BI 358 Lecture 18

I. Announcements Quiz 5 returned at end of lecture. Eye Dissection & Vision lab next Tuesday > Lecture by Dr. Sims! Final Quiz (6) next Thursday, then thoughts on grad schools in medicine & allied health.


III. Eye I: Anatomy & Optics of Vision G&H ch 49 + LS +

IV. Eye II: Retinal Receptor & Neural Function G&H ch 50

V. Eye III: Overview of Visual Pathways & Pathologies G&H ch 51 + LS1 + Silverthorn +...
Wellness Facts

- Smoking worsens the prognosis in men with prostate cancer. In a Harvard study of 5,366 male health professionals with the disease, smokers were much more likely than nonsmokers to have a recurrence and to die from this cancer, cardiovascular disease or any cause. Men who had quit smoking had prostate cancer mortality rates similar to those who had never smoked, unless they were heavy smokers who stopped less than 10 years earlier. Previous research has suggested that smoking also increases the risk of developing prostate cancer.

- More bad news about television viewing: For every two hours watched daily, death rates rise by 13 percent, according to a recent analysis in the Journal of the American Medical Association, which pooled data from eight large studies. That works out to about one extra death each year per 1,000 adults who watch TV two hours a day. And the risk rises with longer viewing hours. The study also found a 20 percent increased risk of diabetes for every two hours watched daily. That’s not surprising, since heavy TV viewing often leads to heavy TV viewers.

The sight-saving diet?
A look behind the eye-health claims made for foods and supplements

No one knows how to prevent the eye disorders that often come with aging, though not smoking and avoiding strong sunlight may help reduce the risk of cataracts. That’s why there has been so much interest in the role of nutrition in eye health, which has generated hundreds of studies in recent years—and many promising leads.

It’s clear that malnutrition harms vision. A shortage of vitamin A, for example, causes night blindness and other problems. Thus, carrots really are good for your eyes, since they’re rich in beta carotene, which the body converts into vitamin A. Vitamin deficiencies can also cause eye disorders such as cataracts in lab animals.

Other nutrients and plant compounds may help protect vision, perhaps by acting as antioxidants and reducing inflammation—the progression of AMD if you do develop it (see page 2).

Eye on research
Here are the nutrients and supplements most often promoted as ways to preserve vision in healthy people and prevent AMD and/or cataracts, along with what the research shows:

- Lutein and zeaxanthin. Most (but not all) observational studies have found that people with high dietary intakes or high blood levels of these carotenoids have a reduced risk of AMD and cataracts. Some small short-term clinical trials have also suggested protective effects in people with healthy eyes, as well as benefits in those who already have AMD. More research is needed.

- Vitamin C and E, selenium, beta carotene and other antioxidants. Again.
Sight-saving Diet?

1. High intakes of lutein & zeaxanthin (carotenoids) may reduce risk of macular degeneration (AMD) & cataracts.

2. Consuming plant-foods rich in antioxidants including vitamins C & E, selenium & β-carotene also may reduce risk of macular degeneration & cataracts.

3. Older vegetarians are 30-40% less likely to develop cataracts compared to daily meat eaters.

4. The above holds for foods, but there is little evidence that anti-oxidant supplements have this effect.

5. Zinc is essential to good vision & is found in the retina & may protect eyes from light damage & inflammation. Get zinc from food (oysters, shrimp, whole grains, yogurt...)

6. High intakes of fish rich in Ω-3 fats also reduce AMD.
Eye: Elaborate sensory receptor $\equiv$ Camera

Aperture + Lens + Film!
Lens Separates Major Compartments

Aqueous Humor → Vitreous Humor/Body
Eye: Anterior View

Lacrimal Gland

Canal for tear drainage

Sclera

Iris

Pupil

L Sherwood 2012
The Blind Spot?

- Optic disk (blind spot)
- Central retinal artery and vein (+ optic nerve)
- Fovea
- Macula

(b)

D. Silverthorn 2010
Convex lens convergence + focal length
Concave lens divergence

Light from distant source

G&H 2011 fig 49-3
Image formation by convex lens

A

Point sources

Focal points

B

G&H 2011 fig 49-7
What's a diopter? Refractive power measurement = $f^{-1}$ or 1m divide by $f$
Refractive index?

Total refractive power = 59 diopters

- Vitreous humor 1.34
- Lens 1.40
- Aqueous humor 1.33
- Cornea 1.38
- Air 1.00

G&H 2011 fig 49-9
Mechanism of accommodation

G&H 2011 fig 49-10
Mini-tramp analogy

- **Lens**: The center of the mini-tramp represents the lens in the eye.
- **Suspensory ligaments**: These are the tension points that control the elasticity of the trampoline, similar to the suspensory ligaments in the eye.
- **Ciliary muscle**: This muscle is responsible for adjusting the shape of the lens, just as the ciliary muscle in the eye controls lens accommodation.

[Image](http://trampolinefiend.com/)
Accommodation $\equiv$ Lens Thickens + Pupils Constrict + Eyes Adduct!
Normal, far- & near-sighted vision

- Emmetropia
- Hyperopia
- Myopia

G&H 2011 fig 49-12
Correcting near- & far-sightedness

Myopia

Hyperopia

G&H 2011 fig 49-13
Astigmatism?

G&H 2011 fig 49-15
Fluid formation & flow

- Aqueous humor
- Iris
- Flow of fluid
- Spaces of Fontana
- Canal of Schlemm
- Ciliary body
- Formation of aqueous humor
- Lens
- Vitreous humor
- Diffusion of fluid and other constituents
- Filtration and diffusion at retinal vessels
- Optic nerve
Aqueous humor formation

G&H 2011 fig 49-20
Glaucoma & intraocular pressure (IOP)?

IOP Normal 12-20 mm Hg
\[ \bar{x} = 15 \pm 2 \text{ mm Hg} \]

Glaucoma \( \geq 25-30 \text{ mm Hg} \)
up to \( 60-70 \text{ mm Hg} \)!
Retinal layers

- Pigmented layer
- Outer nuclear layer
- Outer plexiform layer
- Inner nuclear layer
- Inner plexiform layer
- Ganglion cell layer
- Stratum opticum
- Inner limiting membrane

G&H 2011 fig 50-1

DIRECTION OF LIGHT
Macula & fovea hot spot!

Direction of light

G&H 2011 fig 50-2
Exposed Cones @ Fovea/Macular Region

Peripheral (L) vs. foveal (R) retina

G&H 2011 fig 50-12
Rod & cone functional parts

- Membrane shelves lined with rhodopsin or color pigment
- Mitochondria
- Outer segment
- Inner segment
- Outer limiting membrane
- Nucleus
- Synaptic body

G&H 2011 fig 50-3
Rod & cone outer segments
In rods, light converts cis to trans retinal.

Rhodopsin = Opsin + Retinal
Rhodopsin-retinal visual cycle

Light energy
(Rhodopsin) → Batrhorhodopsin (nsec) → Luminrhodopsin (μsec) → Metarhodopsin I (msec) → Metarhodopsin II (sec) → Scotopsin → Isomerase → all-trans retinal → Isomerase → all-trans retinol → 11-cis retinal → 11-cis retinol

G&H 2011
fig 50-5
<table>
<thead>
<tr>
<th><strong>TABLE 6-2</strong></th>
<th>Properties of Rod Vision and Cone Vision</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>RODS</strong></td>
<td><strong>&gt; 33 x more!</strong></td>
</tr>
<tr>
<td><strong>CONES</strong></td>
<td></td>
</tr>
<tr>
<td>100 million per retina</td>
<td>3 million per retina</td>
</tr>
<tr>
<td>Vision in shades of gray</td>
<td>Color vision</td>
</tr>
<tr>
<td>High sensitivity</td>
<td>Low sensitivity</td>
</tr>
<tr>
<td>Low acuity</td>
<td>High acuity</td>
</tr>
<tr>
<td>Night vision</td>
<td>Day vision</td>
</tr>
<tr>
<td>More numerous in periphery</td>
<td>Concentrated in fovea</td>
</tr>
</tbody>
</table>
Intermediate Colors Are Produced When $1^0$ Colors Are Superimposed
Ratios of cone stimulation determine color interpretation: orange 99:42:0
Color Deficiencies Can Impact Daily Activities, Pleasure & Work!

Red Cone Deficiency = Protanopia
Green Cone Deficiency = Deuteranopia
Blue Cone Deficiency = Tritanopia

http://www.color-blindness.com/coblis-color-blindness-simulator/
Ishihara Chart for Normal (74) vs. Red-Green Color Blindness (21)
Ishihara chart for red-blind protanope (2) vs. green-blind deuteranope (4)

G&H 2011 fig 50-11b
(Viewing brain from above with overlying structures removed)

- Left eye
- Right eye
- Optic nerve
- Optic chiasm
- Optic tract
- Lateral geniculate nucleus of thalamus
- Optic radiation
- Optic lobe

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Visual deficits with specific lesions

1. Left optic nerve
   - Site of lesion:
   - Visual deficit:

2. Optic chiasm
   - Site of lesion:
   - Visual deficit:

3. Left optic tract (or radiation)
   - Site of lesion:
   - Visual deficit:

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Fig 6-24b p 163 LS1 2006
Rods in Darkness → Rhodopsin Not Active, cGMP High, CNG and $K^+$ Channels Open

Rods – 3 Main Cation Channels

1. CNG (Cyclic Nucleotide-Gated) Channel
   Enable Na$^+$ and Ca$^{2+}$ entry into Rod

2. $K^+$ Channel
   Enables K$^+$ to leak out of Rod

3. Ca$^{2+}$-Voltage-Gate Channel
   Enables Ca$^{2+}$ Entry into Synaptic Terminal to Regulate Glutamate Exocytosis
Sodium flows in photoreceptor - A

G&H 2011 fig 50-6a
Sodium flows in photoreceptor - B

- **Dark**: High (cGMP), open channels
- **Light**: Low (cGMP), closed channels

G&H 2011 fig 50-6b
Phototransduction (outer segment)

- Light activates Rhodopsin, leading to the activation of G-Protein Transducin.
- This leads to the hydrolysis of cGMP by Phosphodiesterase, resulting in an increase in 5'-GMP.
- The increase in 5'-GMP causes the opening of a cGMP gated sodium channel, allowing sodium ions (Na⁺) to enter the cell.

G&H 2011 fig 50-7
Summary: Let There Be Light!

Light → Bleaches Rhodopsin → Opsin → cGMP → NT Release → Closes CNG Channel (No more free inflow of Na⁺, Ca²⁺) → Hyperpolarizes Membrane (to -70 mV)