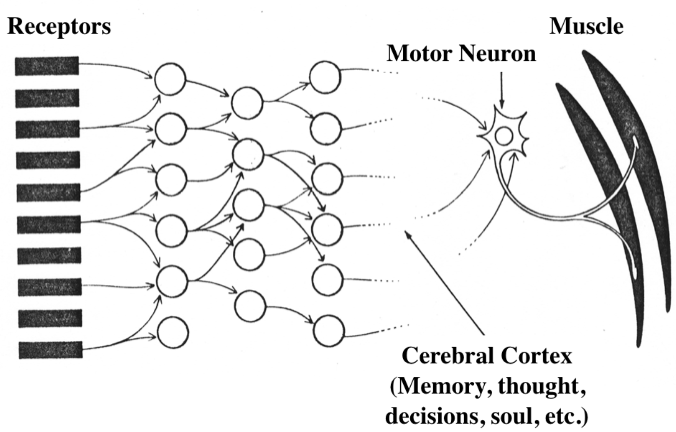
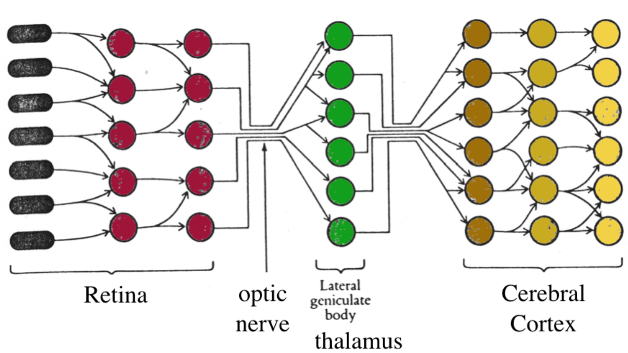
Vision I

- Eye & Retina
- Photoreceptors & Dark Current
- Color Vision
- Ganglion Cells
- Center Surrounds

Sensory Neural Network



Visual Neural Network



Vision

Frequency of Light coming from Objects
Position of Objects in Visual Field
Shape of Objects

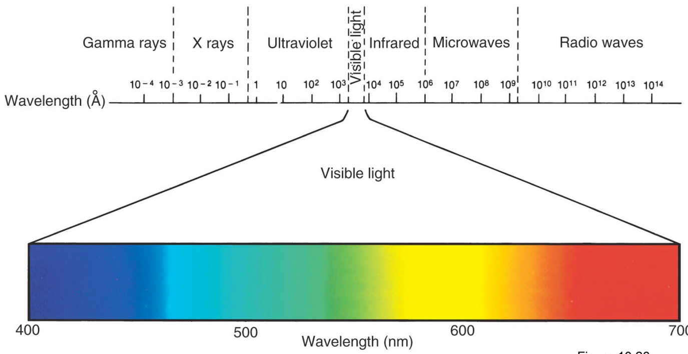
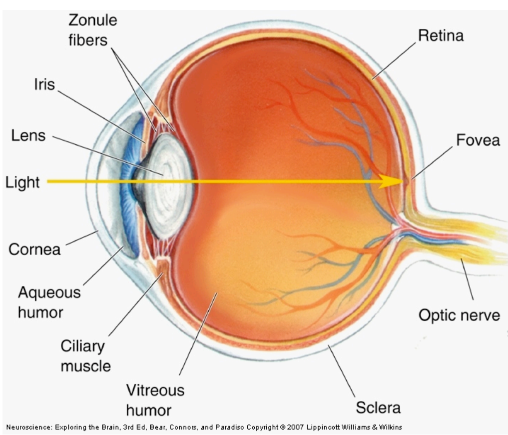


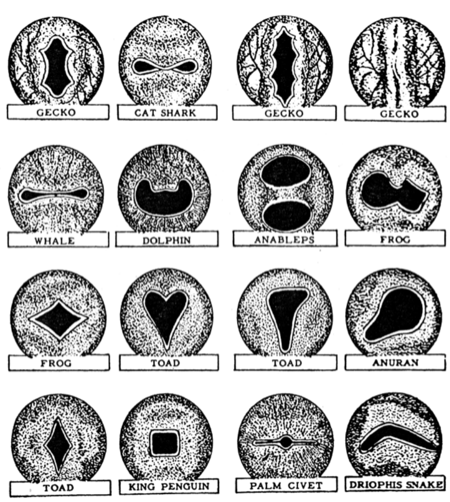
Figure 10.26

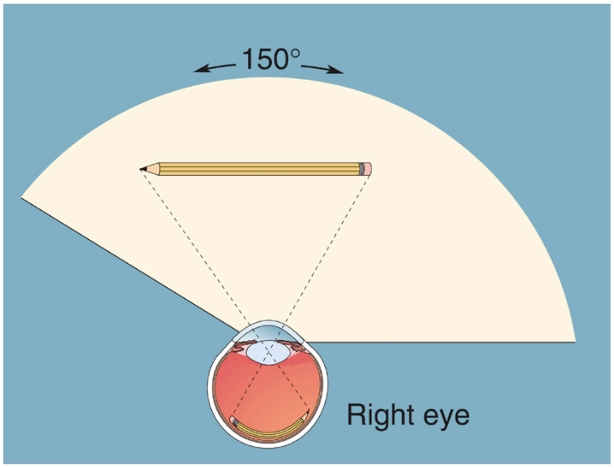
The eye

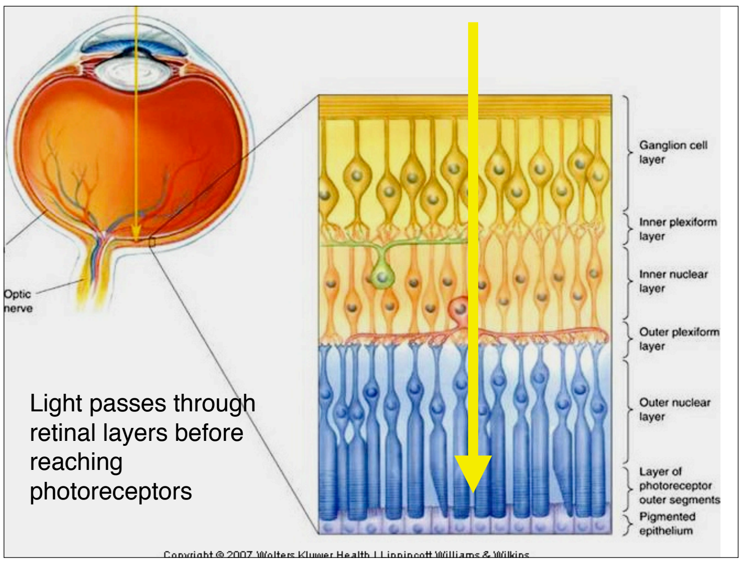


Pupil

can adjust to changes in light levels by about 1 log unit of illumination (1/10th of dark adaptation)




150°
Right eye


Ganglion cell layer
Inner plexiform layer
Inner nuclear layer
Outer plexiform layer
Outer nuclear layer
Layer of photoreceptor outer segments
Pigmented epithelium
Optic nerve
Light passes through retinal layers before reaching photoreceptors
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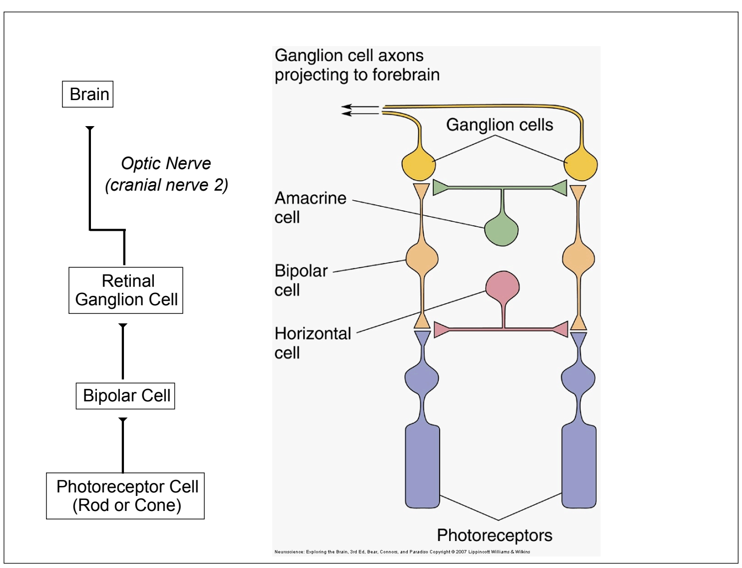
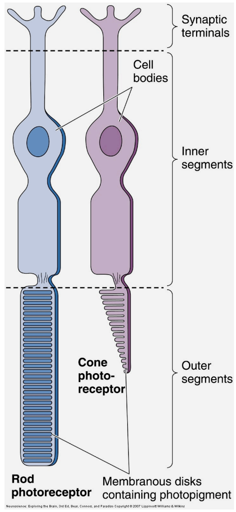

Brain
Optic Nerve (cranial nerve 2)
Retinal Ganglion Cell
Bipolar Cell
Photoreceptor Cell (Rod or Cone)
Ganglion cell axons projecting to forebrain
Ganglion cells
Amacrine cell
Bipolar cell
Horizontal cell
Photoreceptors

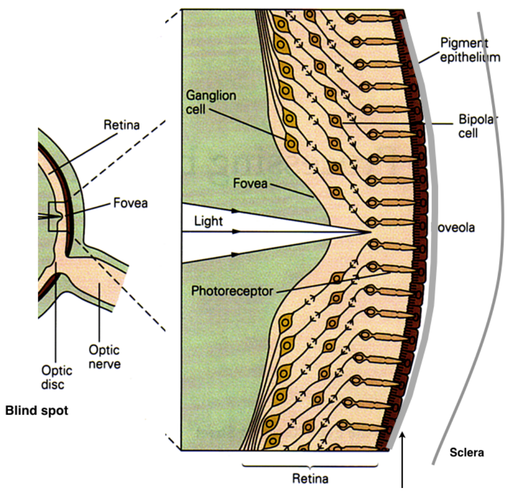
Photoreceptors



cones =
bright light,
color vision

rods =
low light level,
B&W vision

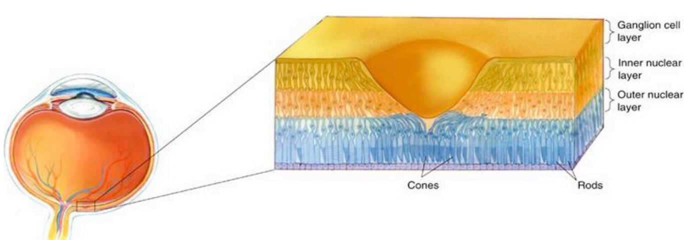
The retina

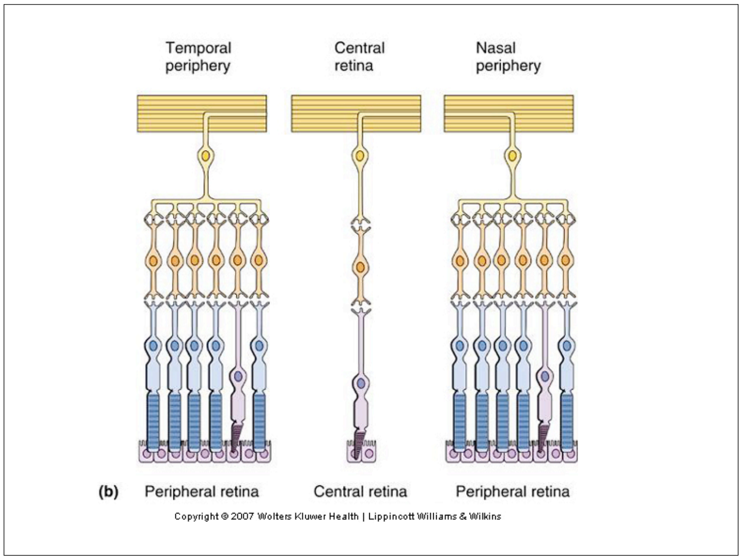


reflective layer in nocturnal animals to harvest more light: tapetum lucidum

Regional Differences in Retinal Structure (Cont'd)

- Cross-section of fovea: Pit in retina where outer layers are pushed aside
  - Maximizes visual acuity
- Central fovea: All cones (no rods)
  - 1:1 ratio with ganglion cells
  - Area of highest visual acuity




Temporal
periphery
Central
retina
Nasal
periphery
(b)
Peripheral retina
Central retina
Peripheral retina
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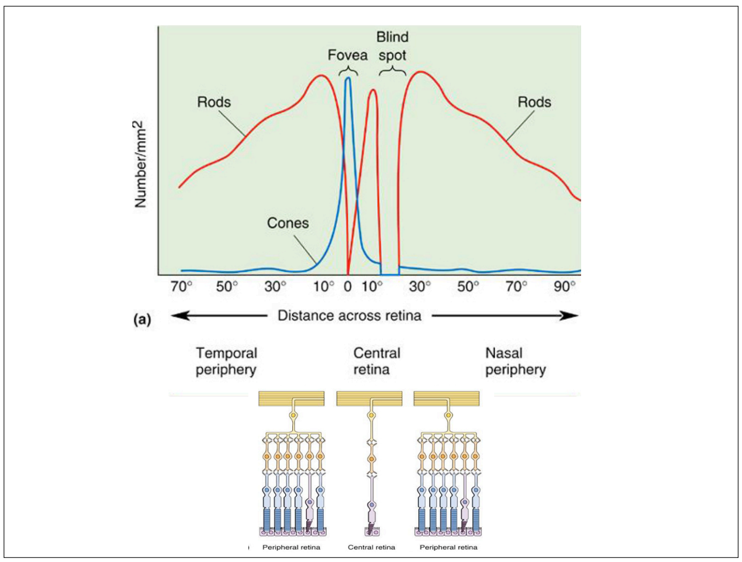
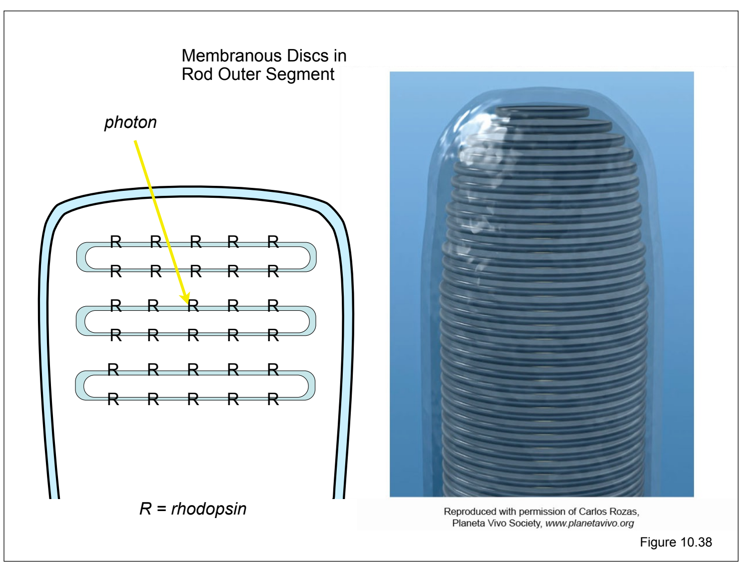

Blind
spot
Fovea
Rods
Rods
Number/mm2
Cones
70° 50° 30° 10° 0 10° 30° 50° 70° 90°
(a)
Distance across retina
Temporal
periphery
Central
retina
Nasal
periphery


Membranous Discs in
Rod Outer Segment
photon
R = rhodopsin
Reproduced with permission of Carlos Rozas,
Planeta Vivo Society, www.planetavivo.org
Figure 10.38

Summary of Dark Current & Activation of Rhodopsin

1. Rod Photoreceptors have **cGMP-gated Na+ channels** on their plasma membrane.

2. In the dark, cGMP levels are high, so Na+ channels are open.

3. In-rush of Na+ depolarizes photoreceptor cell, so it releases **more** neurotransmitter in the dark.

4. Light activates **rhodopsin** in the disk membranes by alterating configuration of **retinal** (vitamin A).

5. Rhodopsin is a **G-protein coupled receptor** (activated by light, not a ligand). Activated G-proteins activate a **phosphodiesterase** that breaks down cGMP.

6. So in light, cGMP levels fall. cGMP-gated Na+ channels close.

7. Photoreceptor cell becomes hyperpolarized, so it releases **less** neurotransmitter in the light.

Rhodopsin is G-protein coupled receptor

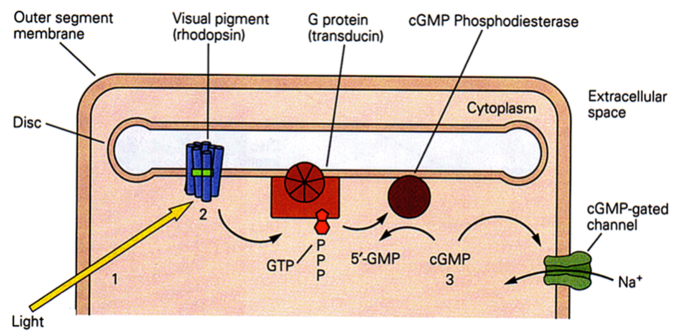
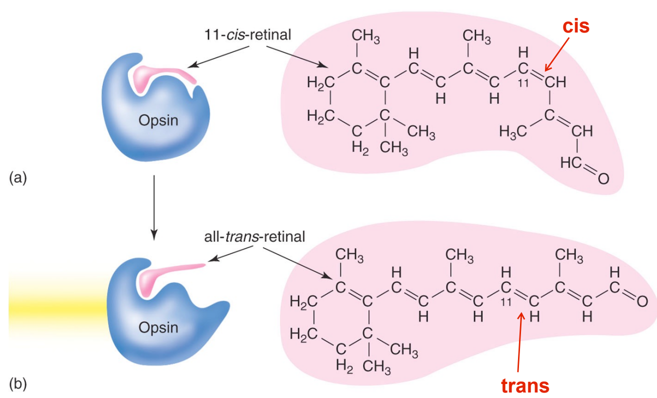
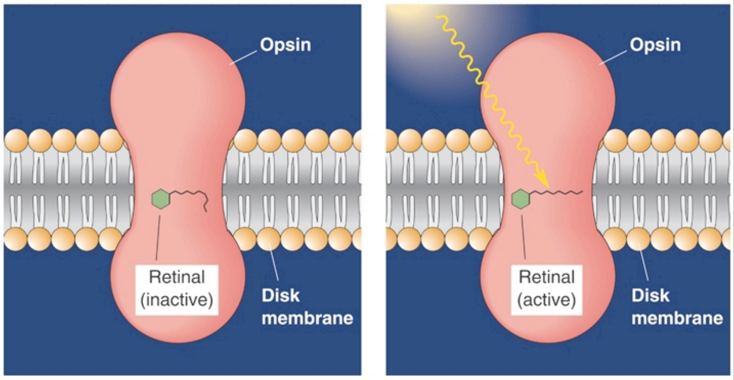


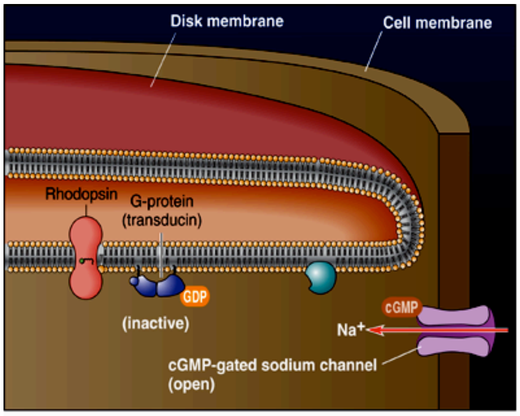
Figure 10.39





Phototransduction in Rods

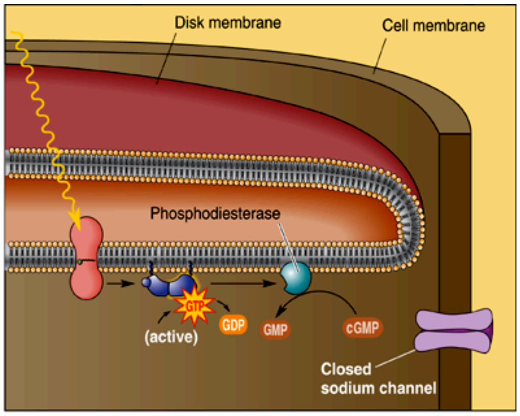
- Light activated biochemical cascade in a photoreceptor
- The consequence of this biochemical cascade is signal amplification



(a) Dark

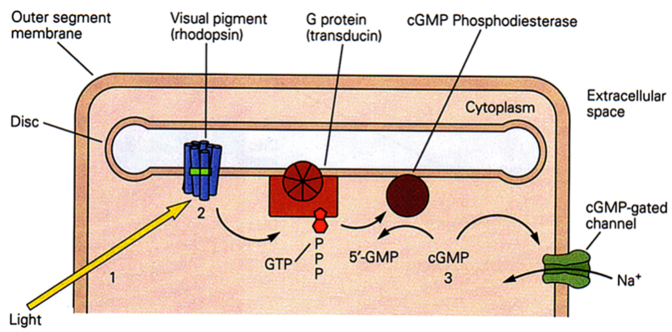
Phototransduction in Rods

- Light activated biochemical cascade in a photoreceptor
- The consequence of this biochemical cascade is signal amplification



(b) Light

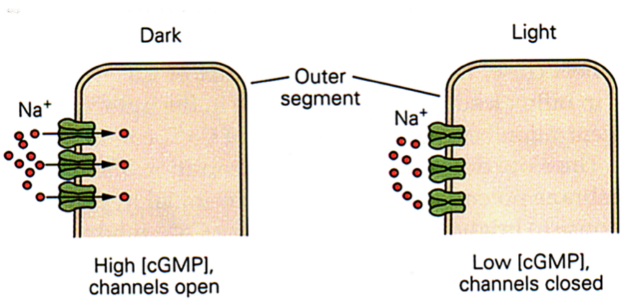
Rhodopsin is G-protein coupled receptor



1 rhodopsin -> 100s of G-proteins -> 100s of phosphodiesterase -> closing of 1,000 Na+ channels/second.

so 1 photon -> blocks 1,000,000 Na+ ions from entering cell

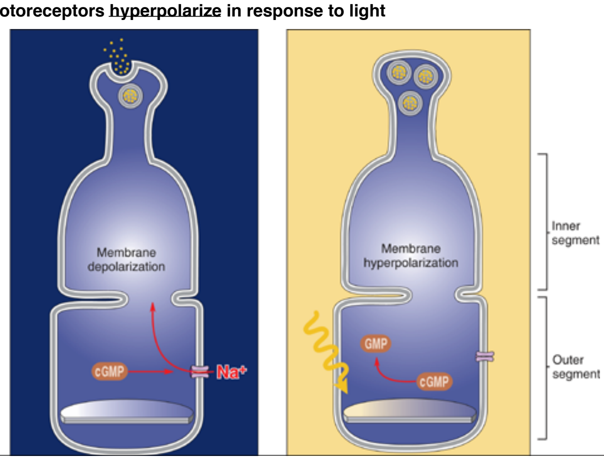
Dark -> high cGMP -> Na+ channels close



Light -> low cGMP -> Na+ channels close

Phototransduction in Rods

- Dark current: Rod outer segments are <u>depolarized</u> in the dark because of steady influx of Na+
- Photoreceptors <u>hyperpolarize</u> in response to light



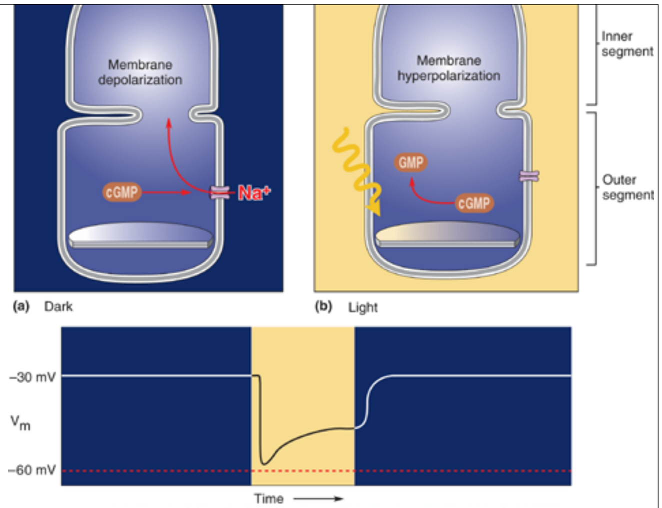
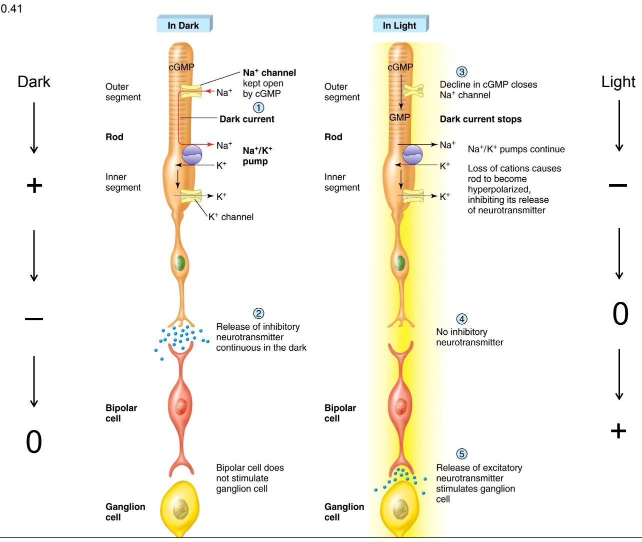


Figure 10.41



Color Pigments

Rhodopsin : rod pigment (grayscale, low light level night vision).

S: short wavelength (blue)

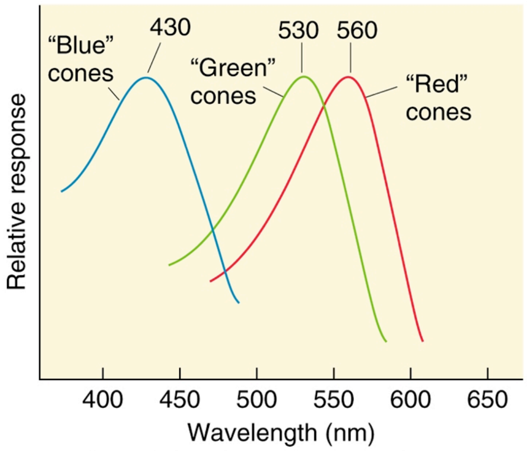
M: medium wavelength (green)

L: long wavelength (red)

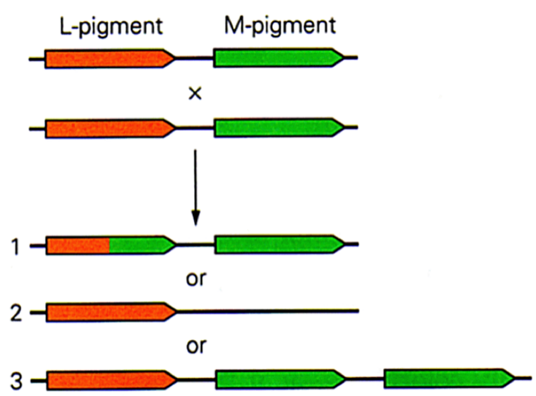
S and M derived from rhodopsin long ago (up to New World monkeys)

L& M diverged only 8 million years ago (New vs Old World monkeys)

Spectral Sensitivity of Pigments



Color Pigment Genes on X chromosome



John Dalton's Color Blindness

Two centuries ago the chemist John Dalton analyzed his own color blindness in his first lecture to the Manchester Literary and Philosophical Society (*1*), and "daltonism" has since become the name for the condition in many languages (*2*). Dalton judged red sealing wax to be a good match for the outer face of a laurel leaf, and a crimson ribbon matched the color that others called "mud" (*1*, *3*). In the solar spectrum he saw only two main hues, one of which corresponded to the normal observer's red, orange, yellow, and green, whereas the second corresponded to blue and violet. He was particularly surprised to observe that the pink flowers of a cranesbill (*Geranium zonale*) (*4*), which appeared "sky-blue" to him by daylight, looked "very near yellow, but with a tincture of red" (*3*) by candlelight (Fig. 1). Of his immediate acquaintances, only his brother shared his astonishment at this failure of color constancy (*3*).

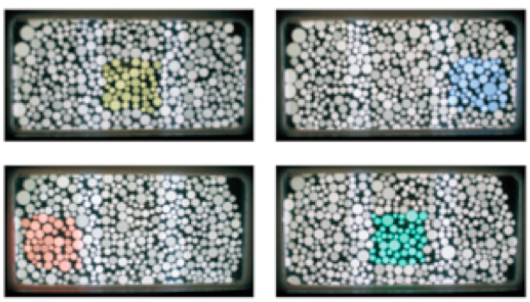
The lenses of Dalton

In an explanation of his color deficiency, Dalton proposed that the vitreous humor of his eye was tinted blue, selectively absorbing longer wavelengths. He instructed that after his death his eyes should be dissected to confirm his hypothesis. He died at age 78 on 27 July 1844, and on the following day an autopsy was done by his medical attendant, Joseph Ransome (5, 6). Ransome collected the humors of one eye into watch glasses and found them to be "perfectly pellucid," the lens itself exhibiting the yellowness expected in someone of Dalton's age (6). He shrewdly left the second eye almost intact, slicing off the posterior pole and noting that scarlet and green objects were not distorted in color when seen through the eye. Thus, Ransome found no support for Dalton's hypothesis that color blindness was due to a preretinal filter. Ransome did not discard the eyes but stored them only in air, and fragments of them have survived to this day (Fig. 2). Originally in the possession of Dalton Hall, the eyes passed into the keeping of the Manchester Literary and Philosophical Society (7) who gave us permission to take small samples for a reexamination of Dalton's color blindness by DNA analysis.

Neitz's team injected their monkeys' eyes with viruses carrying a gene that makes L-opsin, one of three proteins released when color-detecting cone cells are hit by different wavelengths of light. Male squirrel monkeys naturally lack the L-opsin gene (only have M & S); like people who share their condition, they're unable to distinguish between red and green.

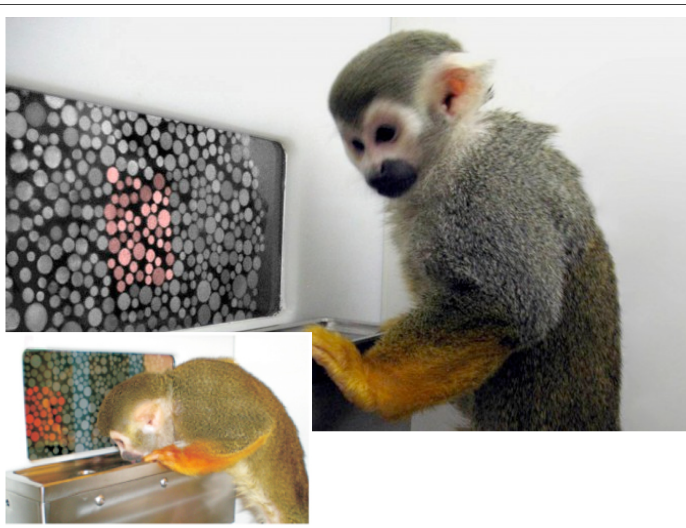
yellow

blue



red

green

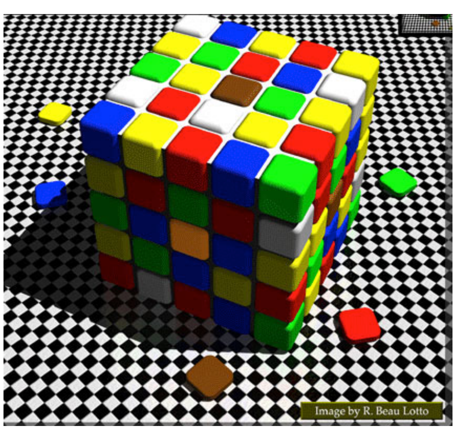


At first, the two monkeys behaved no differently than before. Though quick to earn a grape juice reward by picking out blue and yellow dots from a background of gray dots on a computer screen, they banged the screen randomly when presented with green or red dots.

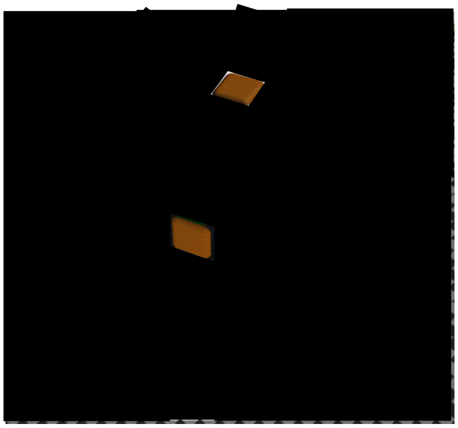
But after five months, something clicked. The monkeys picked out red and green, again and again. At the biological level, Neitz can't say precisely what happened — the monkeys, named Sam and Dalton, are alive and healthy, their brains unscanned and undissected — but their actions left no doubt.

Read More http://www.wired.com/wiredscience/2009/09/colortherapy/#ixzz0fbmdw58J

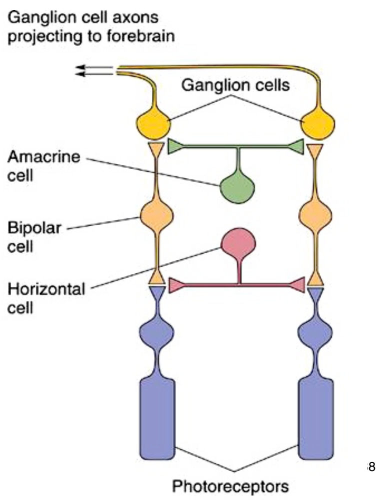
More to color than photoreceptors:



More to color than photoreceptors:

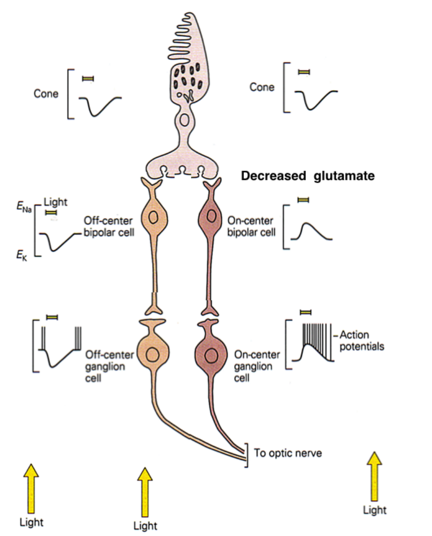


Network in the retina



8

On & Off Bipolar Cells



Receptive Fields of Retinal Ganglion Cells

Photoreceptor cells are coupled to ganglion cells via **bipolar cells**. Input from bipolar cells is modulated by **horizontal cells**.

Receptive field of ganglion cell is based on:

1. **Spatial Location**: A specific spot in the visual field as it is projected onto the retina at the back of the eye.

2. **Contrast**: ganglion cells are either **on-center** or **off-center** cells: they respond to either light surrounded by dark, or dark surrounded by light. This allows ganglion cells to respond well to **high contrast edges** in the visual field.

Ganglion cells activity is influenced by multiple photoreceptors and bipolar cells, which contribute to ganglion receptive field.

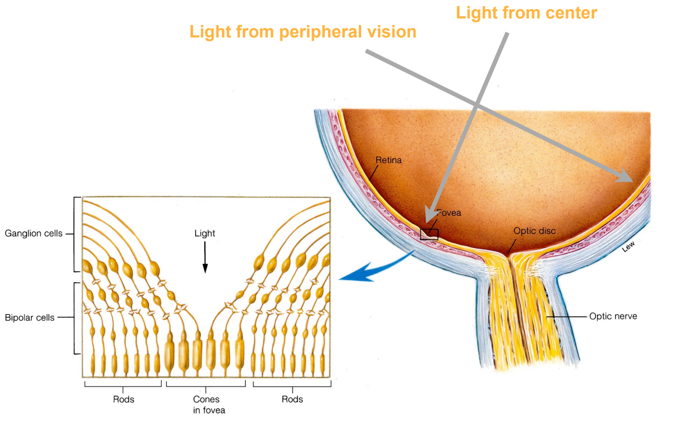
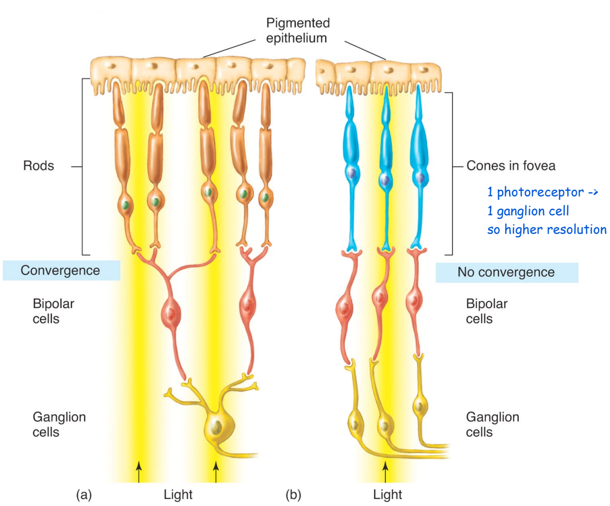
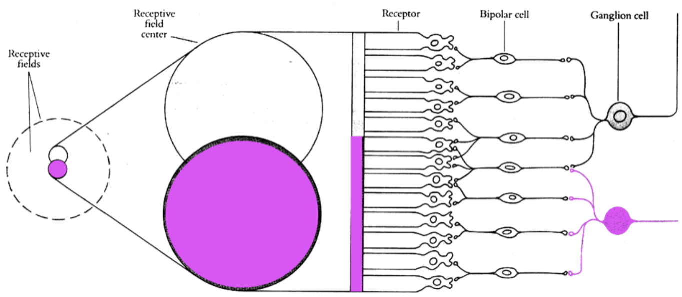


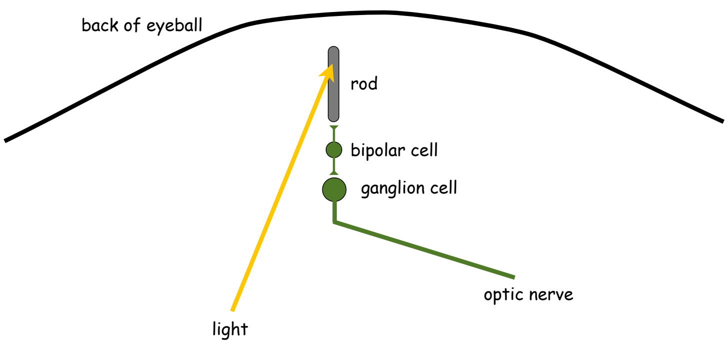
Figure 10.44



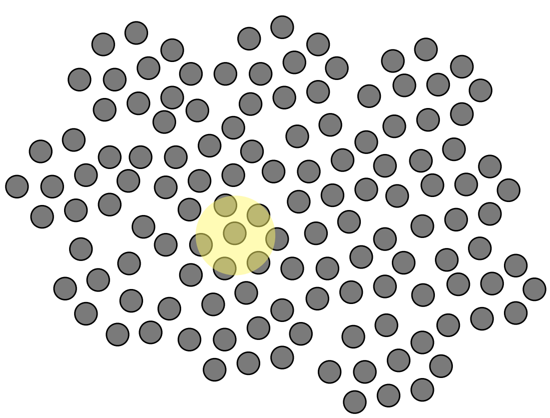
Ganglion cells get input from more than one receptor to form large, overlapping receptive



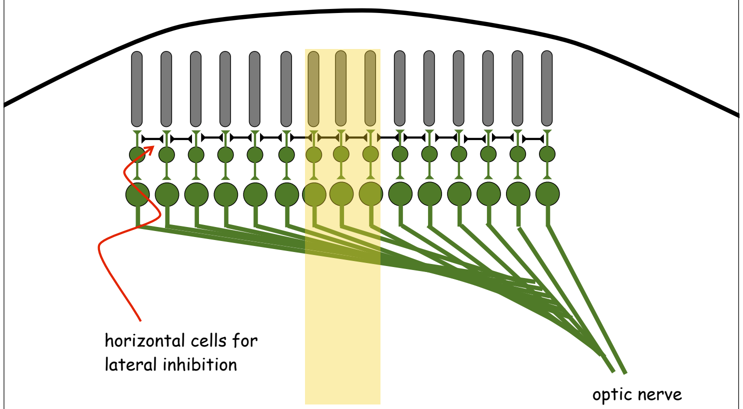
Cross-Section of Retina from side

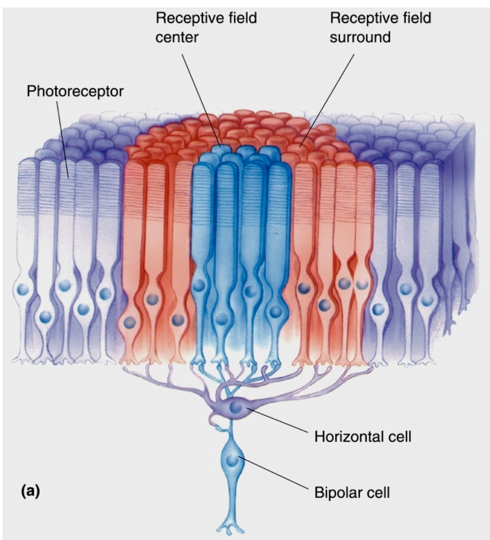


Looking Down on Retina



Cross-Section of Retina from side





On-Center Biopolar/Ganglion Cell

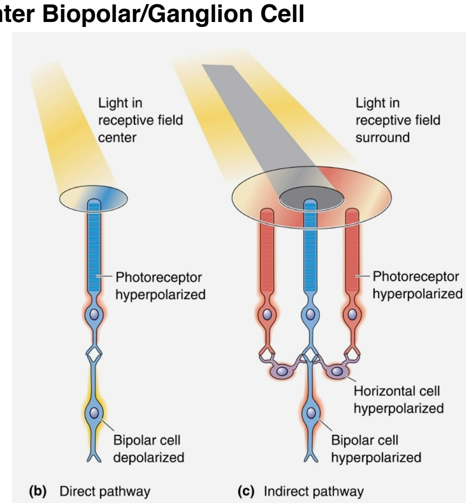
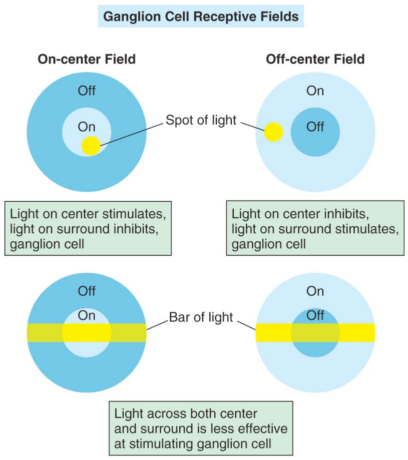
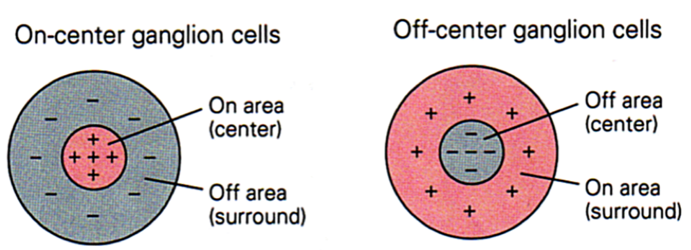
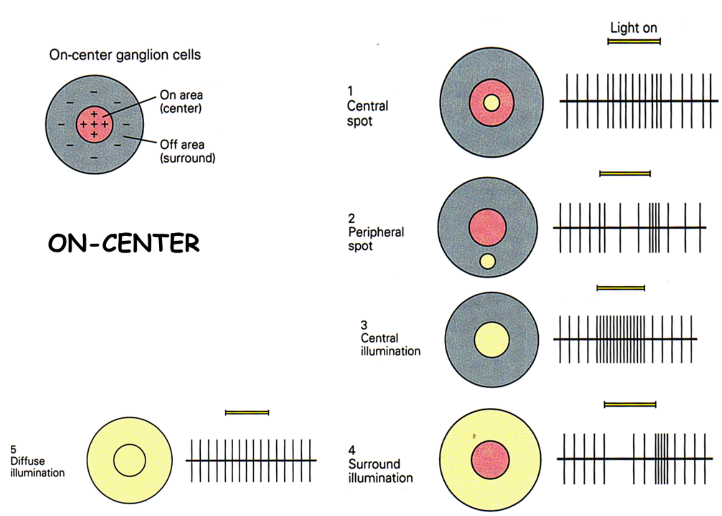


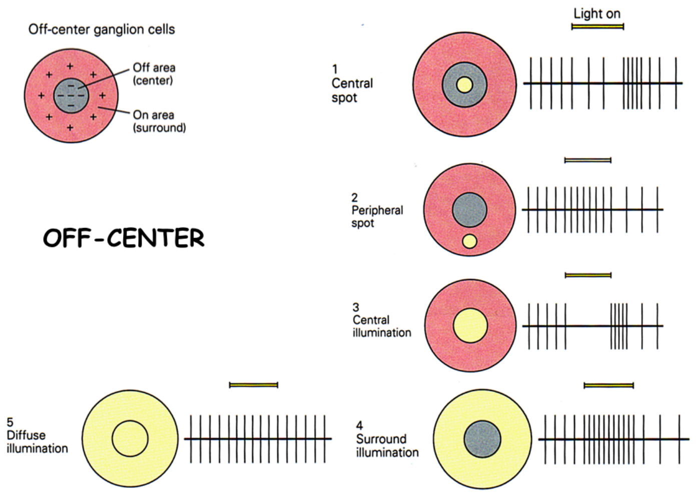
Figure 10.47



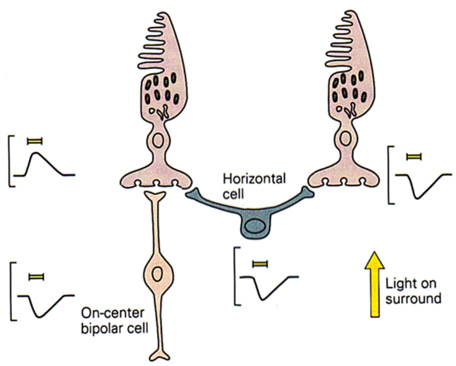
Center/Surround Cells







Horizontal Cells



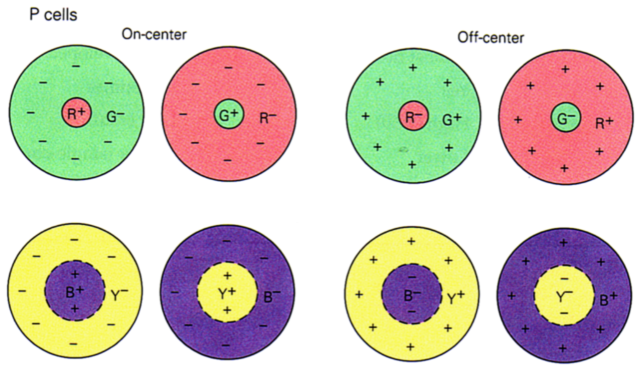
So surround inhibits center

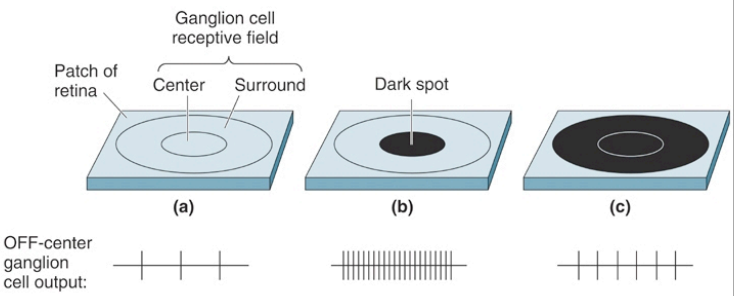
M & P cells

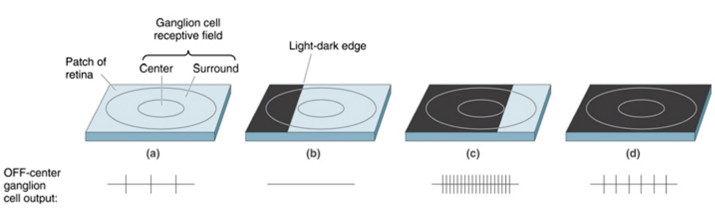
M cells
Ganglion cells that project to magnocellular thalamus
High illuminance contrast (grayscale center/surround)

P cells
Ganglion cells that project to parvocellular thalamus
Contrasting colors (color center/surround)

M & P cells: Color contrast







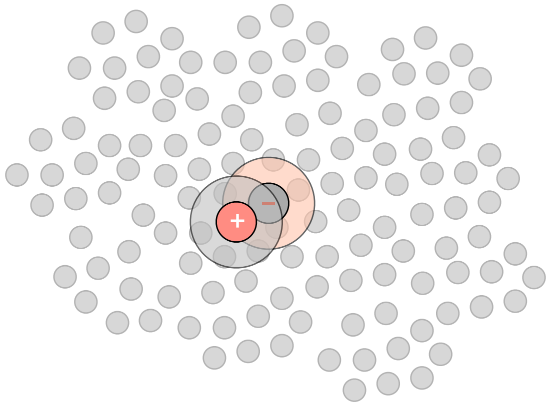
So ganglion cells most responsive to edges:

OFF-center ganglion cells
quiet on light side of edge, active on dark side of edge.

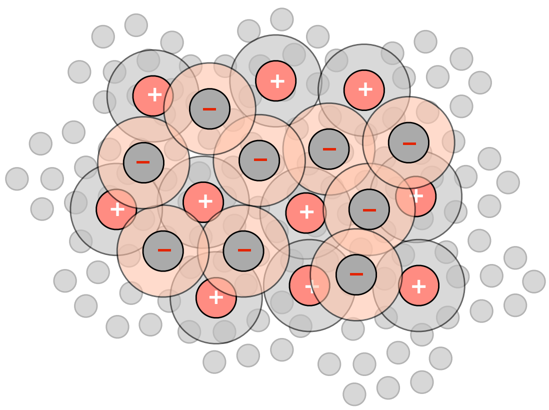
ON-center ganglion cells
active on light side, quiet on dark side of edge.

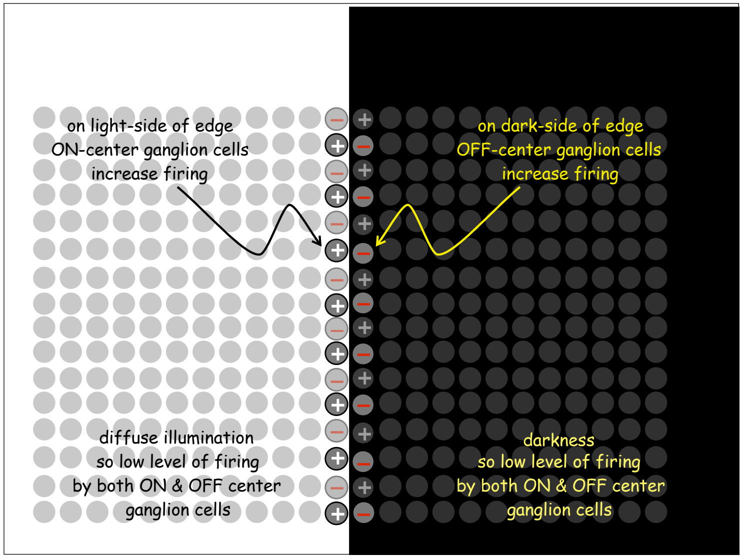
In **constant** field, both types of cells have baseline activity.

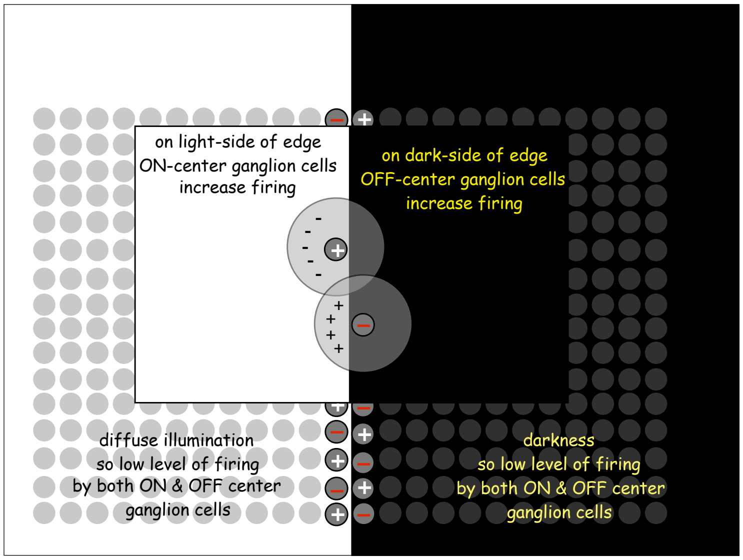
Overlapping Receptive Fields of On-Center & Off-Center Ganglion Cells



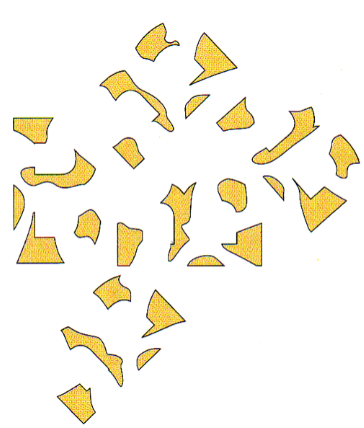
Overlapping Receptive Fields of On-Center & Off-Center Ganglion Cells







More to vision than just edges



More to vision than just edges