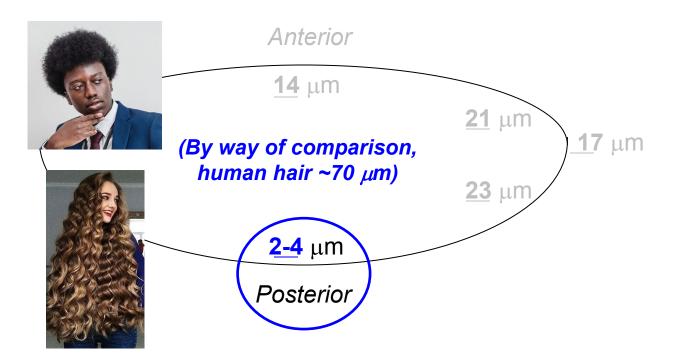
Lens capsule thickness has important clinical and surgical implications.



Key takeaway: While the entire capsule is thin...



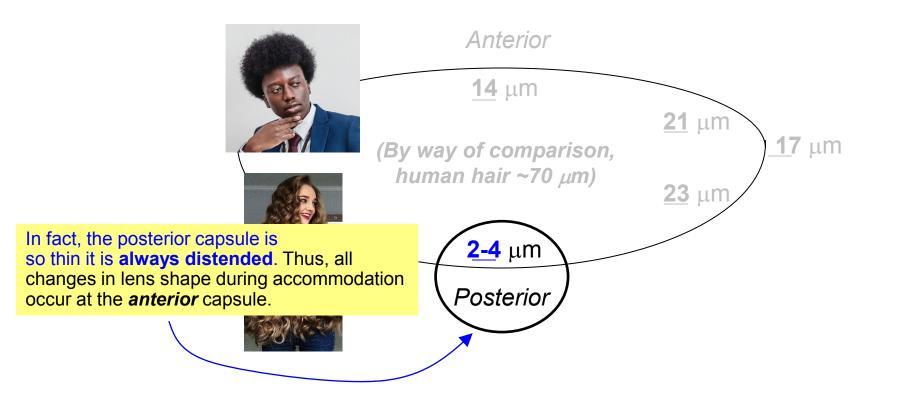
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Key takeaway: While the entire capsule is thin...Its posterior aspect is really thin

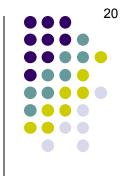


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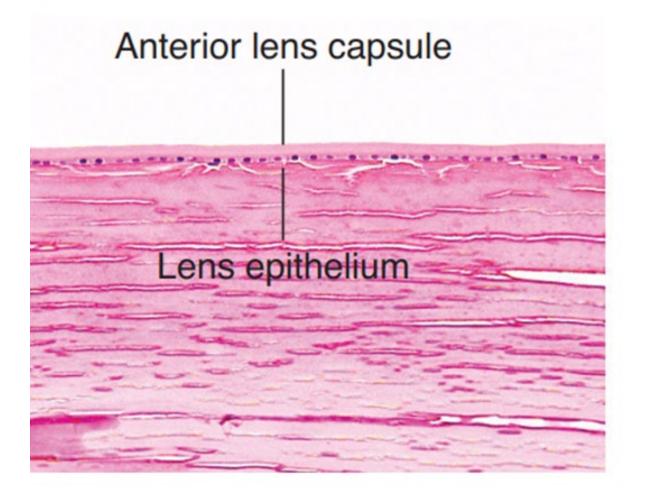


The lens of a 25-year-old woman demonstrated by Scheimpflug photography. The lens is in the nonaccommodative state in *A*, and accommodating in *B*. Note that the anterior radius of curvature is shortened (ie, the surface is more steeply curved) in *B*.

- Anatomy of the mature lens
 - Capsule
 - Type IV collagen
 - Epithelium
 - Single layer of cuboidal cells beneath anterior and equatorial capsule

- Nucleus
- Cortex
- Zonules





Lens epithelium

- Anatomy of the mature lens
 - Capsule
 - Type IV collagen
 - Epithelium
 - Single layer of cuboidal cells beneath anterior and equatorial capsule
 - *Nucleus* Lens epithelial cells are cuboidal in shape, and arranged in a single layer. Their presence is limited to the anterior and equatorial portions of the capsule.
 - Cortex

• Zonules



- Anatomy of the mature lens
 - Capsule
 - Type IV collagen
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 - Single layer of cuboidal cells beneath anterior and equatorial capsule
 - Metabolically active
 - Nucleus
 Lens epithelial cells are cuboidal in shape, and arranged in a single layer. Their presence is limited to the anterior and equatorial portions of the capsule. They are very metabolically active—far more so than the lens fiber cells that are deep to them.
 - Zonules



- Anatomy of the mature lens
 - Capsule
 - Type IV collagen
 - Epithelium
 - Single layer of cuboidal cells beneath anterior and equatorial capsule
 - Metabolically active; mitotically active
 - Nucleus

 Lens epithelial cells are cuboidal in shape, and arranged in a single layer. Their presence is limited to the anterior and equatorial portions of the capsule. They are very metabolically active—far more so than the lens fiber cells that are deep to them. Further, the epi cells are the only lens cells that are *mitotically* active.



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• *Nu* Let's sidebar to consider several important issues related to lens metabolism

Cortex

• Zonules



27

Re lens metabolism, bear these two facts in mind:



2)

Re lens metabolism, bear these two facts in mind:

1) Glucose

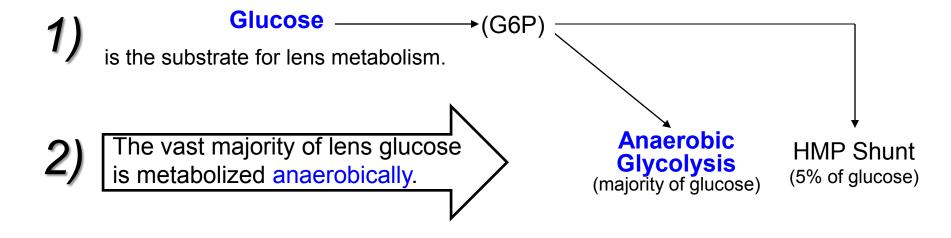
is the substrate for lens metabolism.



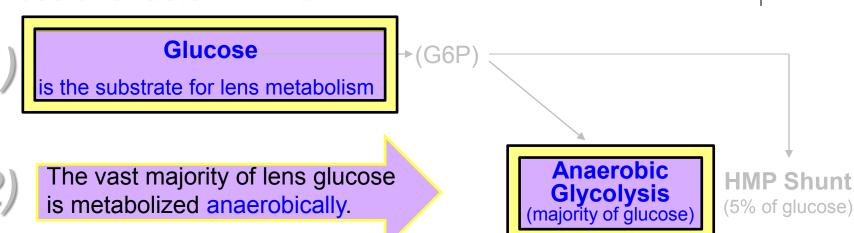


Re lens metabolism, bear these two facts in mind:





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The takeaway point: Lens metabolism is dependent upon the presence of *glucose*, **not oxygen.** Even in a zero-oxygen environment (such as can be created in a lab setting), a lens will remain transparent and viable so long as it has an adequate glucose supply.

►(G6F)

Anaerobic

Glycolysis (majority of glucose)

Re lens metabolism, bear these two facts in mind:

Glucose

is the substrate for lens metabolism

The vast majority of lens glucose

is metabolized anaerobically.

The takeaway point: Lens metabolism is dependent upon the presence of *glucose*, **not oxygen.** Even in a zero-oxygen environment (such as can be created in a lab setting), a lens will remain transparent and viable so long as it has an adequate glucose supply. However, in the reverse environmental situation—that is, one in which oxygen is abundant but glucose is absent—the lens will become cloudy and nonviable in a matter of hours.

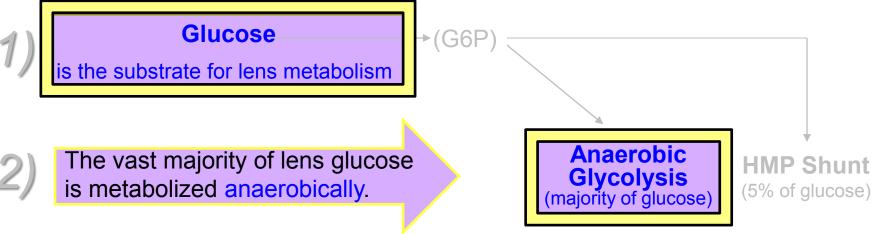
HMP Shunt

(5% of glucose)

31

Re lens metabolism, bear these two facts in mind:



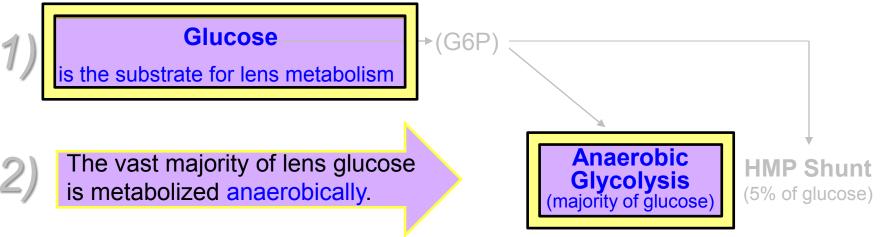


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A secondary (but important!) point concerns the metabolic consequence of anaerobic glycolysis. Because the lens is anaerobic-glycolysis dependent, it creates a great deal of **lactate**.

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34

Re lens metabolism, bear these two facts in mind:

Glucose

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A related metabolic challenge concerns getting the needed glucose (and other metabolic substrates) to its cells. Like every cell in the body, lens fibers must communicate with the 'outside world' to receive metabolic substrates and expurgate metabolic waste. Most (non-lens) cells accomplish this via the circulatory system.

(G6P

35

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G6F

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G6F

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37

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G6F

Now, communicating in this manner is not difficult for the lens epithelium and outermost cortical fibers, as the fluid is just on the other side of the capsule from them. But what of centrally located fibers? To connect *them* to the aqueous and/or vitreous, the lens employs a 'bucket brigade' in which metabolic substrates and waste products are passed cell-to-cell (via gap junctions) to get where they need to go. It should not surprise that *the density of gap junctions in lens-fiber cells is greater than that of any other cells in the body*.

A secondary (but important!) point concerns the metabolic consequence of anaerobic glycolysis. Because the lens is anaerobic-glycolysis dependent, it creates a great deal of **lactate**. The result is that aqueous-humor lactate concentration is **always higher** than its plasma counterpart.

The final metabolism-related issue: The maintenance of **lens transparency**. Lens transparency is exquisitely sensitive to the water content of the lens—a touch too much water and the lens scatters light; a touch more and it becomes opaque. Because of this, intralenticular water levels must be scrupulously maintained. And because water follows osmotic gradients, intralenticular ion levels are tightly controlled. In this regard, the most important ions are **sodium** and **potassium**.



K+

Na²⁺

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<u>Aqueous</u>

K⁺ Plasma(ish)

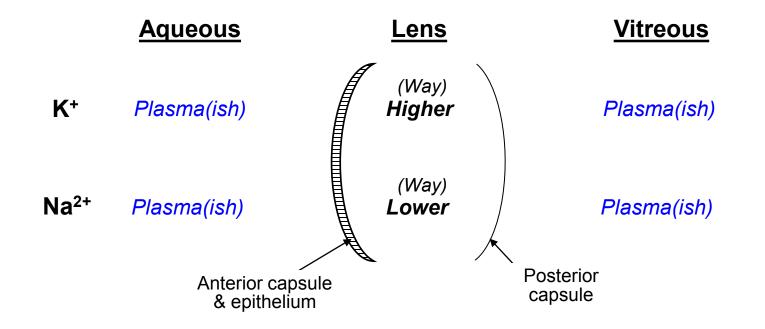
Na²⁺ Plasma(ish)

Vitreous

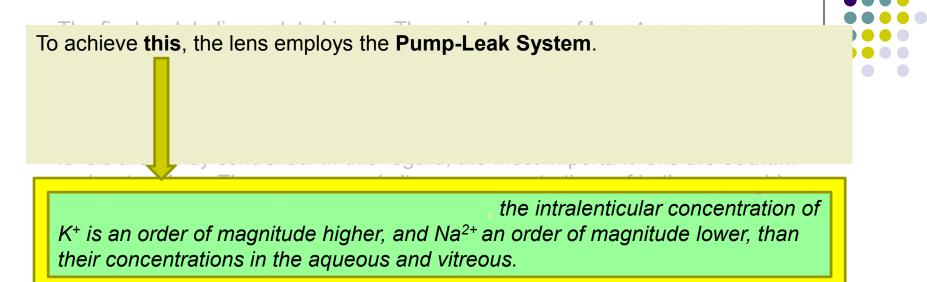
Plasma(ish)

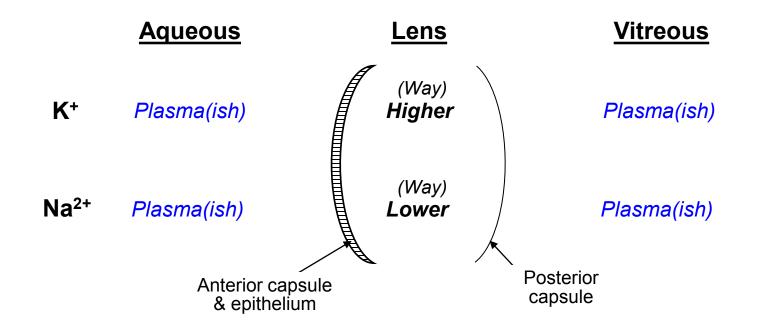
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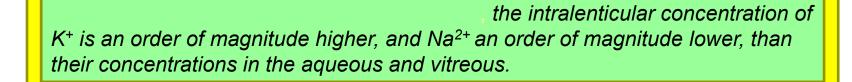


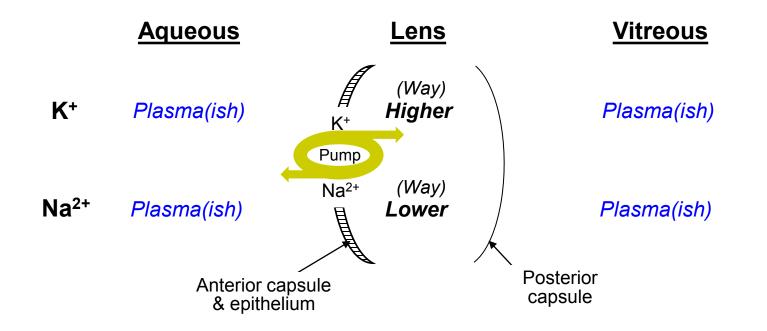






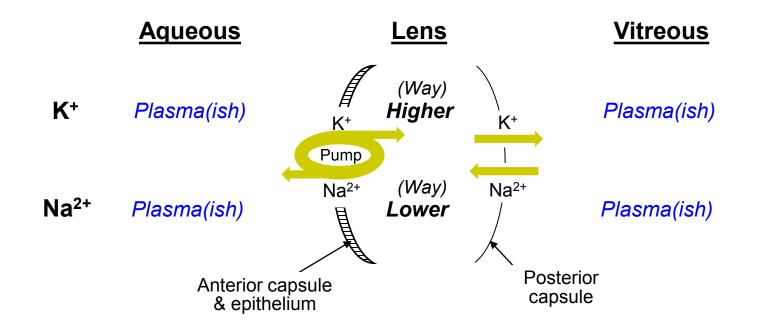
To achieve **this**, the lens employs the **Pump-Leak System**. Lens epithelial cells contain membrane-bound, ATP–powered, sodium-potassium transporters that drive (ie, they 'pump') Na²⁺ out of the lens and K⁺ into it.





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the intralenticular concentration of K⁺ *is an order of magnitude higher, and* Na²⁺ *an order of magnitude lower, than their concentrations in the aqueous and vitreous.*





- Anatomy of the mature lens
 - Capsule
 - Type IV collagen
 - Epithelium



- Single layer of cuboidal cells beneath anterior and equatorial capsule
- Metabolically active; mitotically active
- Give rise to all new lens fibers
- *Nt* After their creation, newly-minted epi cells migrate to the lens's equatorial region, where they begin the process of terminal differentiation into lens fibers. This process is characterized by 1) cell elongation, 2) the creation of new intracellular proteins called *crystallins*, and 3) the loss of intracellular organelles.
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 - Capsule
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- Give rise to all new lens fibers

Nucleus

- Older fibers densely packed in central lens
- Cortex

Zonules

The **nucleus** consists of fibers that are either OGs (ie, those created prenatally) or were created prior to age 20 years or so, and thus have been around long enough to get packed down into the dense structure we call the nucleus.

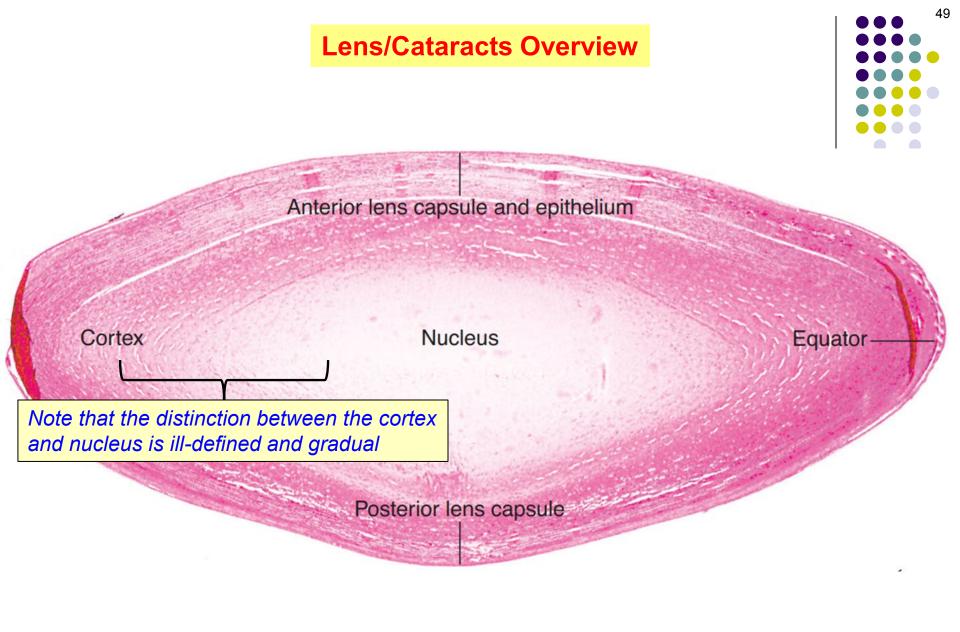
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Zonules

Newer fibers between nucleus and capsule/epithelium

Fibers created after age 20 will live out their lives in the cortex.

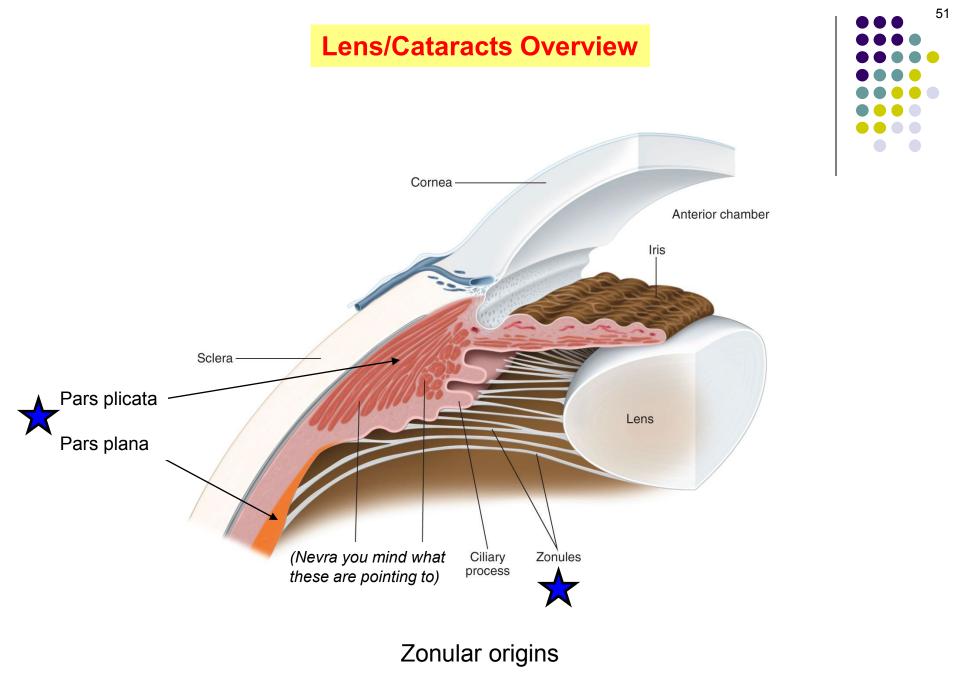




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 - Zonules
 - Originate from the basal lamina of the nonpigmented epithelium of the pars plana and pars plicata of the ciliary body

The **zonules** support the lens, and transmit to it the forces that produce accommodation. (Their origins are as described above.)



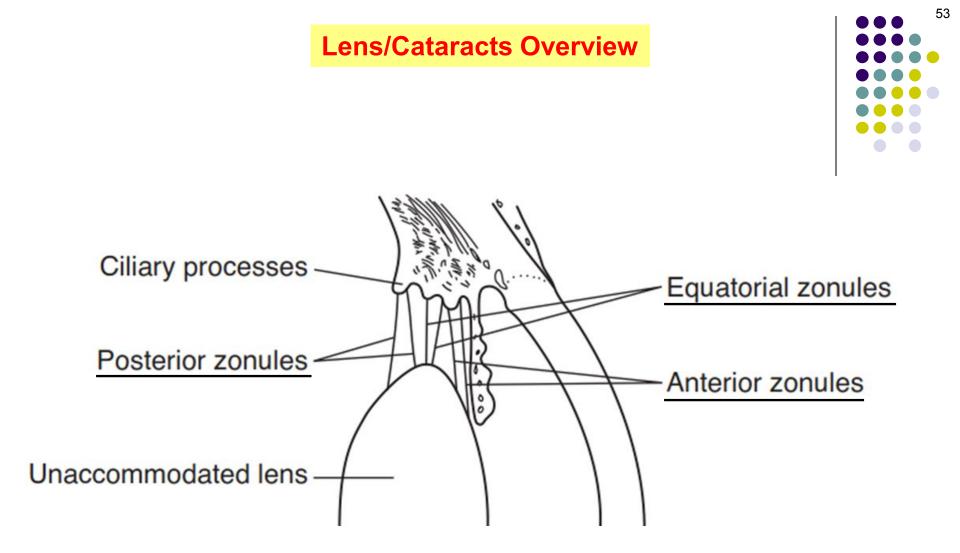


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 - Three sets of fibers:
 - Anterior
 - Equatorial
 - **Posterior**

three sets of fibers: Anterior, equatorial, and posterior.





Zonular insertions on the lens

- Anatomy of the mature lens
 - Capsule
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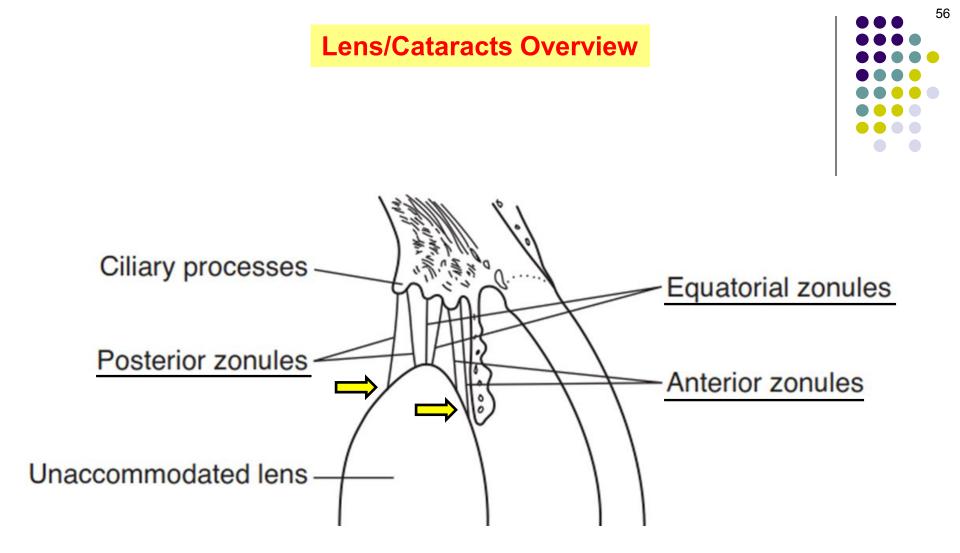
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 - Anterior
 - Equatorial
 - Posterior

Based on their site of insertion, there are three sets of fibers: Anterior, equatorial, and posterior. The equatorial fibers regress, leaving only the anterior and posterior sets.

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 - Three sets of fibers:
 - Anterior: Insert more centrally
 - Equatorial
 - Posterior: Insert less centrally

three sets of fibers: Anterior, equatorial, and posterior. The equatorial fibers regress, leaving only the anterior and posterior sets. The anterior fibers extend farther (ie, insert more centrally) than do the posterior ones.





Zonular insertions on the lens

(Note the relative locations of the insertions of the anterior vs posterior zonules—the anterior insert more centrally than do the posterior)



Lens measurements

- Birth: 6.4 mm equatorial, 3.5 mm anteroposterior
- Adult: 9-10 mm equatorial, 5.0 mm anteroposterior



Lens measurements

- Birth: 6.4 mm equatorial, 3.5 mm anteroposterior
- Adult: 9-10 mm equatorial, 5.0 mm anteroposterior

Probably good enough to ballpark the lens as 6x3 mm at birth and 10x5 mm late in life. (Note that the diameter and depth maintain about a 2:1 relationship.) As the lens never stops creating new fibers, it never stops growing.



- Lens measurements
 - Birth: 6.4 mm equatorial, 3.5 mm anteroposterior
 - Adult: 9-10 mm equatorial, 5.0 mm anteroposterior
- With age...
 - Lens curvature increases $\rightarrow \uparrow$ refractive power
 - Refractive index decreases $\rightarrow \downarrow$ refractive power



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The *Lens* book is confusing re what happens to the refractive index and refractive status of eyes as we age. The above is straight from Chapter 2. However, in Chapter 5 it states that NSCs "cause an **increase** in the refractive index" (emphasis mine) and a "myopic shift." (It goes on to say hyperopic shifts are "rare.") What makes this confusing is that NSCs are very much age-related. Caveat emptor on this score.

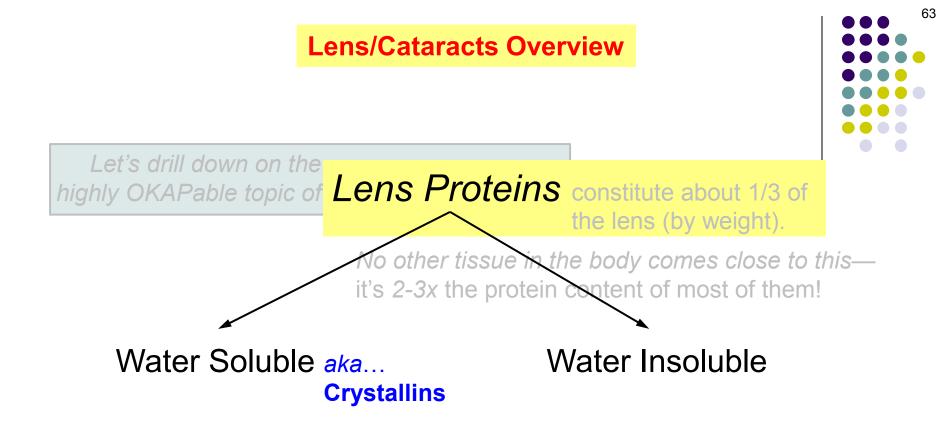
Let's drill down on the highly OKAPable topic of Lens Proteins



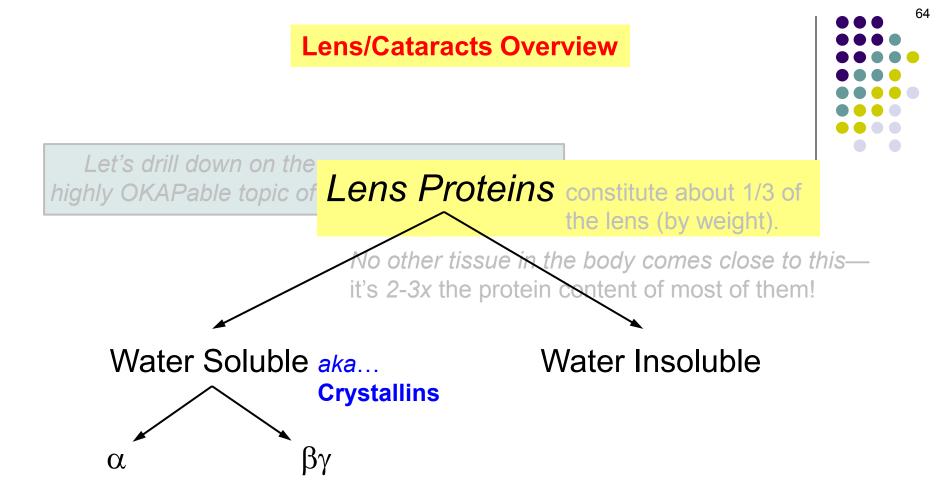


Let's drill down on the highly OKAPable topic of Lens Proteins constitute about 1/3 of the lens (by weight).

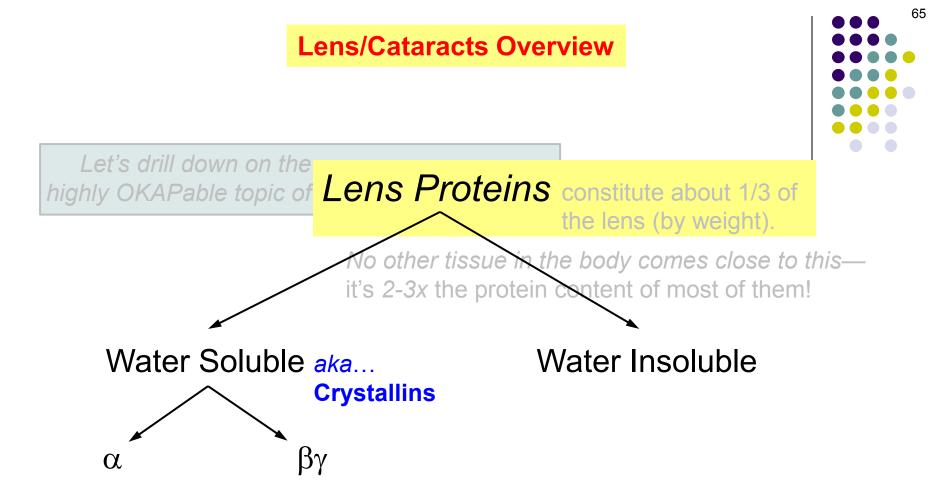
> *No other tissue in the body comes close to this* it's 2-3*x* the protein content of most of them!



Lens proteins come in one of two basic types: Water-soluble (aka the *crystallins*), and water-**in**soluble.



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Let's drill down on the highly OKAPable topic of Lens Proteins constitute about 1/3 of the lens (by weight).

> No other tissue in the body comes close to this it's 2-3x the protein content of most of them!

Next we will look at the **development/embryology** of the lens



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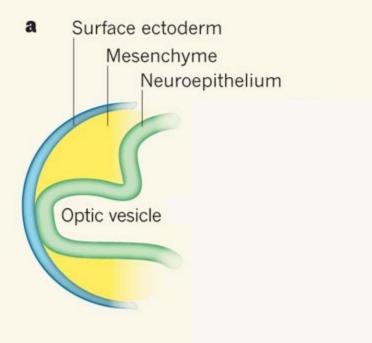


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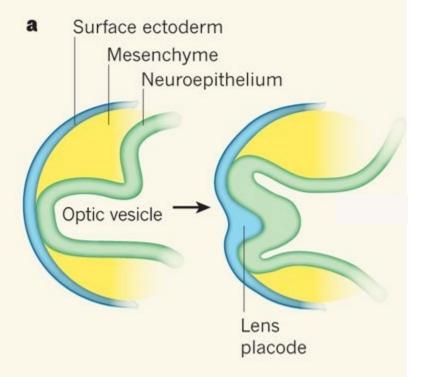




Re surface ectoderm and lens formation:

(Glance at this, then keep going to see the points being made)

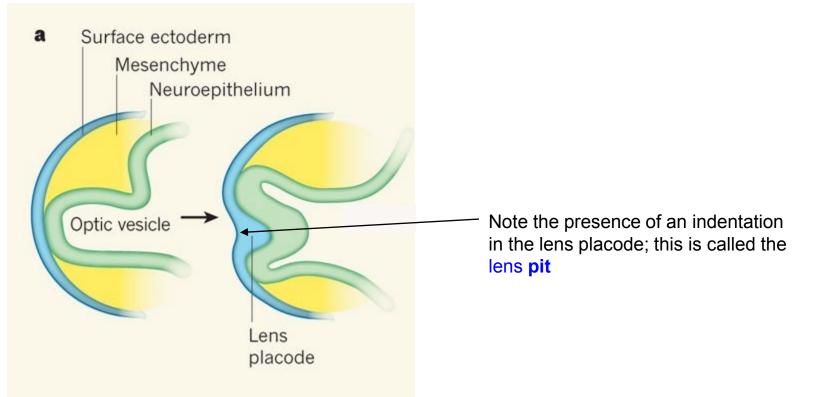




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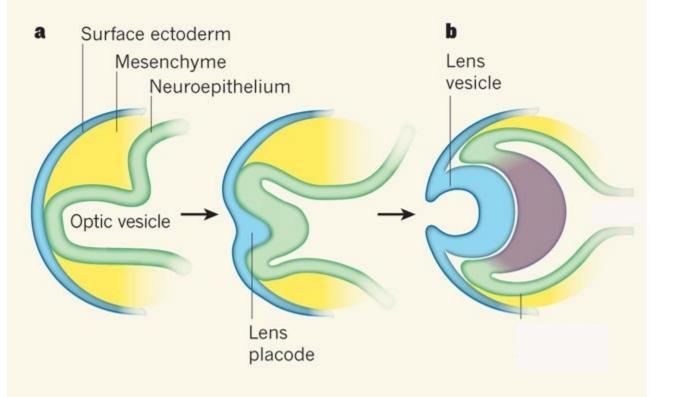
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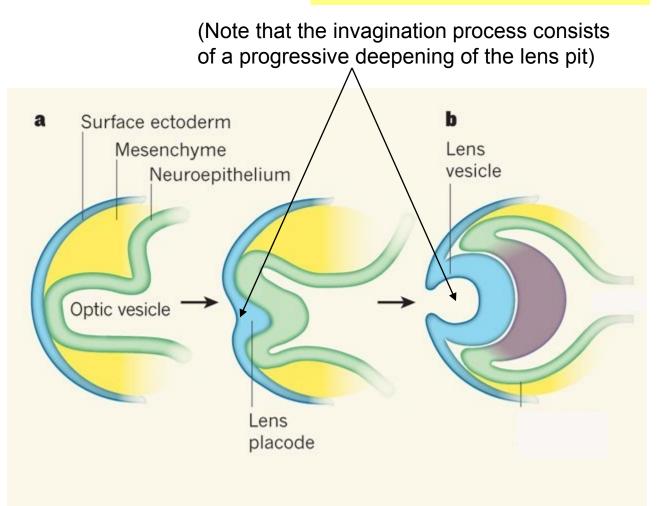


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--A portion of surface ectoderm thickens to form the lens placode

--The placode invaginates to form the lens vesicle





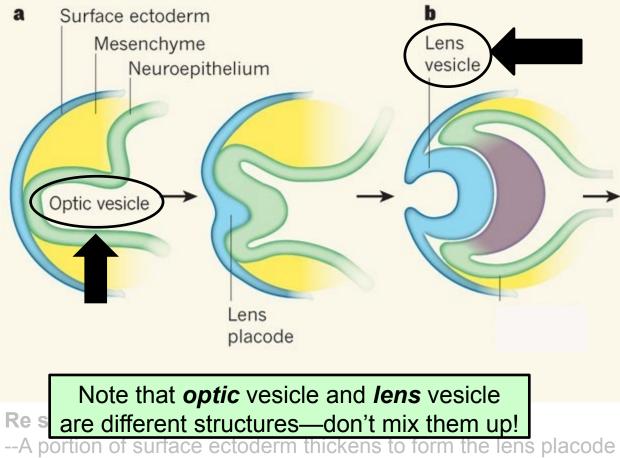
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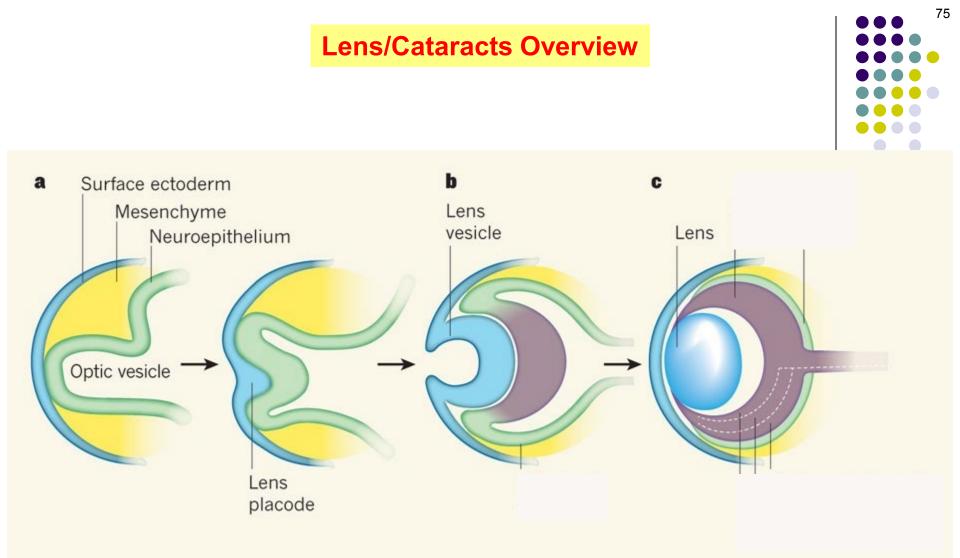
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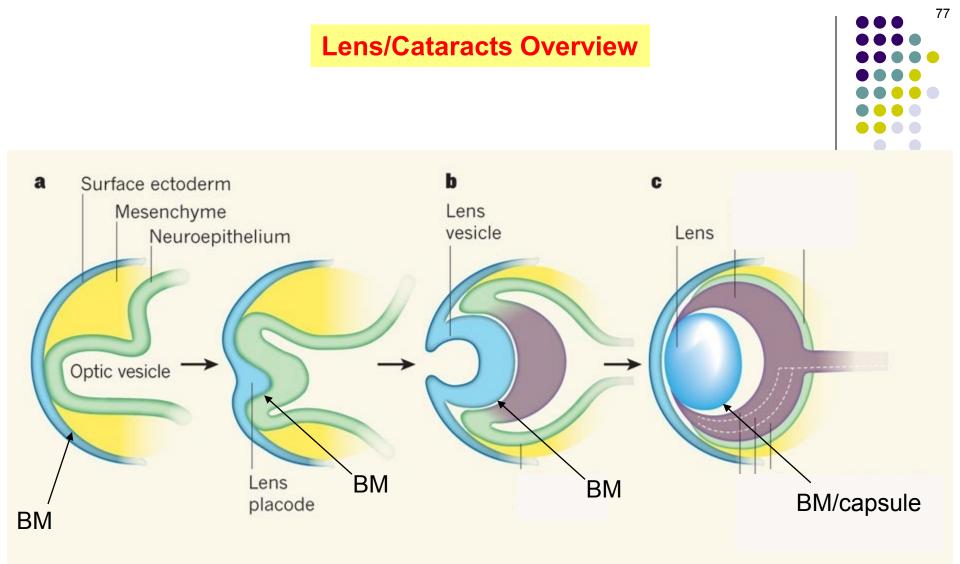


Re surface ectoderm and lens formation:

- --A portion of surface ectoderm thickens to form the lens placode
- --The placode invaginates to form the lens vesicle
- --The lens vesicle goes on to form (eventually; there are intervening steps) the mature lens.



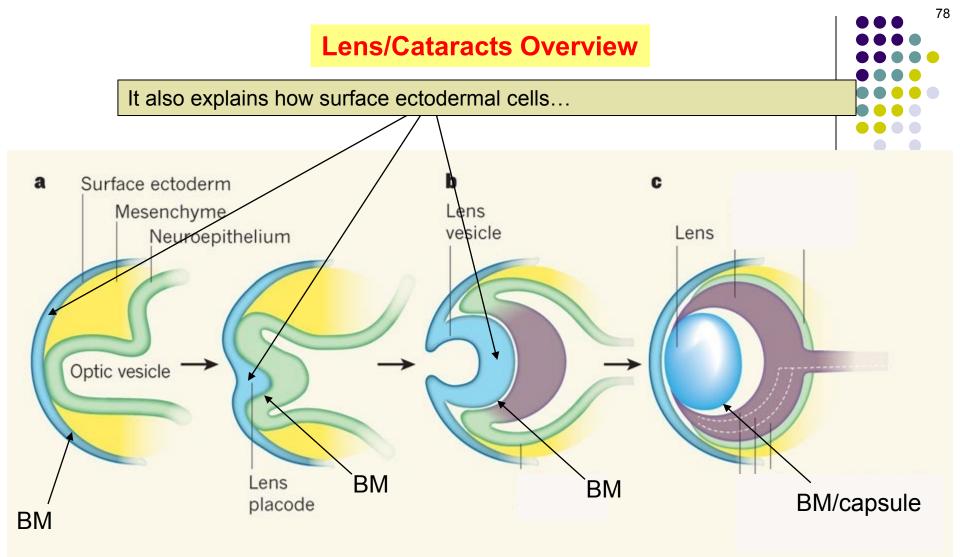
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Res The invagination process leads to the weird result of a structure (the lens) that has its epithelium on its *inside* and its basement membrane on its *outside*.

--The placode invaginates to form the lens vesicle

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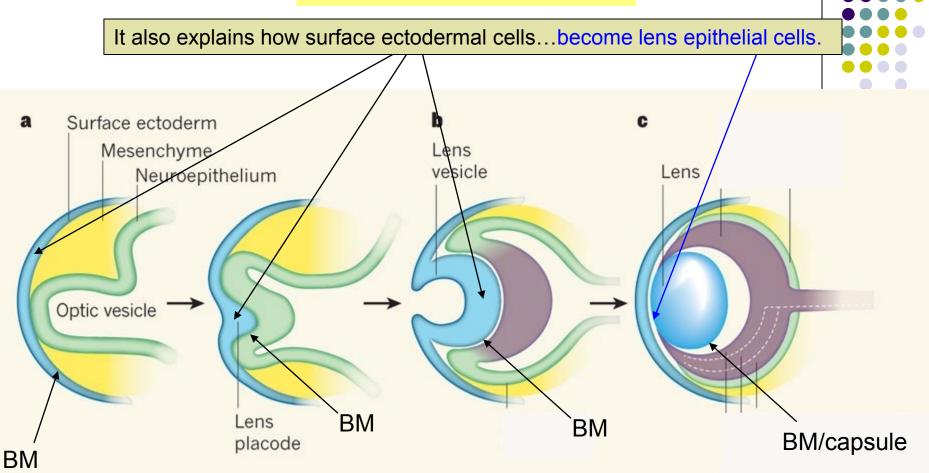


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79



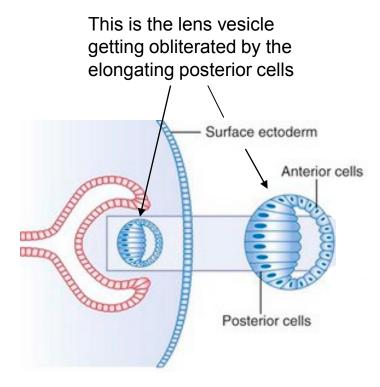
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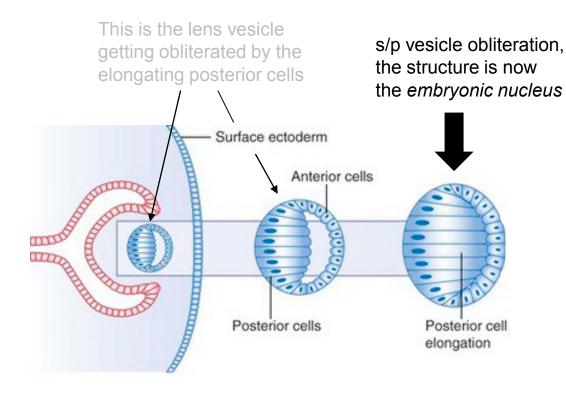


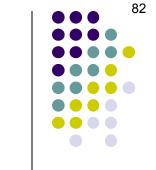
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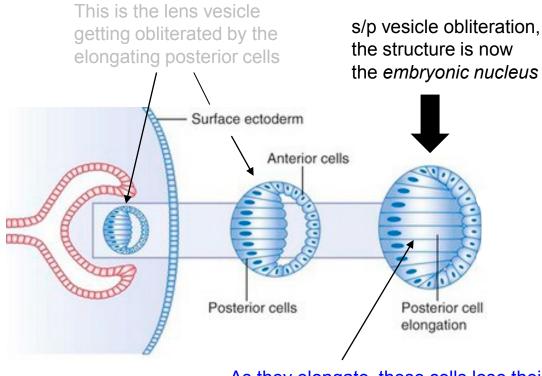


Posterior cells of the lens vesicle elongate to obliterate the vesicle's lumen, thus creating the embryonic nucleus





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As they elongate, these cells lose their organelles and thereby transform into 'fibers.'

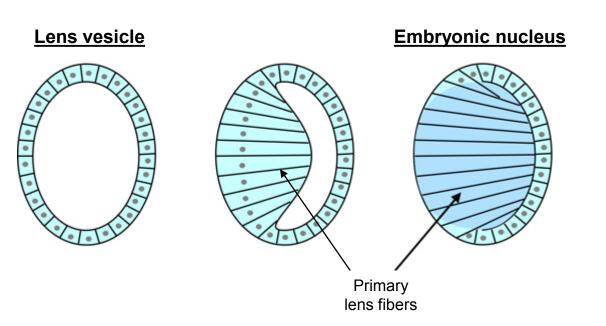
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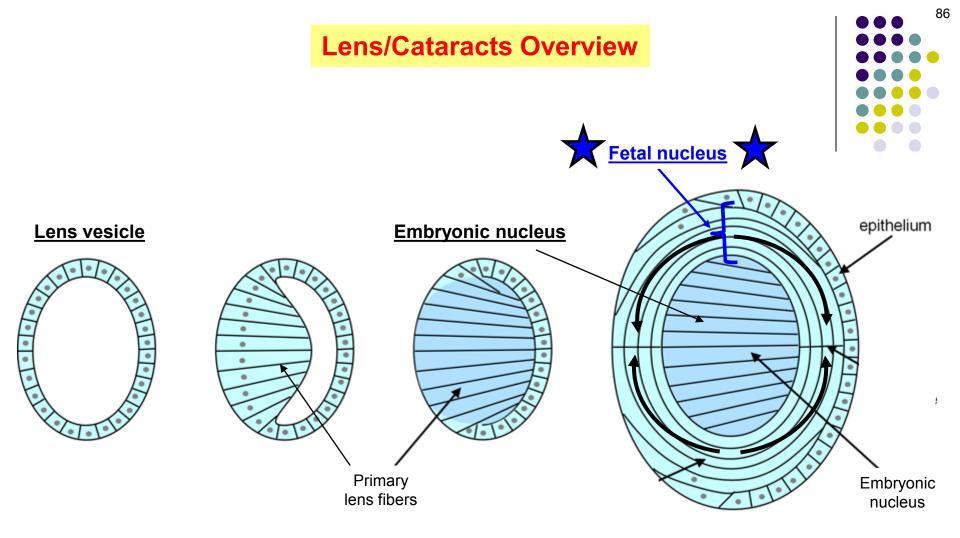


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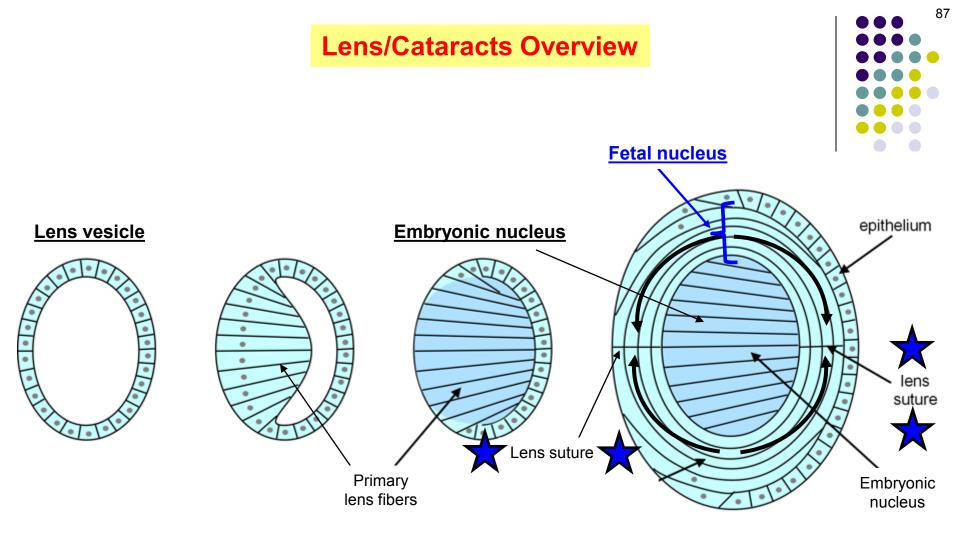




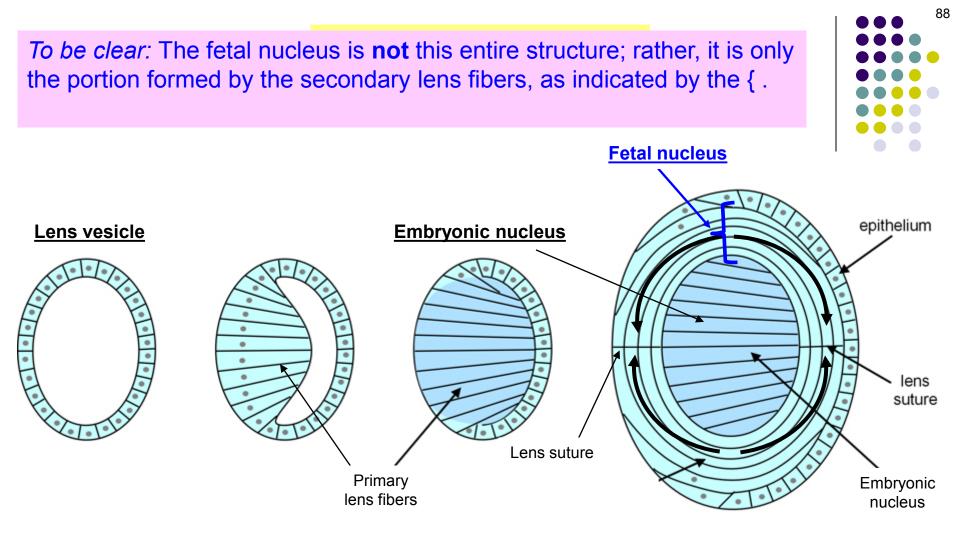
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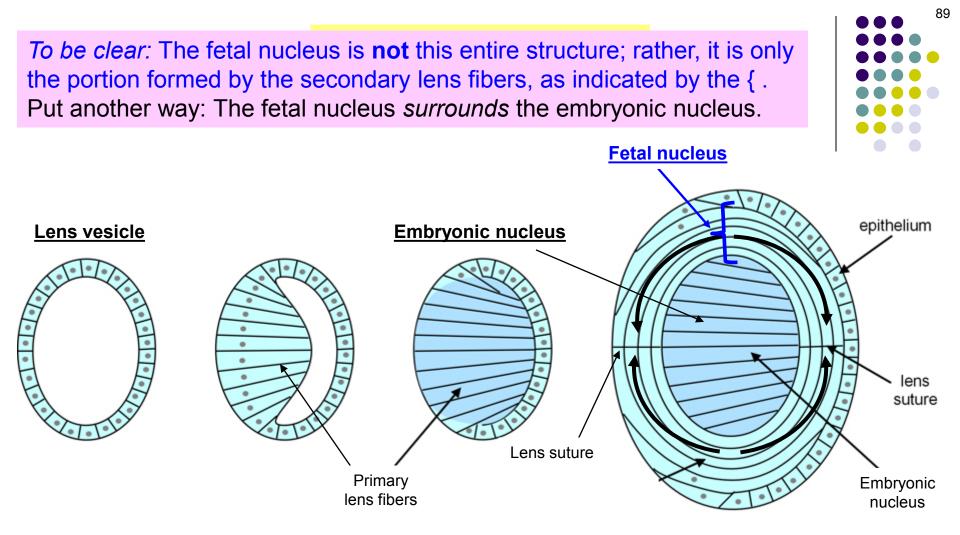
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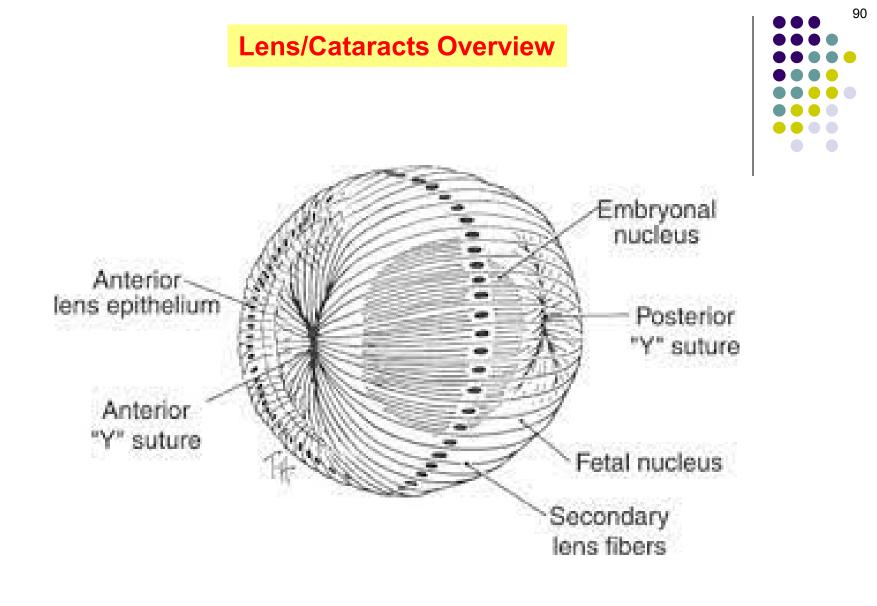
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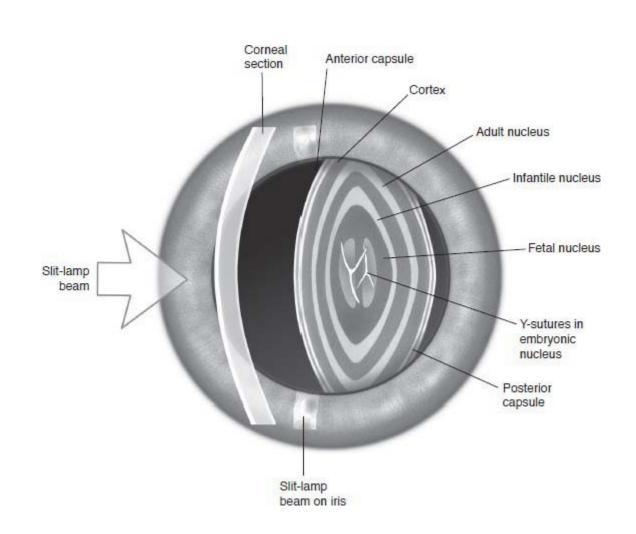


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Y sutures as they might be seen at the slit lamp





Now let's look at the fetal vasculature of the lens

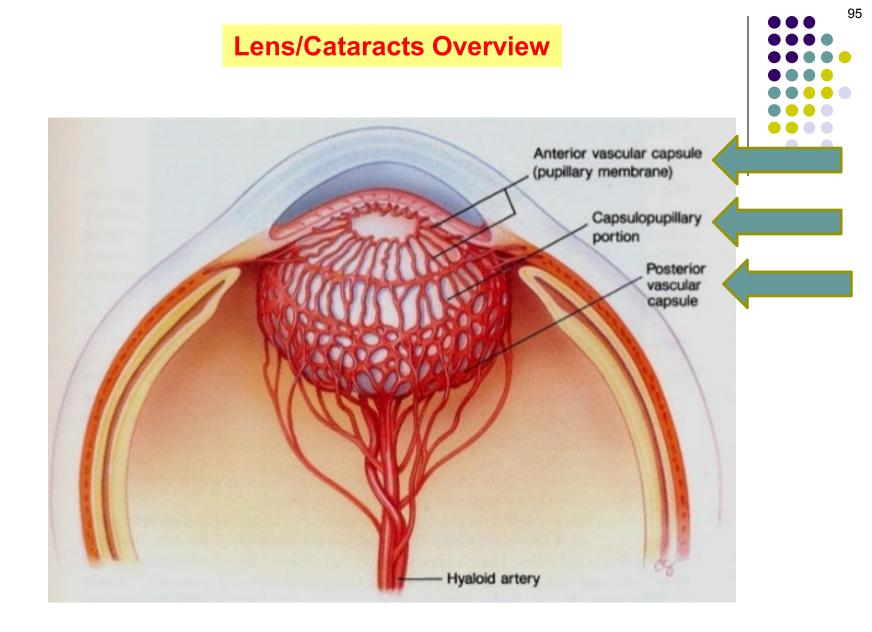


 The vascular supply encapsulating the developing lens is called the tunica vasculosa lentis.

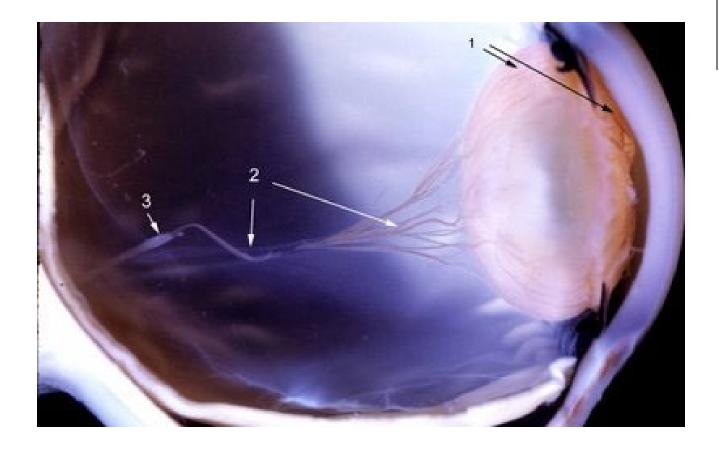


- The vascular supply encapsulating the developing lens is called the tunica vasculosa lentis.
 It has three sections:
 - 1) The posterior vascular capsule

2) The anterior vascular capsule



Tunica vasculosa lentis



In the eye of this very premature infant, the **tunica vasculosa lentis** surrounds the lens (arrows 1).

(We'll get to Arrows 2 and 3 shortly)

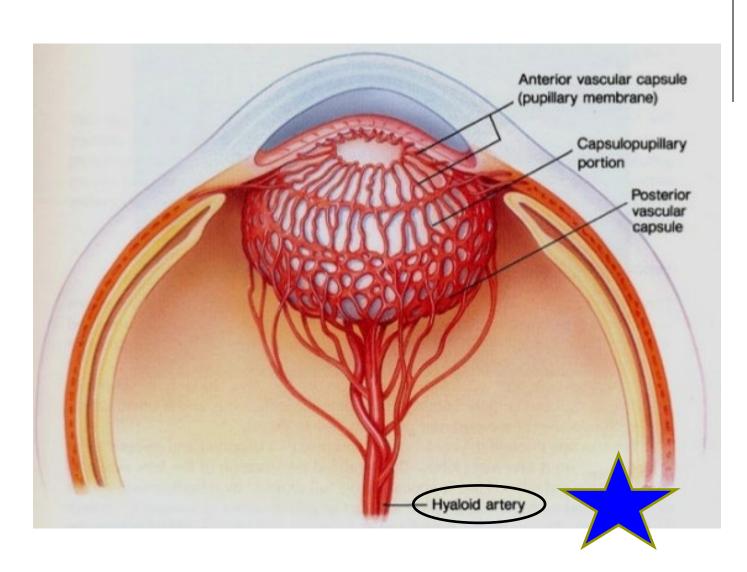




- The vascular supply encapsulating the developing lens is called the tunica vasculosa lentis.
 It has three sections:
 - 1) The *posterior vascular capsule* arises from the hyaloid artery

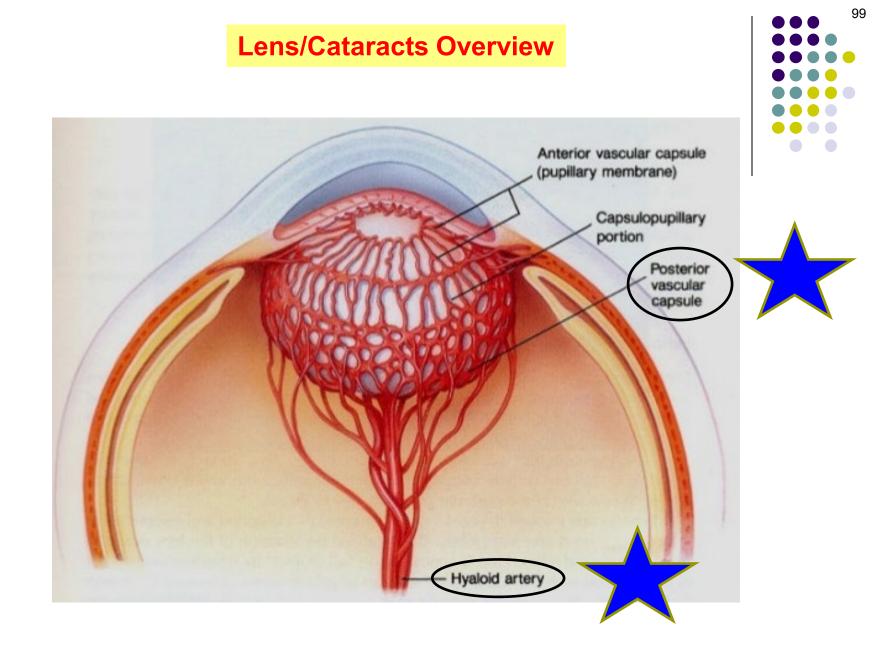
The **hyaloid artery** runs from the optic nerve head to the back of the fetal lens.

2) The anterior vascul

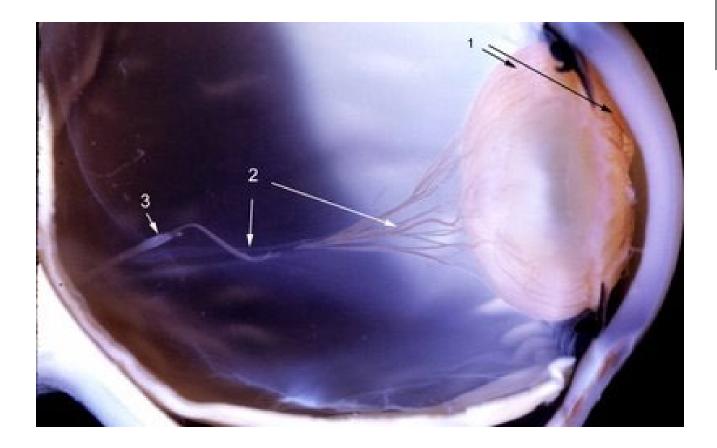


Tunica vasculosa lentis: The hyaloid artery...





Tunica vasculosa lentis: The hyaloid artery...forming the posterior vascular capsule



In the eye of this very premature infant, the **tunica vasculosa lentis** surrounds the lens (arrows 1). It is contiguous with the hyaloid artery and its branches (arrow 2).





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 - A common, clinically insignificant remnant is the Mittendorf dot

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2) The anterior va The Mittendorf dot presents as a small white dot on the posterior capsule of the lens. It is an extremely common finding.





Retroillumination



Direct illumination

Mittendorf dot



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² In PFV (*persistent fetal vasculature*; aka *persistent hyperplastic primary vitreous*, PHPV), the remnant posterior vascular capsule forms a retrolental fibrovascular membrane.



PFV: Retrolental fibrovascular membrane





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2 In PFV (*persistent fetal vasculature*; aka *persistent hyperplastic primary vitreous*, PHPV), the remnant posterior vascular capsule forms a retrolental fibrovascular membrane. This membrane induces a variety of sight-threatening conditions including cataract, progressive AC shallowing with subsequent glaucoma, and retinal detachment.

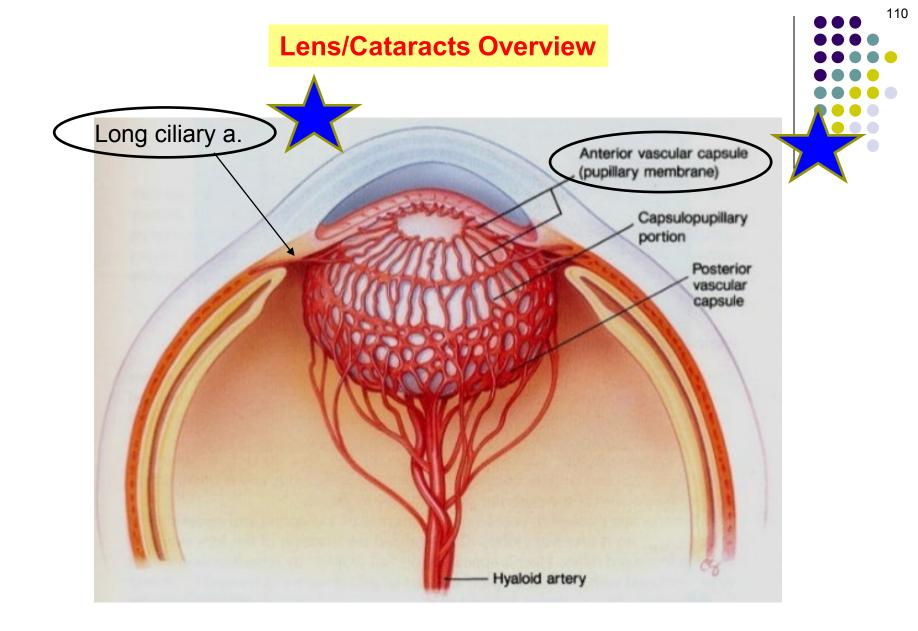


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- 2) The *anterior vascular capsule* derives from the long ciliary arteries

3) The capsulopupillary portion



Tunica vasculosa lentis: Anterior vascular capsule

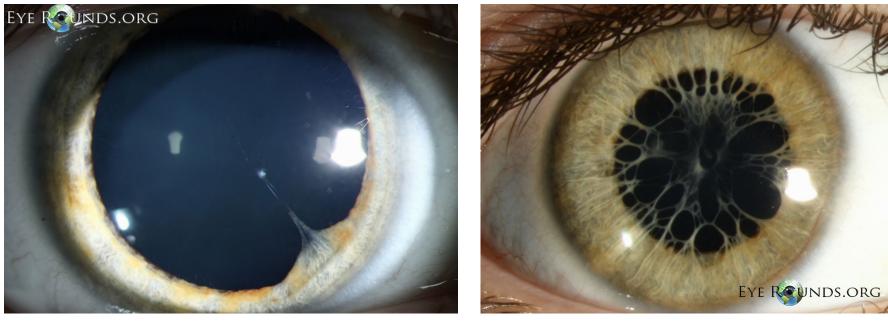


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Trivial case

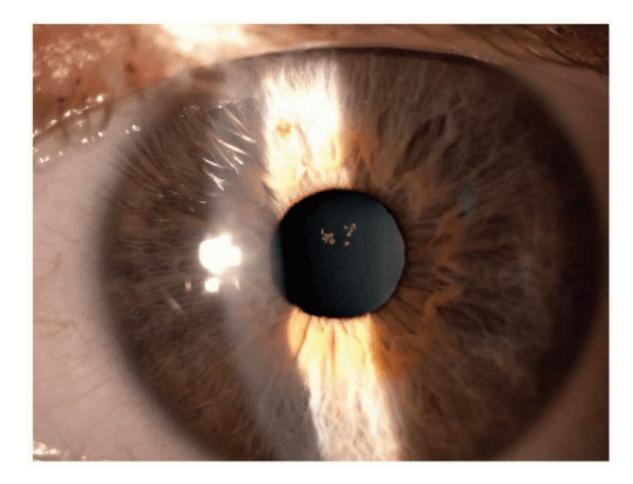
Hey now

Persistent pupillary membrane



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 - Another common remnant is the epicapsular star, colloquially referred to as 'chicken feet' on the anterior capsule

3) The capsulopupillary portion

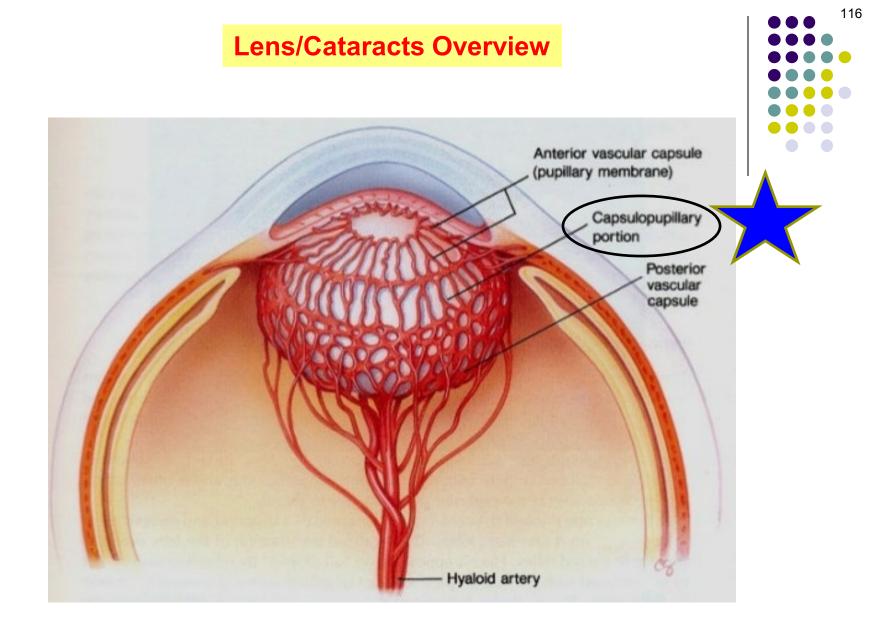




Epicapsular star



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 - 3) The *capsulopupillary portion* anastomoses the anterior and posterior sections of the tunica



Tunica vasculosa lentis: Capsulopupillary portion



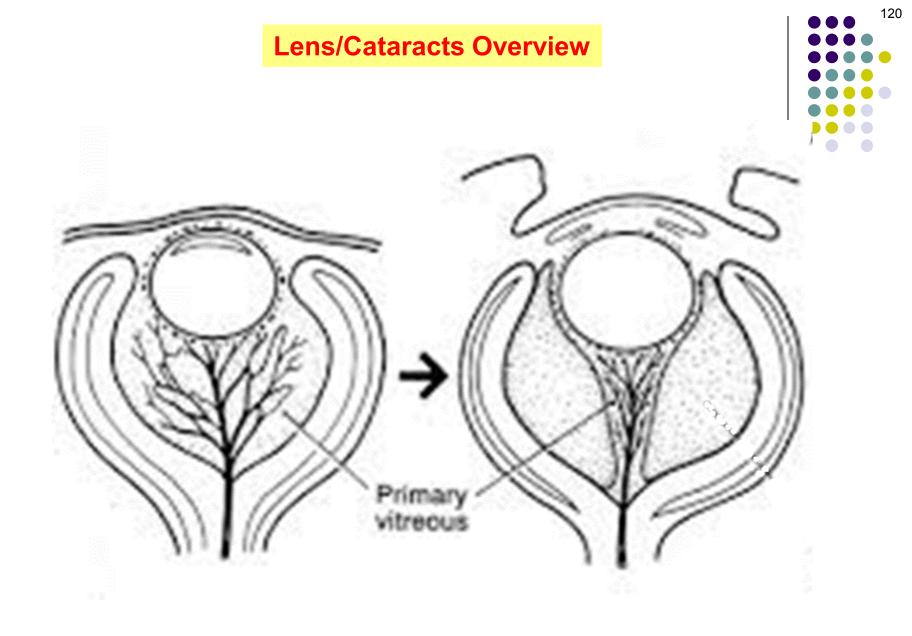
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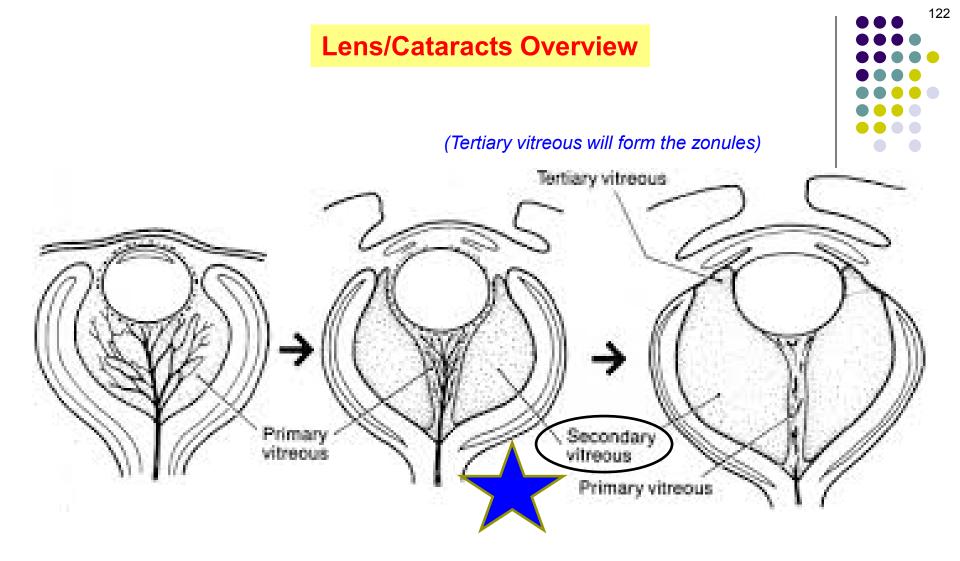
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 - As you already know, the hyaloid vasculature comprises the primary vitreous
 - Hence PFV is aka *persistent hyperplastic <u>primary vitreous</u>*



Primary vitreous



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- Zonules comprise the so-called *tertiary vitreous*
 - As you already know, the hyaloid vasculature comprises the primary vitreous
 - Hence PFV is aka persistent hyperplastic primary vitreous
 - *The secondary vitreous* is the main vitreous body



Secondary vitreous



At long last we're ready to address the cataract portion of the lens/cataract overview. Obviously, cataracts and their extraction are central to the practice of ophthalmology.



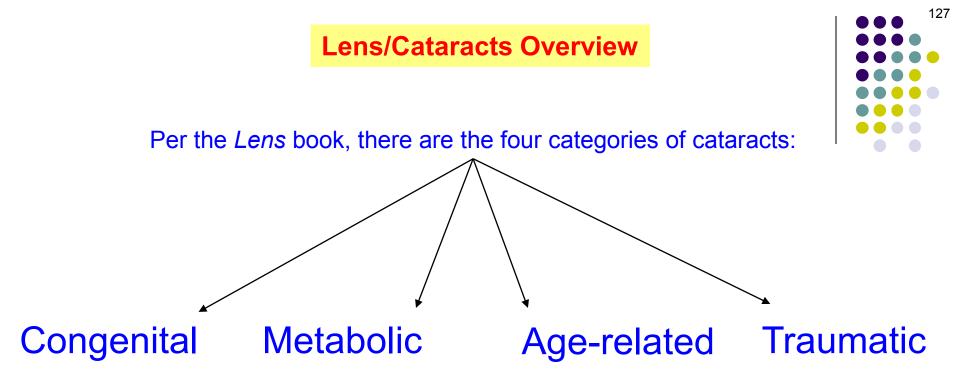
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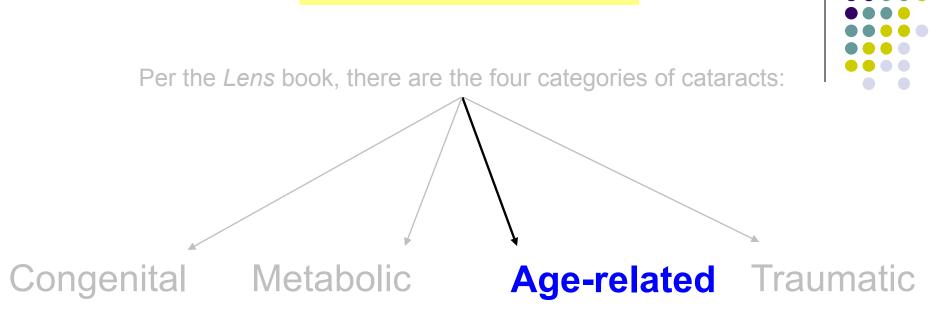
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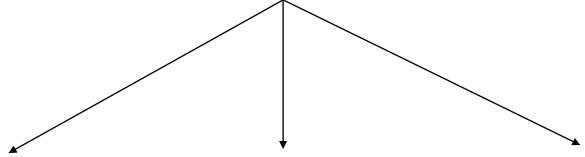


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The remainder of this slide-set will focus on age-related cataracts



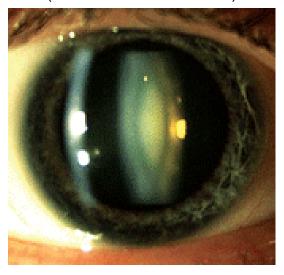




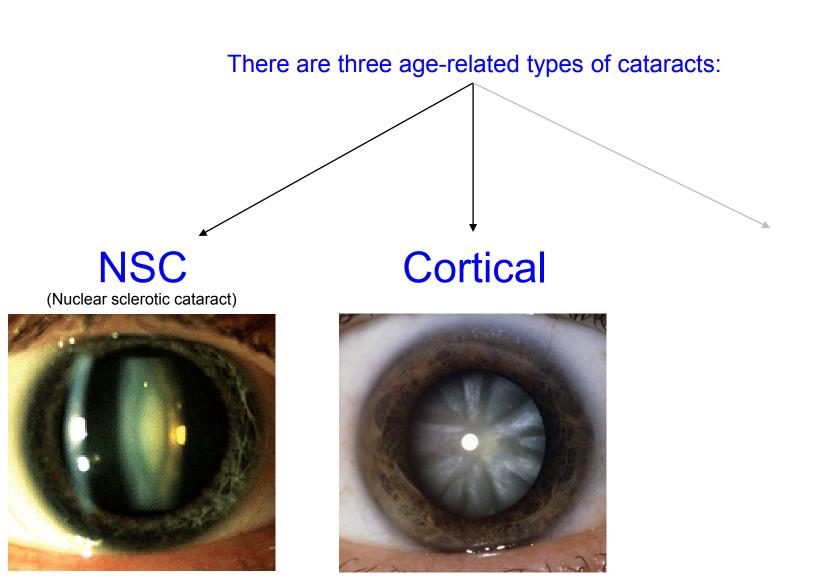




(Nuclear sclerotic cataract)









There are three age-related types of cataracts:



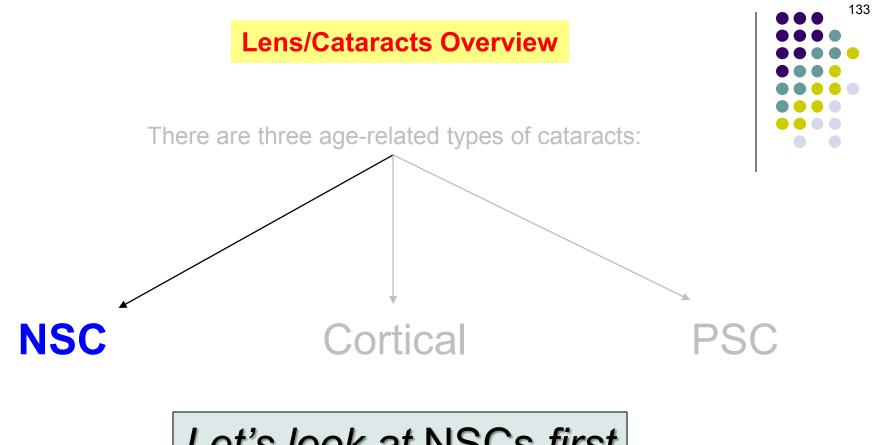
NSC (Nuclear sclerotic cataract)

Cortical



PSC Destariar subsequelar estaract

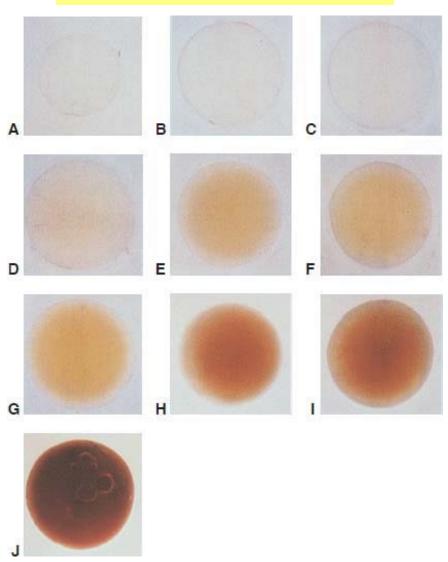


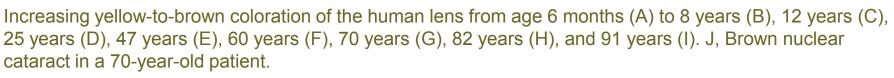


Let's look at NSCs first

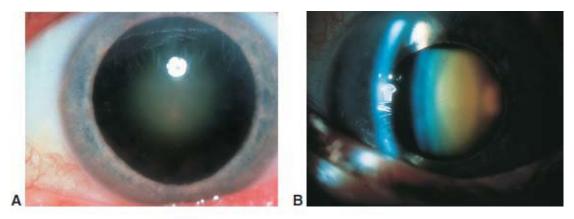


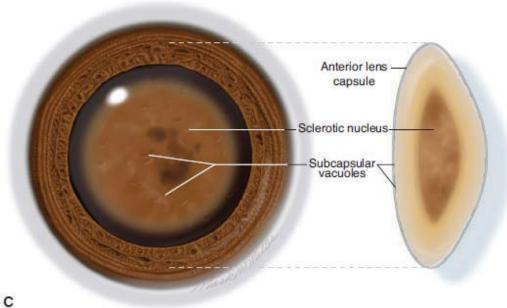
NSCs have two distinctive traits: Their **color** and their **hardness**. Color-wise, NSCs are typically on the **amber-to-brown** spectrum. Why those colors? No one knows. (Per the *Lens* book, the pathogenesis of NSC discoloration is "poorly understood" at this time.) Yellowing of the lens with aging is normal, and is considered pathologic (ie, an NSC) only when it compromises vision.











Nuclear cataract viewed with diffuse illumination (*A*) and with a slit beam (*B*). *C*, Schematic of nuclear cataract





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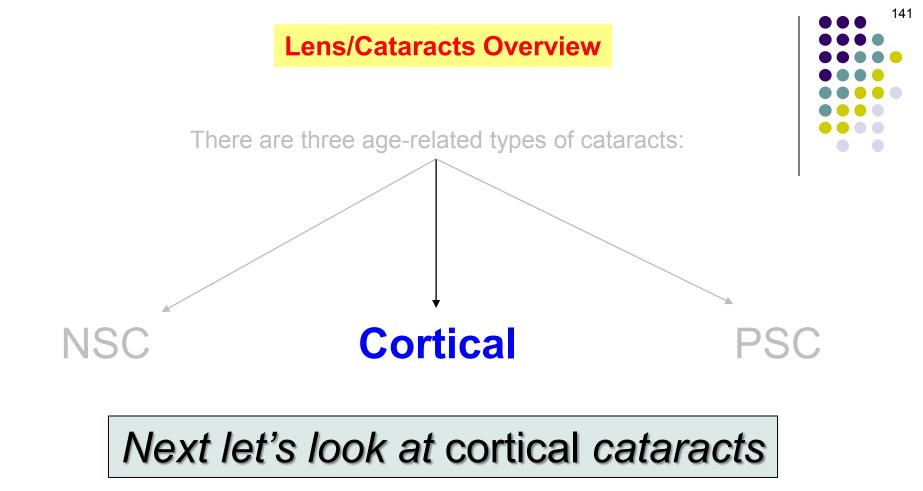
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With respect to vision, pts with NSCs usually c/o difficulty at distance more than near, and in dim light more than bright. As mentioned previously, NSC development is associated with a refractive shift, usually myopic. In some hyperopes and/or presbyopes the myopic shift will temporarily *improve* vision, a phenomenon called *second sight*.



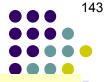
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With respect to vision, pts with NSCs usually c/o difficulty at distance more than near, and in dim light more than bright. As mentioned previously, NSC development is associated with a refractive shift, usually myopic. In some hyperopes and/or presbyopes the myopic shift will temporarily *improve* vision, a phenomenon called *second sight*. In addition to affecting acuity, progressive yellowing/browning of the lens causes patients to have poor color discrimination, especially at the blue end of the spectrum. (In cases of bilateral NSCs, patients are frequently unaware of their altered color discrimination.)





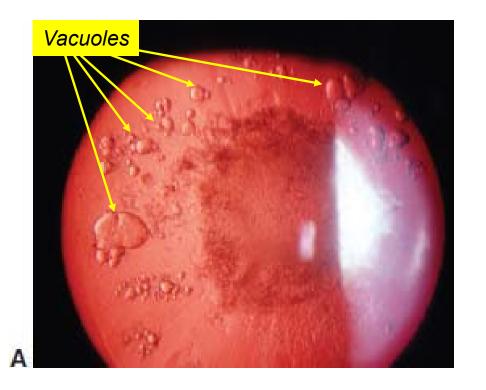
Unlike in NSCs, histopathologic changes *can* be identified in cortical cataracts, as they are characterized by lens fiber swelling and disruption. This loss of cell-membrane integrity leads to protein oxidation and precipitation. This in turn disrupts normal intralenticular osmotic gradients, resulting in an increase in intralenticular water content.



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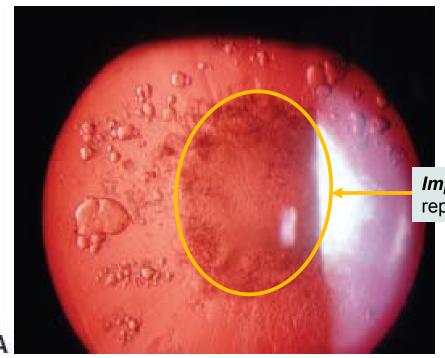
Cortical cataracts pass through a series of defined stages. The first manifestations of an *immature cortical cataract* are the presence of water clefts and vacuoles.





Early cortical cataract development as viewed at the slit lamp using retroillumination. *A*, Vacuoles.





Important: The opacification in this area is **not** representative of cortical changes, but rather is a **PSC**.

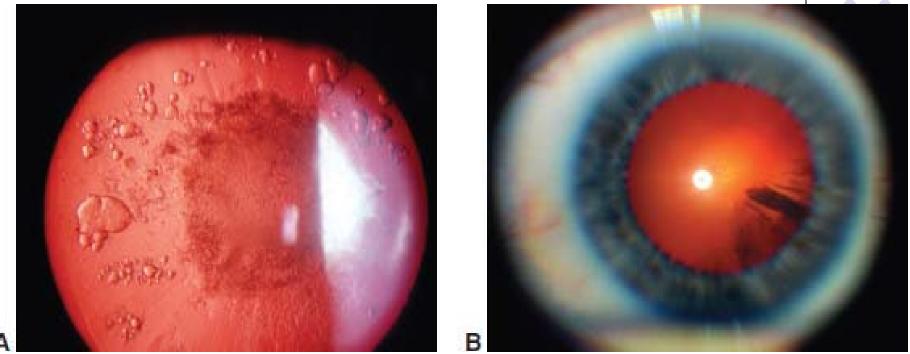
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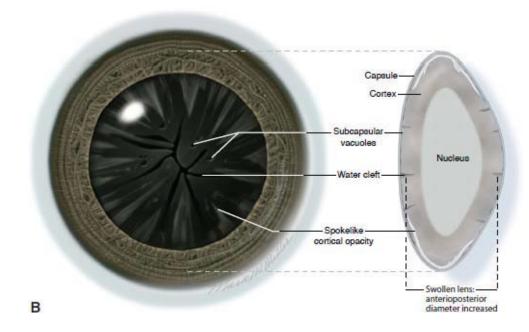




Early cortical cataract development as viewed at the slit lamp using retroillumination. *A*, Vacuoles. *B*, Typical cortical spokes

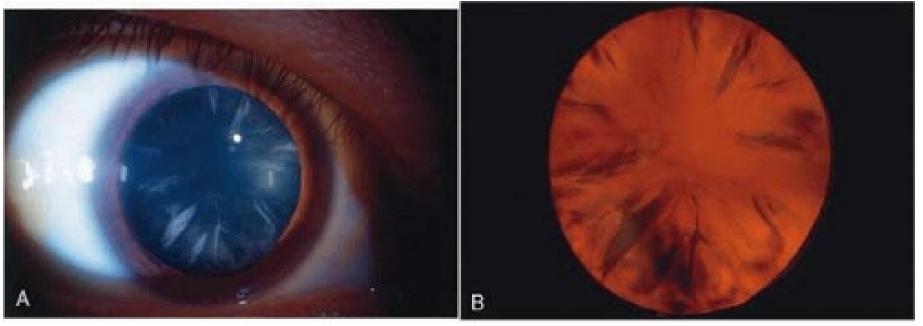






A, Cortical cataract viewed by oblique view at the slit lamp. *B*, Schematic of immature cortical cataract





Direct illumination

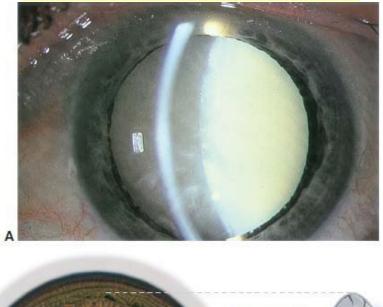
Retroillumination



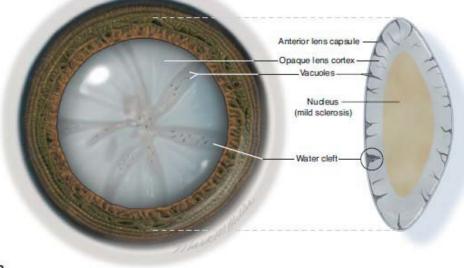
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в

Mature cortical cataract. *A*, Mature cortical cataract viewed at the slit lamp. *B*, Schematic of mature cortical cataract.



Cortical cataract: Mature



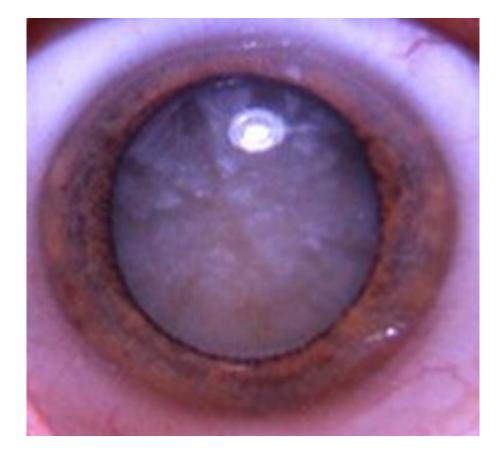


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Many mature cortical cataracts absorb a significant further amount of water. The increase in lens volume enlarges it, often narrowing the angle (and increasing the risk of angle-closure glaucoma) in the process. This stage is called an *intumescent cortical cataract*.





Intumescent cortical cataract

(Lens intumescence isn't really appreciable in a photo, so don't be concerned if it doesn't look significantly different from a mature cataract)



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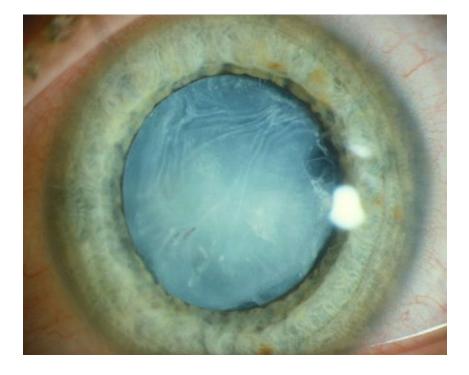
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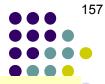
As cortical material continues to degenerate, some will leach through the lens capsule, markedly **decreasing** lens volume. In fact, the volume reduction is significant enough that the previously taut anterior capsule wrinkles in response. This wrinkled capsule is the hallmark of the next stage, the *hypermature* cortical cataract.







Hypermature cataract. Note the capsular wrinkling



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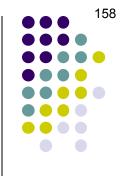
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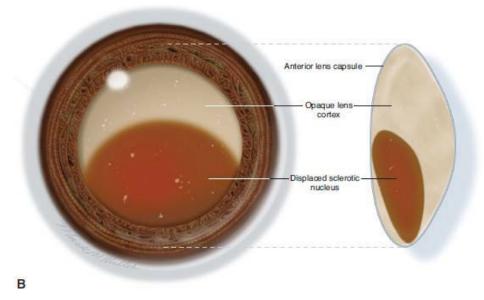
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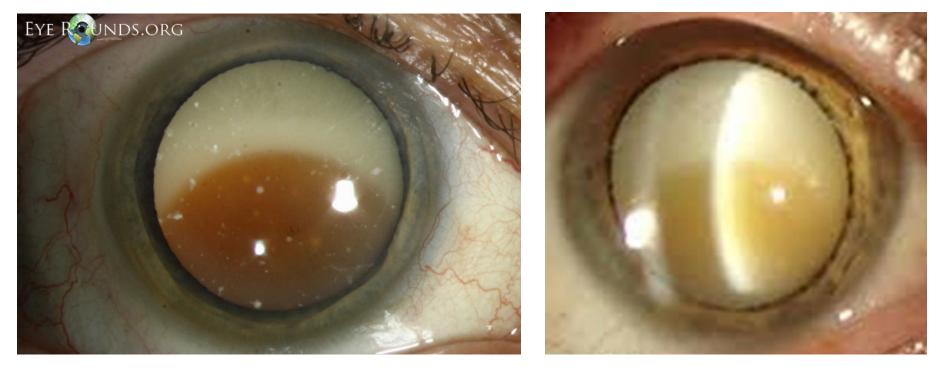






Morgagnian cataract. A, Clinical photo of morgagnian cataract. B, Schematic of morgagnian cataract.





Morgagnian cataract



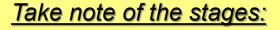
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No red reflex

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Ma For all three stages, the red reflex during cataract surgery is completely obscured. As most cataract surgeons rely on the red reflex to visualize the anterior capsule during capsulorrhexis, this step cannot be performed in a conventional manner.

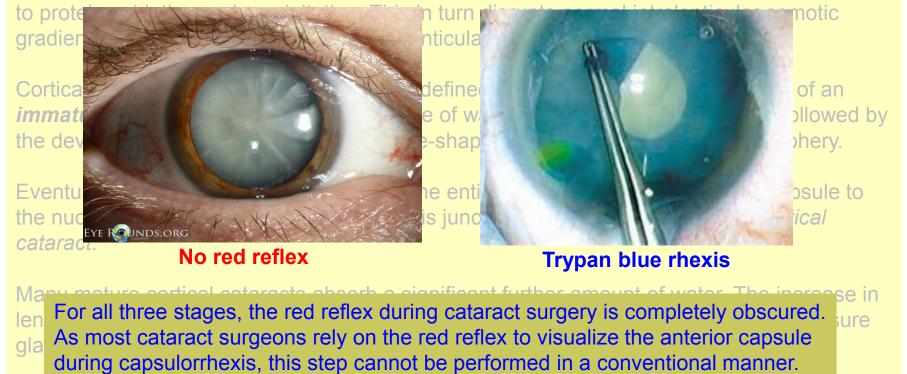
As

Take note of the stages:





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As To facilitate visualization of the anterior capsule during rhexis creation, most surgeons will stain the anterior capsule with **trypan blue**.







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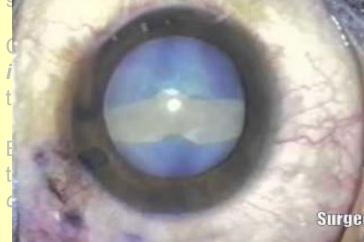
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 Mature cataract
 intumescent cataract
 hypermature cataract

 Cataract absorbs water
 What
 Cataract leaks water



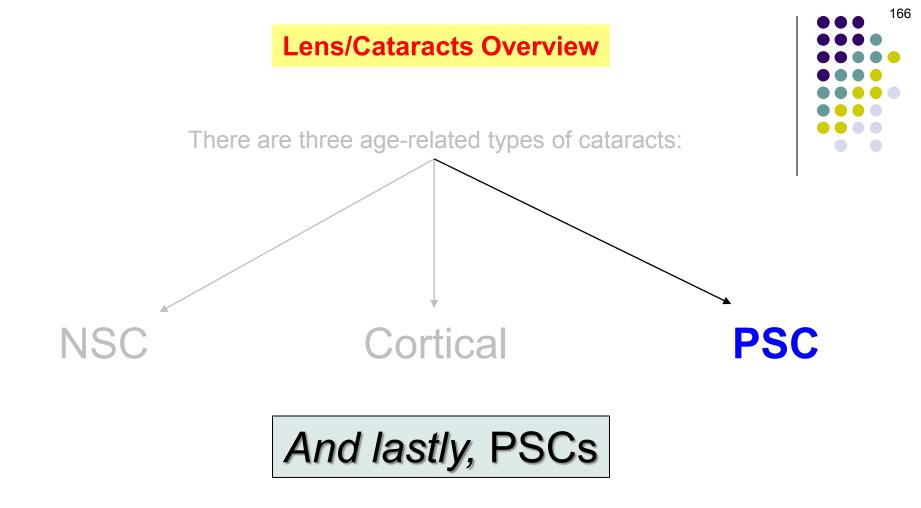
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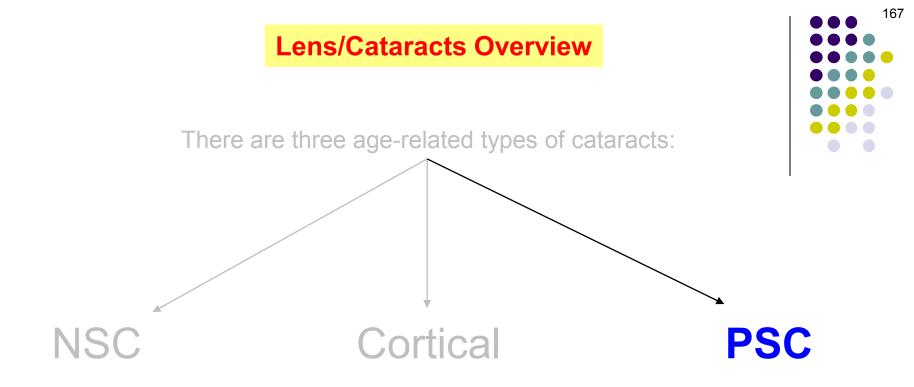


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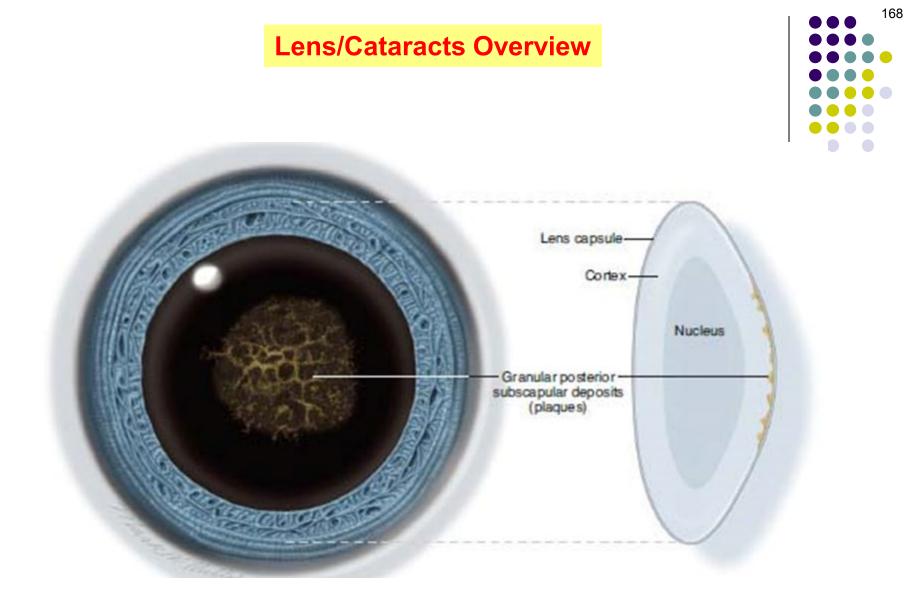
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Cataract **absorbs** water 🔸 _{happens} 🔸 Cataract **leaks** water

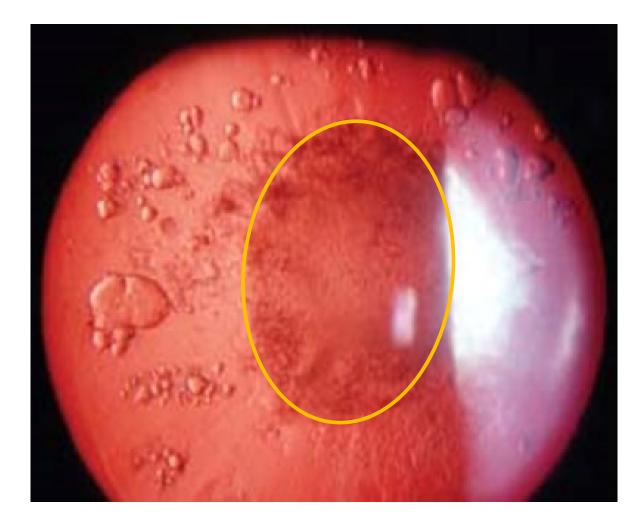




The first and fundamental step in PSC pathophysiology is the migration of equatorial epithelial cells to and across the posterior capsule (PC). As these cells slither across the PC, they swell substantially. These swole cells (called *bladder cells* or *Wedl cells*) cause significant degradation of vision if they're in the visual axis.



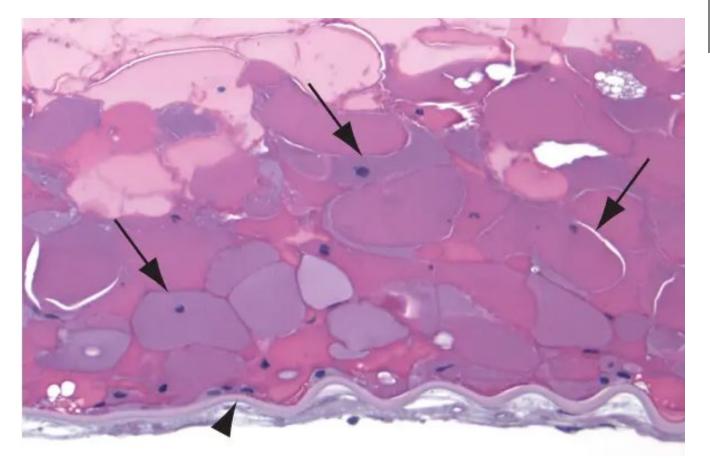
Posterior subcapsular cataract



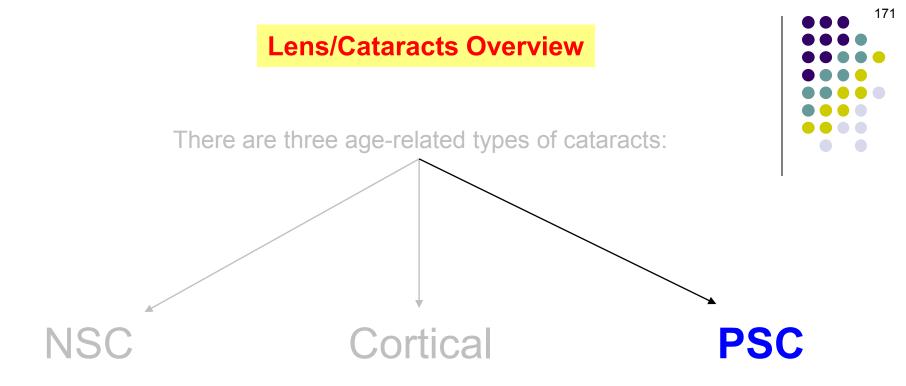
The big ol' gnarly PSC encountered in the cortical cats section



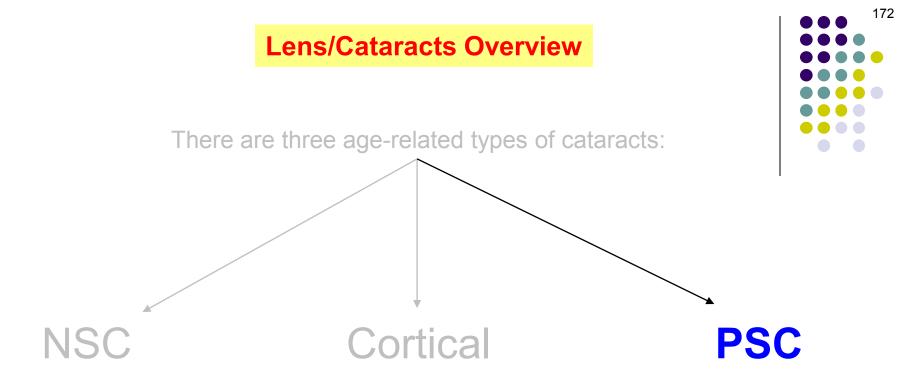




Posterior subcapsular cataract. Oval to round nucleated Wedl cells (*arrows*) and smaller lens epithelial cells line the posterior lens capsule (*arrowhead*).



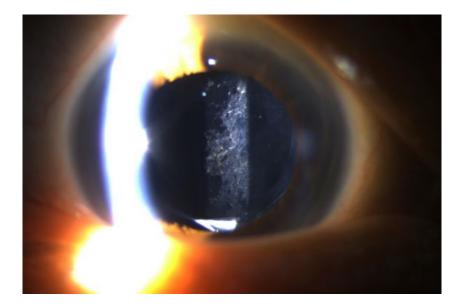
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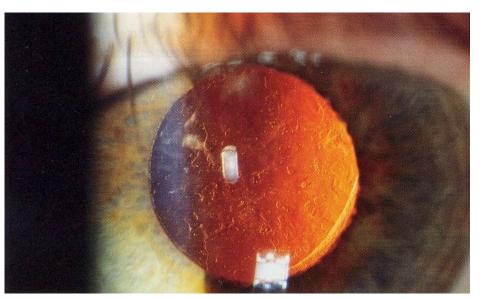


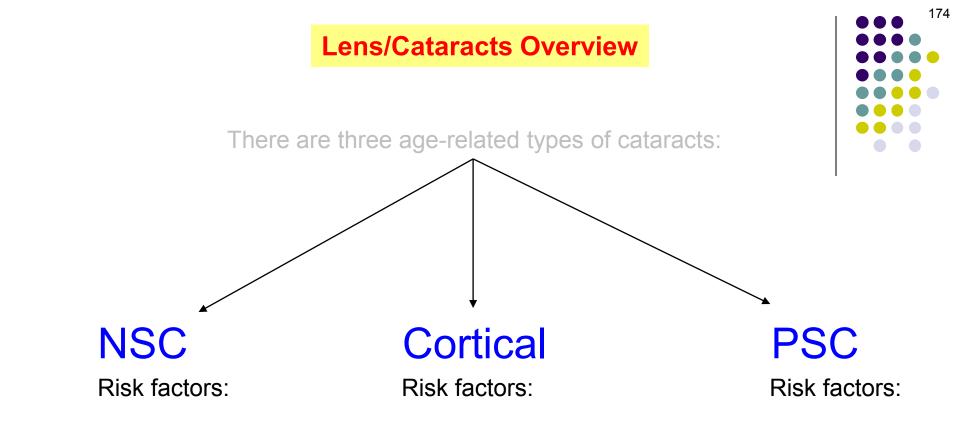
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Important sidebar: The same pathologic process—migration and swelling of equatorial epithelial cells—is responsible for the most common post-CE complication, that being *posterior capsule opacification*. (Up to half of all adult CE pts will develop a PCO, as will essentially **all** peds cases.)

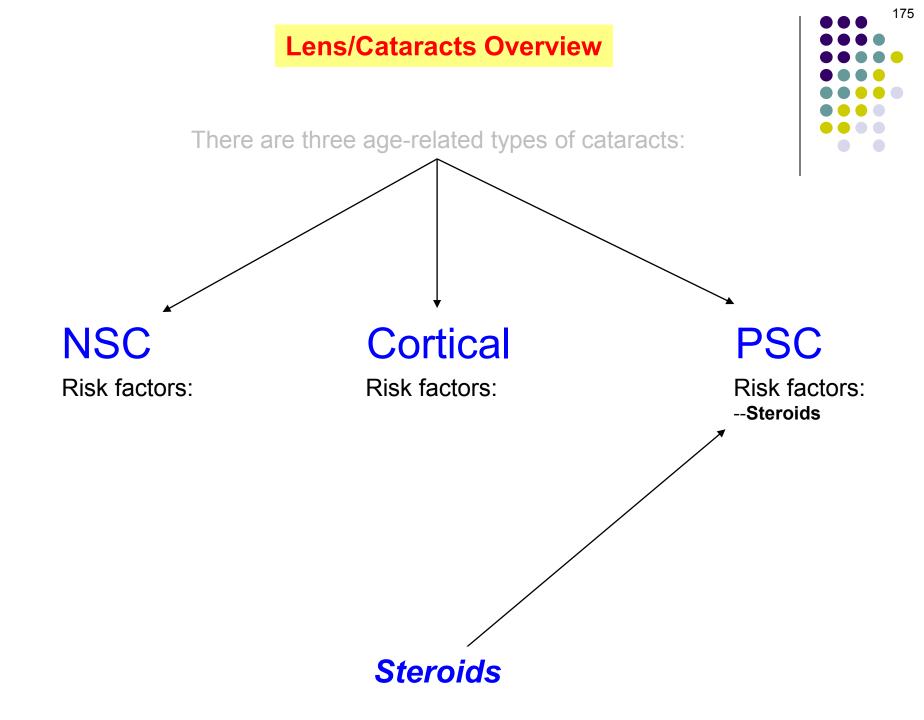


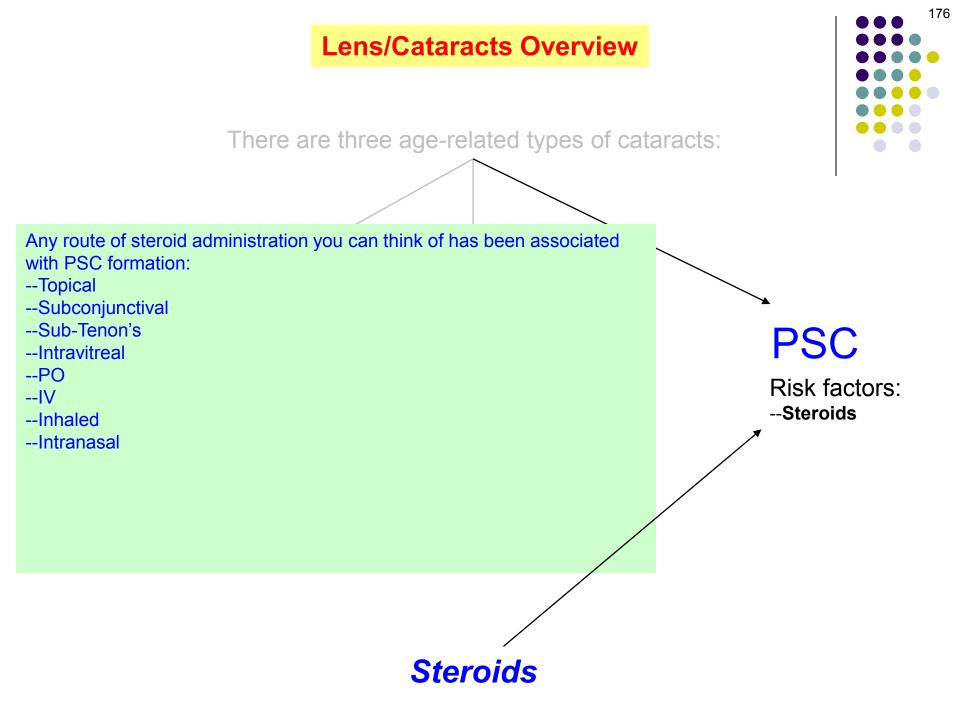






Next we will look at risk factors for each of the age-related cataract types







Any route of steroid administration you can think of has been associated with PSC formation: --Topical --Subconjunctival --Sub-Tenon's --Intravitreal

- --PO
- --IV
- --Inhaled
- --Intranasal

In general, steroid-induced PSCs do not regress with cessation of steroids (with the notable exception of steroid-induced PSCs in *children*).





PSC

--Steroids

Risk factors:

178

PSC

--Steroids

Risk factors:

There are three age-related types of cataracts:

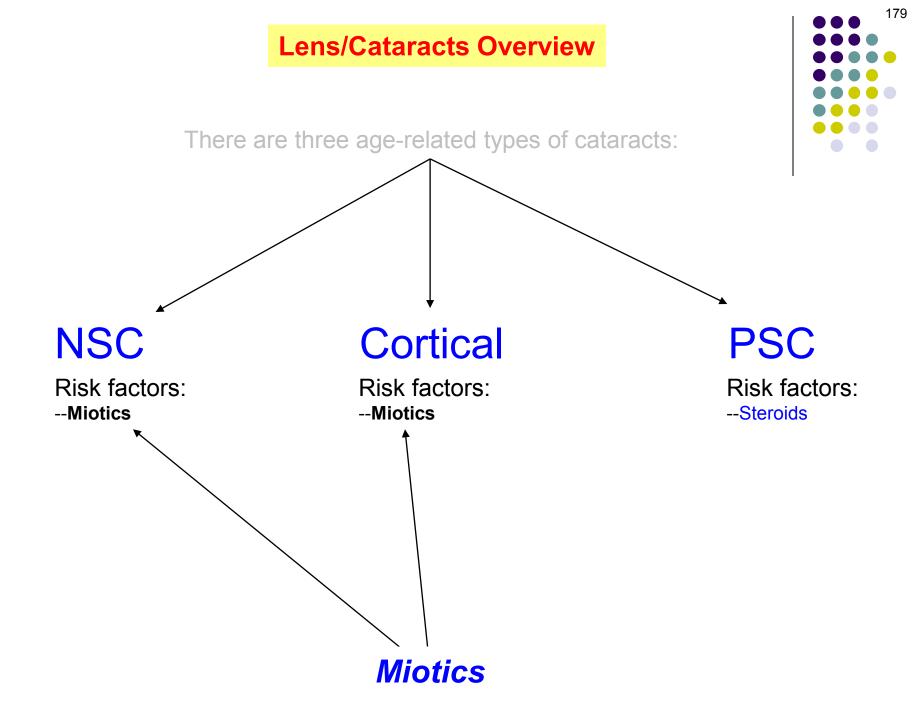
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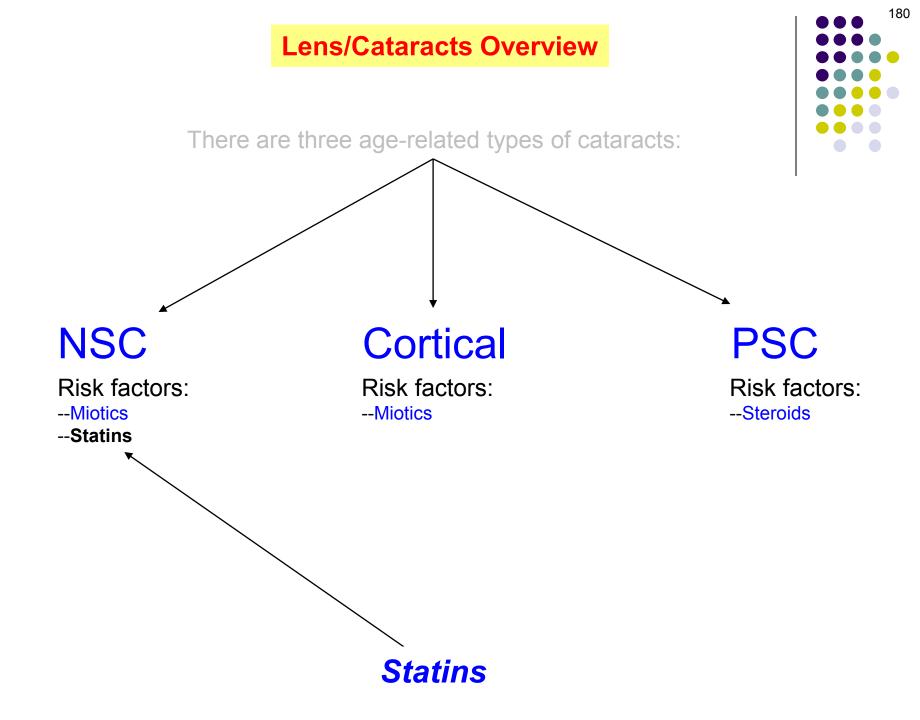
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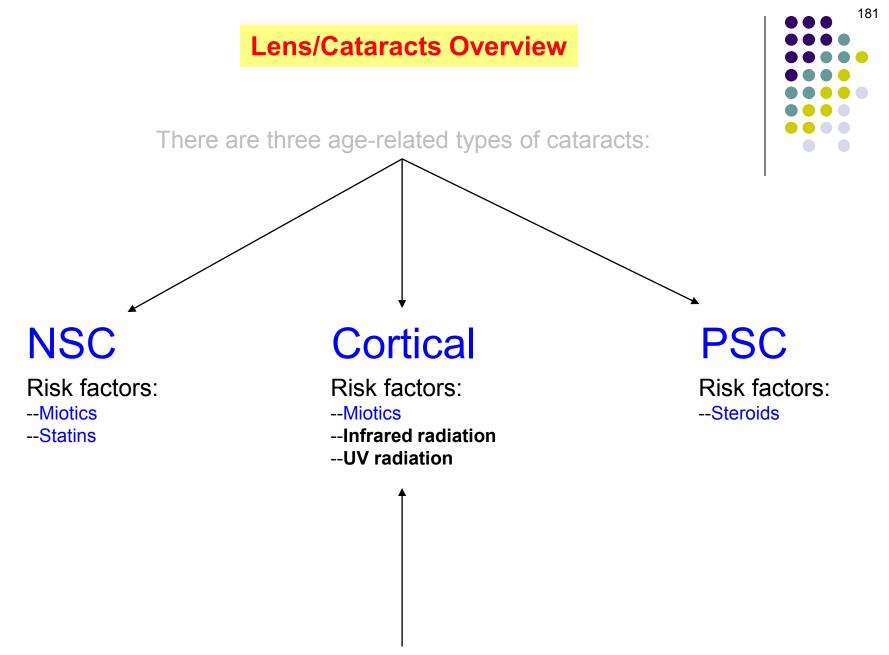
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Interestingly, if a pt has a propensity to develop a steroid-induced PSC, s/he is also at increased risk of steroid-induced ocular hypertension.

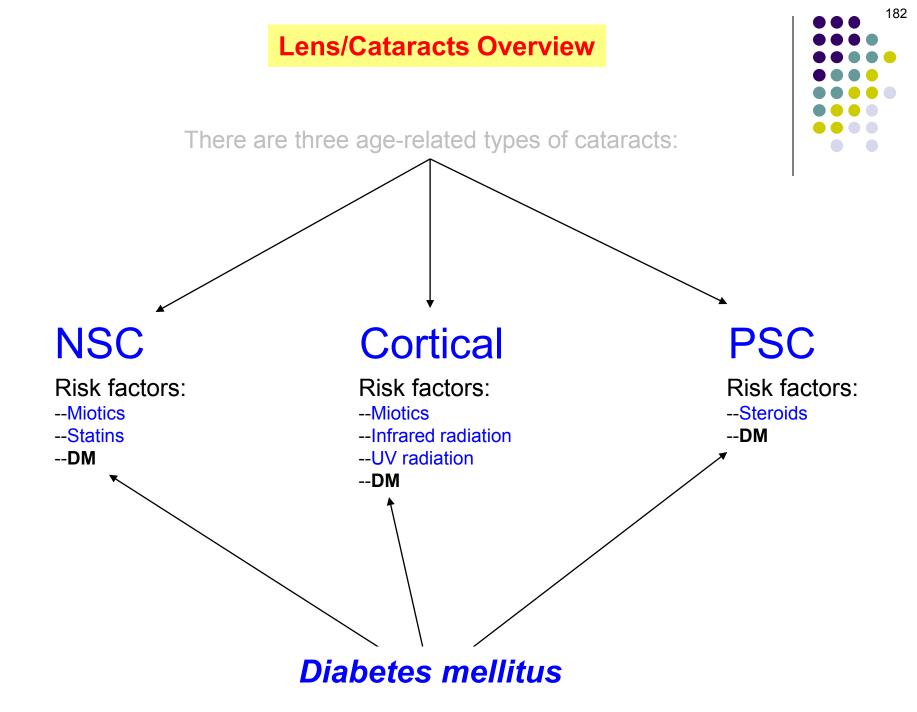
Steroids

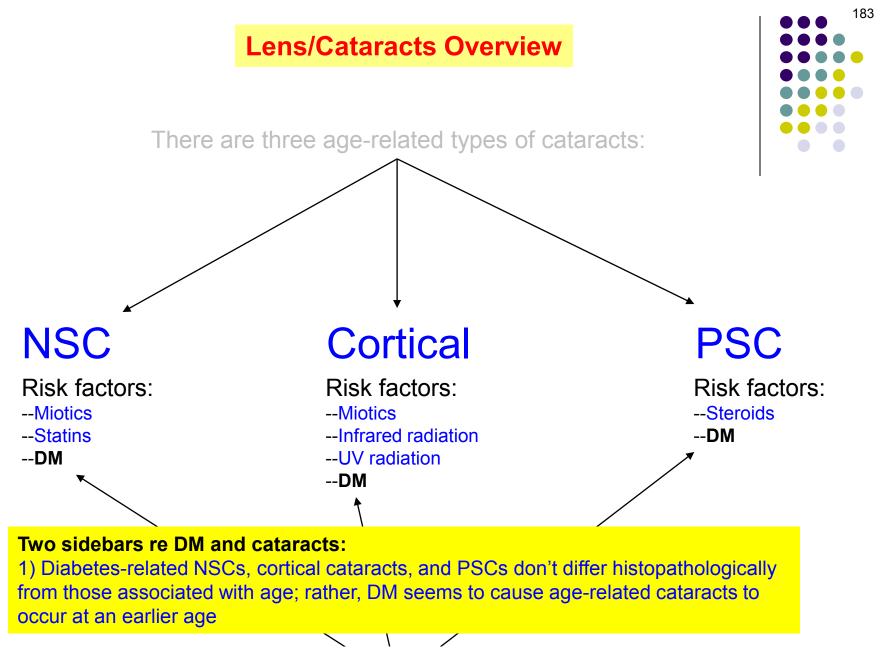




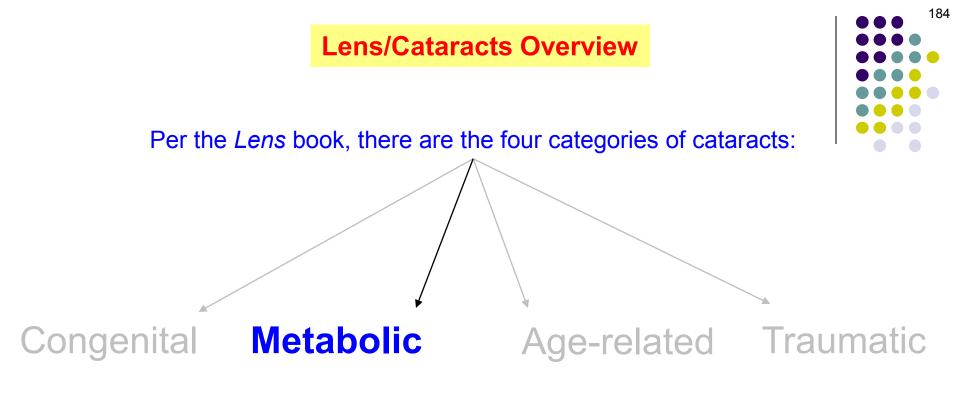


Infrared and/or UV radiation



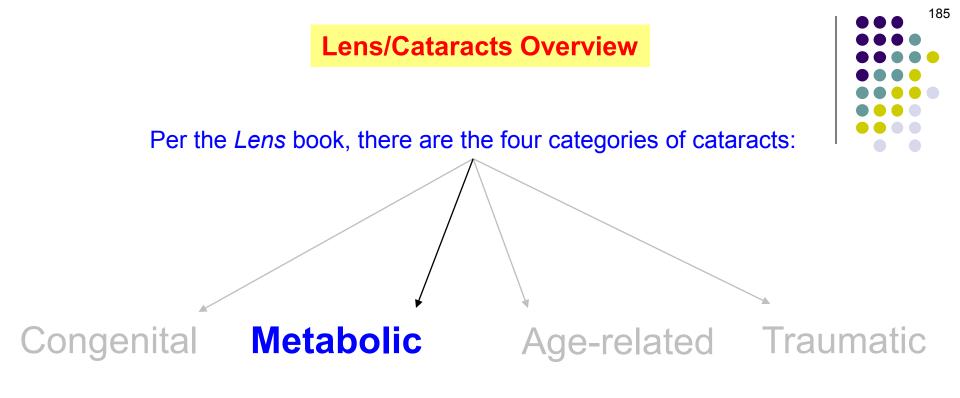


Diabetes mellitus



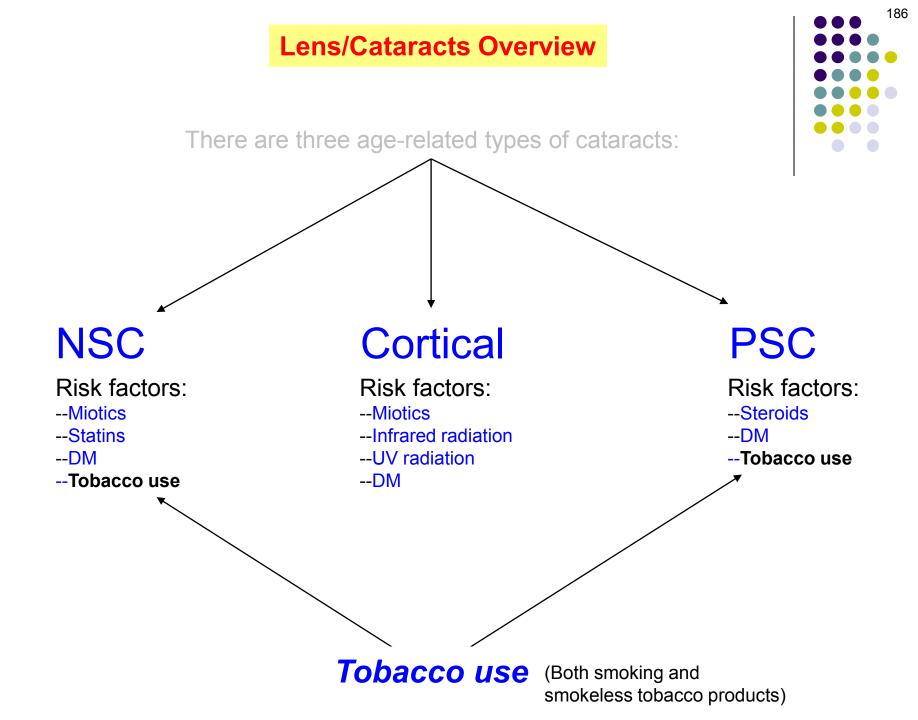
Two sidebars re DM and cataracts:

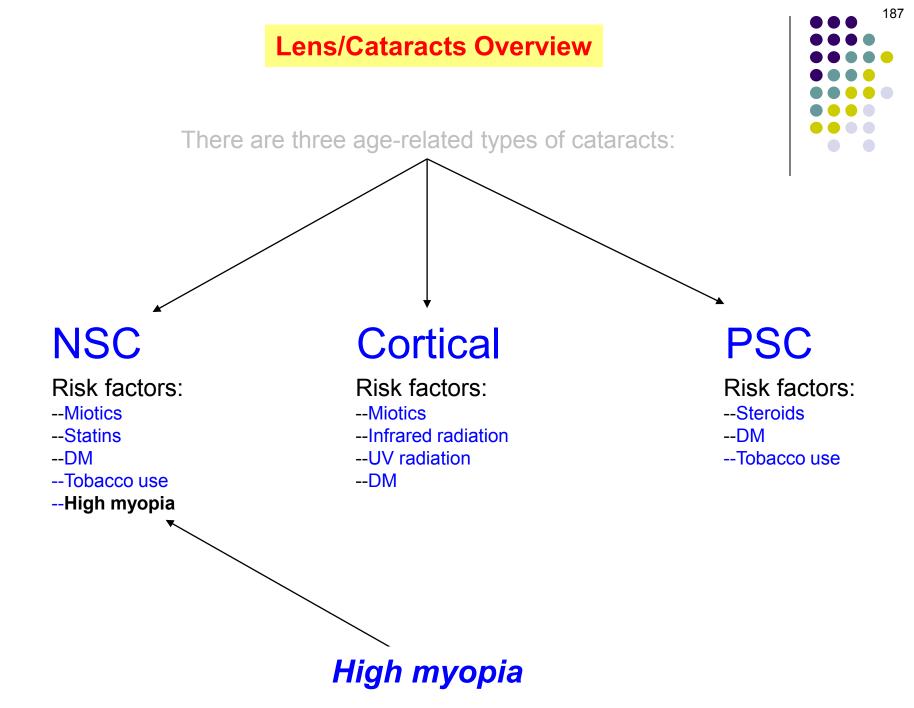
2) Recall that, early in the Cataract section, we noted that cataracts can be **metabolic** in origin. In that regard, diabetes is one of the most common and important causes of metabolic cataract.

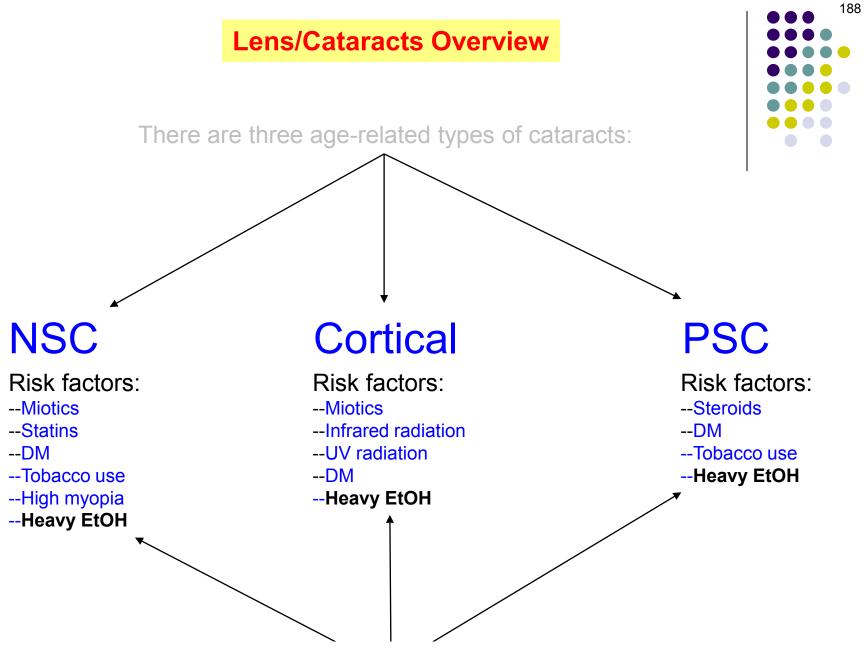


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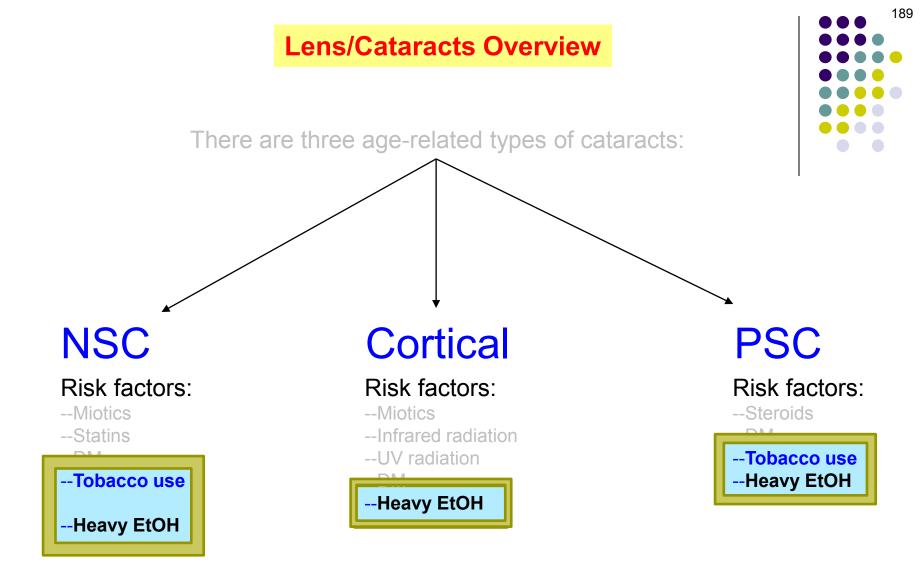
2) Recall that, early in the Cataract section, we noted that cataracts can be **metabolic** in origin. In that regard, diabetes is one of the most common and important causes of metabolic cataract. But note that the pathophysiology underlying a DM-related metabolic cataract (aka a *sugar cataract*) is separate and distinct from DM's role in hastening the development of age-related cataracts.







Heavy EtOH consumption



Note that smoking, the use of smokeless tobacco products, and heavy alcohol consumption are all modifiable risk factors for cataract development



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 *L14*, which covers this material in a Q&A format (and more detail).