**Anatomy of the mature* lens**

- **Capsule**
- **Epithelium**
  - Single layer of cuboidal cells
  - Metabolically active; mitotically active
  - Give rise to all new lens fibers
- **Nucleus**
  - Older, more densely packed fibers in central lens
- **Cortex**
  - Newer fibers between nucleus and capsule/epithelium
- **Zonules**
  - Originate from the basal lamina of the nonpigmented epithelium of the pars plana and pars plicata of the ciliary body
  - Three sets of fibers:
    - Anterior
    - Equatorial
    - Posterior

---

*’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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**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)
These are the surgical layers of the lens...

- **Nucleus**: Opaque, usually with an amber hue. The nucleus is firm, and cannot be aspirated from the eye until/unless it is broken up (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**: 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.
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**Lens/Cataracts Overview**

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It’s unfortunate surgeons settled on **cortex** to refer to the ‘layer-of-tape’ portion of a cataract, as this term already had a (different) meaning regarding lens anatomy.
These are the surgical layers of the lens, and how they behave during cataract surgery:

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It's unfortunate surgeons settled on "cortex" to refer to the 'layer-of-tape' portion of a cataract, as this term already had a (different) meaning regarding lens anatomy. But waddaya gonna do? You just have to keep straight the difference between the anatomic cortex and the surgical cortex.
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**
  - 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.)

- **Nucleus**

- **Cortex**

- **Zonules**
**Lens capsule thickness** has important clinical and surgical implications.

Anterior

- 14 µm
- 21 µm
- 23 µm
- 2-4 µm

Posterior

- 17 µm
**Lens capsule thickness** has important clinical and surgical implications.

Key takeaway: While the entire capsule is thin…
**Lens/Cataracts Overview**

**Lens capsule thickness** has important clinical and surgical implications.

*Anterior*
- 14 µm

*(By way of comparison, human hair ~70 µm)*

*Posterior*
- 2-4 µm
- 21 µm
- 17 µm
- 23 µm

Key takeaway: While the entire capsule is thin... *Its posterior aspect is really thin*
**Lens/Cataracts Overview**

*Lens capsule thickness* has important clinical and surgical implications.

In fact, the posterior capsule is so thin it is *always distended*. Thus, all changes in lens shape during accommodation occur at the *anterior* capsule.

Key takeaway: While the entire capsule is thin… Its posterior aspect is really thin.
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**
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- **Epithelium**
  - Single layer of cuboidal cells beneath anterior and equatorial capsule

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Lens/Cataracts Overview

Anterior lens capsule

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.

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Anatomy of the mature lens

- **Capsule**
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- **Epithelium**
  - Single layer of cuboidal cells beneath anterior and equatorial capsule
  - Metabolically active

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  - 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.

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- **Cortex**

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Anatomy of the mature lens

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Let’s sidebar to consider several important issues related to lens metabolism
Re lens metabolism, bear these two facts in mind:

1)

2)
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1) **Glucose**
   is the substrate for lens metabolism.

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- Anaerobic Glycolysis (majority of glucose)
- HMP Shunt (5% of glucose)
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   \[ \text{Glucose} \rightarrow (G6P) \]

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HMP Shunt (5% of glucose)

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.
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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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1) Glucose is the substrate for lens metabolism

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 expunge metabolic waste. Most (non-lens) cells accomplish this via the circulatory system.

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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.
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**.
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Lens/Cataracts Overview

To achieve this, the lens employs the Pump-Leak System.

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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 $\text{K}^+$ is an order of magnitude higher, and $\text{Na}^{2+}$ an order of magnitude lower, than their concentrations in the aqueous and vitreous.

In this diagram:
- **Aqueous**: $\text{K}^+$ is **Plasma(ish)**, $\text{Na}^{2+}$ is **Plasma(ish)**, both **Higher** and **Lower** compared to the lens.
- **Lens**: $\text{K}^+$ is **Higher**, $\text{Na}^{2+}$ is **Lower**.
- **Vitreous**: $\text{K}^+$ is **Plasma(ish)**, $\text{Na}^{2+}$ is **Plasma(ish)**, both at **Lower** levels than the lens.

**Anterior capsule & epithelium** are shown for $\text{Na}^{2+}$ and **Posterior capsule** for $\text{K}^+$.
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*

- **Nucleus**
  - Older, more densely packed fibers in central lens

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  - Originate from the basal lamina of the nonpigmented epithelium of the pars plana and pars plicata of the ciliary body
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**Lens/Cataracts Overview**

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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- **Cortex**

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Lens/Cataracts Overview
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  - Older fibers densely packed in central lens

- **Cortex**
  - 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.

- **Zonules**
  - Originate from the basal lamina of the nonpigmented epithelium of the pars plana and pars plicata of the ciliary body
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  - Fibers created after age 20 will live out their lives in the cortex.
Note that the distinction between the cortex and nucleus is ill-defined and gradual.
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The zonules support the lens, and transmit to it the forces that produce accommodation. (Their origins are as described above.)
Nevra you mind what these are pointing to

Pars plicata

Pars plana

Cornea

Anterior chamber

Iris

Sclera

Lens

Ciliary process

Zonules

Zonular origins
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Based on their site of insertion, there are three sets of fibers: Anterior, equatorial, and posterior.
Zonular insertions on the lens
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    - **Anterior:** Insert more centrally
    - **Equatorial**
    - **Posterior:** Insert less centrally

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. 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/Cataracts Overview

- 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/Cataracts Overview

- **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** → ↑ refractive power
  - Refractive index **decreases** → ↓ refractive power
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● With age…
  ● Lens curvature increases \( \rightarrow \) ↑ 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**
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Next we will look at the development/embryology of the lens.

Lens proteins come in one of two basic types: Water-soluble (aka the crystallins), and water-insoluble. The crystallins come in three types, but two of them (β and γ) are grouped together, and stand apart from the other (α). As mentioned earlier, crystallins are the proteins created to fill lens cells as they transform into lens fibers.
The **lens** originates as a thickening of surface ectoderm overlying the **optic** (not lens!) **vesicle**, an outpouching of the primitive forebrain destined to become the neurosensory retina, RPE, and ciliary body epithelium (among other things).
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Re **surface ectoderm** and lens formation:

*(Glance at this, then keep going to see the points being made)*
Re **surface ectoderm** and lens formation:

--A portion of **surface ectoderm** thickens to form the **lens placode**
Re surface ectoderm and lens formation:
--A portion of surface ectoderm thickens to form the lens placode

Note the presence of an indentation in the lens placode; this is called the lens pit
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Note that **optic** vesicle and **lens** vesicle are different structures—don’t mix them up!
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.
The lens originates as a thickening of surface ectoderm overlying the optic (not lens!) vesicle, an outpouching of the primitive forebrain destined to become the neurosensory retina, RPE, and ciliary body epithelium (among other things). This thickened area of surface ectoderm is called the lens placode. The placode subsequently invaginates (at the lens pit), eventually forming a fluid-filled sphere containing a single layer of cells; this sphere is the lens (not optic!) vesicle. The outer wall of the lens vesicle consists of the basement membrane of the surface ectoderm cells that line the inner aspect of the vesicle; these cells will become the lens epithelium, and their BM will form the lens capsule.
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Lens/Cataracts Overview
Lens/Cataracts Overview

It also explains how surface ectodermal cells...become lens epithelial cells.

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.

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As we’ve seen, the **embryonic nucleus** is formed when the elongating posterior lens fibers obliterate the vesicle.
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As we’ve seen, the embryonic nucleus is formed when the elongating posterior lens fibers obliterate the vesicle. The fetal nucleus is formed by the equatorial epithelial cells as they elongate both anteriorly (insinuating themselves between the anterior epithelial cells and the lens fibers of the embryonic nucleus) and posteriorly (insinuating themselves between the originations of the embryonic lens fibers and the underlying capsule). When these fibers run into each other at the anterior and posterior poles, they interdigitate to form lens sutures.
To be clear: The fetal nucleus is **not** this entire structure; rather, it is only the portion formed by the secondary lens fibers, as indicated by the { .

As we’ve seen, the **embryonic nucleus** is formed when the elongating posterior lens fibers obliterate the vesicle. The **fetal nucleus** is formed by the **equatorial** epithelial cells as they elongate both anteriorly (insinuating themselves between the anterior epithelial cells and the lens fibers of the embryonic nucleus) and posteriorly (insinuating themselves between the originations of the embryonic lens fibers and the underlying capsule). When these fibers run into each other at the anterior and posterior poles, they interdigitate to form **lens sutures**.
To be clear: The fetal nucleus is **not** this entire structure; rather, it is only the portion formed by the secondary lens fibers, as indicated by the `{`. Put another way: The fetal nucleus *surrounds* the embryonic nucleus.

As we’ve seen, the **embryonic nucleus** is formed when the elongating posterior lens fibers obliterate the vesicle. The **fetal nucleus** is formed by the **equatorial** epithelial cells as they elongate both anteriorly (insinuating themselves between the anterior epithelial cells and the lens fibers of the embryonic nucleus) and posteriorly (insinuating themselves between the originations of the embryonic lens fibers and the underlying capsule). When these fibers run into each other at the anterior and posterior poles, they interdigitate to form *lens sutures*.
Lens/Cataracts Overview

Lens: Y suture formation
Lens/Cataracts Overview

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*

3) The *capsulopupillary portion*
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 \textit{tunica vasculosa lentis}. \textbf{It has three sections:}

1) The \textit{posterior vascular capsule} arises from the \textit{hyaloid} artery

2) The \textit{anterior vascular capsule} derives from the \textit{long ciliary arteries}

3) The \textit{capsulopupillary portion}
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).
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.

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

A common, clinically insignificant (usually) remnant is a persistent pupillary membrane. Another common remnant is the epicapsular star, colloquially referred to as 'chicken feet' on the anterior capsule.

3) The *capsulopupillary portion*
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
   - A common, clinically insignificant remnant is the *Mittendorf dot*

2) The *anterior vascular capsule*

3) The *capsulopupillary portion*
The anterior vascular capsule derives from the long ciliary arteries.

A common, clinically insignificant (usually) remnant is a persistent pupillary membrane.

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

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2. The *anterior vascular capsule*

3. The *capsulopupillary portion*
Lens/Cataracts Overview

Retroillumination

Direct illumination

Mittendorf dot
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**
   - A common, clinically insignificant remnant is the **Mittendorf dot**
   - A less common, clinically devastating remnant is **PFV**

2) The *anterior vascular capsule*

3) The *capsulopupillary portion*
2) The anterior vascular capsule derives from the long ciliary arteries. A common, clinically insignificant (usually) remnant is a persistent pupillary membrane. Another common remnant is the epicapsular star, colloquially referred to as 'chicken feet' on the anterior capsule.

3) The capsulopupillary portion

- 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.
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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.

3) The capsulopupillary portion
PFV: Retrolental fibrovascular membrane

Lens/Cataracts Overview
The vascular supply encapsulating the developing lens is called the **tunica vasculosa lentis**. It has three sections:

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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.

3) The *capsulopupillary portion*
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   - A common, clinically insignificant remnant is the **Mittendorf dot**
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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
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 
   - A common, clinically insignificant remnant is the **Mittendorf dot**
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2) The *anterior vascular capsule* derives from the **long ciliary arteries** 
   - A common, clinically insignificant (usually) remnant is a **persistent pupillary membrane**

3) The *capsulopupillary portion*
Persistent pupillary membrane

Lens/Cataracts Overview

Trivial case

Hey now
The vascular supply encapsulating the developing lens is called the **tunica vasculosa lentis**. **It has three sections:**

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2) The *anterior vascular capsule* derives from the **long ciliary arteries**
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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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2) The *anterior vascular capsule* derives from the long ciliary arteries
   - A common, clinically insignificant (usually) remnant is a persistent pupillary membrane
   - Another common remnant is the epicapsular star, colloquially referred to as ‘chicken feet’ on the anterior capsule

3) The *capsulopupillary portion* anastomoses the anterior and posterior sections of the tunica
Tunica vasculosa lentis: Capsulopupillary portion
● Zonules are secreted by the ciliary epithelium near the end of the third month of gestation
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● Zonules comprise the so-called tertiary vitreous
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As you already know, the hyaloid vasculature comprises the primary vitreous.

Hence PFV is aka persistent hyperplastic primary vitreous.
Primary vitreous
Zonules are secreted by the ciliary epithelium near the end of the third month of gestation.

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

(Tertiary vitreous will form the zonules)
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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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. Cataracts are the #1 cause of blindness worldwide, accounting for an astonishing 1/2 of all cases. Further, it is estimated that a third of the world’s population has at least some degree of visual impairment owing to cataracts. (Cataracts are second only to refractive error as a cause of visual impairment.) Over 10 million cataract surgeries are performed yearly worldwide—but we’re still falling behind.
Per the *Lens* book, there are the four categories of cataracts:

- Congenital
- Metabolic
- Age-related
- Traumatic
The remainder of this slide-set will focus on age-related cataracts
Lens/Cataracts Overview

There are three age-related types of cataracts:
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NSC
(Nuclear sclerotic cataract)
There are three age-related types of cataracts:

- **NSC**
  - (Nuclear sclerotic cataract)

- **Cortical**
There are three age-related types of cataracts:

- **NSC** (Nuclear sclerotic cataract)
- **Cortical**
- **PSC** (Posterior subcapsular cataract)
There are three age-related types of cataracts:

- NSCs
- Cortical
- PSC

*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.
Increasing yellow-to-brown coloration of the human lens from age 6 months (A) to 8 years (B), 12 years (C), 25 years (D), 47 years (E), 60 years (F), 70 years (G), 82 years (H), and 91 years (I). J, Brown nuclear cataract in a 70-year-old patient.
Nuclear cataract viewed with diffuse illumination (A) and with a slit beam (B). C, Schematic of nuclear cataract
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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.)
There are three age-related types of cataracts:

- NSC
- Cortical
- PSC

Next let’s look at cortical cataracts
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.
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.

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.
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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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. This is followed by the development of *cortical spokes*—wedge-shaped opacifications at the lens periphery.
A, Cortical cataract viewed by oblique view at the slit lamp. B, Schematic of immature cortical cataract
Lens/Cataracts Overview

Direct illumination

Retroillumination

Cortical cataract: Early spokes
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Eventually, the cataract will progress until the entirety of the cortex (ie, from the capsule to the nucleus) is opacified and/or white. At this juncture it has become a *mature cortical cataract.*
Cortical cataract: Mature
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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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)
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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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.
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.

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. This is followed by the development of cortical spokes—wedge-shaped opacifications at the lens periphery.

Eventually, the cataract will progress until the entirety of the cortex (ie, from the capsule to the nucleus) is opacified and/or white. At this juncture it has become a mature cortical cataract.

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.

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Take note of the stages:

Mature cataract → intumescent cataract → hypermature cataract

Cataract absorbs water → What happens → Cataract leaks water
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, which in turn disrupts normal intralenticular osmotic gradients, increasing intralenticular water content.

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Many mature cortical cataracts absorb a significant further amount of water. The increase in lens volume often narrows the angle (and increases the risk of angle-closure glaucoma). This stage is called an intumescent cortical cataract.

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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.
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 proteolytic fiber degradation and loss of lens volume. This in turn disrupts the normal intralenticular osmotic gradient, leading to protein oxidation and precipitation. This in turn disrupts the normal intralenticular osmotic gradient, resulting in an increase in intralenticular water content.

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Mature cataract → intumescent cataract → hypermature cataract

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. To facilitate visualization of the anterior capsule during rhexis creation, most surgeons will stain the anterior capsule with trypan blue.
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As if obscuration of the red reflex wasn’t enough, the increased intralenticular pressure of an intumescent cataract poses an additional challenge during capsulorrhexis.
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Cataract absorbs water

What happens

Cataract leaks water
There are three age-related types of cataracts:

- NSC
- Cortical
- PSC

And lastly, PSCs
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.
Lens/Cataracts Overview

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).
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. If a visual-axis PSC is small, its effect on vision may be lessened in dim light, when pupil dilation allows light to go around it. Similarly, because the near reflex induces miosis, PSC pts often complain that near vision is more degraded than distance vision.
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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.)
Lens/Cataracts Overview

PCO
Lens/Cataracts Overview

There are three age-related types of cataracts:

- **NSC**
  - Risk factors:

- **Cortical**
  - Risk factors:

- **PSC**
  - Risk factors:

*Next we will look at risk factors for each of the age-related cataract types*
There are three age-related types of cataracts:

- **NSC**
  - Risk factors: [Steroids]

- **Cortical**
  - Risk factors: [Steroids]

- **PSC**
  - Risk factors: [Steroids]
There are three age-related types of cataracts:

- Age-related nuclear cataract
- Age-related cortical cataract
- Age-related posterior subcapsular cataract

Risk factors:

- Steroids

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

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.
There are three age-related types of cataracts:

Any route of steroid administration you can think of has been associated with PSC formation:
--Topical
--Subconjunctival
--Sub-Tenon's
--Intravitreal
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--IV
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In general, steroid-induced PSCs do not regress with cessation of steroids (with the notable exception of steroid-induced PSCs in *children*).
There are three age-related types of cataracts:

Any route of steroid administration you can think of has been associated with PSC formation:
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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.
There are three age-related types of cataracts:

- **NSC**
  - Risk factors: --Miotics

- **Cortical**
  - Risk factors: --Miotics

- **PSC**
  - Risk factors: --Steroids
There are three age-related types of cataracts:

- **NSC**
  - Risk factors:
    - Miotics
    - Statins

- **Cortical**
  - Risk factors:
    - Miotics

- **PSC**
  - Risk factors:
    - Steroids

**Statins**
Lens/Cataracts Overview

There are three age-related types of cataracts:

**NSC**
- Risk factors:
  - Miotics
  - Statins

**Cortical**
- Risk factors:
  - Miotics
  - Infrared radiation
  - UV radiation

**PSC**
- Risk factors:
  - Steroids

*Infrared and/or UV radiation*
There are three age-related types of cataracts:

- **NSC**
  - Risk factors:
    - Miotics
    - Statins
    - DM

- **Cortical**
  - Risk factors:
    - Miotics
    - Infrared radiation
    - UV radiation
    - DM

- **PSC**
  - Risk factors:
    - Steroids
    - DM

*Diabetes mellitus*
There are three age-related types of cataracts:

**NSC**
- Risk factors:
  - Miotics
  - Statins
  - DM

**Cortical**
- Risk factors:
  - Miotics
  - Infrared radiation
  - UV radiation
  - DM

**PSC**
- Risk factors:
  - Steroids
  - DM

Two sidebars re DM and cataracts:
1) Diabetes-related NSCs, cortical cataracts, and PSCs don’t differ histopathologically from those associated with age; rather, DM seems to cause age-related cataracts to occur at an earlier age.
Per the *Lens* book, there are the four categories of cataracts:

- Congenital
- Metabolic
- Age-related
- Traumatic

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.
Per the *Lens* book, there are the four categories of cataracts:

- Congenital
- **Metabolic**
- Age-related
- Traumatic

**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. 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.
There are three age-related types of cataracts:

- **NSC**
  - Risk factors:
    - Miotics
    - Statins
    - DM
    - Tobacco use

- **Cortical**
  - Risk factors:
    - Miotics
    - Infrared radiation
    - UV radiation
    - DM

- **PSC**
  - Risk factors:
    - Steroids
    - DM
    - Tobacco use

**Tobacco use** (Both smoking and smokeless tobacco products)
There are three age-related types of cataracts:

- **NSC**
  - Risk factors:
    - Miotics
    - Statins
    - DM
    - Tobacco use
    - High myopia

- **Cortical**
  - Risk factors:
    - Miotics
    - Infrared radiation
    - UV radiation
    - DM

- **PSC**
  - Risk factors:
    - Steroids
    - DM
    - Tobacco use
There are three age-related types of cataracts:

- **NSC**
  - Risk factors:
    - Miotics
    - Statins
    - DM
    - Tobacco use
    - High myopia
    - Heavy EtOH

- **Cortical**
  - Risk factors:
    - Miotics
    - Infrared radiation
    - UV radiation
    - DM
    - Heavy EtOH

- **PSC**
  - Risk factors:
    - Steroids
    - DM
    - Tobacco use
    - Heavy EtOH
There are three age-related types of cataracts:

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  - Risk factors:
    - Miotics
    - Statins
    - DM
    - Tobacco use
    - Heavy EtOH

- **Cortical**
  - Risk factors:
    - Miotics
    - Infrared radiation
    - UV radiation
    - DM
    - Heavy EtOH

- **PSC**
  - Risk factors:
    - Steroids
    - DM
    - Tobacco use
    - Heavy EtOH

**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).